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Sleep and Waking Ultradian Rhythms in Hyperkinetic Children

by Keith A. Busby

Thesis submitted to the School of Graduate Studies of the University of Ottawa in partial fulfillment of the requirements for the Doctor of Philosophy Degree in Psychology.

Ottawa, Canada, 1980.

Abstract

The investigation of sleep patterns and waking ultradian rhythms in performance and motility were examined in groups of nonmedicated hyperkinetic (n = 11) and normal control children (n = 11). Sleep recordings were undertaken to examine possible events and relationships obscured by or not present during wakefulness, which may provide evidence of a postulated arousal disorder in HK children. Children (8-12 years) slept in the sleep lab for five consecutive nights during which EEG, EOG, EMG, and SSPR measures were monitored. During the days following the third and fourth nights, subjects underwent testing to detect the possible presence of ultradian variations in performance (detection and false positives) and motility (global body movements and limb movement during the task, and limb movement during off-task periods). Testing was conducted for five minutes every 15 minutes (10-minute "rest periods") over a six-hour period on each day. Phase relationships between the previous night's NREM and REM cycles and waking ultradian rhythms were also examined. Analyses of sleep pattern variables revealed a significantly longer REM onset latency ($p < .03$) and marginally significant ($p < .08$) greater amounts of movement time, body movements, and NREM twitches for the HK group relative to controls. No other sleep parameters differentiated the groups. On waking attention and motility measures, HK subjects made significantly fewer detections ($p < .05$) and were more active ($p < .01$) during off-task periods on both days. Limb movement during the task was marginally significant ($p < .06$) on one day (HK > Control). False positives and global body

movements failed to differentiate the groups. The results for waking performance and motility, and sleep were interpreted within the hyperarousal/hypoarousal theory of HK. With regard to ultradian rhythmicity in these waking variables, some subjects in both groups showed evident ultradian peaks which were present across a wide range of frequencies in one or more variables. Inconsistent results were obtained for phase relationships between waking ultradian rhythms and extrapolated nocturnal REM cycles, prompting the conclusion that multiple oscillators operate during the sleep and waking states.

CURRICULUM STUDIORUM

Keith A. Busby was born October 4, 1951 in Calgary, Alberta. He received the Bachelor of Arts (Honours) degree from Brock University, St. Catharines, Ontario, in 1974. He received the Master of Arts degree in Psychology from the University of Ottawa, Ottawa, Ontario, in 1977. The title of his Master's thesis was The Effects of Transcendental Meditation on Dream Content.

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Chapter I

REVIEW OF THE LITERATURE

Of all childhood psychiatric disorders, hyperkinesis (HK) has probably generated the most research and resultant controversy in recent years. Prevalence estimates have ranged from one to 20 percent for all school-age children (Chess, 1960; Huessy, 1967; Lambert, Sandoval, & Sassone, 1978; Stewart, Pitts, Craig, & Dieruf, 1966; Wender, 1971), and it is one of the most common primary presenting symptoms of children referred for psychological difficulties (Patterson, Jones, Whittier, & Wright, 1965). The study and treatment of this clinical syndrome have been frustrated by ambiguous, global, and often imprecise conceptualizations as to definition, etiology, and diagnosis as evidenced in the introduction over the years of widely discrepant terminology used to identify the syndrome, including minimal brain dysfunction (MBD), minimal brain damage, hyperactivity, hyperkinesis, specific learning disabilities, etc.. In recent years, there has been a question as to whether an entity known as hyperkinesis actually exists, since it has been associated increasingly with other psychiatric and purely medical conditions. Based upon clinical and research literature, definition and diagnosis have primarily relied upon a constellation of symptoms extending along a number of behavioral, perceptual-cognitive, and social dimensions. The classical or core symptomatology includes excessive and often inappropriate activity, short attention span, distractibility, impulsivity, excitability, and poor scholastic performance, despite scoring within the normal range on various intelligence measures. In addition, several secondary or ancillary

signs such as aggressiveness, low frustration tolerance, and poor self-esteem are often present (Clements, 1966; Minde, Weiss, & Mendelson, 1972; Schmitt, Martin, Nellhaus, Cravens, Camp, & Jordan, 1973; Whalen, & Henker, 1976). Although many of these behaviorally-defined deficits also occur in children throughout normal developmental stages, it is the persistence, intensity, and clustering of symptoms which characterizes HK behavior (Minde, Lewin, Weiss, Lavigueur, Douglas, & Sykes, 1971; Wender, 1975). This has resulted in one hypothesis, that these children may suffer from a neuro-developmental lag (Kinsbourne, 1973; Rosenthal, 1973).

The earliest descriptions of HK began to appear shortly after the encephalitis pandemic following World War I. It was thought that the typical behavioral sequelae were directly attributable to the post-infectious consequences of that disease (Hohman, 1922). Since that time, similar behavioral patterns have been shown to be associated with neurological dysfunction resulting from head trauma (Kasanin, 1929), prematurity and pre- or perinatal birth complications (Knobloch, & Pasamanick, 1966; Rogers, Lilenfeld, & Pasamanick, 1955), and lead poisoning (Thurston, Middelkamp, & Mason, 1955). While it became obvious that head trauma and neurological disease could produce HK behavior, subsequent observations made it clear that this could not account for all of the cases. Genetic transmission, implying congenital etiology, has also been proposed. Evidence supporting this notion comes from observations of the presence of minor physical anomalies (Firestone, Lewy, & Douglas, 1976; Rapoport, & Quinn, 1975; Waldrop,

Bell, McLaughlin, & Halverson, 1978; Waldrop, Pederson, & Bell, 1968), as well as reports of increased prevalence of HK within families (Cantwell, 1975; Morrison, & Stewart, 1973; Omenn, 1973; Safer, 1973).

The initial relationship of HK to brain damage was supported by reports of the relatively high incidence of "soft" neurological signs and borderline abnormalities in EEG activity in this group (Clements, & Peters, 1962). However, recent critical evaluation of this research has suggested that only a small subgroup of HK children show an obvious association to overt neurological abnormality (e.g., Dubey, 1976). Thus, a specific organic etiology has yet to be found, yet a biological deficit is still considered to underly this disorder.

The search for postulated differences in psychophysiological processes in HK has been directed toward the more global concept of arousal. As previously mentioned, the original association with brain injury was highly emphasized. Two corollaries were further proposed, namely: 1) that perceptual deficits often observed in these children were the result of an impaired ability to filter irrelevant stimuli and to selectively attend to relevant environmental stimuli; and, 2) that HK was a reaction to excessive sensory stimulation (Strauss, & Kephart, 1955; Strauss, & Lehtinen, 1947). This implies that at a higher cognitive level, there may be a defective attentional mechanism (Douglas, 1972; Tarver, & Hallahan, 1974). The inability to filter and organize stimulus input was looked upon as "stimulus overload", which led in turn, to increased activity. This increased behavioral responsivity was thought to serve no functional utility, but merely acted as undirected

responding to overstimulation. It was argued that the underlying physiological basis for this notion was over- or hyperarousal of the various brain stem and subcortical structures (Laufer, Denhoff, & Solomans, 1957). The obvious treatment to counter this overwhelming stimulation was an attempt to reduce the amount of environmental stimulation. Cruickshank, Bentzen, Ratzeburg, and Tannhauser (1961) attempted to confirm this theoretical approach by showing that over a one year period, HK children made significant performance increments in a reduced stimulus environment. However, these gains were no greater than those for a similarly treated control group. General lack of support together with Cruickshank et al.'s inconclusive results have tended to disconfirm this approach. This lack of support has come primarily from studies showing that increased stimulation under highly novel conditions actually leads to a decrease in HK behavior (Zuk, 1963). Furthermore, in a structured classroom situation, the behavior of HK children is not merely random but appears to be inappropriately goal-directed (Douglas, 1972).

An alternative explanation to account for HK behavior has recently been offered (Zentall, 1975; Zentall, 1977) in which increased motoric activity is viewed as stimulus-seeking behavior. With more movement come concomitant increases in visual, auditory, proprioceptive, and kinesthetic stimulation as well. Thus, adequate stimulation involves the operation of a homeostatic mechanism functioning to maintain a level of stimulation at some optimal level (Berlyne, 1960; Hebb, 1955; Leuba, 1955). The notion

of optimal level of stimulation infers that stimulus-seeking behavior has functional value for the child. Within this context, HK has been viewed as a direct result of under- or hypoarousal.

Considerable empirical support has been gathered for the optimal stimulation hypothesis. Differential HK behavior has been shown to result from a variety of situations. For instance, on simple tasks, HK children do not differ from normals, while on complex structured tasks, HK children show increased activity and problems of attention (Pope, 1970). Similarly, tasks involving decreased stimulation or little movement induce greater motoric activity, while tasks involving increased stimulation through movement (free time, unstructured play) tend not to produce greater activity in HK children compared to controls (Zentall, & Zentall, 1976). HK behavior has been shown to be a function of both amount of stimulation and length of exposure to a particular environment. Initially, the more novel the environment or task, the better the performance and the less the activity (Reardon, & Bell, 1970; Tizard, 1968). Douglas (1972) has demonstrated that performance on a continuous performance task (CPT) deteriorates rapidly and motor restlessness increases with time on task.

Zentall (1975) has recently argued for a strong resemblance between the effects of sensory deprivation on normal human adults and behavioral descriptions observed for the HK syndrome. In typical sensory deprivation paradigms, subjects are placed in an environment where sensory stimulation is minimized. Particularly in those situations where movement is restricted, an inability to concentrate, disorganization of thought and intellectual processes,

and hallucinatory experiences have often been reported (Freedman, & Greenblatt, 1960; Heron, Doane, & Scott, 1956; Scott, Bexton, Heron, & Doane, 1959). Zubek (1963) has further shown that these impairments are somewhat reduced in severity if subjects are allowed to move about. Utilizing optimal level of stimulation theory, subjects who increase their activity level could be viewed as attempting to achieve some homeostatic balance. The behavioral descriptions of sensory deprivation effects are quite similar to those of HK behavior patterns. By logical extension, it could be argued that the heightened activity levels observed in HK children under low stimulation conditions, function to offset the effects of stimulus deprivation by increasing the amount of visual, auditory, kinesthetic, and proprioceptive stimulation.

The effects of stimulant drug treatment (primarily methylphenidate and dextroamphetamine) on HK behavior have also been interpreted within the hyperarousal/hypoarousal framework. Typically, stimulant administration in HK children serves to suppress restless and impulsive behavior while facilitating attentional abilities (Campbell, Douglas, & Morgenstern, 1971; Conners, 1970; Knights, & Hinton, 1969). According to the overstimulation model, these stimulant drug effects act in a paradoxical manner, since in normal adults stimulants function to increase arousal and activity and enhance performance (Sroufe, & Stewart, 1973; Wender, 1971). These drugs might be operating differentially on arousal mechanisms in HK children compared to controls. In HK children, stimulants may be acting on inhibitory systems leading to decreased arousal and activity, whereas, in normal children, stimulants might facilitate excitatory systems.

Within the alternative (hypoarousal) model, the effects of stimulant drug treatment on HK behavior may be analogous in function to activity reduction associated with environmental stimulation increases. Both of these mechanisms might involve facilitation of effective stimulation concomitant with decreases in HK behavior (Zentall, 1975). Stimulant drugs may be seen as affecting all children in the same manner, i.e., an excitatory effect which in HK children serves to elevate level of arousal to an optimal level (Rosenthal, 1973; Satterfield, 1976). Some initial support has been provided for this notion. Studies have recently been reported in which dextroamphetamine effects on electrophysiological, cognitive, and behavioral measures were evaluated in sample groups of normal children. Rapoport, Buchsbaum, Zahn, Weingartner, Ludlow, and Mikkelsen (1978) found that amphetamine decreased activity levels, and improved reaction time, attention on a continuous performance task, and performance on a variety of cognitive tasks (learning, memory, and language tests). Similar findings were obtained on these measures when a HK group and a normal control group were both given dextroamphetamine (Weingartner, Rapoport, Buchsbaum, Bunney, Ebert, Mikkelsen, & Caine, 1980). Furthermore, other studies have demonstrated that barbiturates, which exert a depressant effect in normal human adults, tend to increase the activity level of HK children (Bradley, & Bowen, 1941; Stewart, 1970).

Psychophysiological Parameters

The search for psychophysiological measures which might differentiate HK from normal children has gained increasing popularity over the last decade.

Excellent recent reviews of the psychophysiological literature pertaining to HK have been provided by Ferguson and Pappas (1979), Hastings and Barkley (1978), and Rosenthal and Allen (1978). Many of these investigations have addressed themselves directly to the hyperarousal/hypoarousal question. The term arousal has had an extremely ambiguous connotation with the result that general findings related to this concept have failed to completely clarify this issue as it relates to HK.

Rosenthal and Allen (1978) have differentiated arousal into two categories: 1) physiological arousal (cortical and autonomic), which is mediated by brain stem and sub-cortical brain structures; and 2) behavioral arousal, which is synonymous with general activity level and represents a quantitative dimension of overt responding. Investigations of arousal level have generally focused upon physiological arousal as the fundamental component since it provides a basis upon which overt behavioral responding depends.

Cortical arousal. Cortical arousal in HK children has been examined using clinical electroencephalographic (EEG) and evoked potential measurements. The frequency of abnormal EEG activity has ranged from approximately 18-47% of HK children studied. The major clinical EEG abnormality found has been excessive slow-wave activity, indicative of lower arousal levels (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). When specific EEG frequency bands were studied, Grunewald-Zuberbier, Grunewald, and Rasche (1975) found more alpha

and less beta wave activity (higher amplitude in both) in HK compared to non-HK "maladjusted" children. Sheer (1976) also noted decreased activity in the 40 Hz range in a group of learning-disabled (only some displayed HK behavior) children. Using power spectral analysis techniques, Montagu (1975) reported less power in the 8-10 Hz band in HK compared to normal children. In addition, more within-hemisphere coherence in the 0-8 Hz bands was observed, which Montagu argued was indicative of "diminished cortical processing". Similarly, Satterfield, Cantwell, Lesser, and Podosin (1972) found more power in the 0-8 Hz range in HK children which provided confirmation of the presence of increased slow-wave activity.

Phasic changes in the EEG have also revealed conflicting results in HK children. Grunewald-Zuberbier et al. (1975) found increased latencies to alpha blocking for tone stimuli, shorter durations of the alpha blocking response to light stimuli, and faster habituation to tone stimuli. Satterfield (1973) corroborated these findings by showing longer latency responses to alpha blocking in HK compared to normal children. Similarly, Milstein, Stevens, and Sachdev (1969) reported longer latencies to alpha attenuation, but contrary to the findings of Grunewald-Zuberbier et al., longer duration of alpha activity were noted to light stimuli.

Several studies have investigated the effects of stimulant drugs on the EEG patterns of HK children. Using power spectral analyses, investigations have shown that drug responders increase their alpha frequency (Shetty, 1971). Satterfield et al. (1972), who observed no increase in slow-wave activity in drug responders, reasoned that stimulant drugs prevented lower arousal from

occurring. Finally, Surwillo (1977) noted that EEG patterns of his HK sample were very similar to those typically exhibited by younger-aged normal children. While on stimulant medication, these patterns became normalized. It was suggested that these HK subjects exhibited a maturity-related or developmental lag.

The evoked potential has also been used as a phasic measure of EEM activity in the study of HK. Results of studies comparing HK with normal children and examining stimulant drug/non-drug effects within HK samples have been somewhat equivocal in nature. This have been largely due to differences in subject selection, experimental design; and varying definitions as to outcome. However, general consistency has been found in the direction of underarousal in subgroups of HK subjects. A number of studies have shown that auditory evoked potential (AEP) responses to tone glick stimuli were significantly smaller in amplitude and longer in latency in untreated HK than normal children, (Klorman, Saltzman, Pass, Borgstedt, & Dainer, 1979; Satterfield, Lesser, Saul, & Cantwell, 1973; Shouse, & Lubar, 1978). In a study in which subjects were required, under certainty and uncertainty conditions, to predict whether one or two clicks would be presented, HK subjects demonstrated a larger P300 component in the certainty condition, while a smaller P200 and larger N250 were noted in the uncertainty condition compared to controls. The smaller P200 component was interpreted as indicative of deficit attention, and the larger N250 as an index of hypoarousal (Prichep, Sutton, & Hakerem, 1976). Buchsbaum and Wender (1973) measured inter-AEP variability to a series of tone stimuli and

found that HK subjects exhibited more variability on this measure, perhaps a reflection of the increased slow content of the EEG.

Using the visual evoked potential (VEP), Buchsbaum and Wender (1973) found that MBD children had greater amplitudes and shorter latencies to all VEP components at four 1 Hz square-wave light intensities. In addition, MBD children demonstrated a faster rate of increase in VEP amplitude with increasing stimulus intensity. In contrast, Hall, Griffin, Moyer, Hopkins, and Rappaport (1976) found no differences in latency, amplitude, or amplitude/latency relationship with increasing intensity light stimulus levels in either attention or inattention conditions. This inconsistency of results has been interpreted as a failure in the previous studies, other than that by Hall et al. (1976), to screen for EEG abnormalities in subject samples. An alternative explanation for these differing findings has been proposed by Satterfield and Braley (1977). They found that in normal subjects, EP amplitudes tended to decrease with age, whereas in HK subjects, the P1-N1 component increased and the P2-N2 component remained the same. These authors postulated a delayed maturational factor to account for these results.

Stimulant medication effects on EP measures have also been evaluated in HK children. Those investigations in which positive clinical responses were obtained after drug administration, showed that EP measures were normalized, i.e., response amplitudes, latencies, and variability of EP components were more similar to those exhibited by normal control subjects (Buchsbaum, & Wender, 1973; Conners, 1972; Conners, 1975; Halliday, Rosenthal, Naylor, & Calloway, 1976; Prichep et al., 1976; Saletu, Saletu, & Itil, 1973; Saletu, Saletu, Simeon, Viamontes, & Itil, 1975).

Autonomic arousal. The majority of studies comparing HK to controls on measures of basal or resting heart rate (HR) have reported no group differences on this measure (Barkley, & Jackson, 1977; Boydstun, Ackerman, Stevens, Clements, Peters, & Dykman, 1968; Ferguson, Simpson, & Trites, 1976; Zahn, Abate, Little, & Wender, 1975), although one study did report a faster rate (5-6 bpm) in a HK sample compared to controls (Ballard, Boileau, Sleator, Massey, & Sprague, 1976). The administration of stimulant medication has been generally reported to be followed by significant increases in HR as has been reported for methylphenidate (Aman, & Werry, 1975; Ballard et al., 1976; Boileau, Ballard, Sprague, Sleator, & Massey, 1976; Cohen, Douglas, & Morgenstern, 1971; Knights, & Hinton, 1969; Zahn et al., 1975) and for dextroamphetamine (Zahn et al., 1975). In three studies, there was a failure to observe drug effect differences with pemoline (Knights, & Viets, 1975) and methylphenidate (Barkley, & Jackson, 1977; Porges, Walter, Korb, & Sprague, 1975).

When phasic HR changes in response to stimuli were assessed, smaller phasic cardiac responses in HK compared to control subjects were found in a fixed foreperiod reaction time task (Sroufe, Sonies, West, & Wright, 1973; Zahn, Little, & Wender, 1978), a tone discrimination task (Boydstun et al., 1968), and in a task requiring response to nonsignal tones (Zahn et al., 1975). No differences between groups were observed by Ferguson et al. (1976). Following methylphenidate administration, Sroufe et al. (1973) and Porges et al. (1975) reported increased HR deceleration to nonsignal tones, while Zahn et al. (1975) noted no differences.

Finger pulse volume responses to a tone stimulus have been found to habituate more slowly in HK children than in controls, and the administration of dextroamphetamine in this context increases the rate of habituation (Conners, 1976; Conners, & Rothschild, 1973).

Ballard et al. (1976) have reported the only study to date of blood pressure differences in nonmedicated HK subjects compared to controls. They observed higher systolic, diastolic, and mean blood pressure levels in HK children. A number of studies, however, have assessed stimulant drug effects on blood pressure. Significant increases in this measure have been reported following administration of methylphenidate (Aman, & Werry, 1975; Ballard et al., 1976; Rapoport, Quinn, Bradbard, Riddle, & Brooks, 1974; Rie, Rie, Stewart, & Ambuel, 1976). In contrast, no differential effects have been observed for pemoline (Conners, Taylor, Meo, Kurtz, & Fournier, 1972; Knights, & Viets, 1975), dextroamphetamine (Arnold, Wender, McCloskey, & Snyder, 1972; Conners et al., 1972; Epstein, Lasagna, Conners, & Rodriguez, 1968), or in one study for methylphenidate (Knights, & Hinton, 1969).

Studies of electrodermal activity in HK and normal children have also reported inconsistent results. A number of studies have indicated no differences between groups in basal skin conductance (Boydstun et al., 1968; Cohen, & Douglas, 1972; Ferguson et al., 1976; Firestone, & Douglas, 1975; Montagu, 1975; Satterfield et al., 1972; Spring, Greenberg, Scott, & Hopwood, 1974; Zahn et al., 1975). However, other reports have found lower basal conductance levels in HK subjects, implying lowered arousal levels (Kløve, & Bu, 1976 cited in Ferguson, & Pappas, 1979; Satterfield, & Dawson,

1971; Shouse, & Lubar, 1978). Two studies have noted higher conductance levels (Satterfield, Atoman, Brashears, Burleigh, & Dawson, 1974; Zahn et al., 1978). Conners (1976) also found higher skin conductance levels when HK subjects were required to inhibit responding on a task. Stimulant medication has been followed by reductions (Cohen et al., 1971; Zahn et al., 1975), increases (Satterfield, & Dawson, 1971), and no change (Spring et al., 1974) in skin conductance levels.

Spontaneous galvanic skin responses (GSR) have shown either no differences (Barkley, & Jackson, 1977; Spring et al., 1974; Zahn et al., 1975; Zahn et al., 1978) or fewer and smaller nonspecific responses (Satterfield, & Dawson, 1971) in HK children relative to controls. Stimulant drugs have been reported to increase the number of spontaneous electrodermal fluctuations (Satterfield, & Dawson, 1971; Spring et al., 1974), but Cohen et al. (1971) found no effect on this measure. Surwillo and Quilter (1965) have shown spontaneous GSR activity to be a good predictor of vigilance performance. Since stimulant drugs have been found to improve vigilance in HK subjects (e.g., Sykes, Douglas, Weiss, & Minde, 1971), these studies suggest that some HK subjects may be less autonomically labile (Hastings, & Barkley, 1978; Rosenthal, & Allen, 1978).

Studies of electrodermal activity in response to specific stimuli during a variety of on-task paradigms have also yielded conflicting results. Smaller amplitude phasic responses (Boydstun et al., 1968; Cohen, & Douglas, 1972; Dykman, Ackerman, Clements, & Peters, 1971; Ferguson et al., 1976; Satterfield, & Dawson, 1971; Spring et al., 1974; Zahn et al., 1975) and decreased

specific GSR activity to reaction stimuli (Firestone, & Douglas, 1975; Zahn et al., 1975) have been reported for HK subjects. Although smaller amplitude GSRs to non-signal stimuli have been reported as well (Spring et al., 1974; Zahn et al., 1975), Cohen and Douglas (1972) and Ferguson et al. (1976) found no differences between groups. Stimulant medication was observed to have little or no effect on specific GSR activity (Cohen et al., 1971; Satterfield, & Dawson, 1971; Zahn et al., 1975).

Data on habituation rate of specific GSRs to tones have also been equivocal. Studies utilizing this measure have reported more rapid (Boydstun et al., 1968; Kløve, & Bu, 1976; Spring et al., 1974), slower (Conners, 1976), or similar (Ferguson et al., 1976) rates of habituation in HK children relative to controls. Stimulant drugs are reported to slow habituation rate (Kløve, & Bu, 1976; Spring et al., 1974), but Conners and Rothschild (1973) found that dextroamphetamine increased habituation rate in a group of learning-disabled children.

Pupillometric activity has also been investigated as a measure of arousal in HK children. It is known that pupillary dilitation and constriction are controlled by the sympathetic/parasympathetic nervous systems. Knopp, Arnold, Andras, and Smeltzer (1973) compared pupillometric activation before and after dextroamphetamine administration in a HK group. Several subgroups within their sample exhibited differential responding on this arousal measure. Responses indicative of overarousal were shown in 23%, while underarousal patterns were observed in 36% of HK subjects. Stimulant medication normalized responses of both groups to a level approximating that shown for normal

children. Yoss and Moyers (1971) studied HK children both on and off stimulant drugs and found 25-35% showed underarousal patterns which were increased toward normal levels by stimulant drugs. In contrast, Zahn et al. (1978) found a tendency for greater pupil size on baseline measures, implying sympathetic activation. When pupillary response to a RT task was measured, dilatation occurred; however, some MBD children did show frequent spontaneous constrictions and decreasing base pupil size. In this study, stimulant drugs produced an arousal effect.

In summary, evaluation of physiological indices during wakefulness has not produced clear-cut conclusions regarding the state of arousal (hyper- versus hypoarousal) in HK children. Many of the inconsistent and conflicting results are due in large part to differing subject samples, medication status, and experimental methodologies. Studies which have assessed cortical arousal suggest that some HK children may be underaroused or exhibit patterns which are of an immature nature. Autonomic nervous system activity in HK children is generally not different on resting, baseline measures. However, consistent results on the impact of environmental stimulation seem to furnish evidence of underreactivity or underarousability in some of these children. Stimulant medication is generally arousal-producing, energizing, and has a normalizing influence on many cortical and autonomic measures. In some cases, similar effects have also been observed in normal children, thus adding more credence to the supposition that a strictly organic differentiation between HK and normal children is unlikely (Hastings, & Barkley, 1978).

Several investigators have speculated upon the location or site of dysfunction by proposing relatively specific anatomical or functional models. With regard to the notion of overstimulation or hyperarousal, Laufer, Denhoff and Solomans (1957) invoked a deficit in diencephalic inhibitory mechanisms to account for the inability of HK children to screen irrelevant stimulus input and decrease their activity levels. However, the physiological and environmental stimulation literature has generally failed to confirm the concept of an overarousal disorder in these children.

Decreased activity in the reticular activating system (Satterfield, & Dawson, 1971; Satterfield, Cantwell, & Satterfield, 1974; Zentall, 1975) or dysfunction in forebrain inhibition (Rosenthal, & Allen, 1978) have been offered as possible explanations in accounting for the hypoarousal data. The physiological evidence of lower cortical (ongoing tonic EEG, phasic alpha blocking, EP correlates) and autonomic (phasic skin conductance and HR, pupillometric measures) arousal indices, as well as deficit attentional abilities, are consistent with this position. In addition, the majority of the stimulant drug literature has demonstrated the enhancement of generalized arousal on many measures in HK. However, the inconsistent research results point more to decreased arousability which may be task or situation-specific, than to a general reduced arousal level per se.

Some authors have proffered deficits in specific neurotransmitter substances such as serotonin (Coleman, 1971), acetylcholine (Sheer, 1976), norepinephrine (Wender, 1974), and dopamine (Arnold et al., 1972; Shaywitz, Yager, & Klopper, 1976; Wender, 1971). The evidence in support of any one of

these neurotransmitters has been somewhat limited, often indirect, and as yet not very compelling. The vagueness surrounding many of these models has resulted in correspondingly imprecise research and clinical predictions, and presently no parsiminious arousal theory has been offered to account for the heterogeneity observed in HK children.

