Sleep
12(4):299–308, Raven Press, Ltd., New York

Sleep and Wakefulness in Normal Preadolescent Children

*†Lars Palm, *Elin Persson, *Dan Elmqvist, and †Gösta Blennow

*Departments of *Clinical Neurophysiology and †Pediatrics, University Hospital, Lund, Sweden*

Summary: Eighteen healthy children, 9 boys and 9 girls, between 8 and 12 years of age were examined with polygraphic sleep records, multiple sleep latency tests (MSLTs), and measurements of reaction times. Sleep was recorded at home on Oxford Medilog 9 channel cassette tape recorders (Oxford Medical Systems, Abingdon, U.K.) and sleep staging was performed from the screen of the display unit. Two consecutive nights were recorded. MSLT was done in the laboratory. The subjects were given 30 min to fall asleep on four occasions during the day after the last recorded night of sleep. Reaction times were measured repeatedly between each MSLT trial. More slow wave sleep was found in this study compared to others. Also, the first night effect was slight. It is proposed that this is due to the fact that the recordings were performed at home. The initial sleep cycle was incomplete in almost all subjects. A sleep stage with traits of both rapid eye movement (REM) and non-REM could be seen in this cycle, probably representing an abortive REM period. MSLT confirmed the low daytime sleepiness in healthy preadolescent children. A sleep latency of 10 min or less on two or more sleep trials, or a daily mean sleep latency of less than 20 min, is rarely seen in this age group. The reaction times were within normal limits for the age of the subjects. Nighttime sleep values, daytime sleep latencies, and reaction times were not correlated in these normal-sleeping children. **Key Words:** Sleep—Children—Polygraphic recording—Ambulant monitoring—Multiple sleep latency test.

Sleep disturbances in childhood and adolescence are often coupled to behavioral problems during the daytime, presumably as a sign of disturbed wakefulness. Thus, when investigating daytime behavioral problems, knowledge of the nighttime sleep quality is important. Normal sleep in children and adolescents has been studied with polysomnography by several groups (1–4). These studies have been performed mostly in sleep laboratories where conditions do not always correspond to conditions at home. Home recordings are now possible with portable recording systems (5–7).

Some investigators have related nighttime sleep to daytime wakefulness (4,8,9). Var-

Accepted for publication February 1989.
Address correspondence and reprint requests to Dr. L. Palm, Department of Pediatrics, University Hospital, S-221 85 Lund, Sweden.

ious methods can be used to measure wakefulness (10). The Multiple Sleep Latency Test (MSLT) reflects the immediate craving for sleep (11,12), caused by, for example, sleep deprivation, defective regulation of sleep as in narcolepsy, or influence from hypnotic drugs. The reaction time reflects mental alertness. It decreases with age until the late teens as the central nervous system matures (13). It also seems to be an indicator of unspecified central nervous system pathology as, for example, in attention deficit disorder (14).

This study was performed in order to contribute to the data on sleep and wakefulness in healthy youngsters and to set standards for studies of pathological conditions.

MATERIALS AND METHODS

Subjects

Eighteen healthy children, nine boys and nine girls, between 8 and 12 years of age volunteered to participate. Two subjects were 8 years, three were 9 years, five were 10 and 11 years, and three were 12 years of age. The subjects were mainly children of the hospital staff. Informed consent was obtained. A subsidiary gratification was given. The study was approved by the hospital's ethical committee. The polygraphic sleep records from 14 of these children were also used for a comparison between visual and automatic sleep staging (15).

The parents were asked to complete a questionnaire regarding earlier and present illnesses of the subjects, their physical and mental development, and school performance. Reading and writing difficulties were specifically asked for. They were also questioned about the presence of such difficulties or notable clumsiness among relatives.

None of the children suffered any chronic or acute illness and no one received any medication. Two children were examined simultaneously, most often friends. Two consecutive nights were recorded. The children slept at home. They were advised to keep their ordinary routines, especially ordinary bed times. A thorough physical and neurological examination was performed on each child. Sexual maturation was evaluated according to Tanner (16) and in the boys testicular volume was measured.

