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EEG Power Density during Nap Sleep: Reflection of an Hourglass Measuring the Duration of Prior Wakefulness

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Abstract The relation between the duration of prior wakefulness and EEG power density during sleep in humans was assessed by means of a study of naps. The duration of prior wakefulness was varied from 2 to 20 hr by scheduling naps at 1000 hr, 1200 hr, 1400 hr, 1600 hr, 1800 hr, 2000 hr, and 0400 hr. In contrast to sleep latencies, which exhibited a minimum in the afternoon, EEG power densities in the delta and theta frequencies were a monotonic function of the duration of prior wakefulness. The data support the hypothesis that EEG power density during non-rapid eye movement sleep is only determined by the prior history of sleep and wakefulness and is not determined by clock-like mechanisms.

The distribution of human sleep stages over time is not uniform. Slow-wave sleep (SWS; stages 3 and 4) is predominantly present at the beginning of a nocturnal sleep episode, whereas rapid eye movement sleep (REMS) is abundant at the end of sleep. Both distributions are periodic and can be considered a rhythm (Karacan *et al.*, 1970a; Broughton, 1975; Kronauer *et al.*, 1982). “The notion of a rhythm is sufficiently vague . . . to be useful in listing a wide variety of phenomena that might reflect quite different underlying mechanisms” (Aschoff, 1981, p. 3). The first obvious inquiry into the mechanisms underlying sleep rhythms concerns to what extent sleep is immediately dependent on prior nonsleeping and how sleep stages make up for their prior absence. To answer such questions, sleep has been recorded after various manipulations. Sleep deprivation studies have revealed that homeostatic regulatory mechanisms are involved in the organization of a sleep episode: When one night of sleep is skipped and recovery sleep is initiated, the next evening the amount of SWS is increased and REMS remains unchanged (Williams *et al.*, 1964; Borbély *et al.*, 1981). In the second recovery night, an increase in REMS above baseline levels has been reported (Williams *et al.*, 1964). When subjects are selectively deprived of REMS or SWS, a selective response occurs (Dement, 1960; Agnew *et al.*, 1967). Studies of sleep during naps in daytime have shown that in the afternoon more SWS is present than in the morning (Karacan *et al.*, 1970a) and that naps in the evening contain more SWS than naps in the afternoon (Maron *et al.*, 1964). After a nap in the morning, night sleep is hardly affected, whereas a nap in the afternoon reduces SWS in the following night (Karacan *et al.*, 1970b; Feinberg *et al.*, 1985).

All these data are compatible with the hypothesis that both SWS and REMS are regulated by simple homeostatic mechanisms, in conjunction with a priority of SWS over REMS. There is evidence, however, that REMS is also to a certain extent determined by circadian phase, independent of the immediate prior history. Sleep in the early morning after a night without sleep is characterized by a short latency to REMS, despite the high level of SWS (Endo *et al.*, 1981). Further evidence for a circadian influence on REMS comes from sleep recordings under conditions of temporal isolation. Under these conditions, REMS shifts to the beginning of sleep, as does the minimum of the body temperature rhythm (Czeisler *et al.*, 1980a; Zulley *et al.*, 1981). Analysis of the distribution of REMS during internal desynchronization has revealed that REMS propensity is coupled to the body temperature rhythm (Czeisler *et al.*, 1980b).

The question whether or not SWS is also to some extent determined by circadian phase is not yet settled. In the data of Weitzman *et al.* (1980), there is no solid evidence for a circadian modulation of SWS. Data presented by Webb and Agnew (1971) and Hume and Mills (1977) have been interpreted as evidence for a circadian influence on SWS (see Gagnon *et al.*, 1985), although also in these studies the amount of SWS was primarily determined by the duration of prior wakefulness. Additional evidence for a circadian influence on SWS has been derived from studies in which sleep latency (i.e., the length of the interval between lights-off and the first epoch of sleep) was measured at several times within a single day. The pattern of sleep tendency is essentially bimodal. Shortest sleep latencies were found in the afternoon and in the early morning (Richardson *et al.*, 1982; Carskadon and Dement, 1985). Broughton (1975) has suggested that this bimodal pattern of sleep propensity will be accompanied by a bimodal pattern of deep non-REM sleep (NREMS). In contrast, in the two-process model of sleep regulation (Borbély, 1982; Daan *et al.*, 1984) it is assumed that NREMS intensity is a monotonic function of the duration of prior wakefulness, without any circadian modulation.