Attention and Activity Levels in Hyperkinesis

There is a consensus that the fundamental deficit in HK is cognitive, consisting of an inadequate ability to focus and maintain (sustain) attention. In this view, behavioral hyperactivity has been considered epiphenomenal (Douglas, 1972; Tarver, & Hallahan, 1974). This is reflected to some extent by the new designation of attention-deficit disorder in DSM-III replacing the previous diagnostic classification of hyperkinetic reaction of childhood (DSM-II).

Reaction time (RT) and vigilance tasks have been the most commonly used measures to evaluate attention in experimental situations. In a simple RT task, subjects execute a behavioral act (e.g., pressing a response button or releasing a lever) in response to the onset of a sensory stimulus (usually visual or auditory in nature). On such tasks, HK children relative to normal controls, typically exhibit longer latencies to behavioral response (Barkley, 1977; Cohen, & Douglas, 1972; Douglas, 1972; Firestone, & Douglas, 1975; Firestone, & Martin, 1979; Grunewald-Zuberbier et al., 1975; Klorman et al., 1979; Porges et al., 1975; Spring, Greenberg, Scott, & Hopwood, 1973; Zahn et al., 1975) and greater intraindividual variability in response latency

(Cohen, & Douglas, 1972; Sroufe et al., 1973). Cohen and Douglas (1972) and Spring et al. (1973) have also reported a significant deterioration in RT performance across repeated trials for HK subjects, whereas Sroufe et al. (1973) failed to find any notable intergroup or across session differences. When more complex RT tasks (choice or serial) were evaluated, Sykes, Douglas, and Morgenstern (1972) reported no differences between groups. It has been argued that more complex RT tasks provide more stimulating conditions for HK subjects, thereby obviating attentional deficiencies with such results supporting optimal level of stimulation theory.

Stimulant drugs have been found to significantly improve RT performance (Barkley, 1976; Barkley, 1977; Cohen et al., 1971; Firestone, Davey, Goodman, & Peters, 1978; Firestone, & Douglas, 1975; Sprague, & Sleator, 1977; Spring et al., 1973; Spring, Yellin, & Greenberg, 1976; Sroufe et al., 1973; Ullman, Barkley, & Brown, 1978; Zahn et al., 1975), reduce intrasubject variability (Cohen et al., 1971; Sroufe et al, 1973), and decrease performance deterioration across trials (Spring et al., 1973) in HK children. The normalizing effect of these drugs may not be complete, however, since Spring et al. (1973) found that medicated HK subjects still had inferior RT latencies when compared to controls.

Similar deficits in attentional abilities have been demonstrated in a variety of studies requiring sustained attention (vigilance) to detect rarely-occurring signals against a background of regularly occurring nonsignal stimuli. Relative to control subjects, HK children perform poorly on this type of task, i.e., making fewer correct detections of signals and more

false positive responses (errors of commission) to nonsignal stimuli (Anderson, Halcomb, & Doyle, 1973; Conners, & Rothschild, 1968; Conners et al., 1972; Kaspar, Millichap, Backus, Child, & Schulman, 1971; Keogh, & Margolis, 1976; Klorman et al., 1979; Sykes et al., 1972; Sykes, Douglas, & Morgenstern, 1973; Sykes et al., 1971). Stimulant drugs have been reported to increase correct detections and decrease errors of commission (Conners, & Rothschild, 1968; Sykes et al., 1971; Sykes et al., 1972; Werry, & Aman, 1975). Klorman et al. (1979) found a significant decrease in false positive responding, while Conners et al. (1972) reported no medication effects on vigilance performance.

A third measure used to experimentally assess attention has been to document the amount of time HK children spend on various activities and the number of activity changes during free play (Barkley, 1977; Barkley, & Ullman, 1975; Pope, 1970; Rapoport, Abramson, Alexander, & Lott, 1971). HK children are reported to spend less time on various activities and to make more frequent activity changes relative to control children - a pattern suggesting decreased attention span. Stimulant drug treatment has been found to have no effect (Ellis, Witt, Reynolds, & Sprague, 1974) or to decrease the number of activity changes (Barkley, 1977; Rapoport et al., 1971).

The term "hyperkinesis" has served to highlight the more traditional description of excessive restlessness and increased activity levels noted in many of these children. Consequently, the focus of many studies has been directed toward empirical validation of this behavior. Laboratory investigations of activity levels have used a wide variety of methods to quantify

this construct. Movement has been evaluated using pedometers, actometers attached to subjects' limbs (wrist, leg), grid-marked playrooms, stabilometric cushions, photoelectric cells, ultrasonic sensors, pneumatic pads, video-taping, radio telemetry, and behavioral checklists in such settings as in the classroom, during experimental laboratory testing, and in free play (Barkley, 1977; Barkley, & Ullman, 1975; Davis, Sprague, & Werry, 1969; Montagu, & Swarbrick, 1974; Pope, 1970; Rapoport et al., 1971; Sprague, & Toppe, 1966; Ullman et al., 1978). However, the diversity of these measures and the types of activity they measure (from discrete, segmental limb movements to global body movements) has made interstudy comparisons somewhat difficult and the differentiation of HK from normal children correspondingly inconsistent. Furthermore, the reliability and validity both across measures and within measures across time have compounded difficulties in interpretation of these inconsistencies (Johnson, 1971; Whalen, & Henker, 1976).

In general, the majority of studies have shown that within unstructured situations (free play), HK subjects are no more active than controls, and drug treatment fails to influence this variable in these situations of relatively unrestricted movement (Ellis et al., 1974; Rapoport et al., 1974; Sleator, & von Neumann, 1974). In contrast, in structured settings (e.g., classroom learning and laboratory situations), HK children consistently exhibit increased motor restlessness relative to controls (Juliano, 1974; Sykes et al., 1971), and stimulant drug treatment has been shown to decrease this excessive motoric activation (Barkley, 1977; Sprague, Barnes, & Werry, 1970; Werry, & Aman, 1975). Two recent studies (Barkley, 1977; Ullman et al.,

1978) compared activation levels derived from a number of different movement measures (grid-marked room, wrist and leg actometers, stabilometric chair), assessed in varying situations (free play, movie-viewing, structured testing, and restricted free play). On all activity measures, HK subjects showed more movement compared to controls across all conditions. Activity on all four measures across all situations was significantly reduced following methylphenidate administration. This conflicts with previous research demonstrating situation-specific, as well as measure-specific HK.

In summary, the evidence supports the notion that HK children can be differentiated from normal children by their inability to focus and maintain attention and by their excessive motor activation in relatively structured settings.

Sleep and Hyperkinesis

The psychophysiological literature reviewed above implies that a common feature in some children exhibiting the hyperkinetic behavioral reaction may be a dysfunction in the arousal-producing mechanisms. Since both sleep and waking (arousal) mechanisms are highly correlated, the study of sleep in HK children may furnish evidence of a postulated arousal dysfunction under conditions free from waking confounding variables such as stress, expectations, and undefined variations in arousal level. In addition, the study of sleep in this disorder may reveal HK syndrome-related events and relationships which might be obscured or not present during wakefulness.

The first published study investigating sleep patterns in HK children focused upon the amount of rapid eye movement (REM) sleep (Luisada, 1969). A common behavioral effect of the deprivation of REM sleep in both animals and man has been increased activity as well as irritability (Dement, 1969), and Luisada postulated that HK children should exhibit measurable variations in baseline REM amounts. In this study, the sleep of 11 HK children (mean age = 9.2 years) was compared to a control group (n = 4, mean age = 9.5 years). Comparisons were based on only two sleep measures: REM percentage and percentage of REM awakenings. Significant differences were shown for both, i.e., HK children displayed less REM sleep (20% versus 24%) and more of their REM periods were disrupted by awakenings (21.5% versus 4.5%). Also, a positive correlation was observed between the individual degree of qualitatively observed HK and percentage of REM awakenings.

Small, Hibi and Feinberg (1971) examined the sleep of three markedly HK males with minimal brain dysfunction (MBD) compared to seven age matched normal controls during baseline (five consecutive nights) and medication periods (dextroamphetamine; three consecutive nights, five mg. each morning; three consecutive nights using an optimal dosage of $\geq$ 20 mg./day; three consecutive nights on placebo after medication withdrawal). Comparisons were made on a number of sleep measures including total sleep time, amounts and distribution of sleep stages, and events within sleep stages (eye movements during REM and muscle activity during both REM and NREM sleep). Significant differences during baseline were found on only four measures, three of which were related to muscle activity (HK subjects had a greater

percentage of 20-sec epochs containing muscle activity relative to controls), and the fourth showing a shorter latency to sleep onset in HK children. Medication administration increased sleep onset latency, absolute and percentage amounts of time awake during total bed time, latency to the first REM period, and percentage of slow wave sleep (stages 3 and 4) as a proportion of total NREM sleep. Surprisingly, other sleep pattern measures were not significantly altered.

In a further investigation of amphetamine effects on the sleep of MBD children, Feinberg, Hibi, Cavness, Westerman, and Small (1974) studied eight HK males (three of which were from the above reported study) during baseline (five nights), while taking stimulant drugs (three nights), and during withdrawal (two or three nights). Sleep was compared to six age matched male controls who slept four or five consecutive nights in the sleep lab. No significant differences among sleep measures were reported between MBD and normal children during baseline or medication conditions. With stimulant drug administration, eye movement density and eye movement burst index were significantly increased, while after stimulant withdrawal, sleep latency was reduced and sleep time increased compared to baseline MBD group measures.

Haig, Schroeder, and Schroeder (1974) compared the sleep of six hyperactive boys (ages 6.5-12 years) receiving methylphenidate treatment (dosages of 10-61.5 mg/day) with that of six unmedicated normal controls from a previous study. In the main portion of the study, subjects spent five consecutive nights in the sleep lab, of which the last three were recorded polygraphically. In an extended part of the study, four of the six hyperactive

subjects were recorded three full nights one week subsequent to medication withdrawal. These authors observed a significant increase in sleep onset latency and latency to the first REM period in medicated hyperactive children compared to controls. No significant medication/nonmedication differences with respect to sleep measures were noted for this small sample of children recorded under both conditions.

Nahas and Krynicki (1977) studied the effects of methylphenidate on the sleep of four HK males (ages 8-9 years) in an eight night paradigm: two adaptation nights (electrodes attached but no recordings); two baseline, nonmedication recording nights; two experimental nights with medication administration (20 mg total) occurring on the first and final (21st) days of medication; and, two medication withdrawal recording nights immediately following drug discontinuation. No significant across-medication differences were observed with regard to total sleep time, sleep stage amounts, latency to REM onset, movement time, or number of stage changes. In addition to the above measures, three nights of data, one from each recording condition, were examined for ultradian rhythms known to be present for REM and delta (stages 3 and 4) sleep (Naitoh, Johnson, Lubin, & Nute, 1973). Nocturnal ultradian rhythms were found to be present, and as well, an increase in delta cycle length after drug withdrawal was noted.

Khan and Rechtschaffen (1978) compared the sleep of five HK males (aged 6 to 8 years) with that of seven normal children (5 boys and 2 girls aged 6 to 8 years) over three consecutive nights (1 adaptation and 2 experimental nights). No significant group differences were reported for percentages of

total sleep time or any of the stages, however, the HK group did show decreased 12-14 Hz sleep spindle production relative to control subjects. Spindle scores doubled in frequency and clinical improvement were noted following treatment with methylphenidate. In addition, three older boys (12 to 13 years) who were previously diagnosed as HK but now showed less overt restlessness, had similar spindle scores to those of similar aged controls.

Lastly, Stahl, Orr, and Griffiths (1979) investigated the sleep and nocturnal levels of growth hormone in five nonmedicated HK children of small stature and in nine normal control subjects over four consecutive nights. Growth hormone amounts and peaking occurred within the normal range and no sleep parameters differed significantly between the two groups.

Considered together, these studies have examined sleep patterns in 39 HK children. All studies except one (Luisada, 1969) have essentially agreed that: a) baseline sleep is not remarkably different in HK children relative to normal controls; and b) stimulant medication given to HK children does not significantly alter their sleep patterns. However, a number of limitations exist in these studies. Across studies, symptomatology and subject selection procedures have varied, ranging from diagnoses based solely upon the presence of restlessness to more complex symptom clustering, some of which may have had a strong organic foundation. When stimulant medication has been administered, drugs and dosages have differed making interstudy comparisons difficult. The ability to generalize from these studies is somewhat limited due to: a) small samples of both HK and normal controls; b) in one study the use of extremely limited measures; c) in two studies the lack of control recordings; and, d) differing design paradigms (e.g., number

of nights recorded, medication/nonmedication, etc.). One of the objectives of the present study was to undertake the investigation of the sleep morphology of a relatively large sample of nonmedicated HK and normal children over a substantial baseline recording period. A more homogeneous HK sample was selected, with excessive motor restlessness not being the primary presenting symptom, in order that the more "typical" HK child would be studied. In addition, this study added to the pre-existing literature on sleep patterns in this pediatric age group.

Ultradian Rhythms

Over the past decade, the existence of oscillatory phenomena has been delineated in a number of normal physiological and behavioral, as well as pathological functions in man during sleep and wakefulness. These studies have opened up a new area of research often referred to as chronobiology (Halberg, 1964) which focuses upon the investigation of varying biological time structures. The rhythmicity of events recurring more frequently than once per day (more than one cycle in 20 hours) has been termed "ultradian". Those cycles fluctuating approximately once per day (one cycle in 20-28 hours) are called "circadian", while those less frequently occurring than once per day such as monthly, seasonly, or yearly frequencies have been designated as "infradian" rhythms (Halberg, 1964; 1969). Thus the sleep-waking cycle in adults would be classed as circadian, with the more rapid periodicity of the NREM/REM cycle within sleep being ultradian in nature. Kleitman (1963; 1969) first proposed that the cyclic occurrence of REM sleep was a prime example of the existence of a more basic, fundamental Basic Rest-Activity

Cycle (BRAC) which was believed to be continuous throughout the 24 hour period. Rhythms that had been shown in various waking variables have been observed to recur at about the same rate as the NREM/REM sleep cycle. In young children this cycle length is about 45-60 minutes; in adolescents approximately 70-90 minutes, and in adults about 80-120 minutes (Dement, & Kleitman, 1957; Roffwarg, Muzio, & Dement, 1966).

Investigations of waking ultradian rhythms of physiological, behavioral, and pathological variables have to date been limited primarily to study in adult populations. Initial evidence for waking ultradian rhythms was furnished before intra-sleep periodicity was known. Wada (1922) first described rhythmic gastric contractions during waking occurring at 90-110 minute intervals. With the advent of technological sophistication (EEG) and increasingly detailed behavioral observations enabling a more direct analysis of sleep morphology, came a striking observation. The polycyclic occurrence of sleep-waking periods and the intra-sleep periodicity of sleep stages in neonatal and infant sleep showed definite "rest-activity" cycles (Aserinsky, & Kleitman, 1953; 1955). From these initial discoveries, numerous nocturnal ultradian rhythms have since been noted for many variables during sleep in man and animals. Recurrent rhythms of about 10-20 cycles/day (72-144 minutes) have been shown for rapid eye movement (REM) sleep, non-rapid eye movement (NREM) sleep, stage 4, phasic eye movement activity, and various EEG frequency bands (Globus, 1970; Hilbert, & Naitoh, 1972; Kripke, 1972; Lavie, 1979a; Lubin, Nute, Naitoh, & Martin, 1973; Naitoh, Johnson, Lubin, & Nute, 1972; Sinha, Smythe, & Zarcone, 1972). A similar range of periodicities during

sleep has also been observed for measures such as metabolism (Brebbia, & Altschuler, 1968), brain temperature (Kawamura, Whitmoyer, & Sawyer, 1966; Reite, & Pegram, 1968), gastric contraction and acid secretion (Lavie, Kripke, Hiatt, & Harrison, 1978; Kales, & Tan, 1969), blood pressure (Coccagna, Mantovani, Brignani, Manzini, & Lugaresi, 1971), genital engorgement (Karacan, Hursch, Williams, & Thornby, 1972), and arousal threshold (Pollak, Weitzman, & Kripke, 1967). In addition, several endocrine-related measures have exhibited cycling characteristics within the ultradian range during the 24-hour period, including plasma concentrations of prolactin, ADH, and luteinizing hormones (Boyar, Perlow, Hellman, Kopen, & Weitzman, 1972; Mandell, Chaffey, Brill, Mandell, Rodnick, Rubin, & Sheff, 1966; Rubin, Kales, Adler, Fagan, & Odell, 1972; Weitzman, Schaumberg, & Fishbein, 1966). Growth hormone which is known to reach a secretion peak during stages 3-4, exhibits a 90-100-minute cycle during the beginning of sleep (Parker, Sassin, Mace, Gotlin, & Rossman, 1969; Quabbe, Schilling, & Helge, 1966). VMA (Mandell, Mandell, Rubin, Brill, Rodnick, Sheff, & Chaffey, 1966) and dehydroiso-androsterone (Rosenfeld, Hellman, Roffwarg, Weitzman, Fukushiama, & Gallagher, 1971) also have shown evidence of episodic secretion patterns.

This knowledge concerning the presence of episodic phenomena during sleep piqued interest into the interrelationships among BRAC rhythms, diurnal cycles, and daytime functions. Wada's (1922) first report of waking gastric motility cycles in man has recently been confirmed in a study by Hiatt and

Kripke (1975). Oral activity in humans confined to an observation room and allowed ad libitum access to food, drinks, and cigarettes revealed a prominent 96 minute rhythm (Friedman, & Fisher, 1967). Oswald, Merrington, and Lewis (1970) replicated Friedman and Fisher's original study confirming the presence of cyclic oral behavior (90 minutes). Kripke (1972) added further support to the notion of cyclic orality by demonstrating peaks in operant bar presses for water in a situation in which subjects were monitored both behaviorally and physiologically (EEG). Similar rhythmicity in oral behavior was also observed in a group of schizophrenics (Friedman, 1968). In a group of obese subjects, Friedman (1972) showed that regular feeding peaks occurred more frequently than those for normals (mean period of 78.8 minutes) and that an individual's degree of cycle shortening was significantly correlated with the degree of obesity. Cyclicity (10-20 cycles/day) was also shown for EEG delta activity and other EEG frequencies. This suggests that a correlation might exist between physiological and behavioral rhythms.

Similarly, using complex computer data processing methods, oscillatory activity has been noted for heart rate (Orr, & Hoffman, 1974), body temperature (Orr, Hoffman, & Hegge, 1976), respiration rate (Horne, & Whitehead, 1976), and urine flow reflecting episodic ADH secretion (Lavie, & Kripke, 1977). Other physiological measures which include alertness as measured by pupillometry, (Lavie, 1979b) cerebral responsiveness measured as evoked potentials (Tanguay, Ornitz, Forsythe, Lee, & Hartman, 1973) and gross body movements (conclude evidence is weak) measured telemetrically (Globus, Phoebus, Humphries, Boyd, & Sharp, 1973) have shown some evidence of rhythmicity. In

addition, a number of psychological phenomena such as perceptual illusions using the spiral aftereffect (Lavie, Levy, & Coolidge, 1975; Lavie, 1976; Lavie, 1977), Rorschach responses (Globus, 1968), and performance on a variety of tasks (e.g., Rosvold-Mirsky CPT, verbal and spatial matching, complex detection task) have demonstrated a cyclic nature (Globus, Drury, Phoebus, & Boyd, 1971; Klein, & Armitage, 1979; Orr, & Hoffman, 1974; Orr, Hoffman, & Hegge, 1974; Orr, Hoffman, & Hegge, 1976).

The animal literature is not nearly as extensive with regard to the equivalent biorhythms, but some similar findings have been obtained. In virtually all species for which sleep data are available, most show cyclicity either within sleep (so-called quiet/active sleep, orthodox/paradoxical, or NREM/REM) or across states of rest and activity (Tauber, 1974). Ultradian rhythms of various sleep variables (NREM/REM, EEG frequencies, muscle tonus, and eye movements) have specifically been reported in rhesus monkeys (Kripke, Halberg, Crowley, & Pegram, 1970) and in the cat (Sterman, 1972; Sterman, Lucas, & Macdonald, 1972). In monkeys a 24 cycle/day (60 minutes) rhythm was observed in the NREM/REM cycle. In the cat, a 20 minute NREM/REM rhythm was shown. Within wakefulness, operant EEG responses for food, operant bar presses for food, and operant EEG responses for electrical brain stimulation all have showed a similar 20 minute rhythm in the cat (Sterman, Lucas, & Macdonald, 1972). Maxim and Storrie (1979) have recently reported the presence of a 40-45 minute fluctuation in bar pressing for rewarding brain stimulation in rhesus monkeys. Reports have been published demonstrating 12-18 cycles/day (80-120 minutes) in ingestion, locomotion, exploration, grooming, and resting in a rhesus monkey sample (Bowden, Kripke, & Wyborney, 1978). Also, Maxim, Bowden,

and Sackett (1976) noted cyclicity (multiples of 45 minutes) in social interaction between rhesus monkey pairs and nonsocial (solitary) behaviors such as ingestion, exploration, locomotion, and self-grooming.

After delineating the basic structure as well as some of the psychological concomitants (e.g., mentation) of sleep, an important question arose as to whether some analogous phenomenon to REM might also be occurring during wakefulness. Othmer, Hayden, and Segelbaum (1969) studied subjects in a variety of isolation conditions and demonstrated the presence of periods of rapid eye movement activity and decreases in muscle tone recurring cyclically throughout the 24-hour period. They also reported a correlation of eye movement activity with "dramaturgic" daydreams. However, the results were not supported by formal statistical or spectral analyses. Recently, Kripke and Sonnenschein (1973; 1978) reported a study in which subjects were asked to write down their thoughts every five minutes over a 10-hour period. The mentation reports were than randomized and two raters independently scored them for degree of fantasy content. The findings indicated a dominant 90-100 minute fantasy rhythm. Lavie and Kripke (1975) have shown further, that these fantasy cycles correlated more positively with ocular quiescence, suggesting a shift to internal processes from external stimulation. This contradicts Othmer et al.'s (1969) findings relating ocular activity to daydreams.

Only a small number of studies attempted to document the phase relationships of these waking oscillations to the preceding or subsequent REM period occurrence during sleep. It would seem important in establishing the existence of a fundamental BRAC around the 24-hours to examine phase characteristics between waking and sleep rhythms. Destrooper and Broughton (1969)

reported anecdotal evidence for dramatic shifts in mentation descriptions at about the time of morning when a subject's next REM period might have been expected, had sleep been allowed to continue. Globus (1972) had subjects perform a visual detection task for 150 minutes following morning arousal and found that errors peaked 90-100 minutes after the mid-point of the preceding REM period; likely when the next one would be predicted had sleep progressed. Similarly, Sterman, Lucas, and Macdonald (1972) demonstrated that eating behavior and rewarding electrical brain stimulation in cats paralleled each other in cycling frequency and were phase-related to the preceding NREM/REM alternations. Furthermore, the occurrence of REM during daytime naps (Globus, 1966) and narcoleptic REM sleep attacks (Passouant, Halberg, Genicot, Popoviciu, & Baldy-Moulinier, 1969) are both phase-locked to nocturnal REM periods. Lastly, some evidence has been gathered showing that temporal lobe and typical petit mal absence discharges in the waking state increase rhythmically every 90-100 minutes and occur in the same phase relation to the NREM/REM cycle at night (Stevens, Kodama, Lonsbury, & Mills, 1971; Jovanovic, 1979). These few studies then, provide initial support for the existence of a common BRAC operating throughout both sleep and waking states.

Based upon the above evidence, it appears that many physiological and behavioral (sensations, autonomic variables, hormonal levels, motor activity, and complex cognitive functions) as well as some pathological (narcolepsy, epilepsy) states exhibit ultradian variations. These cyclic fluctuations can be observed across species as well as in man. The significance of these oscillations however, has remained rather speculative. In any case, a few

conceptual suggestions as to function have been offered. Wolff (1967) has proposed that ultradian rhythms supply a controlling function for early motor development in the infant. He suggests that initial spontaneous rhythmical motor activity such as sucking, crying, and kicking may be part of a maturational continuum and that the transition from simple repetitious behaviors into integrated action patterns is but one aspect of development. These early cyclic behaviors might form the basis for later more complex behaviors such as speech which requires increased differentiation of fine muscular movements. However, as yet no direct empirical support has been provided for this position. The earliest appearance of the BRAC has been reported to be present in intrauterine human fetuses (Granat, Lavie, Adar, & Sharf, 1979; Petre-Quadens, 1966; Sterman, 1967; Sterman, & Hoppenbrouwers, 1971). Emde, Swedberg, and Suzuki (1975) noted a definite sleep-wake cycle (3.5-4 hours) and BRAC (39 minutes) in neonates within the first 10 hours after delivery. Furthermore, Stern, Parmalee, and Harris (1973) have shown that in premature infants, the NREM/REM cycle lengthens as the infant grows older (30 minutes in prematures to 55 minutes at eight months). Investigations of the development of the sleep-waking cycle and other rhythms show that with maturation, these periodicities increase (Roffwarg, Muzio, & Dement, 1966). This also appears to be the case with the postulated underlying BRAC (Kripke, 1974).

Support for Kleitman's (1969) notion of the existence of this "basic rest-activity cycle" is based upon evidence of the resemblance of the NREM/REM cycling frequency and waking measure periodicities, the fact that both reflect fluctuations in brainstem and cortical arousal and autonomic activity, and in

some cases, reports of phase relationships between the occurrence of the NREM/REM cycle and waking rhythms (Lavie, 1979). From a developmental perspective, the sleep-waking cycle is at first polyphasic throughout the 24-hour period. However, as maturation progresses, this rhythm begins to consolidate into a single nocturnal sleep period resulting in a lengthening of the waking daytime period. The progression of ultradian rhythms in many variables follows a similar path, i.e., a lengthening of the periodicity from the nocturnal (70-90 minutes) to the waking state (100-120 minutes) (Kripke, 1974).

Broughton (1975) has proposed that a cyclic interhemispheric alternation may be responsible for the subjectively-experienced waking and sleeping oscillations between fantasy-intuitive and verbal-intellectual psychological processes. These qualitative changes in cognitive processing in wakefulness are thought to be analogous to the fantasy-like reports obtained from REM sleep arousals and the more logical expression inherent in reports obtained from NREM sleep. He postulates that the continuation of these rhythmic thought processes into waking represents the continued cyclic alternation of left and right hemispheric activity. This has been supported by a recent study showing a 96 minute rhythm in both verbal and spatial processing but 180^{0} out of phase to each other (Klein, & Armitage, 1979). Broughton has further proposed that NREM sleep (stages 3-4) may provide a primary synchronizing mechanism for the body's biological clock which is extremely sensitive to stress and external environmental time cue disruptions. The psychological and behavioral evidence of these rhythmicities may be manifestations of the postulated underlying BRAC.