Sleep records

The tape recorders were mounted and started in the laboratory during the afternoon of the first recording day by the same two persons. The recorders were run continuously for about 50 h, function control and necessary adjustments being made daily. Silver/silver chloride electrodes were mounted by collodion on the scalp. On bare skin, self-adhesive disposable electrodes were used. Electrodes were applied according to the International 10–20 system (17). Oxford Medilog 9000 nine-channel tape cassette recorders (Oxford Medical Systems, Abingdon, England) were used with TDK 120 min cassettes. Central and parietal electroencephalogram (EEG) (C3–A2, C4–A1, C3–Cz–C4, P3–P4), submental electromyogram (EMG) and two-channel electrooculogram (EOG) were recorded.

Analysis of sleep records

Records obtained with this tape recorder system are processed in a display computer, the Oxford Medilog 9000 Display Unit, and are shown on a monitor at 20–60 times the recording speed, 16 s of the record being visible at one time. Time constants, filters, and

calibrations can be set when displaying. The tapes were visually scored directly from the screen according to the criteria of Rechtschaffen and Kales (18). The EEG time constant was 0.3 s, high frequency filter was 70 Hz, and gain was 100 mV/cm. According to the routine of our laboratory, the movement artifact time was not scored, and stages 3 and 4 were treated together as slow wave sleep (SWS). The parameters measured from each recorded night were defined as follows: total sleep period (TSP) = the time from the first uninterrupted sleep longer than 2 min until the last awakening in the morning; wake after sleep onset (WASO) = time spent awake during TSP (the periods awake were counted and divided into periods longer and shorter than 2 min); actual sleep time (AST) = TSP reduced with WASO; sleep efficiency index (SEI) = AST as a fraction of TSP (%); stage 1, stage 2, SWS, and REM = time (min and % of AST) spent in stages 1, 2, SWS, and rapid eye movement sleep (REM), respectively; sleep onset latency = the time from lights out to the first continuous period of sleep longer than 2 min; stage 2, SWS, or REM latency = the time from sleep onset to the first appearance longer than 2 min of that stage.

A sleep cycle was defined as the interval between the first appearance in that cycle of stage 2 sleep lasting more than 2 min and the return of stage 2 after a period of REM. During some nights, a period lasting a minimum of 5 min of sleep with relatively high frequency EEG and low muscle tone appeared after at least 40 min of SWS, followed again by at least 40 min of deep sleep. We then considered the middle of the interrupting period of superficial sleep as the end of one sleep cycle and the beginning of a new cycle. The length, the amount of REM and SWS, and the time awake within the individual sleep cycles were measured.

Studies of wakefulness

MSLT was performed in the sleep laboratory the day after the last recorded night. Since the expected sleep latencies in preadolescent children are longer than in adults, 30 min was given for each sleep trial, as compared to the 20 min used by Carskadon et al. (11). When no sleep occurred in a trial, a sleep latency of 30 min was counted. The sleep latencies for each trial and a daily mean were calculated. We also analyzed separately the sleep latencies from the trials when sleep really occurred; in this analysis, the occasions during which no sleep was recorded were disregarded.

Four records were done in 1 day, at 10 and 12 a.m. and 2 and 4 p.m. Two soundproofed, air-conditioned dark rooms with controlled temperature and humidity were used for recording. For monitoring during the MSLT, the tape recorders of both subjects were connected via the Oxford Medilog Writer/Coupler Unit to a Siemens Elema polygraph outside the recording rooms.

Reaction time was measured immediately before each sleep trial at the MSLT. The equipment used gives short light blinks at random intervals (4.75–10.75 s) from either of four lamps of different colors, each blink to be responded to by a corresponding color-coded button. The subjects received no information on whether a correct response had been given. Each test lasted for 10 min. The mean reaction time for each minute of the test and for the 10 min together was automatically calculated by the apparatus (19).

RESULTS

Subjects

The neurological findings were entirely normal. Sixteen of the children had no pubertal development; 2 girls aged 10.2 and 11.3 years had the first signs of beginning

puberty (Tanner's stage 2). In eight boys, the testicular volume was 4 ml or less. One boy had a testicular volume of 8 ml, as the only sign of beginning puberty.

The school performances according to the parental interviews were average to superior. None of the children had specific reading or writing difficulties. One parent each of two nonrelated children had suffered such difficulties during their school education. One child had a nonidentical twin with attention deficit.

In one girl aged 9.6 years, the EEG was completely normal when awake, but during drowsiness and sleep, there were solitary spike and slow waves over both hemispheres independently. This girl had no history of convulsions and her behavior and school results were ordinary. She showed no signs of disturbed wakefulness as compared to the other subjects of this study.

