Sleep, 4(4):366–383

Sleep Patterns in Hyperkinetic and Normal Children

*K. Busby, †‡P. Firestone, and *†R. T. Pivik

*Psychophysiology Laboratory, Department of Psychiatry, School of Medicine, Faculty of Health Sciences, and †School of Psychology, University of Ottawa, Ottawa; and ‡Department of Psychology, Children's Hospital of Eastern Ontario, Ontario, Canada

Summary: Sleep patterns in nonmedicated hyperkinetic (n = 11) and normal control (n = 11) male children (8–12 years old) were compared to document possible sleep disturbance in hyperkinetic children. Electroencephalographic, electro-oculographic, electromyographic, and autonomic measures were monitored continuously for five consecutive nights. Analysis of sleep pattern variables revealed a significantly longer rapid eye movement onset latency ($p < 0.05$) and marginally significant greater absolute and relative amounts of movement time ($p < 0.07$) for the hyperkinetic group relative to controls. No other sleep parameters differentiated the groups. It was concluded that baseline tonic sleep parameters do not indicate marked sleep disturbance in hyperkinesis. The results were discussed within the context of hypothesized arousal dysfunction underlying this disorder. **Key Words:** Hyperkinetic children—Hyperkinesis—NREM and REM sleep cycles—Spontaneous skin potential responses.

Of all childhood psychiatric disorders, hyperkinesis (HK) probably has generated the greatest amount of research and controversy in recent years. Prevalence estimates for this disorder have ranged from 1 to 20 percent for all school-age children (Stewart et al., 1966; Wender, 1971; Lambert et al., 1978), and it is one of the most common primary presenting symptoms of children referred for psychological difficulties (Patterson et al., 1965). Definition and diagnosis of HK have relied on a constellation of symptoms extending along a number of behavioral, perceptual-cognitive, and social dimensions. The core symptomatology includes excessive and often inappropriate activity, short attention span, distractibility, impulsivity, excitability, and poor scholastic performance despite scores within the normal range on various intelligence measures. In addition, several secondary signs such as aggressiveness, low frustration tolerance, and poor self esteem are often present (Clements, 1966; Minde et al., 1972; Whalen and Henker, 1976).

Accepted for publication July 1981.
Address correspondence and reprint requests to Dr. Pivik at Laboratory of Psychophysiology, Department of Psychiatry, Ottawa General Hospital, 501 Smyth Road, Ottawa, Ontario K1H 8L6, Canada.

Reports of a relatively high incidence of "soft" neurological signs and borderline abnormalities in electroencephalographic (EEG) activity (Clements and Peters, 1962) have suggested a relationship of HK to brain damage, but recent critical evaluation of this research indicates that only a small subgroup of HK children show overt neurological abnormality (Dubey, 1976). However, although a specific organic etiology has yet to be found, a biological deficit is still considered to underlie this disorder.

The search for postulated differences in electrophysiological processes specific to HK has been directed toward the more global concept of arousal, and in this regard quite opposite explanations for this syndrome have been advanced. On the one hand, HK has been viewed as a reaction to excessive sensory stimulation (Strauss and Lehtinen, 1947). In this regard it has been argued that HK children may be unable to properly filter and organize stimulus input such that "stimulus overload" occurs, resulting in behavioral overactivity. The underlying physiological basis for this notion is thought to be over- or hyperarousal of various brainstem and subcortical structures (Laufer et al., 1957).

An alternative explanation has been offered (Zentall, 1975, 1977) in which increased motoric activity is viewed as stimulus-seeking behavior emanating from central nervous system hypoarousal. This view is based on the premise that homeostatic mechanisms function to maintain a level of stimulation within some optimal range (Hebb, 1955; Leuba, 1955; Berlyne, 1960).

Evaluation of physiological indices during wakefulness has not produced clear-cut conclusions regarding the state of arousal in HK children, although a recent investigation of variations in spinal reflex amplitude during wakefulness and sleep indicates reduced excitability during wakefulness (Mercier et al., 1980; Pivik and Mercier, 1981). Many of the inconsistent and conflicting results may be due to differences in experimental procedures and methodologies, including variations in number and symptomatology of subjects, and types and dosage levels of medication (cf. Hastings and Barkley, 1978; Rosenthal and Allen, 1978; Ferguson and Pappas, 1979). The electrophysiological data gathered during wakefulness, although not strongly supportive of either hyperarousal or hypoarousal, do continue to implicate a disorder in arousal-producing mechanisms as fundamental to the HK syndrome. The intimate relationship between sleep and arousal mechanisms suggests that studies of sleep patterns and events in HK children might provide information pertinent to the postulated dysfunction of arousal mechanisms under conditions free from waking-confounding variables such as stress, expectations, and undefined variations in level of arousal. Furthermore, sleep studies might reveal HK syndrome-related events and relationships obscured by, or not present during, wakefulness.