From a psychophysiological approach, there is a strong correlation between the physiologic REM state and the psychological experience of dream mentation. Morruzzi (1969) has suggested that REM sleep is part of an instinctual consummatory process involved in the satisfaction of basic needs. Waking ultradian rhythms also appear to be expressions of the consummatory phase in the regulation of these appetitive needs. Overt behavioral acts which include eating, movement, sexual expressions etc. and internal processes such as hormonal rhythms and subjective fantasy may be related to a more fundamental ultradian oscillator system (Kripke, 1974). At a more general level, the survival value for the individual and the species of a modulated series of continually waxing and waning behaviors would be more adaptive than each motivational need seeking expression simultaneously. The cyclic oral and gastric activity, genital engorgement, and readiness to respond to significant stimuli which might threaten safety, all would ensure evolutionary adaptation for species survival (Broughton, 1974; Kripke, 1974).

Recently, some evidence has been proposed showing that the BRAC may not be the only oscillatory system. Various nocturnal ultradian rhythms do not parallel each other in occurrence, i.e., these rhythms are not synchronous with the NREM/REM cycle. For example, nocturnal gastric contractions are not concurrent with the REM sleep cycle (Lavie, Kripke, Hiatt, & Harrison, 1978), nor are these gastric cycles related to similar rhythms in fantasy or EEG activity (Hiatt, Lavie, & Kripke, 1975). Lavie (1979) has postulated that the BRAC is, but one of probably multitudinous ultradian rhythms. The BRAC, therefore, may not be the basic fundamental oscillator upon which other

periodicities are superimposed. This implies the existence of more than one oscillator generator system which have periodicities of about 80-100 min in the awake adult human.

Although ultradian fluctuations have been documented in normal adults with respect to perceptual and attentive functions and gross motor activity (Globus et al., 1971; Globus et al., 1973; Orr et al., 1976), the nature of cyclic behavior has not been previously reported for any variable in either normal or clinical pediatric groups. The demonstrated presence of ultradian rhythms in these groups could provide information regarding improved prediction of cyclical periods of inattentiveness, distractibility, and increased activity. These predictions would be especially useful for optimal utilization in the scheduling of various activities, e.g., thos related to learning particularly in HK children.

Hypotheses

Sleep. Based upon previous studies examining sleep patterns in HK children, it is hypothesized that no significant differences in baseline amounts and distribution of the various sleep stages will be obtained between groups, but that phasic activation (e.g., motoric, autonomic, and EM measures) will discriminate HK from control subjects. Relative to control subjects, hyperaroused HK children would have increased activity in these phasic indices, whereas, if HK children are hypoaroused, decreased activity in these phasic measures is predicted.

Waking Performance and Motility. The variables are assessed to confirm and extend (over multiple testing sessions) the existence of an attention deficit and increased motor activity in HK compared to control children. It is predicted that HK subjects will make fewer correct detections and more false positive responses

on the brief visual attention task and will display greater gross body motility and specific limb movements while on task compared to controls. No group differences in limb motility off-task are postulated.

Waking Ultradian Rhythms

Since rhythmicity in perceptual and motility variables has been shown in adults, it is hypothesized that ultradian cyclicity will be demonstrated in attention and activity in both HK and control subjects. However, it may be that by so yet some unknown mechanism, these oscillatory changes may be abnormally accentuated in the HK disorder. These variations then, could contribute to the attentional deficiencies observed in these children. In addition, as suggested by Cromwell, Baumeister, and Hawkins (1963), attentional deficiencies and increased motility may be intimately related. The possibility of rhythmicity in these variables suggests that complex phase relationships may exist between levels and amounts of motor activity and attention. With regard to motility, the possibilities might include any of the following: a) a general overall increase in motility levels at all BRAC points; b) the selective increase of the activity poles of the BRAC; or c) an increase in cycle frequency with activity peaks occurring more frequently. It is hypothesized that the second possibility, i.e., a selective increase in the active poles, is most likely since in many experimental situations HK children display a greater quantity of movement relative to controls.

With regard to relationships existing between sleep and waking ultradian rhythms, if cyclicity in performance and motility exist, it is hypothesized that these waking rhythms will represent an in-phase continuation of the preceding nocturnal ultradian REM sleep cycle.

Chapter II

METHODOLOGY

Subjects

Eleven unmedicated HK male children (8-12 years old) and 11 similar aged unmedicated male control children participated in the study. HK subjects were initially referred by family physicians who suspected hyperkinesis, and referred them for more extensive psychological assessment at the Psychology Department, Children's Hospital of Eastern Ontario. As well as positive indications derived from Conners Parent (1970 - Appendix A) and Conners Teacher (1969 - Appendix B) behavioral rating scales (a score of 15 or more on the hyperactivity index), diagnosis was based upon the following core symptomatology which is generally agreed upon as being persistently on recurrently present in children labelled HK (DSM-III criteria of attention/deficit disorder with hyperactivity): motor restlessness, short attention span, distractibility, impulsiveness, labile emotions, and poor academic performance (Goyette, Conners, & Ulrich, 1978; Renshaw, 1974; Stewart, Pitts, Craig, & Dieruf, 1966; Wender, 1972). All HK subjects displayed this behavioral symptomatology from an early age (before three years of age).

The control group of normal children was recruited from the local school systems. All of these children scored negatively on the Conners Parent and Teacher questionnaires.

All children in the study were living at home with at least one parent. Based upon clinical and physician interviews children with the following symptoms or classifications were excluded from both groups: major psychosis, over-anxious reaction, unsocialized aggressive reaction, peripheral sensory loss, epilepsy, normal constitutional hyperkinesis, mental retardation, post-traumatic organic brain syndrome, encephalitis, toxic organic brain syndrom (drug), or major sleep disturbances (e.g., enuresis, somnambulism, pavor nocturnus).

Prior to acceptance into the study, each child underwent an I.Q. evaluation (WISC-R full scale I.Q., lower limit of 80), as well as a baseline EEG recording to screen for the presence of gross EEG abnormalities. This short recording session (about 30 minutes duration) also served to familiarize each child with the recording environment, procedures, and apparatus. Informed consent (parents or legal guardian signature) was obtained and a full explanation of the study was given to both parents and children. Subject group characteristics are summarized in Table 1.

Polysomnographic Recordings

Subjects reported to the sleep laboratory (Ottawa General Hospital) for electrode application one hour before bedtime. Electroencephalographic (EEG: C_3/A_2), electro-oculographic (EOG: bipolar DC recordings of horizontal and vertical eye movements), and electromyographic (EMG: facial muscle) were obtained using a Grass (Model 78D) polygraph. Spontaneous skin potential responses (SSPRs: volar surfaces of the left middle finger referenced to the forearm) were also recorded. All night sleep recordings were made for five consecutive nights. Total bed time (TBT) was limited to 9.25 hours.

Waking Ultradian Rhythm Measures

Subjects performed multiple repetitions of a brief visual attention task on two consecutive days subsequent to polygraphic sleep recordings (i.e., on the days following the third and fourth nights). On the mornings of these daytime testing days, subjects, after eating breakfast, were transported from the hospital location where the sleep recordings were obtained, to a university laboratory for daytime testing which began approximately 45 minutes after awakening.

Table 1

Mean (S.D.) Age, I.Q., and Behavioral Ratings for Hyperkinetic and Control Groups

Variable	Group	
	HK	CONTROL
Age	10.6 (1.7)	10.6 (1.3)
Full Scale I.Q.	106.4 (10.2)*	123.2 (13.2)
Verbal I.Q.	104.7 (11.3)*	124.6 (12.5)
Performance I.Q.	106.9 (12.0)	116.6 (14.9)
Conners Parent (HI)	19.5 (6.9)**	4.6 (2.1)
Conners Teacher (HI)	19.1 (5.5)**	3.0 (2.8)

*$p < .01$

**$p < .001$

To obtain sufficient data to document a possible BRAC modulation of attention, five-minute test periods were administered every 15 minutes. During the inter-test "rest periods" (10 minute intervals), children engaged in supervised free play. This schedule extended over a six-hour period (24 sessions/day). During the supervised rest periods, subjects were allowed to read, play games, or be involved in other activities of their choice. Bathroom and lunch breaks also took place during these intervals.

The visual attention (sustained vigilance) task used was a modified continuous performance task (CPT) in which responses were made to a preselected sequence of two letters (e.g., AP) appearing in a continuous letter sequence of 12 possible letters and their combinations; the predetermined paired letter signals occurring randomly 30 times during each testing period (Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956). Subjects were seated approximately one metre from a light emitting diode (LED) display. Responses were made with a hand-held button press using the dominant or preferred hand. Letter presentation and task duration were controlled by an electronic micro-processor unit. Each letter appeared for 0.2 seconds at 1.5 second intervals.

While performing on the CPT, subjects were seated on a sensitive electronic movement indicator (stabilometric chair) which provided the frequency (digital display) of global body movements (Farrall Instruments; adapted from Sprague, & Toppe, 1966). In addition, segmental limb movements during the task (LM-T) were measured using a movement/acceleration integrator watch (Selig Electro-magnetic Ltd.) secured around the ankle of the dominant or

preferred leg. Limb movement during the rest or off-task periods (LM-RP) was also measured using the same device. Subjects were trained and familiarized with the CPT and movement recording devices in a training session (one hour duration) on the afternoon before the first full day of testing.

Data Analysis

Polysomnographic recordings. Sleep records were coded and scored blind by two individuals using the standardized criteria of Rechtschaffen and Kales (1968). A greater than 90% reliability was obtained on pilot data prior to actual data scoring. Spontaneous skin potential responses (SSPRs) were scored according to the criteria of Johnson and Lubin (1966) with a one millimeter pen deflection representing a 100 uV potential change. The input impedance of the SPR amplifier was set at one megohm. Rapid eye movement (REM) density measures were scored using Aserinsky's (1969) methodology (number of two-second mini-epochs containing at least one eye movement divided by the total number of REM mini-epochs). Eye movements were defined by a one millimeter pen deflection which represented a five degree visual angle shift.

In addition, several nocturnal motility variables were measured. These included movement time (minutes and percentage), body movements, REM twitches, and NREM twitches. Movement time was scored as a prolonged burst in EMG activity with obscurring of the EEG and EOG patterns. Body movements were scored as phasic increases in EMG activity with a duration greater than one second and an amplitude at least twice that of the preceding tonic EMG tracing. Twitches were scored as phasic EMG bursts of one second or less duration and with an amplitude at least twice that of the preceding tonic EMG activity.

The first two recording nights were considered laboratory adaptation nights and were therefore excluded from group comparison analyses. However, all nights were included in analyses of trends across the five-night recording period. Statistical comparisons between groups (Factor A) and across nights (Factor B) for the various sleep measures were made using individual repeated measures analysis of variance (BMD program P2V with orthogonal component analysis to test for trends), t-tests, and when appropriate, post-hoc Newman-Keuls tests. The .05 level of significance was used for all statistical comparisons.

Ultradian rhythm measures. Five variables were measured at each waking testing trial: 1) number of correct detections; 2) number of false positive responses (FP); 3) frequency of global body movements (GBM - stabilometric chair) during the task; 4) limb movement during the task (LM-T); and, 5) limb movement during the rest periods (LM-RP). Each variable for each subject on both days was plotted as a time series and evidence of periodicity was established using power spectral analysis (BMD program 02T) (Wiener, 1950; Orr, & Naitoh, 1976). The presence of a spectral peak at the critical frequency (19.2 c/day) was assessed statistically using Wilcoxon matched pairs signed-ranks tests and Chi-square analyses.

Finally, possible phase relationships existing between the NREM-REM cycle and waking ultradian rhythms in performance and activity were examined by extrapolating the timing of the onset and midpoints ($\pm$ 1 S.D.) of the REM sleep cycle had sleep continued. This was based upon the previous night's

average REM cycle length for the last three REM periods. The waking rhythms were then superimposed over the extrapolation. Peaks in waking rhythms were analysed to determine whether they were coincident with predicted REM periods or inter-REM time which would represent NREM sleep.

Chapter III

Results

Tonic Sleep Measures

Sleep Stages. Repeated measures analysis of variance (2 x 5) performed on data from all recording nights revealed no group differences for number of minutes or percentage amounts of any of the conventional sleep stages. Summary data for all nights are presented in Tables 2 and 3. Tables 4 and 5 contain mean values for the sleep measures from Nights 3-5 only.

A significant night effect (five nights) was obtained for the following variables: total sleep time (TST); sleep efficiency index (SEI); minutes of total wake time (TWT); minutes of waking after sleep onset (WASO); minutes of REM sleep; number of REM periods (REMPs); percentage of TWT; percentage of WASO; percentage of NREM sleep; and percentage of REM sleep. Table 6 contains the relevant F values for the above variables. Trend analyses of the above variables showed significant linear increases across nights for TST ($p < .01$), SEI ($p < .02$), REM minutes ($p < .001$), REM percentage ($p < .01$), and number of REMPS ($p < .01$), while significant linear decreases were found for minutes of TWT ($p < .005$), minutes of WASO ($p < .005$), percentage TWT ($p < .005$), percentage WASO ($p < .005$), and NREM percentage ($p < .005$). No significant main effects for group or nights were obtained when only Nights 3-5 were analysed.

Post-hoc mean comparisons for variables showing a main effect for night revealed the following relationships ($p < .05$): TST - Night 1 < Night 4; SEI - Night 1 < Night 4; TWT (min) - Night 1 > Nights 3, 4, and 5; WASO (min) -

Table 2

Means (S.E.M.) of Sleep Measures (Minutes)

Variable	Group	Night 1	2	3	4	5
TBT	HK	557.21(6.25)	556.61(5.91)	562.41(6.39)	564.77(4.05)	562.18(4.76)
	C	543.68(9.91)	552.07(6.58)	547.46(7.47)	554.50(6.72)	548.45(8.74)
SPT	HK	528.02(10.90)	529.70(8.29)	531.50(10.70)	543.66(3.42)	540.76(4.76)
	C	516.41(11.12)	523.85(8.98)	528.20(6.33)	534.56(6.49)	523.19(9.55)
TST	HK	513.41(12.35)	519.02(8.52)	520.59(14.14)	534.29(3.90)	529.71(6.00)
	C	493.91(14.44)	508.57(7.62)	515.44(7.48)	533.16(6.56)	517.92(9.78)
SEI(TST/TBT)	HK	0.92(0.02)	0.93(0.01)	0.93(0.02)	0.95(0.01)	0.94(0.01)
	C	0.91(0.02)	0.92(0.02)	0.94(0.01)	0.96(0.01)	0.94(0.01)
TWT	HK	34.06(7.19)	26.14(4.98)	26.16(7.98)	20.02(4.38)	19.39(3.87)
	C	43.84(11.33)	34.59(9.16)	23.45(6.28)	15.36(3.15)	20.01(2.80)
WASO	HK	12.36(3.11)	7:52(2.36)	7.43(4.21)	6.11(1.94)	7.76(2.65)
	C	20.29(6.58)	13.00(3.36)	10.85(4.68)	1.96(0.46)	3.43(0.79)
S1	HK	33.71(3.62)	35.16(4.94)	29.62(4.55)	34.07(5.24)	40.05(6.05)
	C	43.55(7.01)	32.14(5.52)	37.90(5.75)	32.08(5.20)	32.24(4.24)
S2	HK	311.47(14.02)	297.82(10.67)	309.48(12.73)	313.59(8.39)	306.44(12.31)
	C	276.11(13.15)	286.46(9.65)	289.96(13.35)	305.06(7.33)	284.65(15.09)
S3	HK	30.48(3.07)	28.47(2.58)	30.62(2.87)	35.73(3.40)	27.75(3.28)
	C	32.66(2.94)	26.68(4.68)	33.20(4.85)	25.73(4.66)	28.16(3.18)
S4	HK	52.03(7.80)	55.55(5.42)	50.98(4.32)	50.80(7.83)	55.26(6.86)
	C	61.47(10.14)	63.48(8.05)	56.43(6.31)	64.41(9.82)	63.51(10.46)
NREM	HK	427.70(11.24)	416.98(6.63)	420.71(14.68)	434.20(3.57)	429.48(7.01)
	C	413.77(13.58)	408.85(8.88)	417.46(11.68)	427.27(7.60)	408.55(8.88)
SW	HK	82.52(6.67)	84.02(4.63)	81.61(5.38)	86.53(5.69)	83.00(6.40)
	C	94.12(10.41)	90.16(6.95)	89.62(5.52)	90.15(8.70)	91.65(10.16)
REM	HK	85.71(2.45)	102.03(5.32)	99.84(3.77)	100.09(1.44)	100.23(4.70)
	C	80.14(5.09)	99.82(3.59)	97.96(6.06)	105.89(6.02)	109.38(4.78)

Table 3

Means (S.E.M.) of Sleep Measures (percentage)

Variable	Group	Night				
		1	2	3	4	5
TWT	HK	6.18(1.33)	4.70(0.90)	4.71(1.46)	3.52(0.77)	3.45(0.69)
	C	8.07(2.10)	6.18(1.63)	4.25(1.14)	2.77(0.57)	3.66(0.51)
WASO	HK	2.38(0.62)	1.42(0.45)	1.53(0.93)	1.12(0.36)	1.43(0.49)
	C	4.03(1.33)	2.43(0.62)	2.04(0.88)	0.36(0.08)	0.67(0.16)
S1	HK	6.67(0.84)	6.83(1.03)	5.64(0.85)	6.40(0.99)	7.61(1.17)
	C	8.77(1.39)	6.33(1.07)	7.42(1.19)	5.98(0.93)	6.19(0.77)
S2	HK	60.41(1.53)	57.41(1.91)	59.32(1.55)	58.67(1.40)	57.79(2.05)
	C	55.81(1.94)	56.33(1.69)	56.11(2.14)	57.32(1.59)	58.84(2.46)
S3	HK	5.91(0.58)	5.48(0.47)	5.83(0.46)	6.68(0.64)	5.21(0.61)
	C	6.65(0.58)	5.21(0.85)	6.38(0.89)	4.79(0.83)	5.46(0.61)
S4	HK	10.25(1.65)	10.69(1.00)	9.87(0.90)	9.52(1.50)	10.46(1.30)
	C	12.52(2.21)	12.45(1.49)	10.97(1.24)	12.07(1.80)	12.39(2.03)
NREM	HK	83.24(0.54)	80.40(0.86)	80.65(0.93)	81.27(0.24)	81.07(0.87)
	C	83.75(0.99)	80.33(0.80)	80.87(1.36)	80.15(1.12)	78.88(0.85)
SW	HK	16.13(1.38)	16.16(0.79)	15.69(0.99)	16.21(1.08)	15.68(1.18)
	C	19.18(2.30)	17.65(1.22)	17.34(0.99)	16.86(1.53)	17.84(2.05)
REM	HK	16.76(0.54)	19.61(0.86)	19.35(0.93)	18.74(0.24)	18.93(0.87)
	C	16.25(0.99)	19.69(0.80)	19.11(1.35)	19.85(1.12)	21.13(0.85)

Table 4

Means (S.E.M.) of Sleep Measures (minutes) for Nights 3-5

Variable	Group	
	Hyperkinetic (N = 11)	Control (N = 11)
Total Bed Time (TBT)	563.12 (2.89)	550.13 (4.24)
Sleep Period Time (SPT)	538.64 (4.04)	528.65 (4.33)
Total Sleep Time (TST)	528.20 (5.21)	522.18 (4.71)
Sleep Efficiency Index (TST/TBT)	0.94 (0.01)	0.95 (0.01)
Total Wake Time (TWT)	21.86 (3.24)	19.61 (2.51)
Waking After Sleep Onset (WASO)	7.10 (1.73)	5.41 (1.69)
Stage 1	34.58 (3.06)	34.07 (2.89)
Stage 2	309.84 (6.35)	293.22 (2.46)
Stage 3	31.36 (1.88)	29.03 (2.46)
Stage 4	52.35 (3.66)	61.45 (5.10)
NREM	428.13 (5.47)	417.76 (5.50)
Slow Wave (SW)	83.71 (2.00)	90.47 (4.67)
REM	100.05 (3.28)	104.41 (3.27)

Table 5

Means (S.E.M.) of Sleep Measures

(percentage) for Nights 3-5

Variable	Group	
	HK	Control
Total Wake Time (%)	3.89(0.59)	3.56(0.45)
Waking After Sleep Onset (%)	1.36(0.36)	1.02(0.32)
Stage 1 (%)	6.55(0.59)	6.53(0.56)
Stage 2 (%)	58.59(0.95)	56.09(1.18)
Stage 3 (%)	5.91(0.34)	5.54(0.45)
Stage 4 (%)	9.95(0.70)	11.81(0.97)
REM (%)	19.01(0.42)	20.03(0.65)
Slow Wave (%)	15.86(0.61)	17.35(0.89)

Table 6

F Values for Variables Showing a Significant Across Night Effect

Variables	F value (df = 4,80)	p value
TST	3.47	.01
SEI	3.44	.01
TWT (min)	4.27	.01
WASO (min)	4.50	.01
REM (min)	9.45	.001
REMPs	2.64	.05
TWT (%)	4.39	.01
WASO (%)	4.54	.01
NREM (%)	6.90	.001
REM (%)	6.91	.001

Night 1 > Nights 4 and 5; REM (min) - Night 1 < Nights 2, 3, 4, and 5; TWT (%) - Night 1 > Nights 3, 4, and 5; WASO (%) - Night 1 > Nights 4 and 5; NREM (%) - Night 1 > Nights 2, 3, 4, and 5; and REM (%) - Night 1 < Nights 2, 3, 4, and 5.

Cycle Analysis

No significant differences were observed between groups or across nights for Nights 3-5 for REM cycle length (waking included), REM cycle length with waking excluded, NREM cycle length (waking included), or NREM cycle length with waking excluded. With Nights 1 and 2 included, REM cycle length (waking time subtracted) showed a significant main effect for night ($F(4,80) = 2.82$, $p < .05$) with a significant linear trend increase across nights ($p < .03$). Post-hoc tests revealed only a shorter REM cycle length on Night 1 relative to Night 5 ($p < .05$). Summary values of the cycle analysis are presented in Tables 7 and 8.

Successive sleep cycles across the night were subjected to trend analyses. These cycles were defined using NREM (stage 2) and REM onsets. In this way, all recorded sleep could be utilized without neglecting the first cycle which has often been excluded in the cycle analyses of previous studies which have considered only the REM cycle. The methodology of Feinberg and Floyd (1979) was used to compute these NREM and REM cycle lengths, i.e., NREM cycle length was measured as the duration of total sleep (time awake subtracted) from initial stage 2 onset in the first cycle to stage 2 onset in the second cycle, from the onset of stage 2 in the second cycle to stage 2 onset in the third cycle, etc. Similarly, REM cycle was measured as the duration of total sleep

Table 7

Cycle Analysis

(min; Mean and S.E.M. for Nights 3-5)

Variable	Groups	
	HK	Control
REM Cycle Length	89.21(2.06)	92.91(2.81)
REM Cycle Length (real time)	93.03(2.22)	95.35(2.79)
NREM Cycle Length	108.18(3.21)	104.96(3.49)
NREM Cycle Length (real time)	111.90(3.50)	107.17(3.51)

Table 8

Cycle Analysis

(min; Means (S.E.M.) for Nights 1-5)

		Night				
		1	2	3	4	5
REM Cycle Length	HK	85.23(3.20)	91.48(4.33)	84.55(3.10)	90.44(3.28)	92.66(4.10)
	C	84.34(2.81)	81.20(3.19)	91.17(3.35)	92.19(2.90)	95.37(7.41)
REM Cycle Length (real time)	HK	88.13(3.31)	95.08(5.00)	89.95(4.18)	93.12(3.12)	96.03(4.25)
	C	91.26(3.90)	84.55(3.72)	94.83(3.51)	93.42(2.92)	97.82(7.28)
NREM Cycle Length	HK	108.43(6.13)	112.85(5.07)	108.28(3.70)	100.38(3.46)	115.87(7.83)
	C	107.33(2.62)	96.16(2.64)	102.81(3.94)	107.66(4.73)	104.46(8.83)
NREM Cycle Length (real time)	HK	112.51(6.81)	116.29(5.71)	113.03(5.11)	102.86(3.23)	119.81(8.15)
	C	112.03(2.56)	99.46(3.06)	106.16(4.37)	108.64(4.74)	106.75(8.71)

recorded (time awake subtracted) from the first REM onset to the onset of the second REMP, fromthe second REMP onset to the third, etc... The cycles were also measured with waking time included (real time).

Results were limited to the first four cycles of alternating NREM and REM sleep from Nights 3-5, since complete data could be obtained from all subjects for these nights. Table 9 presents the successive REM and NREM mean cycle durations and trend analyses. Significant trends across cycles were present for both REM and NREM cycle durations. Figures 1 and 2 present successive mean durations of the first four cycles for REM and NREM onsets, respectively, in HK and control subject groups. A strong linear trend toward shorter REM cycle lengths across the night was found, although, the control group evidenced an increase in the third REM cycle which was equal in duration to the first. With regard to the NREM cycle duration, the cubic component reached statistical significance. Both groups showed a lengthy first NREM cycle with the HK group showing this to a significantly greater extent. In addition, the HK group evidenced a slight increase in the duration of the third cycle.

When time awake was included, allowing real-time values of sleep cycles to be compared to real-time periods of other physiological and behavioral measures, the trends of these real-time REM and NREM cycles were very similar in appearence to those exhibited for both REM and NREM cycle durations. A significant linear trend was again noted for the real-time REM cycle, and a significant cubic trend was found in the real-time NREM cycles across the night.

Table 9

Sleep Cycle Means (S.E.M.) and Trend Analyses

Sleep cycle Definition	Group	Cycle 1	2	3	4
REMC	HK	96.96(3.85)	91.48(6.36)	85.44(2.47)	79.75(4.93)
	C	97.29(4.78)	94.03(4.83)	97.30(5.53)	78.71(5.30)
REMC (real time)	HK	100.35(4.56)	93.94(6.81)	91.95(2.34)	83.24(4.74)
	C	101.02(5.05)	95.68(4.61)	99.90(5.58)	80.38(5.36)
NREMC.	HK	150.76(8.40)	99.66(5.50)	103.99(5.33)	87.59(2.81)
	C	128.38(10.36)	104.37(4.59)	104.17(3.88)	98.36(7.12)
NREMC (real time)	HK	152.97(8.67)	103.16(6.60)	108.33(6.00)	89.85(3.04)
	C	129.04(10.33)	108.16(4.75)	106.01(3.88)	101.08(7.27)

	F Values			
	Among	Linear	Quadratic	Cubic
REMC	5.65**	14.10***	2.44	1.26
REMC (real time)	5.74**	12.39**	2.04	1.86
NREMC	22.99***	41.31***	9.96**	9.67*
NREMC (real time)	19.99***	38.52***	7.08*	8.10*

*$p < .01$
**$p < .005$
***$p < .001$

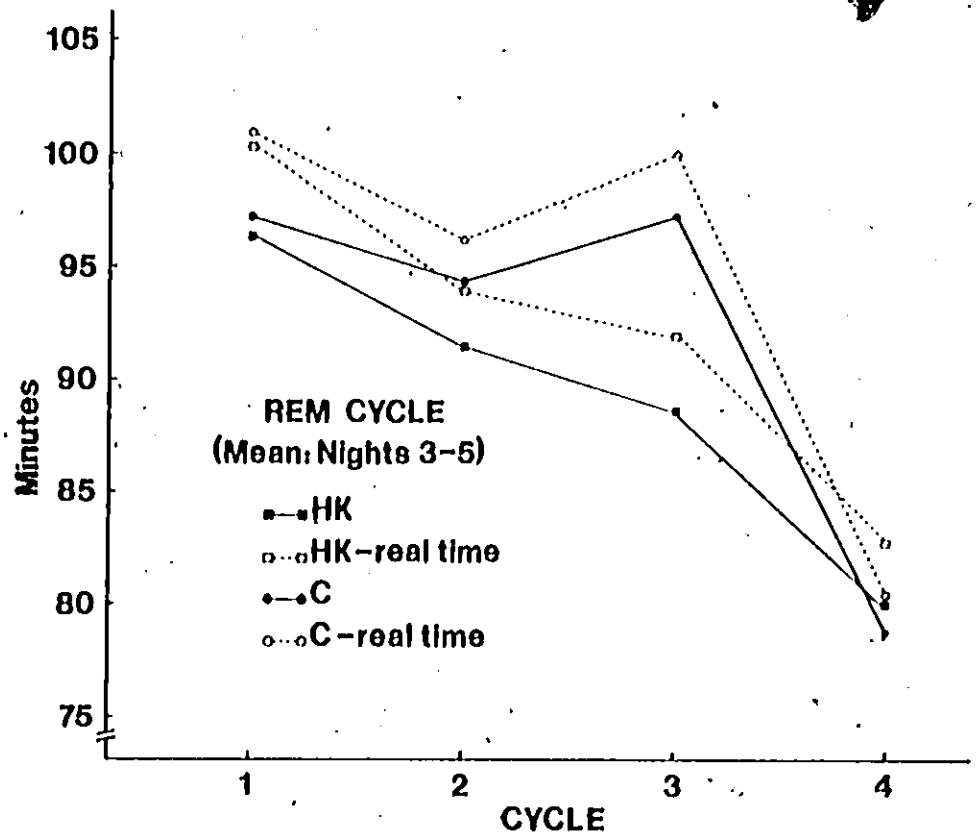


Figure 1. Mean durations of successive REM cycles within nights for the HK and Control groups.