Sleep

Complete sleep records were obtained from 16 of the 18 children. Cable breakage or stop of band transport spoiled the records from both nights of one boy and one night of one girl. Less prominent artifacts seen were caused by defective skin–electrode connection, most often in the EMG channel. Two girls slept restlessly during the first part of one night each and therefore the first sleep cycles of these two nights could not be clearly defined for comparison. These nights were excluded from the sleep cycle analysis but were included in the other parts of the study. One girl had an extremely long sleep onset latency the first night, 177 min, after going to bed unusually early. Her sleep latency was therefore considered to be out of the normal range and excluded from the analysis of the sleep latencies.

Data from the two nights are presented in Table 1. The total sleep periods of the first

TABLE 1. *Sleep data—mean (SD)—from full first nights, first nights after abbreviation to awakening time of second night (see text), and from second nights*

Variable	First night		Second night
	Full data	Truncated data	
TSP, min	581.4 (55.2)[b]	540.9 (58.6)	544.5 (54.5)
AST, min	549.1 (45.7)[a]	510.3 (60.7)	523.7 (53.2)
SEI, %	94.6 (6.3)	94.5 (7.1)	96.0 (2.9)
Stage 1, min	29.2 (20.4)[a]	26.3 (20.8)[c]	35.3 (20.4)
%	5.2 (3.7)[b]	5.1 (3.7)[b]	6.6 (3.9)
Stage 2, min	186.2 (31.9)	177.1 (34.1)	181.1 (34.6)
%	34.2 (6.3)	35.0 (6.7)	34.8 (7.1)
SWS, min	210.8 (47.9)	202.8 (50.0)	201.6 (48.6)
%	38.2 (7.3)	39.5 (6.9)	38.3 (7.5)
REM, min	122.6 (27.1)[a]	108.2 (25.0)	105.9 (28.7)
%	22.2 (4.2)[b]	21.2 (3.8)	20.0 (4.2)
SOL	8.2 (5.0)[c]		13.5 (8.3)
Stage 2 latency	3.6 (4.1)		4.0 (4.1)
SWS latency	11.0 (7.3)		10.0 (4.4)
REM latency	117.6 (57.2)[b]		158.9 (51.4)
Number of awakenings			
>2 min	2.8 (1.9)	2.4 (1.8)	3.1 (3.1)
<2 min	7.3 (3.8)	6.6 (3.6)	7.2 (4.6)
WASO, min	23.0 (24.2)	21.3 (23.1)	22.6 (17.7)

Abbreviations, see text. Significances in comparison first–second and first revised–second.

[a] $p < 0.1$, [b] $p < 0.05$, [c] $p < 0.01$.

nights were longer than those of the second, because the children had to come to the laboratory at 8 a.m. the second morning. The first morning they were free to sleep on. To enable a comparison between the two nights, the first nights were truncated at the wake-up time of the second nights after an initial analysis, and then a second analysis was done. On the first night, the sleep onset latency was significantly shorter and there was less stage 1 sleep than on the second night. REM latency was shorter on the first than on the second night.

The sleep cycle length first increased until cycle number 3 and it then gradually decreased during the rest of the night (Fig. 1). The content of SWS and REM gradually changed in opposite directions—SWS decreasing and REM increasing towards the end of the night (Fig. 2). There were no differences in cycle length and sleep stage content between the first and second nights.

In 21 of the recorded 32 nights, the first sleep cycles did not contain any REM but only a shift from deep SWS to stage 2 or 1. In three children, this was the case with both the first and the second cycles. In 21 of these 24 incomplete cycles, there was a period of a dissociated sleep state characterized by a low muscle tone and a mixed frequency EEG of low amplitude. There were no rapid eye movements and sometimes sleep spindles and K complexes could be seen.

Wakefulness

At MSLT, only 2 of the 18 children fell asleep on all occasions; 5 did not fall asleep at all. Thirteen children fell asleep within 30 min at least once. No REM was recorded. The sleep latencies are presented in Table 2. Although not statistically significant, a diurnal influence could be seen in that more children fell asleep around noon, and they did so in a shorter time than in the morning or afternoon (Fig. 3). The average daily mean sleep latency of the two children who fell asleep on all trials was 22.8 min, compared with the six children who fell asleep in one trial only and at that trial had an average sleep latency of 16.2 min. The shortest single sleep latency, 9 min, was recorded in a boy 12.5 years, one of those who did only fall asleep once. There were no differences between boys and girls.