Sleep laboratory investigations of HK consist of seven studies which have reported on sleep patterns in a total of 39 HK children (Luisada, 1969; Small et al., 1971; Feinberg et al., 1974; Haig et al., 1974; Nahas and Krynicki, 1977; Khan and Rechtschaffen, 1978; Stahl et al., 1979). With one exception all studies agree that: (a) baseline sleep is not remarkably different in HK children relative to normal controls; and (b) stimulant medication administered to HK children does

not significantly alter their sleep patterns. In the one dissenting study, Luisada (1969) reported less rapid eye movement (REM) sleep and more REM sleep disruptions during baseline recordings. Across studies, symptomatology and subject selection procedures have varied. Diagnoses, for example, have ranged from those based solely on the presence of excessive restlessness to more complex symptom clustering, some of which may have had a strong organic foundation. In experiments in which stimulant medication was administered, varying types and dosage levels of drugs were employed making inter-study comparisons difficult. Differing design paradigms (e.g., number of nights recorded, medication/nonmedication, etc.) have further confounded cross-study comparability.

The purpose of the present study was to provide additional comparative information regarding sleep morphology in a relatively large sample of nonmedicated HK and normal children over a substantial baseline recording period. A 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.

METHODS

Subjects

Eleven nonmedicated HK male children (8–12 years old; $\bar{X}$ = 10.6, SD = 1.7) and 11 similarly aged nonmedicated male control (C) children ($\bar{X}$ = 10.6, SD = 1.3) participated in the study. Diagnosis of HK was based on positive indications (>15 on hyperactivity index) on Conners Parent (1970; $\bar{X}$ = 19.5, SD = 6.9) and Conners Teacher (1969; $\bar{X}$ = 19.1, SD = 5.5) behavioral rating scales, as well as the persistent or recurrent presence of the following core symptomatology (DSM-III criteria of attention-deficit disorder with hyperactivity): motor restlessness, short attention span, distractibility, impulsiveness, labile emotions, and poor academic performance (Stewart et al., 1966; Renshaw, 1974; Wender, 1972; Goyette et al., 1978). All HK subjects displayed this behavioral symptomatology prior to the age of three.

The control group of normal children was recruited from local school systems. All of these children scored negatively on the Conners Parent ($\bar{X}$ = 4.6, SD = 2.1) and Conners Teacher ($\bar{X}$ = 3.0, SD = 2.8) behavioral rating scales.

All children in the study were living at home with at least one parent. Children with the following symptoms or classifications were excluded from the study: major psychosis, over-anxious reaction, unsocialized aggressive reaction, peripheral sensory loss, epilepsy, normal constitutional hyperactivity, mental retardation, post-traumatic organic brain syndrome, encephalitis, toxic organic brain syndrome (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, lower limit of 80) and a baseline EEG recording. This EEG recording session served to screen for the presence of gross EEG abnormalities and to familiarize each child with the recording environment, procedures, and apparatus.

A full explanation of the study was given to parents and children and informed consent was obtained (parent's or legal guardian's signature).

Polysomnographic Recordings

Subjects reported to the sleep laboratory 1 hr before bedtime for electrode application. EEG (C_3/A_2), electro-oculographic (EOG; bipolar DC recordings of horizontal and vertical eye movements), and electromyographic (facial muscle) activity were recorded using a Grass (Model 78D) polygraph. Spontaneous skin potential responses (SSPR; volar surface 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 was limited to 9.25 hr, with "lights out" occurring at approximately 9:30–10:00 each night. It was communicated to both children and their parents that napping should not occur during the course of the study.

Data Analysis

Sleep records were coded and scored blind by two individuals using standardized criteria (Rechtschaffen and Kales, 1968). High interrater agreement (≥90%), established on pilot data prior to actual data scoring, was periodically checked and found to be consistent throughout data analysis. The first two recording nights were considered laboratory adaptation nights and separate group comparison analyses were made with data from these nights included and excluded.

The method of Feinberg and Floyd (1979) was used to compute non-REM (NREM) and REM cycle lengths within nights. Specifically, NREM cycles were measured as the duration of total sleep time (TST; 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. REM cycle length was measured as the duration of total sleep recorded (time awake subtracted) from the first REM period (REMP) onset to the onset of the second REMP, from the second REMP onset to the third, etc. These cycle durations were also measured with waking time included (real time).

Movement time during sleep was scored using the criteria of Rechtschaffen and Kales (1968), i.e., epochs in which EEG and EOG activity channels were obscured by muscle tension and/or amplifier blocking artifacts produced by subject movement lasting for more than half the epoch.

SSPRs were scored according to the method of Johnson and Lubin (1966); i.e., 1 mm pen deflection = 100 μV potential change, with amplifier input impedence = one megohm. SSPR data from the first cycle of sleep on nights 1, 4, and 5 were pooled and within-group response rates/min determined for stages 2, 4, and REM. These rates were based separately on the total amount of scorable activity divided by the number of min of a particular stage, and the frequency of SSPR activity during 15 min samples (one sample/stage/subject) of stages 2 and 4. For the latter analysis, equivalent samples from REM sleep could not be obtained because initial REMPs were often less than 15 min in duration.