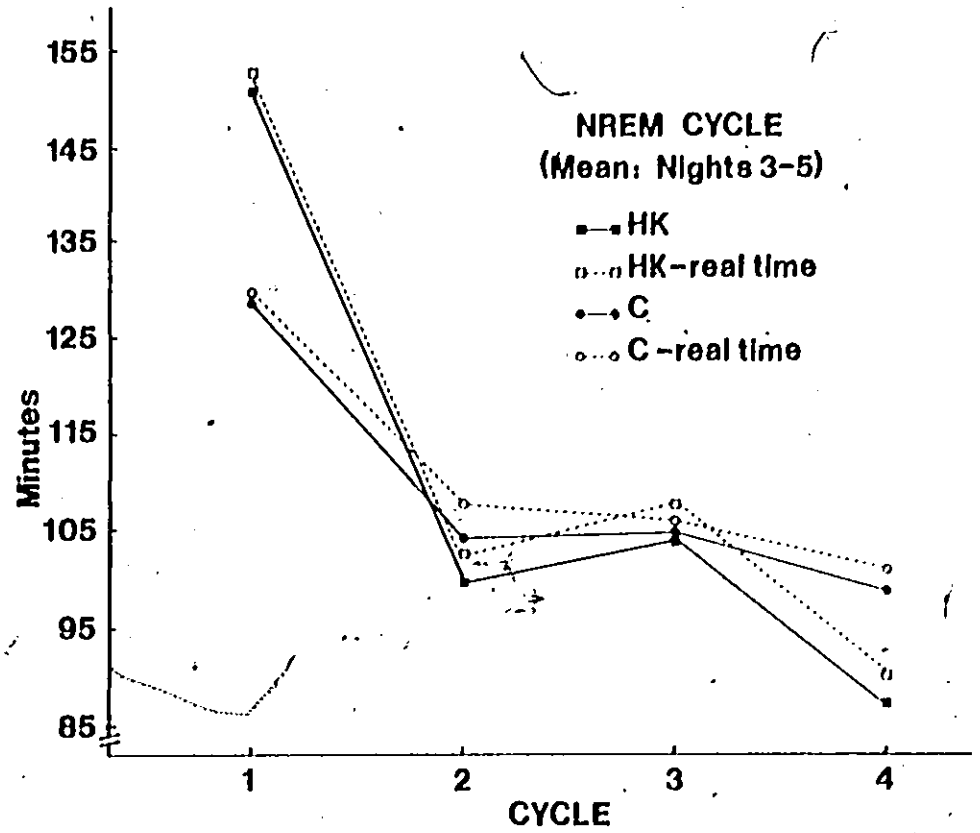


Figure 2. Mean durations of successive NREM cycles within nights for the HK and Control groups.

Latencies

A significant main effect for group {F (1,20) = 4.46, $p < .05$} was present for REM onset latency on Nights 3-5; with the HK group showing the longer latency. When Nights 1 and 2 were added to the analysis, the significant group effect was maintained {F (4,80) = 7.08, $p < .05$} and a significant night effect {F (4,80) = 2.69, $p < .05$} was present, with Night 1 > Night 5 ($p < .05$). Mean values of REM onset latency across nights for both groups are presented in Figure 3. No significant group or night differences were obtained for latencies to sleep onset or any NREM sleep stage. Summary data for latency variables are presented in Tables 10 and 11. To examine whether increased REM latencies might be associated with longer durations of the first REMP, a correlation (Pearson r) was computed between these two measures (Nights 3-5). A significant positive correlation ($r = 0.62$, $df = 21$, $p < .01$) was obtained.

Motor Activity During Sleep

HK subjects exhibited greater amounts of movement time (minutes and percentage) compared to controls ($p < .08$) across all nights (Figure 4). Body movements on Nights 1, 3, and 5 (Figure 5) were also greater for the HK group relative to controls ($p < .08$). There was, moreover, a significant main effect for nights {F (2,40) = 7.07, $p < .01$} with body movements increasing across nights ($p < .01$).

The frequency of REM twitches on Nights 1, 3, and 5 did not differ between groups, but a significant night effect {F (2,40) = 7.59, $p < .01$;

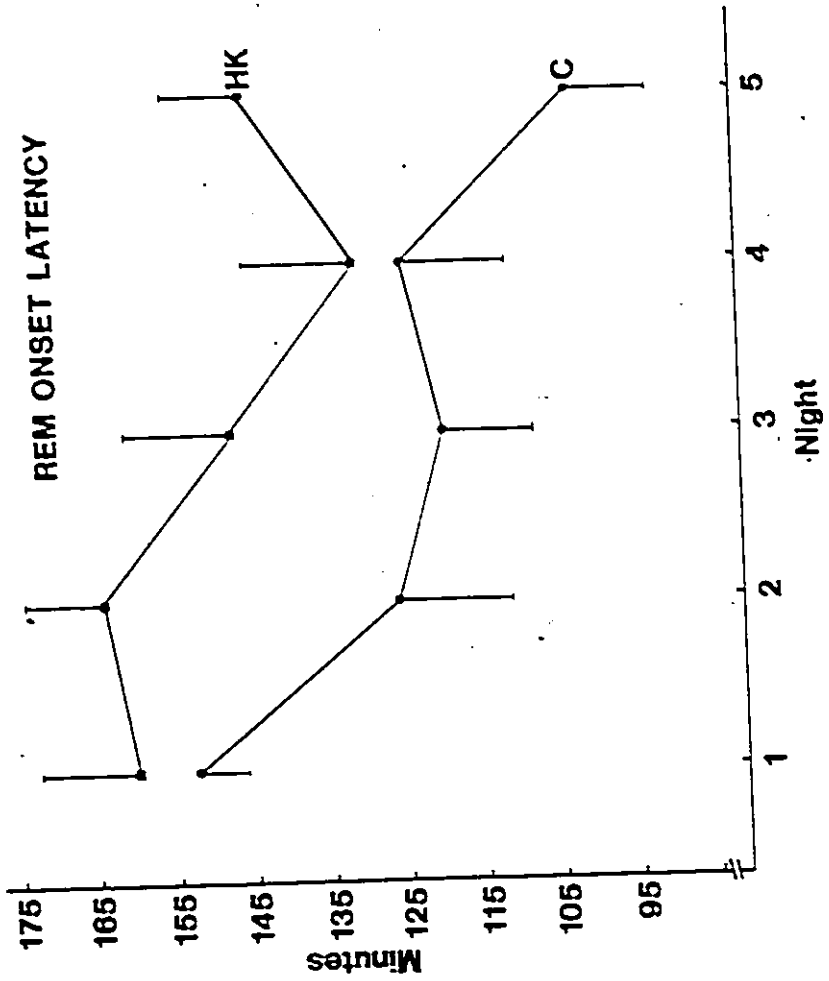


Figure 3. Mean (S.E.M.) REM onset latencies across nights for the HK and Control groups.

Table 10

Mean Latencies (S.E.M.) for Nights 3-5

Variables	Groups	
	HK	Control
Lights Out to Sleep Onset	15.17(2.60)	11.43(1.60)
Lights Out to Stage 1	14.20(0.44)	10.48(1.54)
Sleep Onset to Stage 2	3.99(0.42)	3.11(0.24)
Sleep Onset to Stage 3	14.38(0.63)	14.94(0.73)
Sleep Onset to Stage 4	21.41(1.08)	20.74(1.14)
Sleep Onset to 1st REMP	140.13(7.48)*	115.39(6.71)

*$p < .05$

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

Table 11

Latencies (min; Means (S.E.M.) for Nights 1-5)

Variable	Group	Night 1	2	3	4	5
Lights Out to Sleep Onset	HK	22.38(6.83)	18.53(4.21)	19.56(6.63)	14.00(3.58)	11.94(2.22)
	C	18.18(5.38)	11.55(2.11)	9.77(1.73)	12.17(3.12)	12.36(3.38)
Lights Out to Stage 1	HK	22.29(6.85)	17.85(4.31)	18.93(6.62)	12.18(3.05)	11.48(2.18)
	C	15.18(5.37)	10.46(1.72)	9.05(1.31)	11.12(3.08)	11.26(3.35)
Sleep Onset to Stage 2	HK	3.77(0.84)	4.20(0.93)	2.84(0.64)	4.03(0.73)	5.11(0.71)
	C	2.48(0.67)	3.06(0.47)	3.57(0.58)	2.78(0.23)	2.96(0.38)
Sleep Onset to Stage 3	HK	16.41(2.54)	14.70(1.23)	12.86(1.14)	15.76(1.28)	14.52(0.64)
	C	14.41(1.98)	13.77(1.25)	16.44(1.29)	14.35(1.32)	14.05(1.18)
Sleep Onset to Stage 4	HK	22.57(2.57)	20.57(1.35)	19.89(1.21)	23.59(2.82)	20.76(0.98)
	C	21.86(3.71)	20.09(1.52)	20.82(1.32)	21.00(1.75)	20.39(2.78)
Sleep Onset to 1st REMP	HK	159.70(12.45)	162.03(11.76)	146.91(14.02)	130.66(14.09)	142.82(11.32)
	C	152.41(6.17)	125.83(14.30)	119.56(11.81)	124.25(12.69)	102.36(10.24)

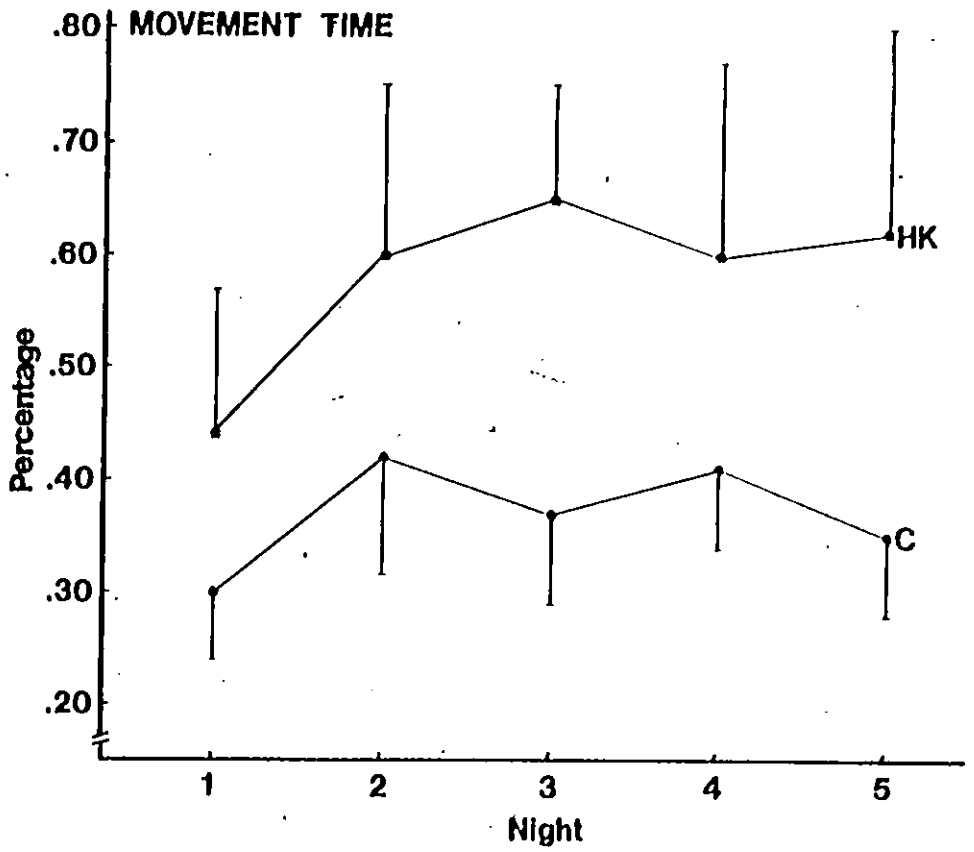


Figure 4. Mean (S.E.M.) percentage values for movement time across nights for the HK and Control groups.

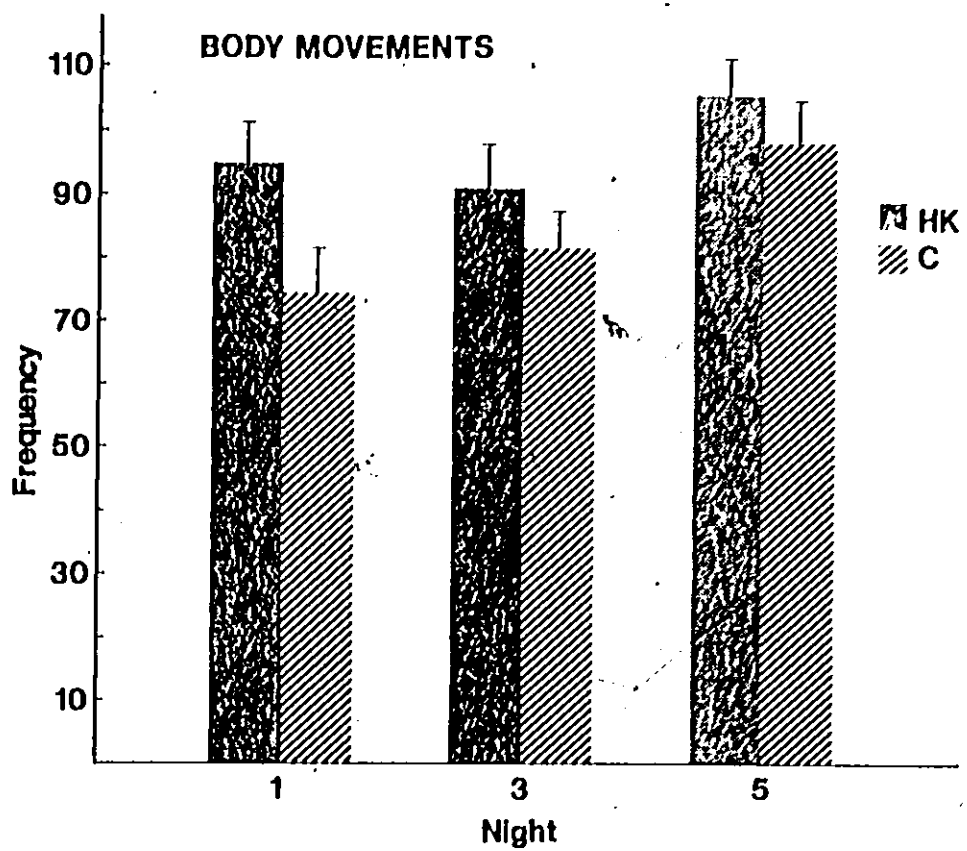


Figure 5. Mean (S.E.M.) frequency histograms of body movements for the HK and Control groups for nights 1, 3, and 5.

quadratic trend, $p < .005$). HK subjects exhibited an increased frequency of NREM twitches (Figure 6) compared to controls ($p < .07$), and both groups showed across night differences ($F(2,40) = 8.70$, $p < .001$; quadratic trend, $p < .05$).

To investigate whether motor activity in the first cycle of sleep may have contributed to the significantly longer REM onset latency by disrupting sleep, comparisons were made of movement time (min), number of body movements, and frequency of NREM twitches between groups (Nights 3 and 5). No group differences were found in movement time (HK: $\bar{X} = 6.64$; C: $\bar{X} = 4.77$, $t = 1.15$, N.S.) or number of body movements (HK: $\bar{X} = 16.18$; C: $\bar{X} = 13.32$, $t = 1.08$, N.S.). However, the HK group had significantly more NREM twitches (HK: $\bar{X} = 81.27$; C: $H = 41.55$, $t = 2.44$, $p < .05$). These motility measures were then correlated (Pearson r) to REM onset latency. Movement time and number of body movements were not significantly related to REM onset latency, however, a positive correlation ($r = 0.50$, $df = 21$, $p < .02$) was found for NREM twitches.

Autonomic Activity

Technical difficulties limited SSPR data to the first cycle of sleep for both groups. Response rates per minute for Stage 2, Stage 4, and REM were based on: 1) the total amount of scorable SSPR activity divided by the number of minutes of a particular stage in the first cycle; and, 2) the frequency of SSPR activity over 15-minute samples (one sample per subject) of Stage 2 and Stage 4. In the latter case, equivalent samples from REM were not obtained since the initial REMPs were often of short duration.

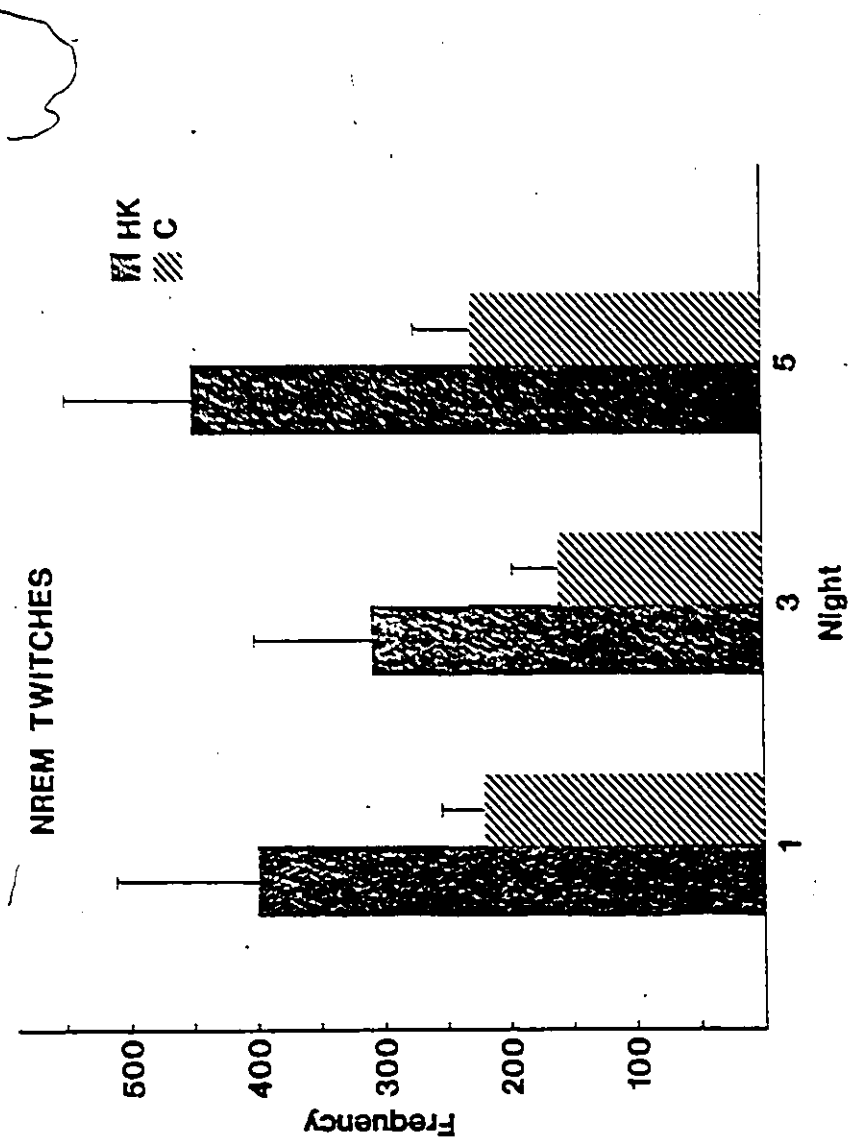


Figure 6. Mean (S.E.M.) frequency histograms of NREM twitches for the HK and Control groups for nights 1, 3, and 5.

Mean response rates per minute (Nights 1, 4, and 5) using the first method compared the HK and control groups using t-tests for independent samples. No significant group differences were found in SSPR frequency in Stage 2, Stage 4, or REM. Mean rates per minute were then compared between stages for all subjects using t-tests for correlated samples. Mean response rates per minute for Stage 2, Stage 4, and REM were 3.30 (S.D. = 1.67), 5.08 (S.D. = 2.49), and 1.71 (S.D. = 1.38) respectively. Significant differences were found between Stage 2 and Stage 4 ($t = 5.89$, $df = 21$, $p < .001$), Stage 2 and REM ($t = 5.30$, $df = 21$, $p < .001$), and Stage 4 and REM ($t = 7.25$, $df = 21$, $p < .001$). Calculated mean response rates per minute for 15-minute samples of Stage 2 and Stage 4 showed no group differences. A comparison of pooled subject mean rates per minute for Stage 2 ($\bar{X} = 3.27$, S.D. = 2.15) and Stage 4 ($\bar{X} = 5.27$, S.D. = 2.63) revealed a significant difference ($t = 4.85$, $df = 21$, $p < .001$).

Eye Movement Activity

Mean REM density measures were computed for all REM periods from Nights 4 and 5 but did not reveal any group differences (HK: $\bar{X} = 0.46$, S.D. = 0.11; C: $\bar{X} = 0.50$, S.D. = 0.11). Since REMP durations tend to lengthen across the night, analyses were performed on intra-night REM density to determine changes in density patterns across the night. Accordingly, REM density was analysed across the first four REMPs, this being the number of REMPs all subjects completed. A significant main effect for REMP {$F(3,60) = 22.49$, $p < .001$; linear trend ($p < .001$)} with increasing REM density across the night

(Figure 7). Similar analyses of REM density for the initial five minutes of these REMPs also revealed a significant main effect for REMP {$F(3,54) = 4.28$, $p < .01$; linear trend ($p < .001$)} showing increasing REM density (Figure 8).

Since REMP durations vary across the night and density may be influenced by longer REMPs, density measures were calculated over the first 15 minutes of each of the first four REMPs (Nights 4 and 5) in five-minute blocks (Figure 9) to control for the influence of REM duration on eye movement density. The across REMP increase in REM density is again quite clearly evident. Within REMPs, a relatively rapid increase in density occurs by the second five-minute block of REM sleep.

Eye movement density within longer REMPs was also examined. Figure 10 presents mean REM density in five-minute blocks for the HK and control groups across a 30-minute REMP (mean density obtained from nine HK and nine C subjects - one 30-minute REMP per subject). Although no group differences were found, density did change with time into REM {$F(5,80) = 6.93$, $p < .001$; quadratic trend ($p < .001$)}. Density peaked after the first 10 minutes of REM, decreased and plateaued for the next 10 minutes, and then rapidly declined until the end of the REMP. Eye movement density during the first five-minute block was significantly less than during the second, and the sixth block was significantly less than all blocks except the first ($p < .05$).

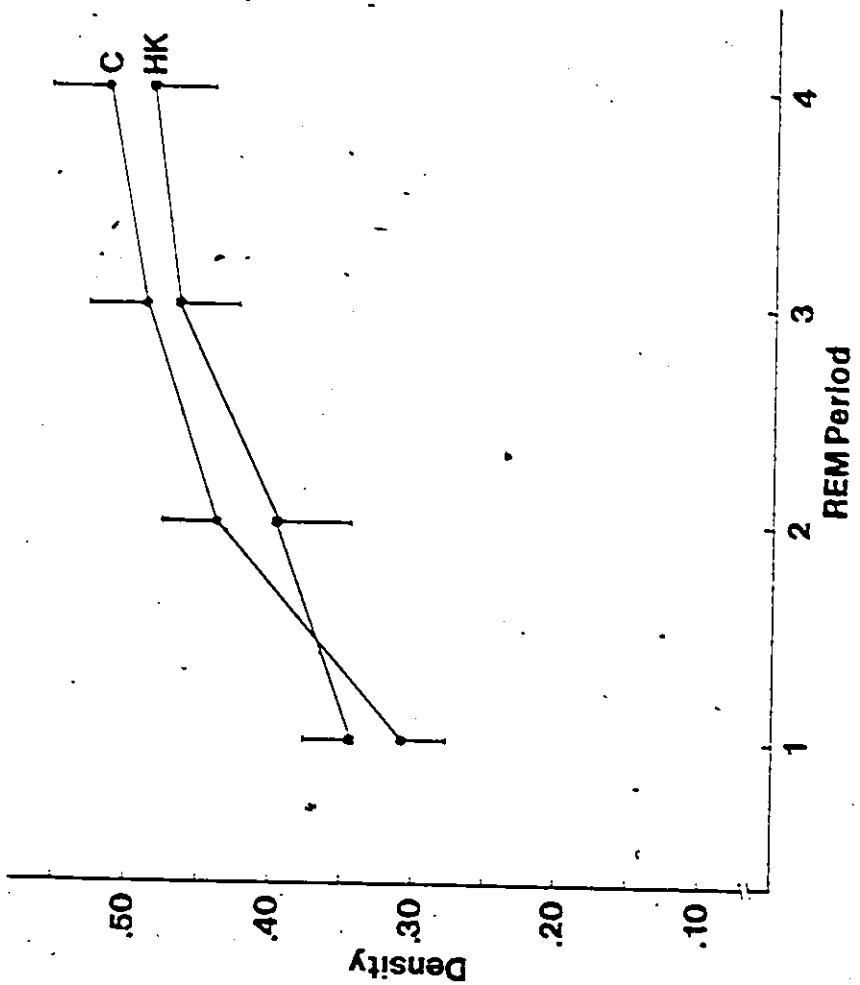


Figure 7. Mean (S.E.M.) EM densities across successive REMPs for the HK and Control groups.