The night sleep data from the children who fell asleep at MSLT only once or less were compared to corresponding data from those who fell asleep twice or more. There was

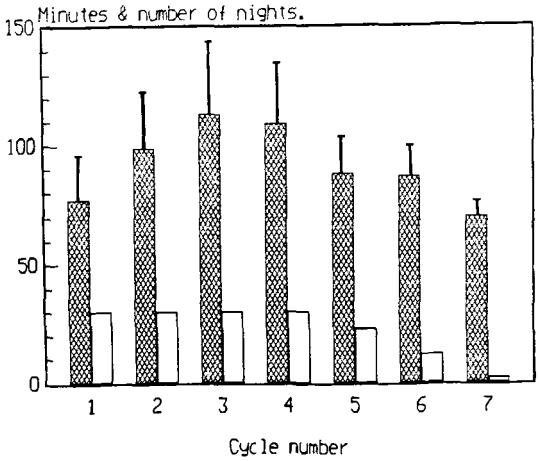

FIG. 1. The length (means and SD, min, shaded bars) of the individual sleep cycles from all 30 nights presented with the number of nights during which the indicated number of cycles were completed (open bars). Between four and seven sleep cycles were completed per night.

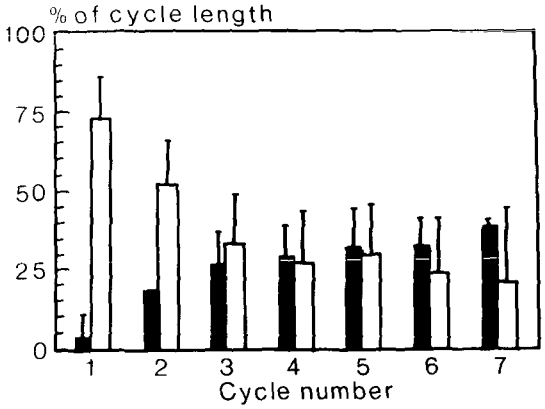

FIG. 2. The amount of REM sleep (closed bars) and slow wave sleep (open bars) in the individual sleep cycles (means and SD, % of the cycle lengths).

a tendency for more SWS during the first night in the former group, and more REM during the second night in the latter. The groups, however, are too small for conclusions.

Reaction time

The subjects responded faster the older they were. The mean reaction time of all subjects was 0.64 s. All subjects fell within the normal range for this method found by Frisk (19). No significant diurnal variation could be found over the examined hours.

DISCUSSION

Measurements of vigilance and sleep should be evaluated together. However, the different parts of this study also merit separate discussion. To our knowledge, this is the first published study in which sleep recorded at home on a portable cassette recorder has been analyzed directly from the screen of the replay system (20). The results of our sleep study therefore should be compared with those achieved with conventional techniques.

Sleep

We found much more slow wave sleep and less stage 2 sleep than in studies performed with other techniques (2–4,21) (Fig. 4). An explanation for this may be that our children slept at home. It is a common observation that mental tension and an unfamiliar environment influence sleep quality. Åkerstedt et al. (22) found that in the cumulative EEG–frequency spectrum of ship's engineers sleeping on call, the delta power

TABLE 2. *Multiple Sleep Latency Test Results*

Examination at (h)	Mean sleep latency all subjects (N = 18), min (SD)	Number of subjects falling asleep	Mean sleep latency in subjects sleeping, min (SD)
10.00	27.5 (4.0)	5	22.1 (3.7)
12.00	26.8 (6.3)	6	18.8 (7.3)
14.00	25.6 (6.3)	7	18.6 (4.8)
16.00	25.8 (5.7)	6	20.3 (3.6)
Daily mean	26.4 (2.8)		18.5 (4.8)