To distinguish between these two hypotheses, sleep tendency and NREMS intensity have to be measured in the same experiment at several times of day. For a quantification of NREMS intensity, the classical scoring of the sleep EEG is not sufficiently sensitive; the changes induced by manipulating prior wakefulness are incompletely reflected by scoring of stages. In a sleep deprivation experiment in which spectral analysis of the sleep EEG was combined with the classical scoring, an increase in power density in the lower frequencies was found within SWS. These changes in power density were not limited to SWS, but were also present in stage 2 and REMS. The insensitivity of visual scoring was further demonstrated by the changes over a sleep episode. Apart from the well-known decay in the amount of SWS and the increase in stage 2 toward the end of the sleep episode, an attenuation of power density in the lower frequency range within stage 2 was observed (Borbély *et al.*, 1981). In a first approximation of the underlying process, the average integral EEG power density (0.75–15.0 Hz) per NREM–REM cycle has been used. This measure decays exponentially over successive NREM–REM cycles during undisturbed sleep and increases after prolonged wakefulness, as in sleep deprivation (Borbély *et al.*, 1981). It is not known whether this is a monotonic, noncircadian increase, although

this has been assumed in our model of the sleep timing process (Daan *et al.*, 1984). In this model, the timing of sleep and wakefulness results from an interaction between a homeostatic "Process S" and a circadian "Process C." Process S is only determined by the prior history of sleep and wakefulness. It has been assumed that Process S is reflected in the EEG power density during sleep. Under this assumption, EEG power density should only be determined by the duration of prior wakefulness, and not by the circadian phase at which sleep occurs.

We have therefore investigated how power densities between 0.25 and 15.0 Hz, in naps taken at different times of day, depend on prior wakefulness. The data presented here are consistent with a monotonic increase of EEG power density between 0.25 Hz and 8.0 Hz with increasing duration of prior wakefulness; hence they support the hypothesis that EEG power density in NREMS reflects a homeostatic control process, not circadian variability.

METHODS

Subjects were six female students (age 23 ± 3 years) who did not habitually nap. They were free of sleep complaints as assessed by a general sleep quality scale (Mulder-Hajonides van der Meulen and Van den Hoofdakker, 1984). They signed a written informed consent in which they were instructed not to nap (except for the scheduled naps) and not to use alcohol or other drugs for the duration of the experiment. All subjects used oral contraceptives. EEG recordings were made only during nonmenstrual periods. All naps were preceded by night sleep in the laboratory. The subjects were allowed to go to bed at their habitual time (range: 2300–2400 hr), but were awakened at 0800 hr. Naps were scheduled at 1000 hr, 1200 hr, 1400 hr, 1600 hr, 1800 hr, 2000 hr, and 0400 hr. So, at the beginning of the naps the duration of prior wakefulness was 2, 4, 6, 8, 10, 12, and 20 hr, respectively. At the beginning of the baseline sleep preceding a nap, subjects had been awake for 15–16 hr. Three to six of these baseline nights were recorded in each subject. In two subjects, a nap was recorded thrice at each time; in these two subjects, the average of the data points for each nap was calculated and used in further computations. In the remaining subjects, each nap was recorded once. To prevent contamination between naps, the interval between two consecutive naps was at least 3 days. In each subject, the order of naps was such that possible order effects could not influence the group average substantially. To standardize the conditions before a nap, subjects entered the laboratory 90 min before the start of the nap. In the case of the "nap" at 0400 hr, the subjects entered the laboratory at midnight and were under continuous surveillance to prevent sleep. Subjects were awakened from a nap when they had completed one NREM–REM cycle. The nap starting at 0400 hr was not terminated but lasted until the subjects awakened spontaneously in the morning. All sleep recordings were made in a completely darkened room.

The EEG was derived from C3–A2 and C4–A1 (Jasper, 1958) and low-pass-filtered at 25 Hz (24 dB/oct). EEG, EMG, and EOG were recorded at a paper speed of $10 \text{ mm} \cdot \text{sec}^{-1}$. Paper recordings were scored according to the criteria of Rechtschaffen and Kales (1968). All signals were digitized with a sampling rate of 64 Hz and

stored on magnetic tape. Both EEG signals were subjected to spectral analysis by using a fast Fourier transform subroutine on a PDP 11/34 computer. The epoch length was 4 sec. Power spectra between 0.25 and 15.0 Hz were calculated. Data were reduced to 1-Hz bins by adding powers over adjacent 0.25-Hz bins. The EMG was rectified and stored on disks together with the EEG power spectra. The visual scorings were also fed into the computer. This enabled us to calculate power densities per sleep stage or per NREM-REM cycle. In all data presented here, epochs of stage 0, stage 1, REMS, and movement time during sleep were excluded. Epochs in which the EEG signal was disrupted by brief movement arousals were removed when the value of the rectified EMG exceeded a predetermined threshold.