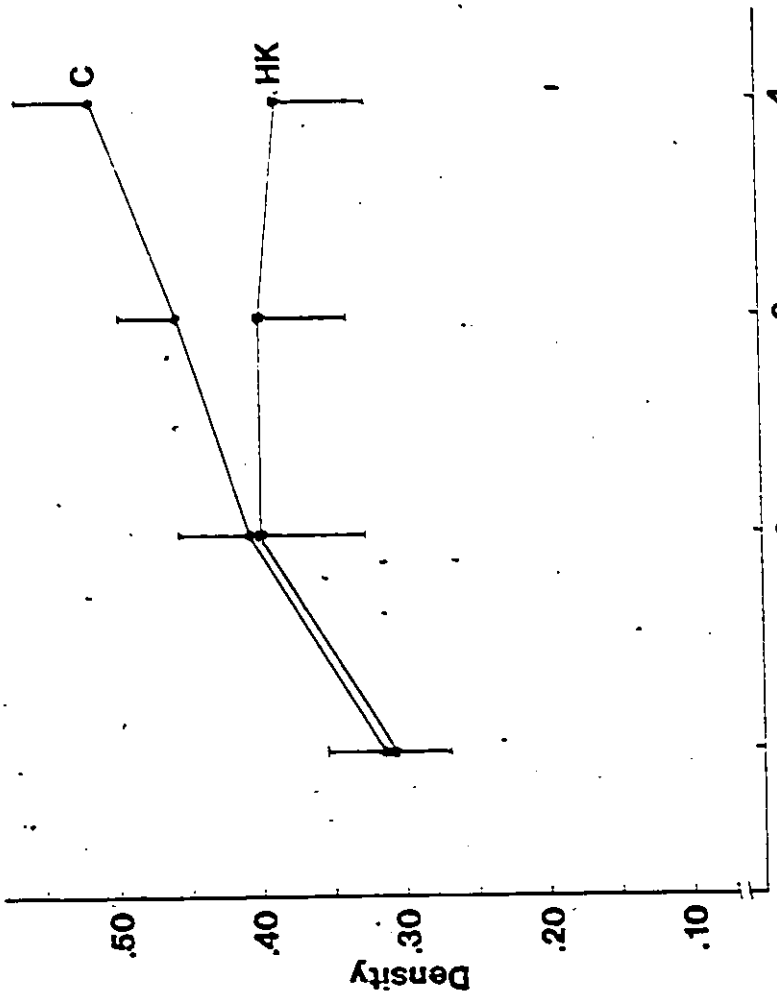


Figure 8. Mean (S.E.M.) EM densities for the first five minutes of each successive REMP for the HK and Control groups.

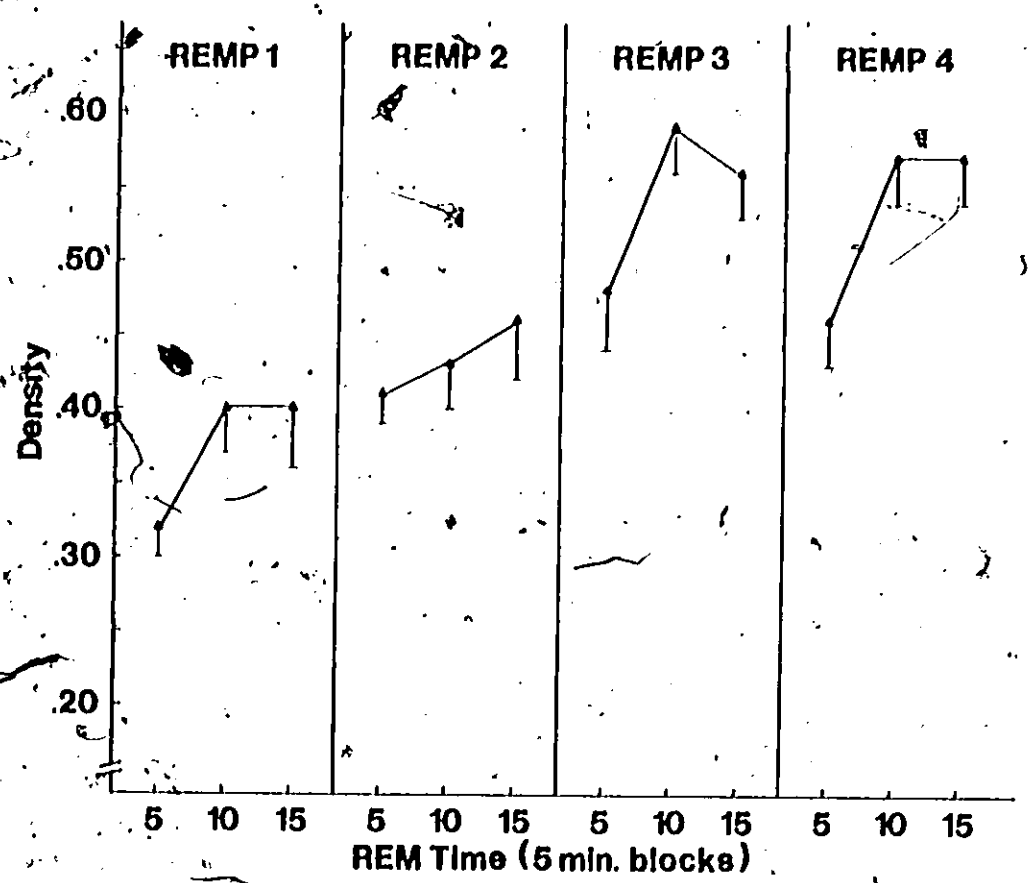


Figure 9. Mean (S.E.M.) EM density (pooled subjects data) across the first fifteen minutes (five minute blocks) of successive REMPs.

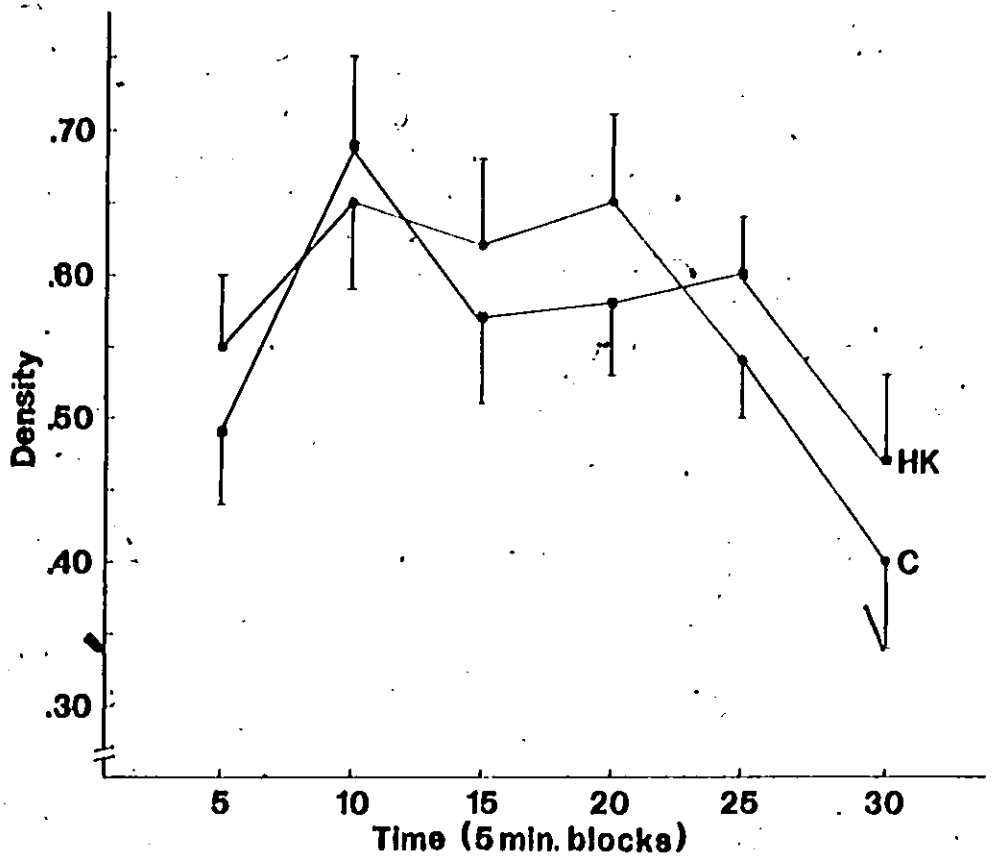


Figure 10. Mean (S.E.M.) EM density across 30-minute REMPs for the HK and Control groups.

Attention and Activity Measures

General findings. Mean values for each variable (i.e., across 24 trials) were computed for each subject on each day and groups were compared using t-tests for independent samples. Due to occassional technical difficulties with the equipment, measures on some variables were not able to be obtained for some subjects.

On both days of testing, HK subjects made significantly fewer correct detections relative to controls. Similarly, the HK group was significantly more active on the segmental limb movement measure during the rest periods on both days compared to the control group. Limb movement during the task approached significance on Day 2 only, with HK subjects showing greater motility on this measure. No significant differences between groups were found for either false positive responses or global body movements during the task on the two days of testing. Table 12 presents the mean values for each group on each variable for the two testing days. Figures 11 and 12 show the pooled subject data across trials for detections and limb movement during the task respectively.

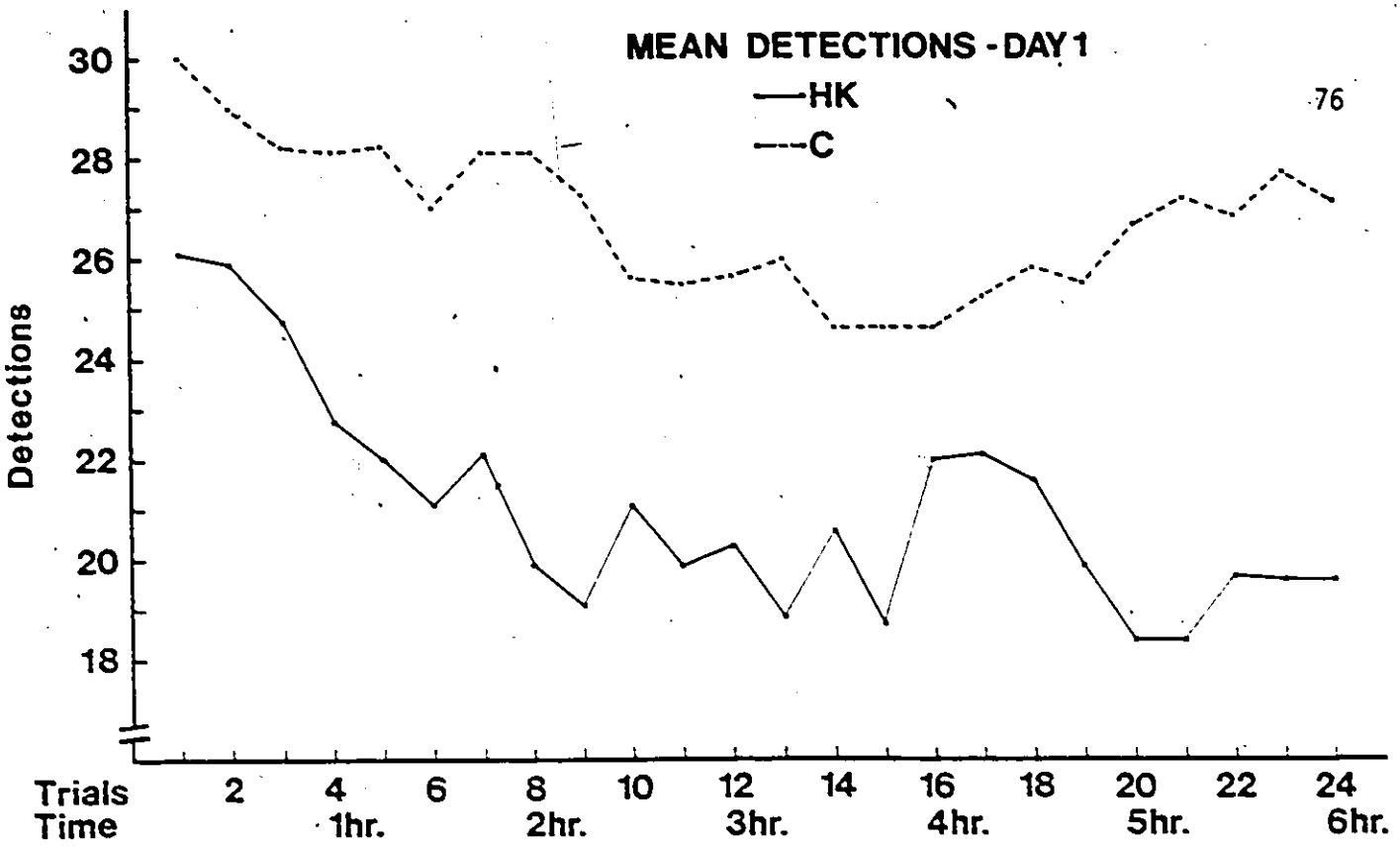
The relationship of attention to I.Q. yielded a significant positive correlation (Pearson $r = 0.67$, $df = 16$, $p < .005$) showing that the higher I.Q. control group may have contributed to a slightly inflated difference between groups on the attention measure. Correlation between attention and age was 0.61 ($df = 16$, $p < .005$), and between age and I.Q. was 0.29 (N.S.). However, this disparity in I.Q. would seem insufficient to explain the proportionally greater test results.

Table 12

Performance and Activity Measures
(Mean S.E.M.)

Variable	Hyperkinetic		Control		t-value	Probability
Day 1						
Detection	21.02	2.64(N=7)	26.77	1.05(N=6)	1.89	$p < .05$
False Positives	4.05	1.16(N=7)	3.46	0.57(N=6)	0.43	N.S.
Global body movement (on task)	124.27	32.20(N=8)	67.47	28.02(N=10)	1.34	N.S.
Limb movement (on task)	35.04	9.28(N=9)	29.09	17.98(N=11)	0.27	N.S.
Limb movement (rest period)	334.91	51.96(N=9)	202.46	15.70(N=11)	2.66	$p < .01$
Day 2						
Detection	20.62	1.93(N=9)	25.73	1.80(N=7)	1.89	$p < .05$
False Positives	3.73	0.69(N=9)	2.75	0.68(N=7)	1.00	N.S.
Global body movement (on task)	182.04	49.44(N=9)	111.33	41.35(N=9)	1.10	N.S.
Limb movement (on task)	54.69	18.98(N=10)	20.40	6.76(N=10)	1.70	$p < .06$
Limb movement (rest periods)	370.81	74.62(N=10)	216.66	22.24(N=10)	1.98	$p < .05$

Figure 11. Mean detections for the HK and Control groups across trials on Day 1 and Day 2.


MEAN DETECTIONS - DAY 1
HK
C
Detections
30
28
26
24
22
20
18
Trials
Time
2
4
1hr.
6
8
2hr.
10
12
3hr.
14
16
4hr.
18
20
5hr.
22
24
6hr.

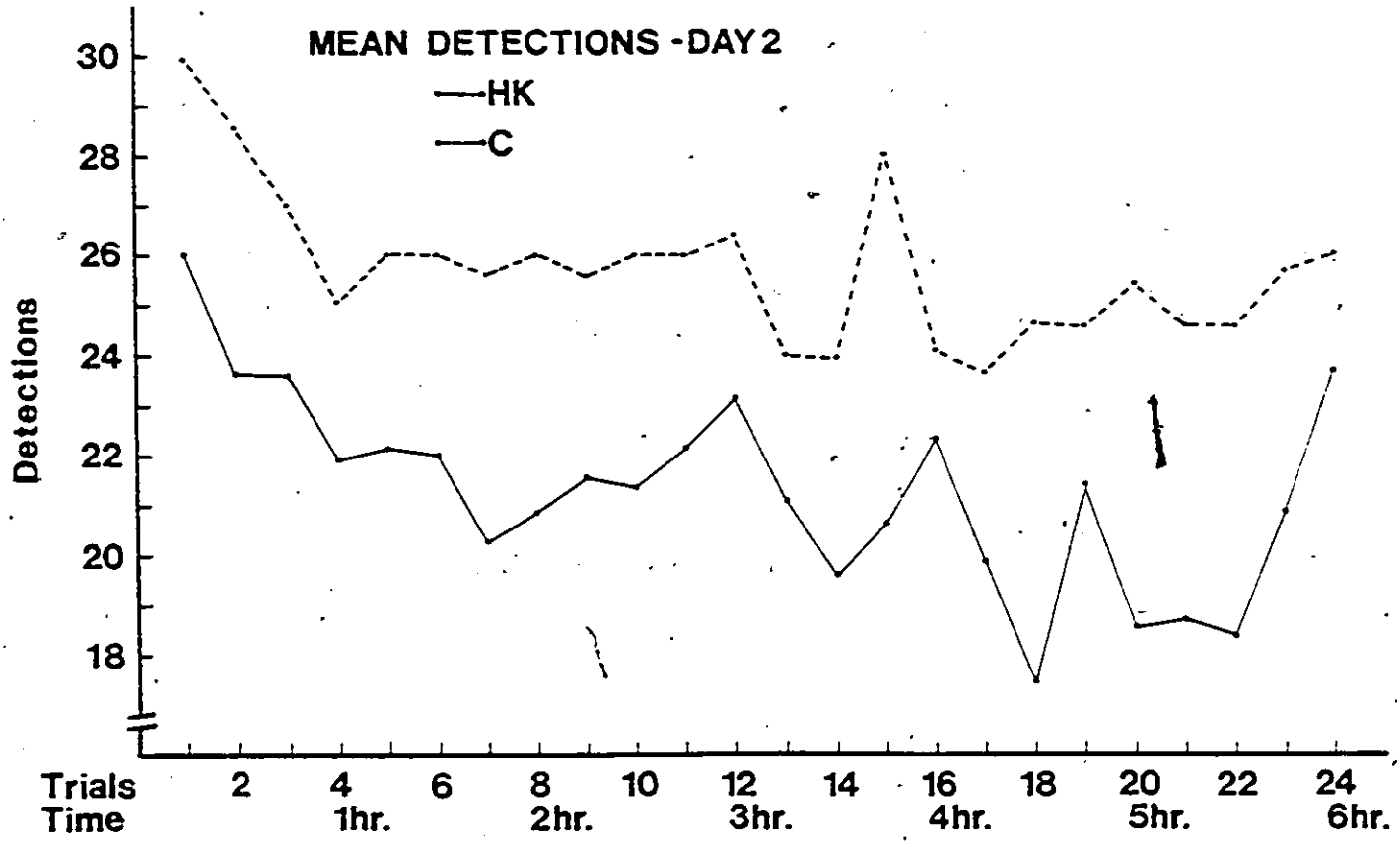
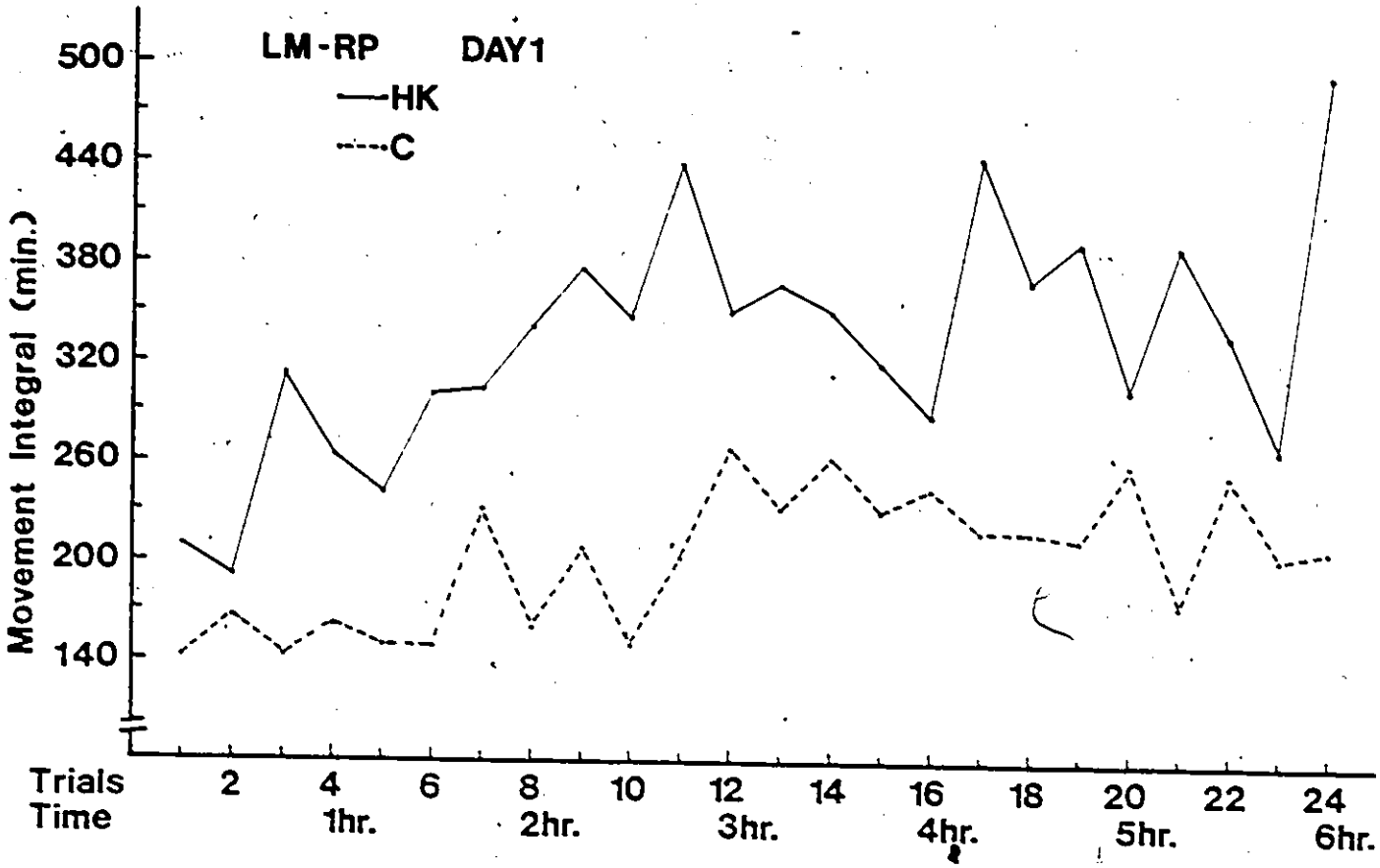
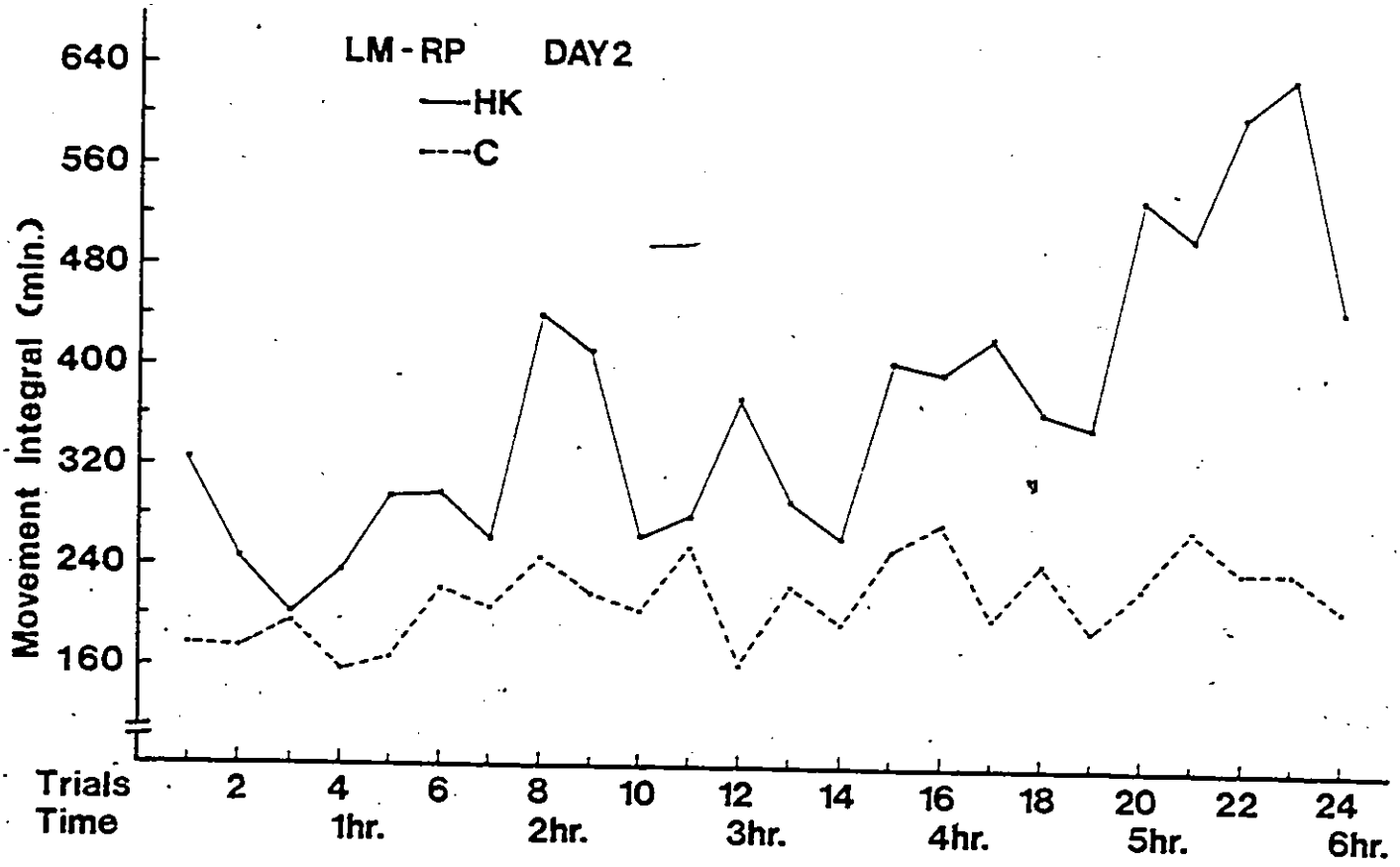

MEAN DETECTIONS - DAY 2
HK
C
Detections
30
28
26
24
22
20
18
Trials
Time
2
4
1hr.
6
8
2hr.
10
12
3hr.
14
16
4hr.
18
20
5hr.
22
24
6hr.

Figure 12. Mean values for limb movement during the rest periods (LM-RP) for the HK and Control groups across trials on Day 1 and Day 2.


LM-RP DAY1
—HK
----C
Movement Integral (min.)
500
440
380
320
260
200
140
Trials
Time
2
4
1hr.
6
8
2hr.
10
12
3hr.
14
16
4hr.
18
20
5hr.
22
24
6hr.


LM-RP DAY2
—HK
---C
Movement Integral (min.)
640
560
480
400
320
240
160
Trials
Time
2
4
1hr.
6
8
2hr.
10
12
3hr.
14
16
4hr.
18
20
5hr.
22
24
6hr.

Correlational analysis was also performed among the performance and activity measures on each testing day. Table 13 presents this correlation matrix of variables. On both days, a significant negative correlation between detections and false positive responses was obtained, indicating that as detections increased, false positives decreased, and conversely as detections decreased, false positives increased. On Day 2, a significant positive correlation was shown between global body movements and limb movements while on task.