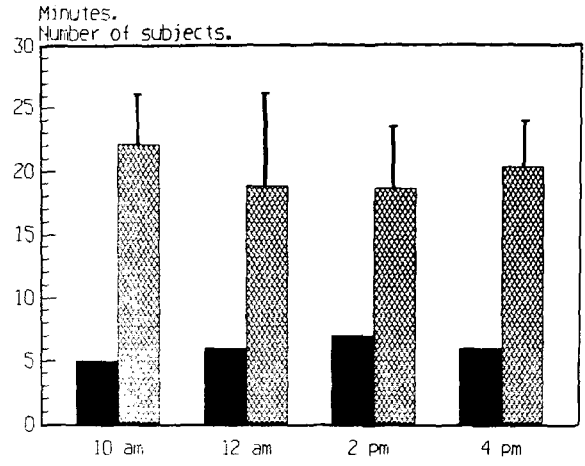

FIG. 3. Multiple sleep latency test. Sleep latencies (means and SD, min, shaded bars) from children that really fell asleep at the trial, and the number of these sleeping children (closed bars).

diminished as compared to their sleep when off duty in the same environment, even though they were not called upon. The amount of slow wave activity, the delta power, is considered an indicator of the restoring component of sleep. The greater amount of SWS in our subjects would imply a better sleep quality in subjects sleeping at home with the tape recorder than is usual in a sleep laboratory. In the study on the influence of noise on sleep by Eberhardt (21), the investigated children were recorded at home as well. During quiet, nonexposed nights, the children in that study spent less time in SWS than ours, but more than, for example, those of Coble (2). Nights with noise exposure alternated with quiet nights, and it is possible that the experimental situation as well as the recording equipment used influenced sleep in a way similar to the on-call situation.

Differences between our results and others could depend on variations between the

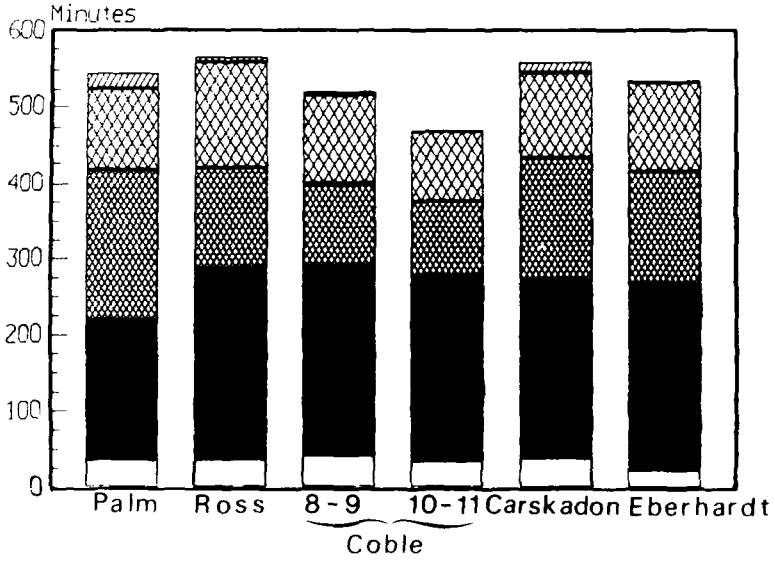

FIG. 4. Comparison between sleep results in the present study with data published by Ross et al. (3), Coble et al. at ages 8–9 and 10–11 years, respectively (2), Carskadon et al. (4), and Eberhardt (21). WASO (▨), REM (▩), SWS (▩), stage 2 (■), stage 1 (□). For abbreviations, see the text.

groups studied. It seems less likely, however, that middle-class Swedish children should differ fundamentally from children from, for example, the U.S. Since the amount of SWS was even greater when these records were analyzed with the automatic sleep stager, it is unlikely that the differences were due to misinterpretation of the screen picture (15). The time constant used, 0.3 s, should not contribute to any false increase of slow wave activity.

The first night of a sleep study may be disturbed by the equipment and the unfamiliar surroundings (23). When sleep was examined with a portable tape recorder at home, no first night effect was found in adults (24). We found only slight first night effects. Our subjects actually fell asleep faster the first night, and there was less stage 1 sleep during the first than during the second night. REM appeared somewhat later during the second night than during the first. When several nights are recorded, REM has been noted to appear successively later (21). The fact that sleep was restricted on the second morning but not on the first may have some implication—the expectancy on the length of the night may influence the organization of the cycles. Also, the prospect of additional laboratory investigations the last day may have had an impact on the sleep of the second night.