RESULTS

SLEEP LATENCIES

All six subjects succeeded in falling asleep at all scheduled times of the day. (Two additional subjects failed to fall asleep within 30 min on the first two occasions and left the experiment.) The sleep latencies—the times between lights-off and the first epoch of stage 2 (i.e., sleep onset)—varied considerably (Fig. 1). The overall variation over the day was significantly nonrandom (Friedman nonparametric analysis of variance with repeated measures [Siegel, 1956], $\chi^2 = 27.1$, $df = 7$, $p < 0.001$). However, the latency to stage 2 was not a monotonic function of the duration of prior wakefulness. There was a steep decrease in sleep latency from 1000 hr to 1600 hr, followed by an increase till midnight. When wakefulness was extended till 0400 hr, sleep latency dropped again to values below those obtained for the baseline nights.

SLEEP STAGES IN THE FIRST NREM-REM CYCLE

Table 1 summarizes the data obtained from the visual scoring of the first NREM-REM cycle of the naps and the baseline nights. REM sleep failed to occur in only

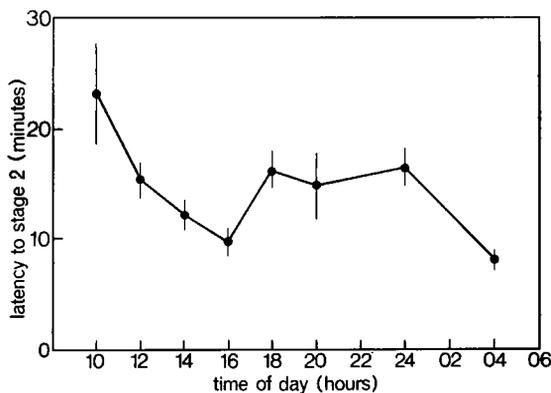


FIGURE 1. Average latencies to stage 2 at eight times of day. Bars indicate 1 SEM.

EEG POWER DENSITY DURING NAPS

TABLE 1 Steep Stages during the First NREM-REM Cycles of Naps Starting at Different Times of Day

Time of nap	Stage								
	0	1	2	3	4	MT	R	RL	CD
1000 hr	1.9 (1.8)	8.3 (8.5)	31.8 (14.0)	1.2 (2.4)	1.4 (3.5)	0.9 (0.8)	14.4 (9.0)	44.5 (14.7)	59.9 (19.4)
1200 hr	3.6 (7.2)	8.8 (8.7)	36.9 (14.2)	5.9 (5.1)	9.3 (10.0)	0.8 (0.5)	22.0 (4.2)	64.9 (19.4)	87.3 (16.8)
1400 hr	0.9 (0.4)	5.4 (3.4)	27.7 (8.6)	10.2 (2.5)	20.0 (16.1)	0.6 (0.3)	18.0 (8.3)	63.9 (15.4)	82.9 (17.0)
1600 hr	1.0 (0.8)	4.9 (3.5)	43.7 (26.3)	10.4 (5.5)	14.6 (8.4)	0.7 (0.6)	20.9 (13.2)	73.6 (34.2)	96.2 (36.0)
1800 hr	0.5 (1.0)	5.2 (4.3)	36.6 (19.0)	7.8 (3.5)	15.7 (13.5)	0.8 (0.5)	11.0 (7.9)	66.1 (28.9)	77.6 (37.0)
2000 hr	2.6 (2.9)	8.7 (6.0)	53.0 (29.4)	12.2 (2.4)	24.0 (9.5)	1.9 (1.1)	20.4 (9.4)	101.2 (37.5)	122.8 (47.4)
Baseline	0.1	3.8	29.3	12.7	36.3	1.2	11.0	80.9	94.4
2300-2400 hr	(0.1)	(3.5)	(11.0)	(2.8)	(8.1)	(0.5)	(6.7)	(12.3)	(11.9)
0400 hr	0.1 (0.2)	4.6 (7.4)	39.8 (27.5)	14.6 (7.3)	46.1 (14.9)	2.1 (1.8)	15.1 (7.9)	101.1 (47.0)	122.4 (54.6)

Note. Six subjects in all cases, except for sleep starting at 1400 hr, where $n = 5$. All values are in minutes. Values in parentheses represent standard deviations. MT, movement time; R, REM sleep; RL, REM latency; CD, first cycle duration.

one nap; this nap was excluded from the analysis of sleep stages in the first NREM-REM cycle. The duration of the first NREM-REM cycle varied considerably. On average, shortest cycles were observed at 1000 hr, whereas longest cycles occurred at 2000 hr. The large variation in cycle length at 0400 hr may be due to so-called "skipped" first REM periods. Time spent in stages 3 and 4 during the first NREM-REM cycle gradually increased from 2.6 min at 1000 hr to 60.7 min at 0400 hr. This variation over time was preserved when the percentage of time spent in stages 3 and 4 per NREM-REM cycle was calculated. When an exponential saturating curve was fitted to the percentage of stages 3 and 4, 48.8% of the total variance could be explained. Although at 1400 hr the percentage of time spent in stages 3 and 4 was somewhat higher on average than at 1600 hr, no significant variation over time remained after removal of the monotonic trend, $F(7, 35) = 1.29, p > 0.05$.