Waking Ultradian Rhythms

Power spectral analysis was performed on each available time series for the five variables in each subject. As above, due to technical difficulties, time series data for all variables for all subjects was not complete (total number of time series = 174). Only six frequency bands were chosen due to the relatively short duration of the time series obtained. Resolution, therefore, was sacrificed in favor of increased stability of the spectra. Table 14 summarizes the major findings with regard to the distribution of spectral peaks in performance and activity. The frequency of primary interest (i.e., containing the expected BRAC for this age range) was 19.2 c/day (± 2 c/day), which is approximately equal to 75 minutes (68 to 84 minutes). As can be seen from the table, there was a relatively even distribution across frequencies with no one frequency (specifically not 19.2 c/day) showing consistent peaking. A number of observations were made with regard to the presence versus absence of a peak at the critical frequency and the varying

Table 13

Correlation Matrix of Waking Performance and Activity Measures

Day 1

	Det	FP	GBM	LM-OT	LM-RP
Det	-	-.57*(N=13)	-.17(N=11)	-.35(N=12)	-.09(N=12)
FP		-	-.07(N=11)	.34(N=12)	.18(N=12)
GBM			-	.05(N=18)	.07(N=11)
LM-OT				-	.35(N=12)
LM-RP					-

*$p < .05$

Day 2

	Det	FP	GBM	LM-OT	LM-RP
Det	-	-.49*(N=16)	-.06(N=14)	-.23(N=15)	-.05(N=15)
FP		-	-.20(N=14)	-.11(N=15)	.28(N=15)
GBM			-	.59**(N=18)	.32(N=14)
LM-OT				-	.19(N=15)
LM-RP					-

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

Det = Detections; FP = False Positives; GBM = Global body movement (on task)
LM-OT = Limb movement (on task); LM-RP = Limb movement (rest periods)

Table 14

Distribution of Spectral Peaks in Performance and Activity

Variable	Group	Day 1 N	Day 1 Frequency (c/day) 0	9.6	19.2	28.9	38.5	48.1	Day 2 N	Day 2 Frequency (c/day) 0	9.6	19.2	28.9	38.5	48.1
Detections	HK	7	3	3				1	9	6		3			
	C	6	8	1	1	1			7		3	2	2		
FP	HK	7	1		1	3	1	1	9	2		2	1	1	3
	C	6			1	3		2	7			2	2		3
GBM	HK	8	1		3		3	1	9	6		1	1	1	
	C	10	1	4	1	1		3	9	2	2	1	1		3
LM-T	HK	9	2		1	2	1	3	10	2	1	2	3		2
	C	11	3	2	4			2	10	1	3	4			2
LM-RP	HK	9	2	1	2	1	1	2	10	1	2	3	2		2
	C	11	2	4	1	2		2	10	1	1	3	1		4

Table 14.

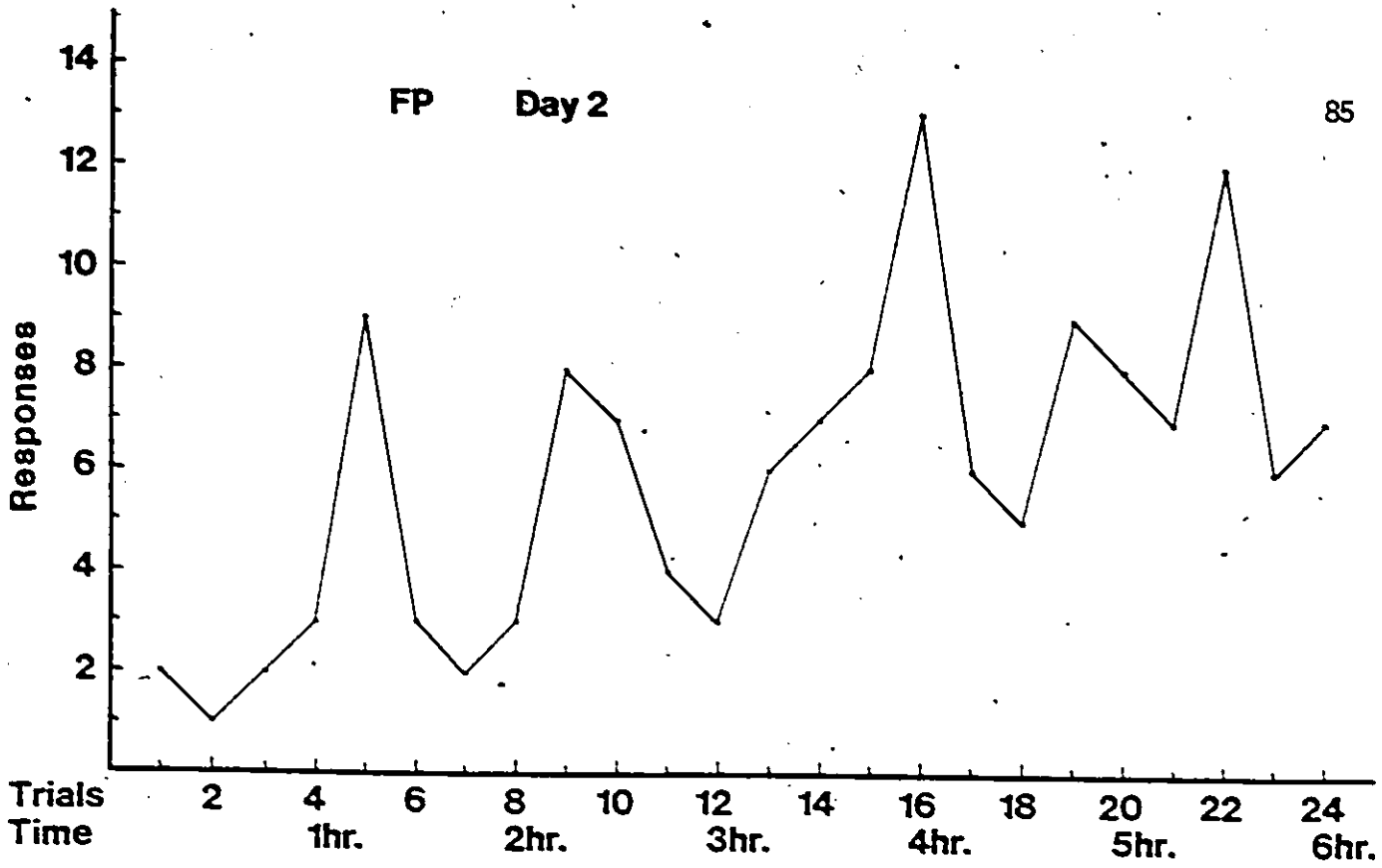
Distribution of Spectral Peaks in Performance and Activity (cont'd)

Variable	Group	N	Total (%) Frequency (c/day) 0	9.6	19.2	28.9	38.5	48.1
Detections	HK	16	56.3	18.8	18.8			6.2
	C	13	23.1	30.8	23.1	23.1		
FP	HK	16	18.8		18.8	25.	12.5	25.
	C	13			23.	38.5		38.5
GBM	HK	17	41.2		23.5	5.9	23.5	5.9
	C	19	15.8	31.6	10.5	10.5		31.6
LM-T	HK	19	21.0	5.3	15.8	26.3	5.3	26.3
	C	21	19.0	23.8	38.1			19.0
LM-RP	HK	19	15.8	15.8	26.3	15.8	5.3	21.0
	C	21	14.3	23.8	19.0	14.3		28.6
	$\bar{X}$		25.0	21.4	21.7	19.9	11.7	22.5

cycle frequencies obtained. Subjects in both groups displayed wide variations in spectral peak locations. Some subjects showed rhythmicity on some variables but not on others on any particular day of testing. Similarly some subjects evidenced well developed cyclicity at the BRAC frequency for a particular variable on one day but not on the next. Lastly, some subjects showed a peak for a given variable on the first day at one frequency (e.g., at 9.6 c/day) but at a different frequency (e.g., 19.2 c/day) on the second day. Generally then, consistency of results within one particular frequency were not obtained either from one variable to another or from one day to the next.

Some representative data can be presented. Figure 13 contains examples of raw data time series obtained. The upper graph shows a quite distinct 75 minute rhythm in false positive responses in a control subject, while the lower graph shows relatively more variable responding with no obvious cyclicity in another control subject. Figure 14 shows the results of power spectral analysis, with a peak occurring at 19.2 c/day (75 minutes) in limb movement (on task) on the left hand side of the upper (HK subjects) and lower (control subjects) graphs and to the right, no peak showing at the critical frequency in the same variable. Figure 15 shows averaged power spectra for each variable for the HK and control groups on Day 1. No peaks at 19.2 c/day are evident. Similarly, Figure 16 shows averaged power spectra for each variable for the HK and control groups on Day 2. Only limb movement (during the task) shows slight peaking at the critical frequency, while the other variables do not show a reliable peak at this frequency. Statistical evaluation (Wilcoxon matched pairs signed-ranks test) revealed no significant peak at 19.2 c/day compared to adjacent frequencies on any variable on either day.

Figure 13. Examples of raw data time series for false positives (FP responses in two control subjects showing a 75-minute rhythm (upper graph) and no obvious cyclicty (lower graph).


FP Day 2
Responses
14
12
10
8
6
4
2
Trials
2 4 6 8 10 12 14 16 18 20 22 24
Time
1hr. 2hr. 3hr. 4hr. 5hr. 6hr.

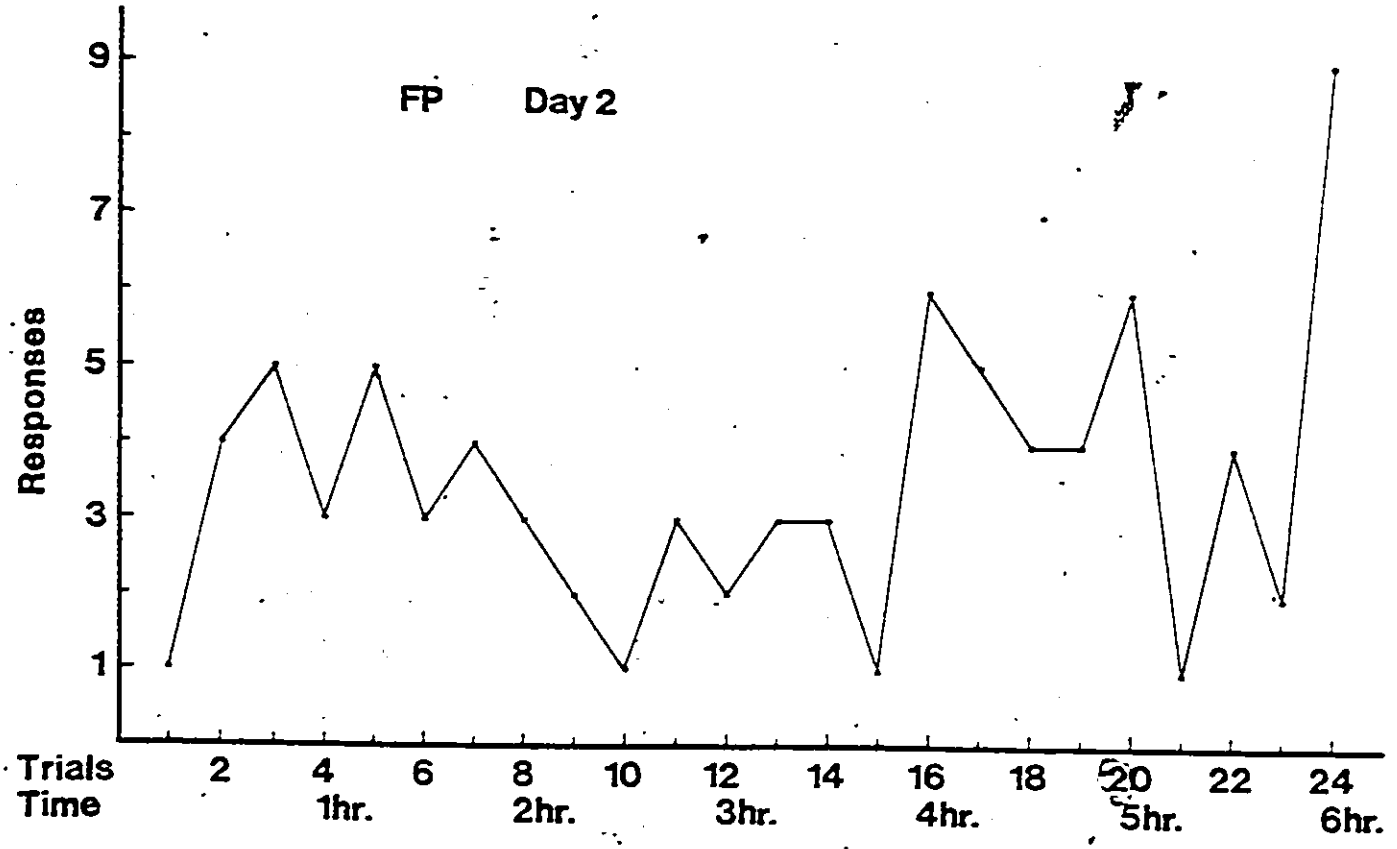

FP Day 2
Responses
9
7
5
3
1
Trials
2 4 6 8 10 12 14 16 18 20 22 24
Time
1hr. 2hr. 3hr. 4hr. 5hr. 6hr.

Figure 14. Examples of individual power spectral plots for limb movement (on task) showing a peak at 19.2 c/day for a HK subject (upper left) and a control subject (lower left) and examples of no peak at this frequency in a HK subject (upper right) and a control subject (lower right).

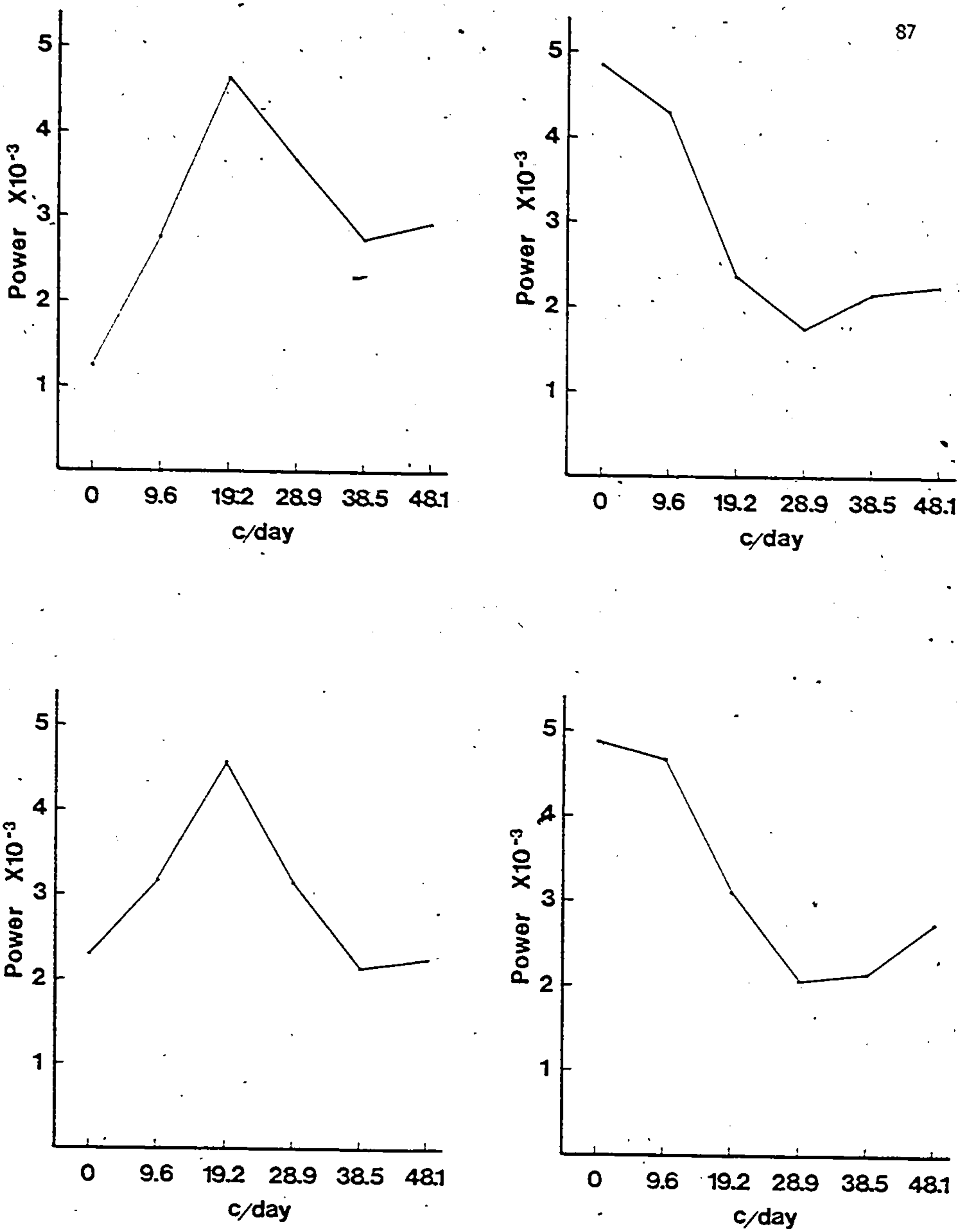
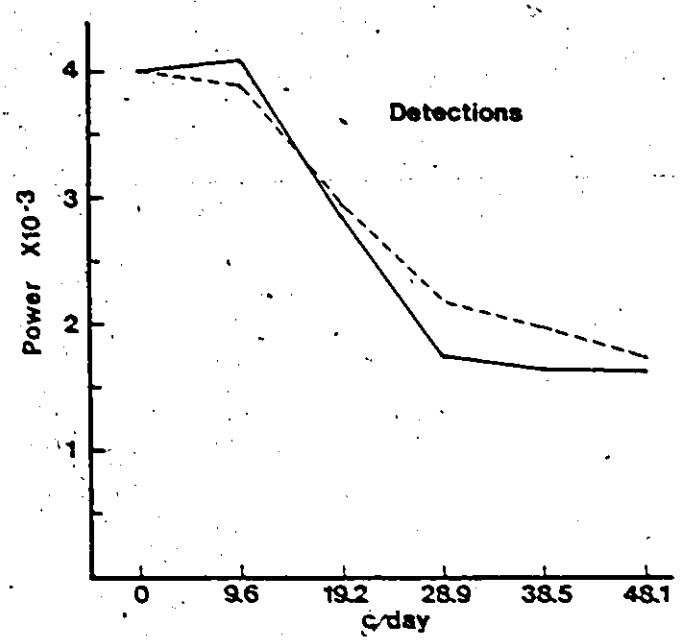
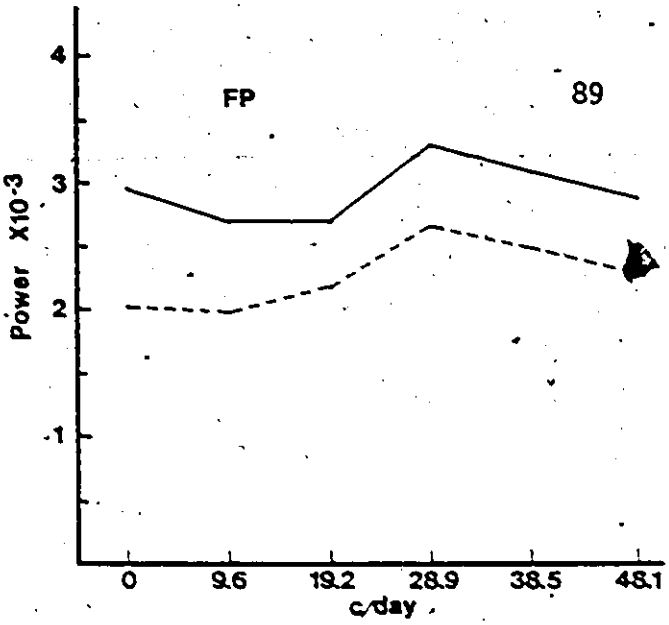
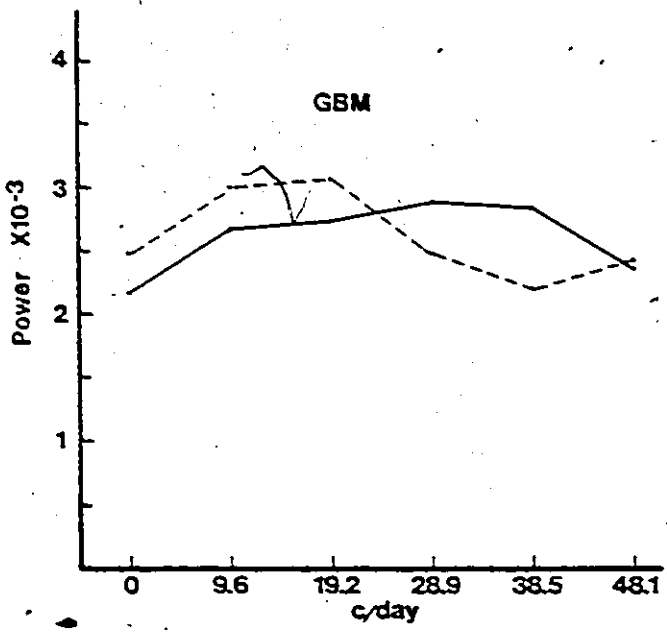
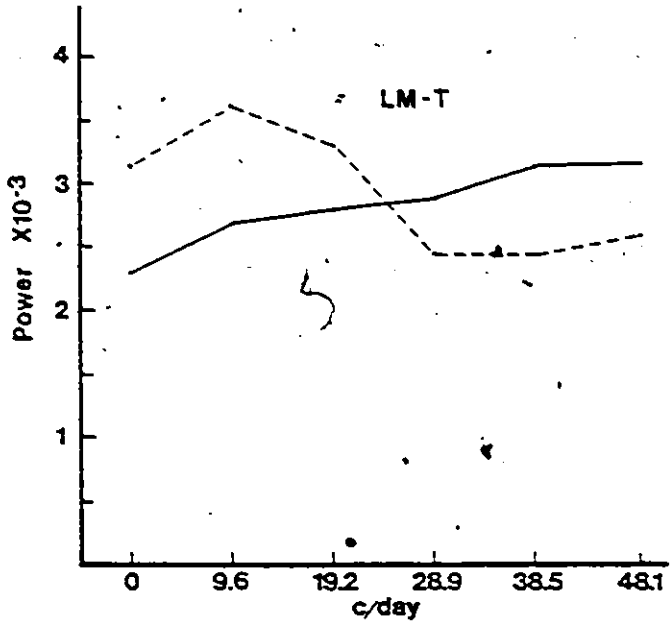

Power X10⁻³
5
4
3
2
1
0
9.6
19.2
28.9
38.5
48.1
c/day
Power X10⁻³
5
4
3
2
1
0
9.6
19.2
28.9
38.5
48.1
c/day
Power X10⁻³
5
4
3
2
1
0
9.6
19.2
28.9
38.5
48.1
c/day
Power X10⁻³
5
4
3
2
1
0
9.6
19.2
28.9
38.5
48.1
c/day

Figure 15. Averaged power spectra for performance and activity measures on Day 1 (HK - solid line, C - dashed line).


Detections
Power X10-3
4
3
2
1
0
9.6
19.2
28.9
38.5
48.1
c/day


FP
Power X10-3
4
3
2
1
0
9.6
19.2
28.9
38.5
48.1
c/day


GBM
Power X10-3
4
3
2
1
0
9.6
19.2
28.9
38.5
48.1
c/day


LM-T
Power X10-3
4
3
2
1
0
9.6
19.2
28.9
38.5
48.1
c/day

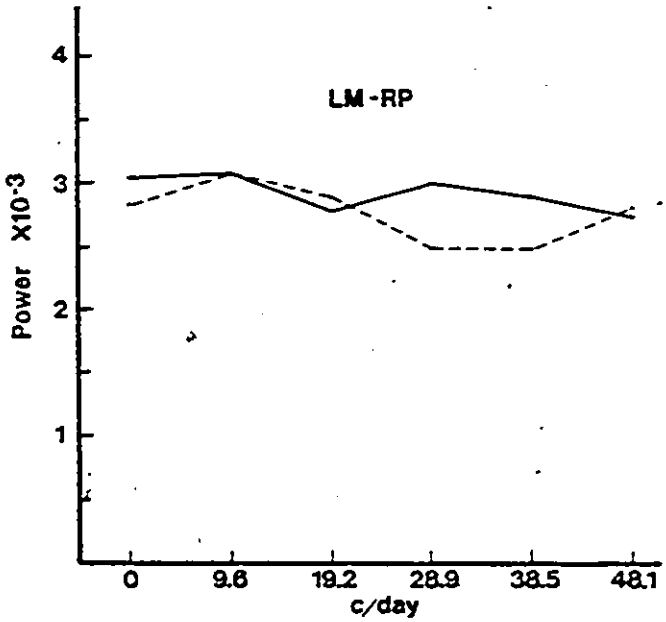
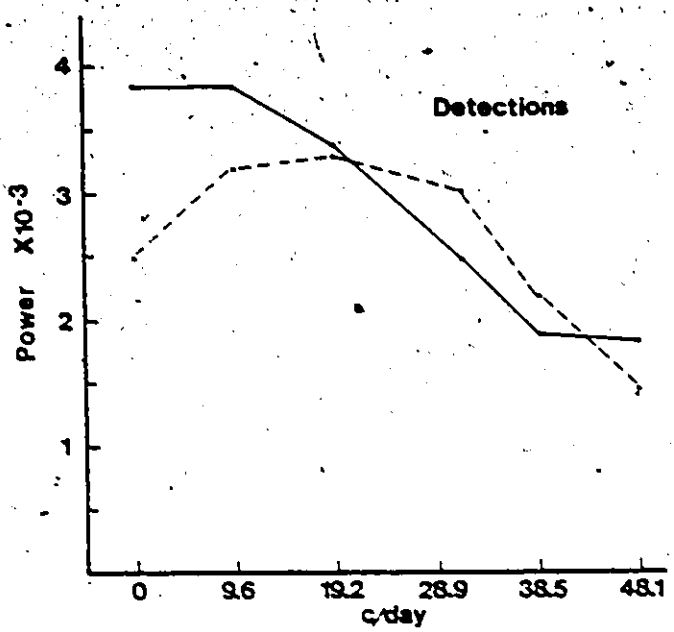
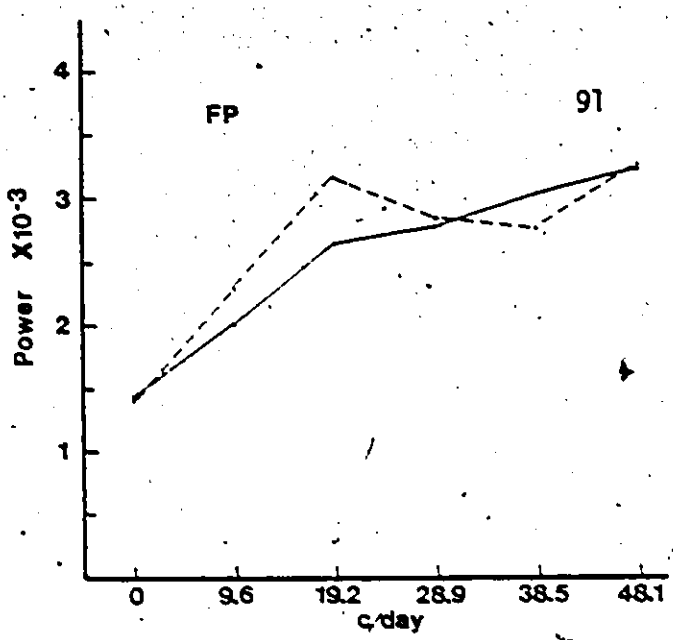
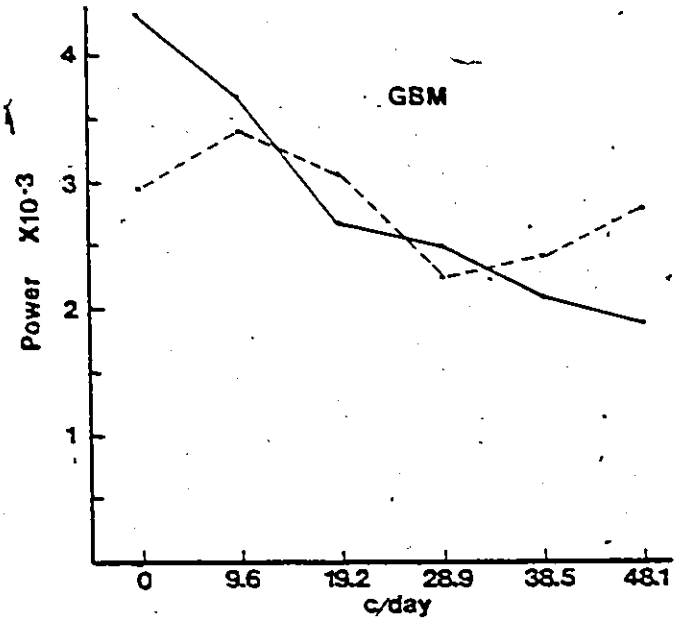
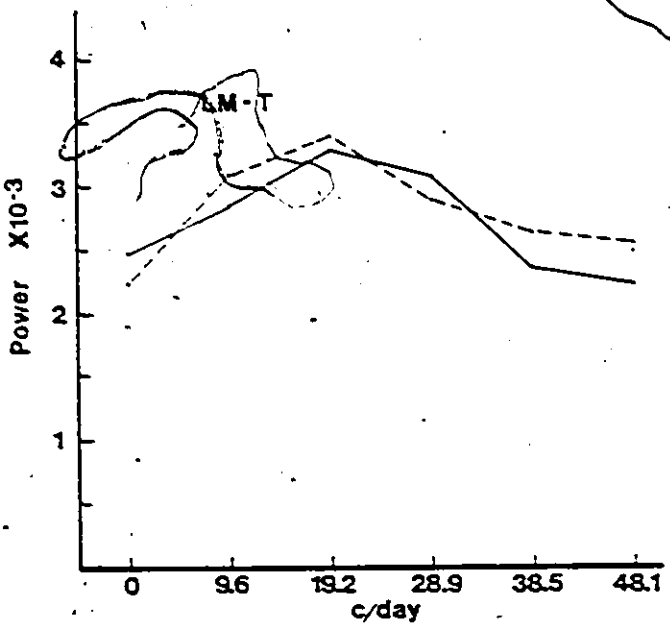