When falling asleep, the children went rapidly into a period of deep slow wave sleep evident both in the first and the second sleep cycles. Consequently, there was almost no REM during these first cycles. All children but one lacked a complete REM cycle in the beginning of at least one of the nights. In 67% of the nights, the first cycle was incomplete, and in 9% this was the case with the two first cycles. This phenomenon occurred as often during the first as during the second night. In 88% of the incomplete sleep cycles, an abortive EEG sleep pattern was found with traits specific to REM as well as to non-REM. In some children, this abortive REM appeared for a while before a short period of real REM. It is well established that children may skip the first sleep cycle (25) and the atypical sleep pattern found in this study seems to represent an abortive REM period (mainly no rapid eye movements).

In terms of Borbély's (26) two process model of sleep regulation, the impression is that the process S-associated SWS, which depends on the accumulated need for sleep, dominates heavily in the beginning of the night so that REM, associated with the chronologically founded process C, is only partially expressed. On the contrary, the cycles towards the end of the night contained about 50% REM and no or very little slow wave sleep.

MSLT

The multiple sleep latency test results confirm that children who have stopped napping but have not yet entered puberty have a very low tendency to fall asleep during daytime. A diurnal variation of vigilance with a period of physiological sleepiness around noon could be seen, although it was not significant. The boy with the shortest single sleep latency had an increased testicular volume. He may thus have been under the influence of beginning puberty, during which daytime sleep tendency increases (8).

Our results on the daytime sleep tendency in children agree with those of other authors (4,12). Our conclusions on the MSLT results are that (a) a sleep latency of 10 min or less on two or more occasions, or (b) a daily mean of all sleep latencies of 20 min or less at 30 min tests, or less than 15 min at 20 min tests, is rarely found among normal, healthy preadolescent children. One implication is that children of this age who fall

asleep during routine EEG recordings probably are sleep deprived or suffer from hypersomnia!

Reaction time measurements in this study reflected the age of the children and merely confirmed the normality of the subjects.

Sleep restriction is well known to reduce sleep latencies at MSLT (27) and also to some extent to increase reaction time (28). In this study of healthy children, nighttime sleep seems to have been adequate and not very much disturbed by the equipment. There was no correlation between the results from night sleep and MSLT or reaction time. Thus, children with a greater tendency to fall asleep at MSLT could not be proved to have slept less or with a poorer sleep quality than their comrades with longer sleep latencies.

The girl with paroxysmal EEG changes during drowsiness and sleep had an otherwise normal sleep pattern and no increased daytime sleep tendency. Although no diagnostic EEG record was obtained, the EEG finding was that of benign epilepsy of childhood with Rolandic spikes (29,30). An earlier study of sleep in children with epilepsy (31) included children with benign epilepsy of childhood who were also found to have normal sleep patterns.

Sleep staging directly from the replay screen is about as time consuming as the conventional staging from paper records. However, our technique saves the time and cost for transferring the records to paper. Further studies are needed to evaluate the results obtained with this method compared to paper record staging. For screening investigations and for rapid check of sleep quality, the method is at least as fast as the traditional technique.

Acknowledgment: This study was supported by the Margaretahemmet Foundation, the First of May Flower Annual Campaign for Children's Health and by the Swedish Medical Research Council (grant no. B8814X0084-24A).

REFERENCES

1. Williams RL, Karacan I, Hursch CJ. *EEG of human sleep: clinical applications*. New York: John Wiley & Sons, 1974.
2. Coble PA, Kupfer DJ, Taska LS, Kane J. EEG sleep of normal healthy children. Part 1: findings using standard measurement methods. *Sleep* 1984;7:289–303.
3. Ross JJ, Agnew HW, Williams RL, Webb WB. Sleep patterns in preadolescent children: an EEG–EOG study. *Pediatrics* 1968;42:324–35.
4. Carskadon M, Keenan Sh, Dement WC. Nighttime sleep and daytime sleep tendency in preadolescents. In: Guilleminault C, ed. *Sleep and its disorders in children*. New York: Raven Press, 1987.
5. Crawford Ch. Sleep recording in the home with automatic analysis of results. *Eur Neurol* 1986;25(suppl 2):30–5.
6. Hoelscher TJ, Erwin W, Marsh GR, Webb MD, Radtke RA, Lininger A. Ambulatory sleep monitoring with the Oxford–Medilog 9000: technical acceptability, patient acceptance, and clinical indications. *Sleep* 1987;10:606–7.
7. Sewitch DE, Kupfer DJ. Polysomnographic telemetry using Telediagnostic and Oxford Medilog 9000 systems. *Sleep* 1985;85:288–93.
8. Carskadon MA, Harvey K, Duka P, Anders TF, Litt IF, Dement WC. Pubertal changes in daytime sleepiness. *Sleep* 1980;2:453–60.
9. Anders TF, Carskadon MA, Dement WC, Harvey K. Sleep habits of children and the identification of pathologically sleepy children. *Child Psychiatry Hum Dev* 1978;9:56–63.
10. Koella WP. Vigilance–local vigilance—the vigilance profile: a new concept and its application in neurobiology and biological psychiatry. *Acta Neurol Scand* 1984;69(suppl):35–41.
11. Carskadon M, Dement WC. The Multiple Sleep Latency Test: what does it measure? *Sleep* 1982;5:S67–72.
12. Carskadon M, Dement WC. Sleepiness in the normal adolescent. In: Guilleminault C, ed. *Sleep and its disorders in children*. New York: Raven Press, 1987.