LM-RP
Power X10-3
4
3
2
1
0
9.6
19.2
28.9
38.5
48.1
c/day

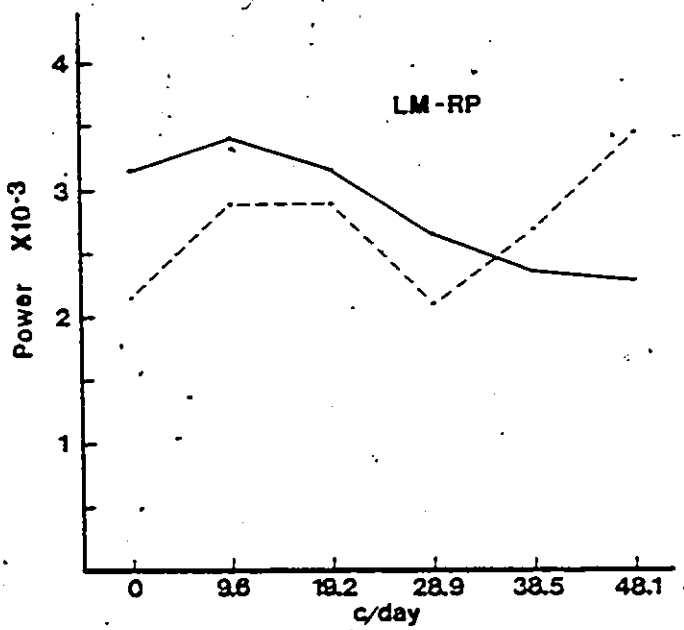
Figure 16. Averaged power spectra for performance and activity measures on Day 2 (HK - solid line, C - dashed line).


Detections
Power X10-3
4
3
2
1
0
9.6
19.2
28.9
38.5
48.1
c/day


FP
Power X10-3
4
3
2
1
0
9.6
19.2
28.9
38.5
48.1
c/day


GSM
Power X10-3
4
3
2
1
0
9.6
19.2
28.9
38.5
48.1
c/day


LM-T
Power X10-3
4
3
2
1
0
9.6
19.2
28.9
38.5
48.1
c/day


LM-RP
Power X10-3
4
3
2
1
0
9.6
19.2
28.9
38.5
48.1
c/day

Further analysis using the Chi-Square statistic was undertaken to investigate whether either group showed an increased presence versus absence of rhythmicity at the critical frequency (19.2 c/day). All analyses yielded non-significant results for all variables on both days. When this analysis was repeated including a wider range of frequencies (i.e., if a spectral peak occurred at 9.6, 19.2, or 28.9 c/day), the control group showed a greater number of spectral peaks in these pooled frequencies for detections on Day 2 compared to the HK group ($X^2 = 6.22$, $p < .02$). No other variables reached significance using the pooled frequency method.

Relationships between daytime variables and preceding nocturnal sleep

Descriptive analysis of the superimposition of waking rhythms over the predicted occurrence of REM periods had sleep continued showed inconsistant results. Figures 17, 18, and 19 show representative data (four examples each) for detections, global body movements, and limb movement during the rest periods respectively. All of the examples were based on raw data exhibiting a 68-84 minute ultradian rhythm. Examples for detections (Figure 17) show peaking occurring within the inter-REM (NREM) phase had sleep continued. For global body movements (Figure 18) peaking also occurs predominantly in the predicted inter-REM portion. The location of the peaks within this phase vary from near the beginning or onset, to the middle portion, and toward the end of the NREM portion. With regard to limb movement during the rest periods (Figure 19) mixed results are obtained. The top graph shows peakes approximately in the middle of the predicted

Figure 17. Four examples of the relationship between peaks in detections during wakefulness and the predicted occurrence of REMPs into waking. Darkened portion represents the duration of the last REMP of the preceding night's sleep, W - waking from sleep, O - predicted REMP onset, M - predicted REMP midpoint.

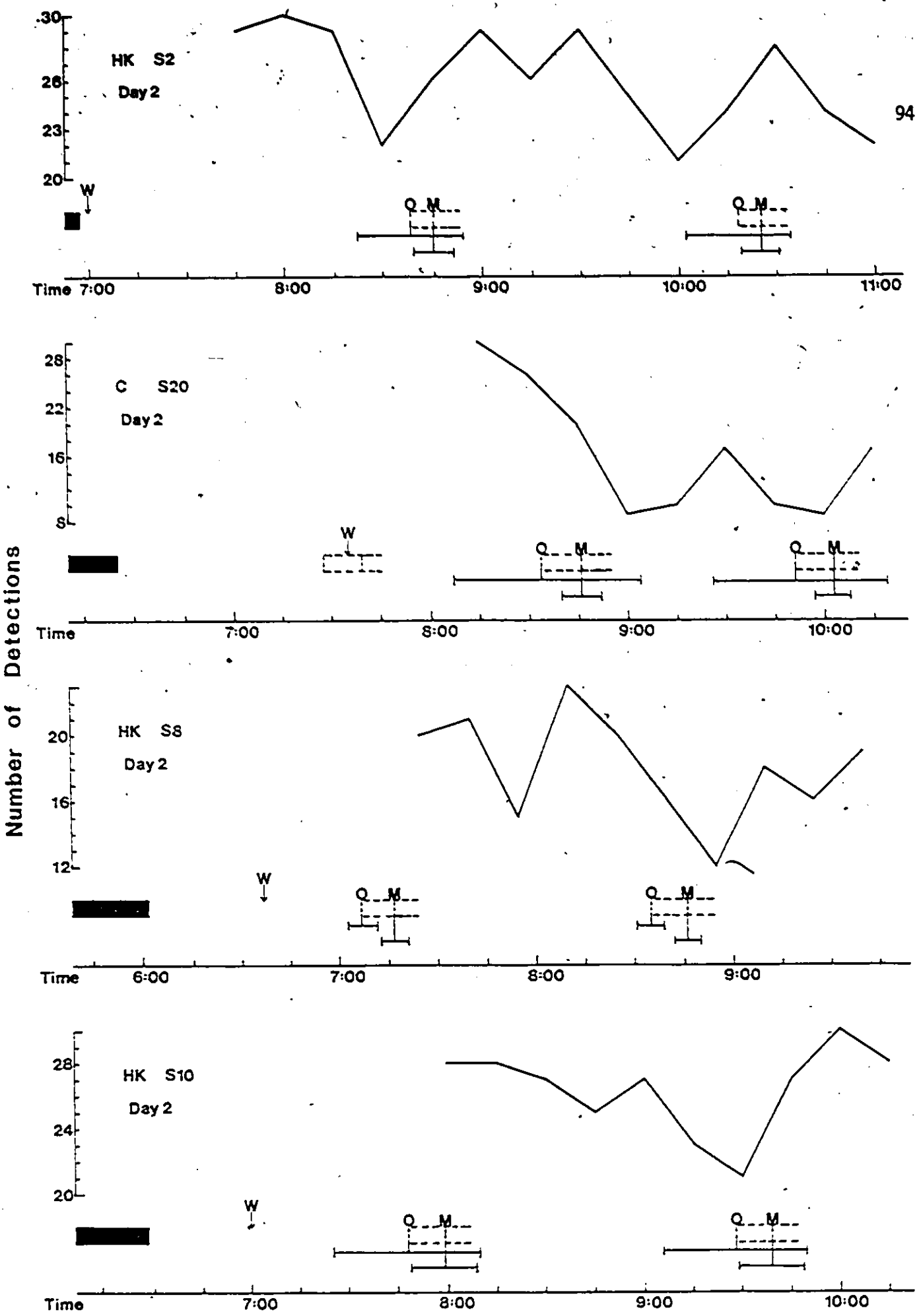

HK S2
Day 2
30
26
23
20
W
Q M
Q M
Time 7:00
8:00
9:00
10:00
11:00
C S20
Day 2
28
22
16
8
W
Q M
Q M
Time
7:00
8:00
9:00
10:00
HK S8
Day 2
20
16
12
W
Q M
Q M
Time
6:00
7:00
8:00
9:00
HK S10
Day 2
28
24
20
W
Q M
Q M
Time
7:00
8:00
9:00
10:00
Number of Detections

Figure 18. Four examples of the relationship between peaks in global body movements during wakefulness and the predicted occurrence of REMPs into waking. Darkened portion represents the duration of the last REMP of the preceding night's sleep, W - waking from sleep; O - predicted REMP onset, M - predicted REMP midpoint.

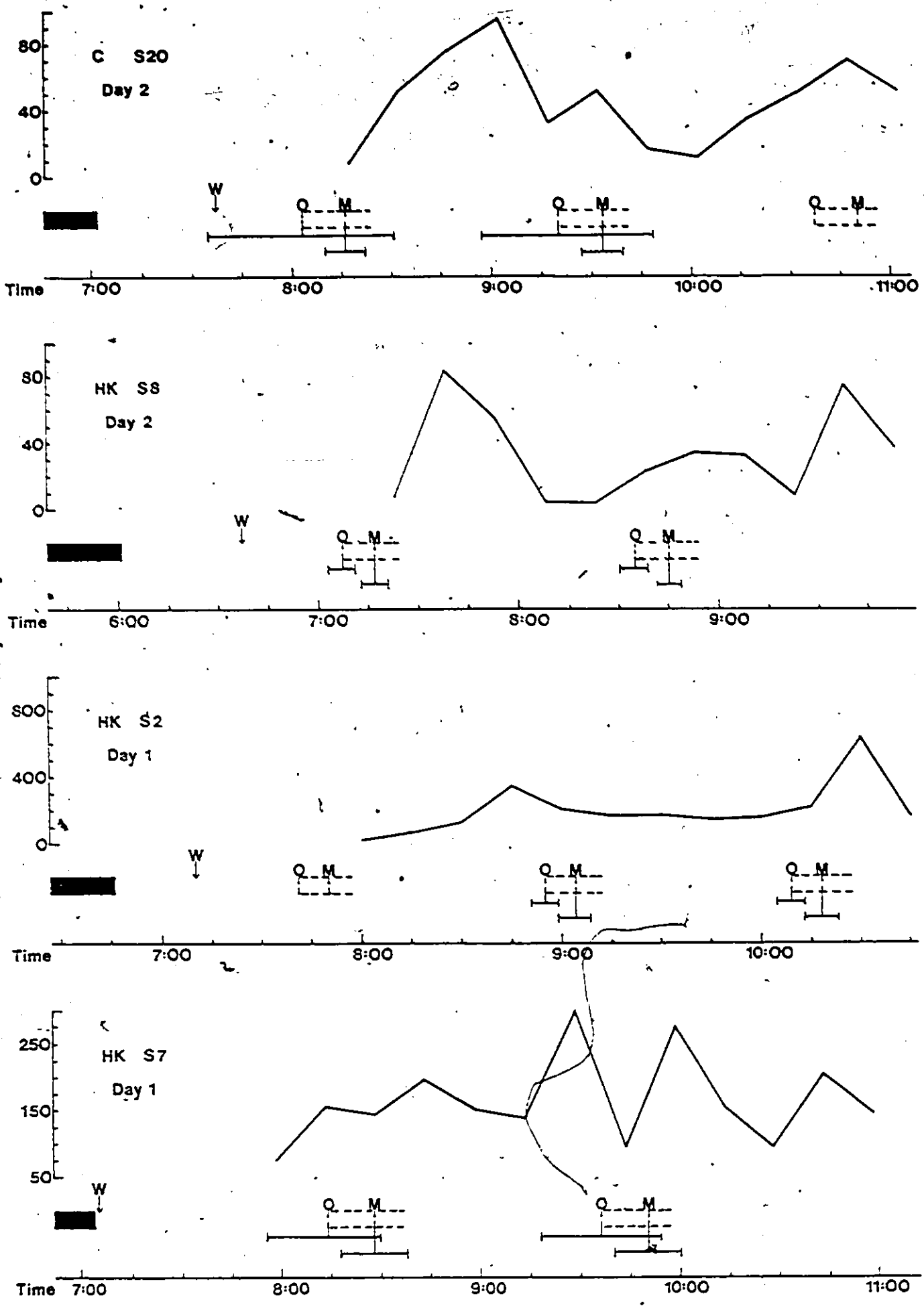
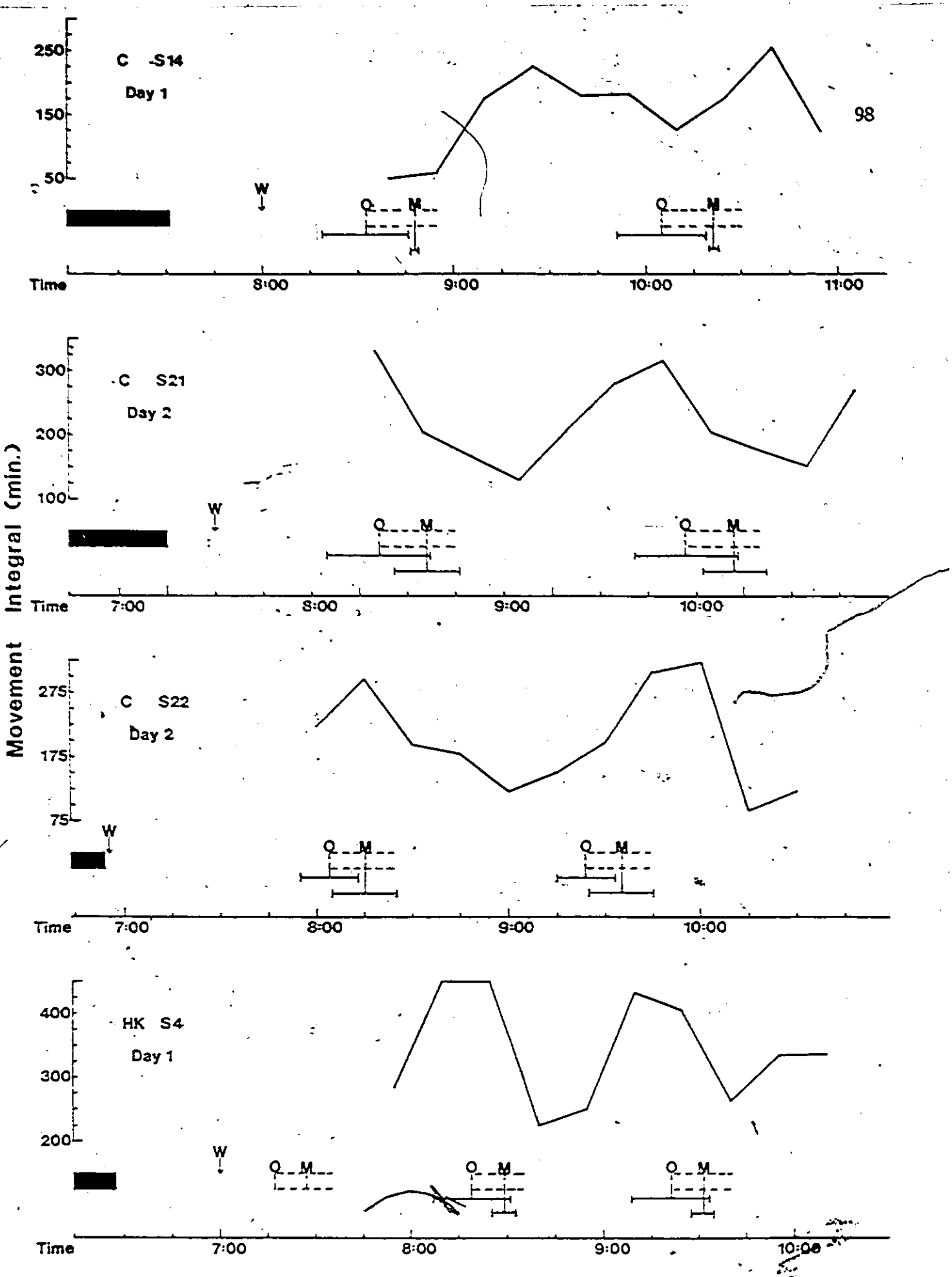

C S20
Day 2
80
40
0
W
Q M
Time 7:00 8:00 9:00 10:00 11:00
HK S8
Day 2
80
40
0
W
Q M
Time 6:00 7:00 8:00 9:00
Frequency
HK S2
Day 1
500
400
0
W
Q M
Time 7:00 8:00 9:00 10:00
HK S7
Day 1
250
150
50
W
Q M
Time 7:00 8:00 9:00 10:00 11:00

Figure 19. Four examples of the relationship between peaks in waking limb movement during the rest periods and the predicted occurrence of REMPs into wakefulness. Darkened portion represents the duration of the last REMP of the preceding night's sleep, W - waking from sleep, O - predicted REMP onset, M - predicted REMP midpoint.


C S14
Day 1
250
150
50
W
Q M
Time
8:00
9:00
10:00
11:00
98
C S21
Day 2
300
200
100
W
Q M
Time
7:00
8:00
9:00
10:00
Movement Integral (min.)
C S22
Day 2
275
175
75
W
Q M
Time
7:00
8:00
9:00
10:00
HK S4
Day 1
400
300
200
W
Q M
Time
7:00
8:00
9:00
10:00

inter-REM phase. The second and third graphs show peaks coincident with the onset of predicted REM periods and then gradual shifting. The fourth graph shows peaks occurring at roughly the predicted REM onset time. The general conclusion drawn from this limited data suggests non-stationarity displayed by these waking rhythms with respect to the predicted occurrence of REM periods. Due to the lack of consistency in occurrence of these waking rhythms with respect to the predicted REM and NREM phases and no published documentation of mathematical evaluation, no statistical tests were attempted.

A further example of sleep-waking relationship in rhythms is presented in Figure 20 in which the total previous night's sleep and waking rhythms in detections and false positive responses are graphed. Detections peak in the NREM phase while false positive responses are in phase with the occurrence of predicted REM. This also graphically represents the relationship of detections to false positives which are approximately 180^{0} out of phase to each other in this HK subject, supporting the significant negative correlation between these variables as reported earlier.

To further investigate the relationships which might exist between nocturnal sleep and waking variables, motor activity variables during these two states were correlated. The correlations amongst these variables are presented in Table 15. Significant correlations were obtained between REM twitches and limb movement during the rest periods ($r = 0.57$, $p < .01$) and between NREM twitches and limb movement during the rest periods ($r = 0.45$, $p < .05$). No other correlations between waking and sleep measures of motility reached significance.

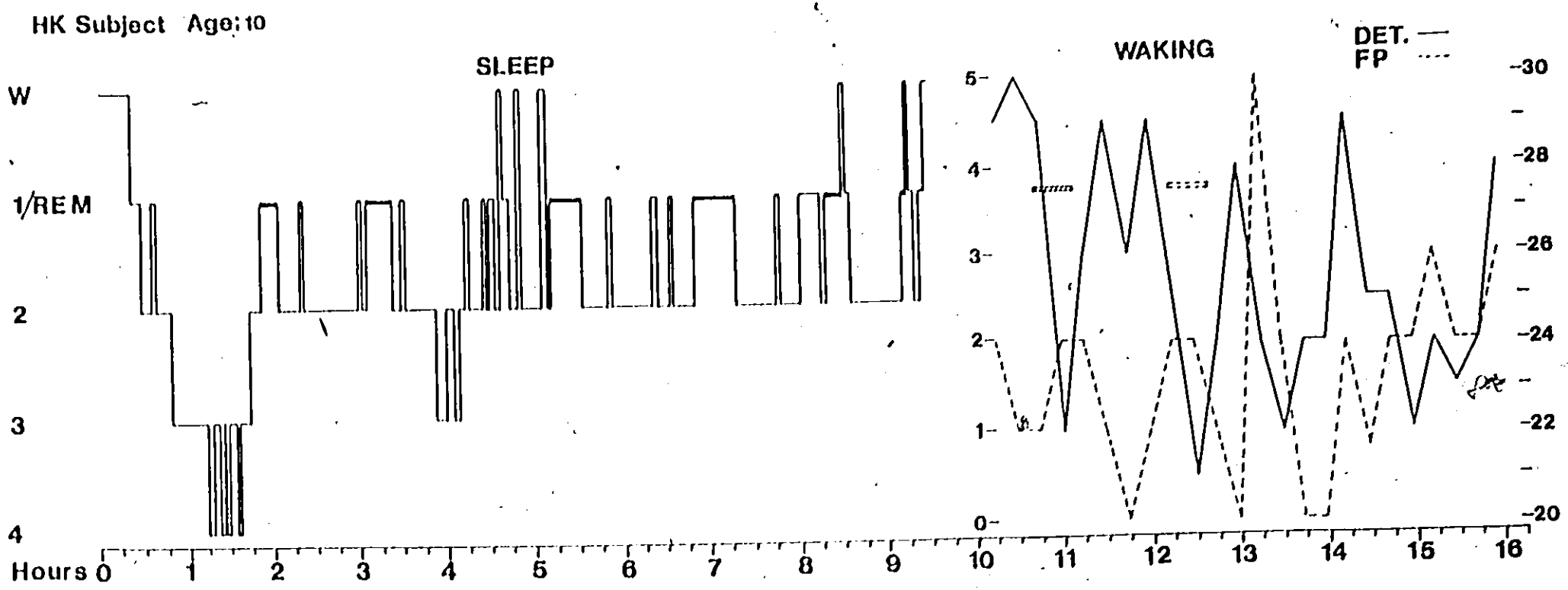


Figure 20. Previous nights' sleep histogram with waking ultradian rhythms. (75 minutes) in detection and false positive responses. Hash-marked bars represent predicted REMPs had sleep continued.

Table 15

Correlation Matrix of Waking and Sleeping Motility Measures

Waking Variable	Preceding Nocturnal Sleep Variable				
	MT(min)	MT(%)	BM	REMT	NREMT
GBM	-0.02	0.01	-0.07	-0.05	0.12
LM-T	0.32	0.35	0.09	0.10	0.08
LM-RP	0.20	0.18	0.30	0.57**	0.45*

*$p < .05$

**$p < .01$

MT(min) = movement time in minutes
MT(%) = movement time in percentage
BM = body movements
REMT = REM twitches
NREMT = NREM twitches
GBM = global body movement
LM-T = limb movement on task
LM-RP = limb movement during rest periods

Chapter IV

DISCUSSION

Sleep

Stages. The findings of this study support and extent results obtained in the previous investigations of baseline sleep patterns of nonmedicated HK children. In agreement with previous reports (except Luisada, 1969) no significant differences in absolute (minutes) or percentage amounts of the various sleep stages were found between HK and normal control children.

The pattern of across night differences observed for several sleep variables (e.g., decreasing TWT, WASO, and NREM, and increasing TST, SEI, and REM) suggest the occurrence of the common phenomenon of sleep laboratory adaptation. This well-known "first-night effect" (Agnew, Webb, & Williams, 1966; Dement, Kahn, & Roffwarg, 1965; Rechtschaffen, & Verdone, 1964; Schmitt, & Kaelbling, 1971) was indexed in the present study by the reduced amounts of REM sleep and greater amounts of waking time on the first night in the sleep laboratory compared to subsequent recording nights. Since previous reports examining the sleep of HK and normal children have routinely discarded data from the first one or two nights, this observation provides initial data on the existence of a first-night effect in nonclinical samples of this age group as well as in HK children.

Cycles. No significant group differences were found for either NREM or REM average cycle durations, suggesting that the mechanisms governing the periodic alternation of NREM and REM sleep are intact in these HK children.

The trends in sleep cycle duration across the night agree with previous findings for human sleep indicating that intra-sleep cycles are not constant in duration (Feinberg, 1974; Feinberg, & Floyd, 1979; Feinberg, Koresko, & Heller, 1967). However, the results from the present study do differ from previous reports with regard to REM cycle duration across the night. Feinberg and Floyd (1979) reported a curvilinear ("umbrella-shaped") trend in this measure, with the first and fourth cycles being shorter in duration than the middle two, which were of nearly equivalent durations. The same analysis applied to REM cycle data in the present study did not yield a curvilinear trend configuration. Instead, the HK group evidenced a steadily declining linear trend in REM cycle duration across the night, while the control group's REM cycles decreased in duration from the first to the second cycle, lengthened in the third cycle, and finally decreased again in the fourth cycle. The longer first REM cycle length shown for both groups may be related to REM onset latency. A positive relationship was found between REM onset latency and the duration of the first REMP, such that the longer the latency to REM onset, the longer the duration of the first REMP. The increased first REMP duration may have affected the subsequent first REM cycle length by extending its duration.

The NREM cycle durations across the night are more consistent with those observed in previous reports, i.e., with the first cycle showing a longer duration typical of children's sleep (Feinberg, & Floyd, 1979; Feinberg et al., 1971; Williams, Karacan, & Hursch, 1974). This first NREM cycle duration was exaggerated in the HK group who showed a significantly longer REM onset

latency. The addition of wake time to these analyses increased the absolute values, but did not change the configuration of these REM and NREM cycle durations across the night.

There has been a consuming interest in the REM sleep cycle to the exclusion of adequate considerations of the NREM portion of sleep. Feinberg and Floyd (1979) have argued for a comprehensive description of the cyclicity within sleep. As is evident from the intra-sleep cycles in the present study, REM cycle durations (mean = 90.5 minutes) are much shorter than NREM cycle durations (mean = 109.7 minutes). The different rates of alternation or periodicity of these two sleep states may indicate varying functional properties for these two rhythms and point out the need for comprehensive analyses of these intra-sleep rhythms to determine the general significance of these variable oscillatory phenomena and their involvement in the diagnosis of disorders of initiating and maintaining sleep (insomnias), as well as in disorders of the sleep-wake schedule (time zone changes, shift work, delayed and advanced phase syndromes).

Latencies. In the present study, latencies to the onset of the first REMP were significantly longer in the HK group relative to controls. Although in many cases of younger children's sleep (e.g., 7-12 years old) the first REMP appears to be "missed" (Roffwarg, Muzio, & Dement, 1966), the differential distribution of latencies in the present study, together with previous reports of extended REM onset latencies in HK subjects (Haig et al., 1974), warrants further consideration of this variable.

A number of naturally occurring and experimentally-induced conditions are known to influence latency to the first REMP. With regard to reduced REM latency,

normative data on the evolution of sleep structure and morphology following the course of developmental maturation show that shorter REM onset latencies are present in neonates and infants (Roffwarg, Muzio, & Dement, 1966). In the classic REM deprivation paradigm, subjects increase the number of attempts at initiating REM (reduced latencies) during deprivation and display reduced latencies to the first REMP during recovery sleep (Dement, 1960). Two clinical conditions are also known to be related to reduced REM onset latency. In both depression and narcolepsy, significantly reduced latencies to REM have been consistently observed (Kupfer, 1976; Montplaisir, Billiard, Takahashi, Bell, Guilleminanlt, & Dement, 1978). Within the context of the ontogenetic and REM deprivation literature as mentioned above, increased CNS excitability is thought to be associated with reduced REM onset latencies and increased REM amounts, which might possibly relate to the presence of a hyperaroused state. Applying this reasoning to the increased REM latencies of HK children in the present study suggests the opposite interpretation, i.e., that these children may be centrally hypoaroused.

Extended or delayed REM onset latencies are known to be influenced by maturation. Feinberg et al. (1967) reported a mean REM onset latency of 140 minutes for a sample of six-year old children and 123 minutes for a sample of 10-year old children. In the present study, REM onset latencies in the HK group were more similar to those of these younger-aged normal children. This could be interpreted as suggestive evidence for a developmental lag, although the remainder of their sleep was relatively normal compared to similar-aged controls.

Various drugs such as amphetamines (Rechtschaffen, & Maron, 1964) barbiturates (Kales, Malstrom, Scharf, & Rubin, 1969), and alcohol (Johnson, Burdick,

& Smith, 1970) are also known to extend REM onset latency. These drugs act primarily as REM suppressing agents. In addition, experimentally-induced total sleep deprivation and slow-wave (specifically stage 4) sleep deprivation increase REM onset latency during recovery sleep (Agnew, Webb, & Williams, 1964; Berger, & Oswald, 1962). Also, the temporal placement of sleep within the sleep-waking cycle can influence REM latency. Naps taken in the morning show decreased REM latencies and contain more REM sleep, while naps in the late afternoon or early evening exhibit longer latencies to REM and contain more NREM sleep (Moses, Hord, Lubin, Johnson, & Naitoh, 1975). Studies examining the effects of exercise on sleep have generally reported enhanced NREM sleep (specifically slow-wave) after exercise (Baekeland, & Lasky, 1966; Hobson, 1968; Matsumoto, Nishisho, Suto, Sadohiro, & Miyoshi, 1968; Shapiro, Griesel, Bartel, & Jooste, 1975; Zloty, Burdick, & Adamson, 1973). Several of the above studies, as well as others which failed to find exercise effects on sleep stages, examined REM onset latencies subsequent to exercise. Of these, three (Baekeland, & Lasky, 1966; Hauri, 1968; Walker, Floyd, Fein, Cavness, Lualhati, & Feinberg, 1978) found no effect while five (Baekeland, 1970; Browman, & Tepas, 1976; Desjardins, Healey, & Broughton, 1974; Hobson, 1968; Matsumoto et al., 1968) reported significantly longer REM onset latencies. Horne and Porter (1976) observed longer latencies to the first REMP in three of their eight subjects. There is some support, then, for the association of increased pre-sleep activity with longer REM onset latencies. This interpretation of activity effects on subsequent sleep remains largely speculative since no evaluation or

experimental control of presleep activity levels was provided in this study. However, it is notable in this regard, that during the daytime testing off-task periods, HK subjects did exhibit significantly greater amounts of limb movement.

Thus, several factors seem to influence the latency of the first REMP. These include the effects of age (increased NREM sleep in the first cycle with maturation), time of day, and amounts of SW sleep preceding the occurrence of REM sleep. It remains unclear as to whether the longer REM onset latencies displayed by HK children in the present study are due to the naturally-occurring longer periods of sustained arousal during the daytime, increased motoric activity and the opportunity for more environmental interaction, maturational factors influencing CNS development and differentiation, or a combination of these elements. As previously stated, the increased diurnal motility in HK children may be responsible for delaying REM onset, such that the postulated restorative function of NREM sleep must first be satisfied before mechanisms responsible for the initiation of REM are triggered. Alternatively or in addition, there may be unknown factors influencing events occurring specifically within sleep which may selectively alter SW or REM sleep initiation and maintenance mechanisms in the first cycle of sleep.

The latency data has implications for differentiating between the hyperarousal and hypoarousal theories of HK. If HK children are hyperaroused, then minimizing external stimulation such as occurs when environmental

stimulation is reduced in preparation for falling asleep, should induce shorter sleep onset latencies relative to those exhibited by normal children. Alternatively, sleep onset latencies in hypoaroused HK children could be either shorter, since further reducing external stimulation would be conducive to initiating sleep, or longer, since these children might persist in seeking stimulation and thus delay the onset of sleep. Of the three studies examining sleep onset latencies in HK children, Feinberg et al. (1974) found no differences, Haig et al. (1974) found longer latencies, and Small et al. (1971) reported shorter latencies. The present study found no significant differences, although mean values were longer for the HK subjects. No significant effects of exercise on latency to sleep onset have been reported other than Hobson (1968) noting a sleep enhancing effect with moderate exercise in cats.

Motility during sleep. The results regarding motility during sleep in HK children support the findings obtained by Small et al. (1971). They found significantly greater amounts of muscle activity during both REM and NREM sleep. In the present study, greater amounts of movement time, body movements and NREM twitches were observed in HK subjects relative to controls. The relatively stronger effect observed by Small et al. may derive from the selection of HK subjects primarily on the basis of overt motoric restlessness, whereas selection of HK subjects in the present study was not based solely on this

symptom. This greater restlessness during sleep could indicate the existence of a possible continuity relationship between HK children's waking-over-activity carried over into sleep.

These results could also be interpreted within the context of the hyper/hypoarousal theories. Since REM sleep is a period of relatively intense physiological activity, hyperaroused HK children might be expected to show greater motility (twitches) during this state, in that the intense endogenously produced activity would further facilitate excitatory systems. However, if HK children are hypoaroused, then falling asleep would further reduce arousal level. Since external environmental stimulation is minimized, these children would have to rely upon endogenously-produced stimulation. This might be manifested in a greater amount of NREM motor activity (twitches), since NREM sleep is a state of relatively reduced physiological activity. Excessive motoric activation during REM sleep might not be evidenced since the physiological activation provided by this state might provide sufficient endogenous activation. A greater number of NREM twitches was found in the HK group, while no group differences in REM twitch frequency were found in this study. The results of the present study, with respect to motility would be congruent with the hypoarousal interpretation.

Autonomic activity. This is the first study to report on an autonomic activity index during sleep in HK children. However, SSPR activity failed to provide evidence of an autonomic physiological differentiation between HK and control children. The finding of no group differences in SSPR frequency during the various sleep stages complements the previous literature investigating

spontaneous or nonspecific SSPR activity during wakefulness. Furthermore, these observations parallel those observed for children and normal young adults in previous sleep studies (Broughton, Poiré, & Tassinari, 1965; Johnson, & Lubin, 1966; Koumans, Tursky, & Soloman, 1968; Lester, Burch, & Dossett, 1967). All of these studies, the present one included, found the highest SSPR frequency in Stage 4, with Stage 2 next, and finally, the lowest rates occurring during REM sleep. The results add further developmental data concerning the maturation of autonomic nervous system response patterns in this age range showing them to be comparable to those displayed by normal adults.

Eye movement activity. Only two of the seven studies examining sleep characteristics in HK children have reported eye movement (EM) density measures during REM sleep (Feinberg et al., 1974; Small et al., 1971). Both of these studies calculated EM density based on the percentage of four-second REM mini-epochs containing EM. They observed no significant differences between groups on this measure during baseline sleep (Feinberg et al.: HK = 43.9%; Control = 38.3%; Small et al.: HK = 43.1%; Control = 34.2%). The present study calculated EM density on the basis of the percentage of two-second REM mini-epochs containing EM. Therefore, the resolution of the EM density analysis in this study was enhanced relative to that previously reported. Still, the results of this more precise analysis were comparable to those reported above (HK = 42.1%; Control = 43.6%). It has been suggested that various measures of EM density during REM sleep may provide an index of

the overall amount of CNS activation (Aserinsky, & Kleitman, 1955; Dement, Ferguson, Cohen, & Barchas, 1969). One might have expected that if HK children are centrally hyperaroused, higher EM densities would be manifested, or alternatively, if centrally hypoaroused, lower EM density during REM would be evidenced. However, since no significant group differences in EM density were found, differential CNS activity as indexed by this measure is not indicated in these HK children compared to normal children.

Further analyses of EM density both across and within REMPs in this study revealed results comparable to those previously reported for normal young adults and for children (Aserinsky, 1969, 1971, 1973; Feinberg, 1974). Using an extended night sleep paradigm, Aserinsky (1969, 1973) noted a progressive increase in EM density with successive REMPs, with a steep rise between 7.5-10 hours of sleep. Thereafter, with additional sleep, REM density remained at this high level with little variability. Since there are usually no more than five REMPs during the course of a night's sleep, after some critical level of sleep accumulation, REM density may approach a maximum. Aserinsky suggests that physiological processes related to sleep reach a threshold level which then triggers waking and prevents any additional rise in EM density.

Feinberg's (1974) results on changes in EM density across REMPs in children are consistent with those obtained in this study. For children with a mean age of 7.4 years, EM densities of 0.56, 0.48, 0.52 and 0.53 were found for the first four REMPs respectively. For children with a mean age of 13.8

years, these densities were 0.34, 0.46, 0.63, and 0.54. The present study (mean age 10.6 years) found mean densities of 0.32, 0.41, 0.48, 0.50 across REMPs (pooled data across groups).

The suggestion that prior accumulation of REM sleep influences EM density was examined in this study by investigating the progression of this measure within REMPs. It would be expected that as REM duration increases, density would also. Aserinsky (1971) observed a consistent pattern of EM activity within the first 20 minutes of REMPs. EM density peaked 5-10 minutes following REM onset and significantly decreased in the latter 10 minutes. These findings were confirmed in the present subsample of children in which there was a significant increase from the first to the second five-minute block of each REMP (Figure 9) and in a longer REMP of 30 minutes duration (Figure 10). Aserinsky also observed an oscillatory phenomenon in longer REMPs, i.e., a 40-minute REMP had two peaks and a 60-minute REMP evidenced three peaks. Lavie (1979) and Krynicki (1975) have supported this observation by finding a 10-20 minute periodicity in EM within REMPs in normal adults using spectral analyses.

The results of the EM analyses revealed no significant group differences in this measure as a mean value for total REM, across successive REMPs, or within REMPs. Moreover, these measures are comparable to those reported in previous studies of sleep in children and adults.

In summary, the sleep of HK and normal children does not differ significantly with respect to amounts of conventional sleep stages, cycling characteristics, autonomic nervous system activity, or phasic EM activity. The

increased REM onset latency and greater motility during sleep evidenced by the HK children in this study may be best accommodated by the hypoarousal theory of HK, but based upon comprehensive sleep pattern evaluation, the existence of a major arousal dysfunction or abnormal brain function in these children does not appear to be indicated. These conclusions support those of Hastings and Barkley (1978) that despite the psychophysiological examination of a wide variety of arousal states in these children, a reliable index differentiating HK from "normal" children has not been found.

Performance and Activity in Hyperkinesis

HK children perform poorly on tasks requiring focused, sustained attention in relatively structured settings (Anderson, Halcomb, & Doyle, 1973; Douglas, 1972; Sykes et al., 1971). This deficiency is evident in experimenter-controlled situations which are long in duration, such as vigilance tasks. The finding in the present study that HK subjects made fewer correct detections on the CPT is consonant with the interpretation of a deficiency in sustained attention. There were, however, no between group differences in false positive responding -- a measure which has been interpreted as an index of impulsivity.

It is interesting to note that when Sykes et al. (1971) manipulated the interstimulus interval (using the same CPT), HK and normal children made just as many errors of commission at a fast (letter stimulus every 1.0 second) rate of presentation, but controls made significantly fewer false positive responses at a slower (stimulus every 1.5 seconds) presentation rate. They suggested that normal children used the additional time to

assess each stimulus more efficiently and, therefore, could inhibit responding to nonsignal stimuli. In the present study in which the slower presentation rate (1.5 second interstimulus interval) was used, the HK subjects apparently utilized this time just as effectively as controls since they made just as many commission errors as control subjects. The significant negative correlations (pooling data from both groups) obtained on the two testing days between detections and false positives are also noteworthy. This confirms the reciprocal relationship between these two measures, i.e., as number of detections increases, commission errors decrease.

These performance results can be considered within the postulated hyper/hypoarousal framework. Hyperaroused HK children, being unable to filter stimulus input effectively, might respond inefficiently and make fewer correct detections. However, this model would also predict increased commission errors. Since no group differences were found in false positive responses, hyperarousal does not seem adequate to explain these results. On the other hand, if HK children were hypoaroused, fewer detections would be expected assuming that HK subjects might not find the task stimulating enough and would display lapses in attentive abilities which would not effect false positive responding.

The nature of results from the activity measures were unexpected. In past studies, global body movement and specific limb movement measures have indicated increased restlessness in HK children while performing structured tasks (Barkley, 1977; Juliano, 1974; Sykes et al., 1971; Ullman et al., 1978). In fact, Cromwell, Baumeister, and Hawkins (1963) have argued for a causal relationship between the inability to sustain attention and heightened activity

levels. In the present study, no differences were found in global body movement during task performance and on only one day (Day 2) did HK subjects exhibit greater (marginally significant) specific limb motility at this time. Furthermore, no significant correlations were obtained between performance measures on the attention task and on-task measures of global body or limb movement. However, a positive correlation was found between these two activity variables on Day 2 of testing. The lack of correlation between attention and activity suggests that in this case, poor performance is not related to motor restlessness. The positive relationship between the two activity measures is not surprising, since greater global body movement often incorporates increased limb movement.

The lack of group differentiation on movement measures during the task may have been influenced by the scheduling imposed by the experimental testing situation. The task was only of five minutes duration with frequent "rest periods" between testing trials, and HK subjects may have been able to effectively inhibit excessive movement for these short periods of time. This may account for the results of Day 1 testing. However, on Day 2, HK subjects, already familiar with the schedule of repeated testing, might have been less able to inhibit this motility.

The significantly greater activity displayed by the HK group in limb movement during the rest period was also unexpected. Typically, group differences have not been found on movement indices during periods of free play or in unstructured settings, and if the supervised off-task periods can be equated to "free play" situations in previous experiments (Ellis et al., 1974; Sleator, & von Neumann, 1974), then the above results are clearly

contrary to those previously reported. However, the supervision present during these off-task intervals may have introduced an element of structure into the situation and HK children may have perceived the situation as being less under their own control and more exogenously initiated. The off-task condition may have, therefore, been more equivalent to what Barkley (1977) and Ullman et al. (1978) have termed "restricted free play". In this context, the present results are in support of the above two studies showing greater motility under such conditions.

Another possible explanation of greater movement off-task may be based on the nature of the experimental setting. HK subjects may have been aware of the need to inhibit movement and did this effectively while performing on task since task duration was short (five minutes). During the off-task periods, this inhibition of movement may have been relaxed eventuating in heightened activity levels. According to hyperarousal theory, HK subjects would have been expected to show excessive motility under all conditions. This was not found in the present study. The results are more comparable with a hypoarousal interpretation according to which the HK subjects may have found the task sufficiently stimulating and, therefore, found no need to increase proprioceptive and kinesthetic stimulation. They may have been able to focus attention on the task, but manifested an inability to do so effectively, thus accounting for the decreased number of detections. However, the monotony of the off-task periods may have prompted increased motility in HK subjects.

Since HK children's ability to sustain attention seems to be subject to frequent lapses, it has been suggested that material to be learned be distributed across a number of sessions (Sykes et al., 1973). In the present study,

even with the introduction of frequent rest periods, the attentional deficit indexed by fewer number of detections, persisted, but false positive differences between groups were minimized. It is possible that this method of presentation might be effective with extended testing since the experimental setting and repeated performance on the CPT would eventually lose its novel nature. This setting cannot be equated to typical learning situations such as those which occur in the classroom, especially the social component inherent in this setting which may be a necessary condition for HK behavior to be displayed. The length and self-paced nature of learning tasks undoubtedly influences learning effectiveness in HK children. Deficiencies in functional mechanisms may become more evident (decreased attention, increased activity) only when self-control is expected or demanded as in exogenously manipulated situations.

Waking Ultradian Rhythms

Results indicated the presence of waking ultradian rhythms in performance and motility in both HK and normal children. The presence of these waking rhythms has not been previously documented in this pediatric age group. There are, moreover, several interesting features in these waking rhythms. Since the intra-sleep (NREM/REM) cycle length in this age group is 70-90 minutes, the frequency of primary interest was 19.2 c/day (68-84 minutes). Spectral peaks in performance and activity occurring specifically within this frequency range were inconsistently observed, i.e., some subjects in both groups showed no rhythm while others did, and some subjects evidenced cyclicity at one frequency on one day but at another frequency on the next day in a particular

variable. These observations produced an extremely random pattern (Table 13) with nearly equal distribution of spectral peaks across the chosen frequency ranges. These variable results may have derived from effects of motivation and information feedback. These processes may mask or obscure the expression of endogenous rhythms in waking performance and motility. For instance, subjects who displayed high motivation to perform well on the CPT, and were interested in how they were performing may have been able to suppress the possible cyclic expression inherent in these waking processes. In contrast, subjects with less motivation and little interest in outcome may allow more variability in response to be manifested and, consequently, these rhythms may be more clearly expressed.

The saliency or expression of these waking rhythms may also be affected by the amount of noise (variability of response) in the system such that greater noise levels mask the possible presence of waking ultradian rhythms. Evidence of these rhythms may also be influenced in the present study, by the sampling frequency and overall length of the testing interval (time series). Only 24 data points defined the time series, and this may not have been sufficient to detect the presence of ultradian periodicity in performance and activity. It may be that only after several hours of performing on task would cyclicity begin to be clearly manifested. Orr et al. (1976) have suggested that cognitive processes may become more susceptible to the effects of an endogenous rest-activity rhythm when fatigue increases and arousal level decreases. Initially when the novelty of the task and situation are high, performance is optimal, implying optimal arousal as well. During this time, one might expect little

variability in responding and, therefore, little room for oscillation in performance. In this way, rhythmicity could be obscured by an optimal combination of CNS arousal and task difficulty. With increasing time on task and a consequent decrease in arousal, performance would deteriorate and become more variable. At some threshold or zone of transition, the endogenous rhythm would no longer be obscured.

It might also be that waking ultradian rhythms are not present in more complex human behavior. However, in light of the mounting evidence for ultradian rhythms in many physiological and behavioral variables, it seems highly unlikely that this is the case. Several factors may interfere with partialling out the rhythm of interest. They might include situational and environmentally-induced determinants, influences of variable internal states (arousal, fatigue), or a complex combination of both. The presence versus absence of waking rhythms and the shifts in frequency observed in this study may reflect the influence of these little-explored factors.

Due to the variability of results obtained in the present study, nothing definitive can be said with regard to differential cyclicity for HK compared with normal children. However, perhaps this very fact of a wide range of cycling frequencies points in the general direction of no differentiation. Perhaps longer time series with more sensitive indices would reveal ultradian cyclicities which are highly correlated to the basic components of hyperkinetic behavior and more generally, to human behavior.

Relationships between waking rhythms and preceding sleep. Kleitman (1963, 1969) initially hypothesized the existence of an ultradian "oscillator",

of which the BRAC was the most fundamental component, operating throughout the 24-hour period. Support for the existence of this BRAC, which was thought to modulate activity in a number of physiological and behavioral variables, came from studies showing that sleep and waking processes oscillate with a similar period length. However, few studies have examined the phase relationships between previous or subsequent sleep and waking ultradian rhythms. To establish that a possible single periodic generator exists, one might expect phase continuity across states. For example, REMPs during the night which contain relatively greater physiological activity might be in phase with ocular activity, optimal performance (high arousal) or greater motility during waking. Similarly, NREMPs during sleep which are relatively lower in physiological activity might be in phase with ocular quiescence, lowered arousal or reduced motility during wakefulness.

In attempting to explore these relationships in the present study, REMP predictions into wakefulness were made and waking ultradian rhythms were descriptively analysed with respect to whether waking peaks in performance and activity were coincident with predicted extensions of REM or NREM sleep periods. These analyses revealed that waking peaks occurred nearly randomly in relation to the extrapolation of predicted REMPs into wakefulness. Some waking rhythms coincided with an increasing slope of transition, others with a decreasing slope of transition, and still others peaked either in phase with the predicted REMP or with what would have been NREM had sleep continued. Furthermore, some rhythms peaked with the occurrence of REM in the first predicted REMP but then shifted out of phase on the second predicted REMP.

Studies which have investigated sleep in an extended paradigm have not routinely reported REM cycle lengths throughout the extended sleep period (Aserinsky, 1971; Aserinsky, 1973; Verdone, 1968; Webb, 1978) which makes it difficult to assess what the predicted occurrence of REM would be beyond the normal 7-8 hours of sleep. In addition, further limitations are imposed on the ability of subjects to maintain sleep effectively once satiation has been reached. However, some data from these studies indicate that cumulative REM sleep in extended night paradigms is linearly related to the total amount of sleep, although Verdone (1968) reported a slight decline (nonsignificant) in the length of REMPs after the fourth REMP. Furthermore, the diurnal periodicity is an important determinant of the occurrence and duration of REM sleep.

The results from the present study seem to support the notion of multiple oscillators operating throughout the 24-hour period. The wide distribution across the various spectral frequencies argues against a single periodic generator with rhythms which might be related to the primary frequency or harmonics of this basic rhythm. Moreover, the present results provide evidence for the nonstationarity of waking ultradian rhythms. This is contrary to Kleitman's proposal of a BRAC which is characterized as having little variability in period length. It may be that physiological mechanisms which trigger wakefulness after a certain optimal accumulation of sleep also influence those mechanisms responsible for cyclicity during wakefulness (reset mechanism). This may explain the nonstationarity and lack of consistent phase relationships between sleep and waking rhythms. Even during sleep, nonstationarity is observed for both delta sleep (shortens as night progresses) and REM sleep

which lengthens as the night progresses (Webb, 1971). Taub and Berger (1973) have provided additional support for nonstationarity during sleep by observing that NREM/REM cycles were shorter during sleep periods between 4:00-12:00 (81.5 minutes) than during sleep periods from 20:00-4:00 (115.5 minutes). Also, several studies have demonstrated a circadian modulation of ultradian rhythms. Lavie (1977) reported a circadian influence on the perception of the spiral aftereffect which showed an ultradian rhythmicity of 14.4-21.6 c/day in the morning and 10.8-14.4 c/day in the evening. It was suggested that this shift in frequency during waking may have been the result of an accumulation of fatigue.

Finally, the positive correlations obtained between REM and NREM twitches during sleep and motility during the rest periods in wakefulness requires interpretation. As previously mentioned, REM sleep reflects increased arousal relative to NREM sleep. Similarly, one might interpret performance on detection tasks and increased motility during the task as being equivalent to a period of heightened arousal in relation to periods of off-task behavior which might be equated to states of lower arousal. In other words, it might be argued that on-task behavior is to some extent similar to REM in being an aroused state and off-task behavior similar to NREM sleep, i.e. less activated. Within the hypoarousal model of hyperkinesis, HK subjects may be receiving sufficient stimulation during REM (endogenous) and during the waking task (exogenous) to keep their arousal at an adequate level. However, during NREM sleep and off-task periods during waking, the need for stimulation becomes manifested. This is reflected in sleep by increased phasic motor activation (twitches) and in waking by an increase in motility during the rest periods.

Phasic responsivity during both sleep and wakefulness rather than tonic levels of activation may more accurately characterize the state of arousal in these HK subjects.

The existence of multiple oscillators operating at differing frequencies and having the ability to shift from frequency to another may imply that mechanisms which control rhythmicity during one physiological state (e.g., sleep) may be independent of those governing rhythmicity in another state (e.g., waking processes). This would argue against rhythms which occur in sleep and wakefulness being part of the same basic underlying BRAC. Since REM sleep reflects a state of increased arousal (EEG and autonomic changes, phasic eye movements, twitches, etc.) in relation to NREM sleep, it remains unclear as to whether waking rhythms in heightened (increased oral activity, ocular activity etc.) and lowered (detection errors, ocular quiescence etc.) arousal reflect an equivalent counterpart with similar periodicity to the alternation of REM and NREM sleep.

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Appendix A

Parent Behavioral Rating Scale

PARENT RATING SCALE

Child's Name: Date:

Information obtained by: _____

Please ANSWER ALL ITEMS with a check mark.

Observation	Degree of Activity			
	Not al all	Just a little	Pretty much	Very much
1. Restless or overactive				
2. Excitable, impulsive				
3. Disturbs other children				
4. Fails to finish things he starts - short attention span				
5. Constantly fidgeting				
6. Inattentive, easily distracted				
7. Demands must be met immediately - easily frustrated				
8. Cries often and easily				
9. Mood changes quickly and drastically				
10. Temper outbursts, explosive and unpredictable behavior				

Comments:

Appendix B

Teacher Behavioral Rating Scale

TEACHER RATING SCALE

Listed below are descriptive terms of behaviour. Please 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 BEHAVIOUR				
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 behaviour.				
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				