13. Andersen K, Starck L, Rosén I, Svensson E. The development of simple acoustic reaction time in normal children. *Dev Med Child Neurol* 1984;26:490–4.
14. Gillberg C, Frisk M, Carlström G, Rasmussen P. "Complex reaction times" in so-called minimal brain dysfunction. *Acta Paedopsychiatr* 1987;48:253–63.
15. Palm L, Elmqvist D, Blennow G. Automatic versus visual EEG sleep staging in preadolescent children. *Sleep* 1989;12:150–6.
16. Tanner JM. *Growth at adolescence, edition 2.* Oxford: Blackwell Scientific Publications, 1962.
17. Jasper HH. The ten–twenty electrode system of the International Federation. *Electroencephalogr Clin Neurophysiol* 1958;10:371–3.
18. Rechtschaffen A, Kales A, eds. *A manual of standardized terminology, techniques, and scoring system for sleep stages of human subjects.* Los Angeles: Brain Information Service/Brain Research Institute, University of California, 1968.
19. Frisk M, Velasco A. Psychophysical inadequacy expressed as prolonged complex reaction time, EEG deviation and slow mental processing in the etiology of child psychiatric problems. Report to the Swedish Medical Research Council, 1985.
20. Duplicate of reference 6. Deleted in proofs.
21. Eberhard JL. The disturbance by road traffic noise of the sleep of prepubertal children as studied in the home. In: *The influence on sleep of noise and vibrations caused by road traffic.* Thesis, Institute of Environmental Health, Lund University, Sweden, 1987.
22. Torsvall L, Åkerstedt T. Disturbed sleep while being on-call: an EEG study of ship's engineers. *Sleep* 1988;11:35–8.
23. Agnew HW Jr, Webb WB, Williams RL. The first night effect: an EEG study of sleep. *Psychophysiology* 1966;2:263–6.
24. Sharpley AL, Solomon RA, Cowen PC. Evaluation of first night effect using ambulatory monitoring and automatic sleep stage analysis. *Sleep* 1988;11:273–6.
25. Roffwarg HP, Muzio JN, Dement WC. Ontogenetic development of the human sleep–dream cycle. *Science* 1966;152:604–19.
26. Borbély AA. A two process model of sleep regulation. *Human Neurobiol* 1982;1:195–204.
27. Carskadon MA, Harvey K, Dement WC. Sleep loss in young adolescents. *Sleep* 1981;4:299–312.
28. Lisper H-O, Kjellberg A. Effects of 24-hour sleep deprivation on rate of decrement in a 10-minute auditory reaction time task. *J Exp Psychol* 1972;96:287–90.
29. Dalla Bernardina B, Bondavalli S, Colmaria V. Benign epilepsy of childhood with Rolandic spikes (BERS) during sleep. In: Sterman MB, Shouse NN, Passouant P, eds. *Sleep and epilepsy.* New York: Academic Press, 1982:495–506.
30. Heijbel J. Benign epilepsy of children with centrotemporal EEG foci. Umeå University Medical Dissertations, New Series No. 17, Umeå, Sweden, 1976.
31. Rosén I, Blennow G, Risberg AM, Ingvar DH. Quantitative evaluation of nocturnal sleep in epileptic children. In: Sterman MB, Shouse NN, Passouant P, eds. *Sleep and epilepsy.* New York: Academic Press, 1982:397–409.