EEG POWER DENSITY DURING THE FIRST 30 MIN OF NREM SLEEP

Since a considerable variation in the duration of the first NREM-REM cycle was observed, power densities were calculated over the first 30 min of sleep. This procedure also allowed us to include the single nap that was terminated spontaneously before any REMS had occurred. In Figure 2, power densities are expressed relative to power

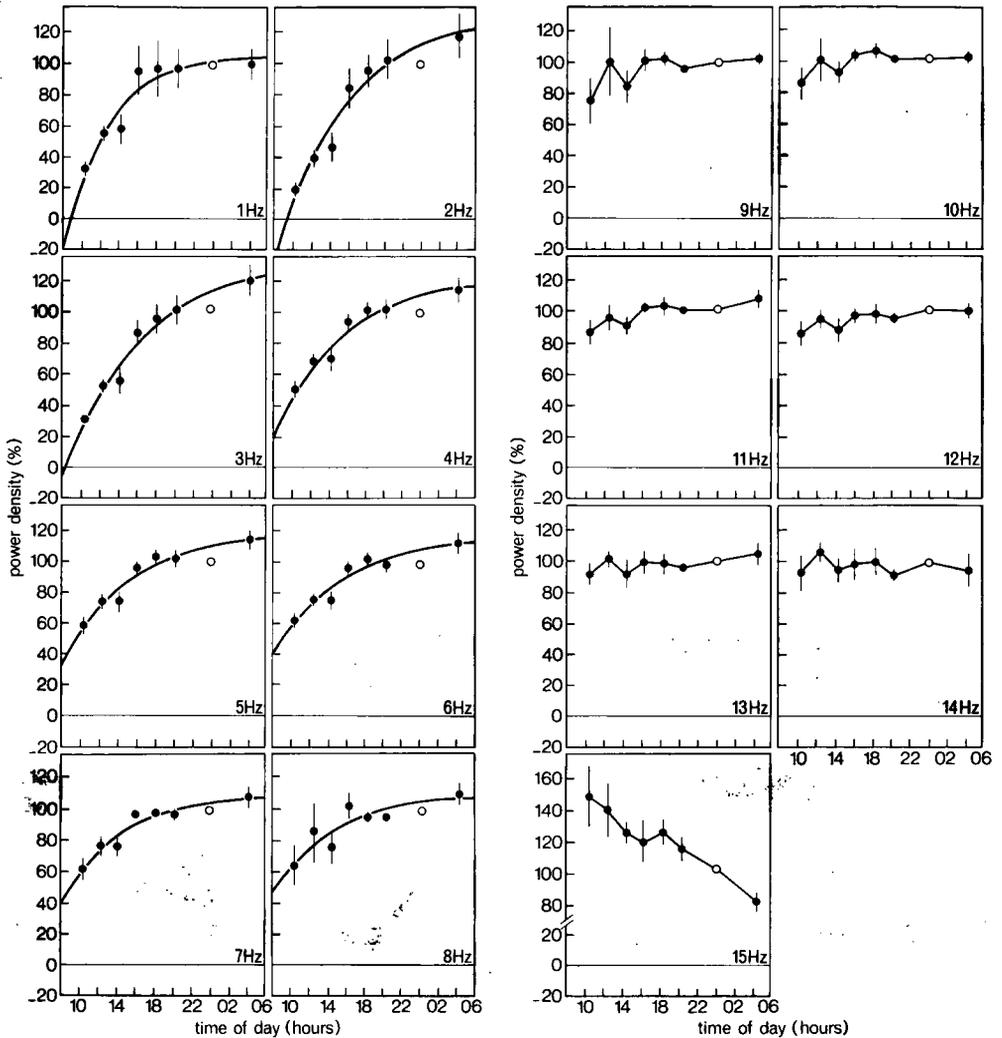


FIGURE 2. Average power densities during the first 30 min of sleep at eight times of day, plotted against the average time of sleep onset. For the first eight frequency bins, the line fitted to the data is also shown. The open circles represent the 100% values (i.e., EEG power densities during the first 30 min of sleep in the baseline nights). These values were not included in the fitting procedure. Bars indicate 1 *SEM*.

densities during the first 30 min of baseline sleep and plotted against clock time. An analysis of variance with repeated measures (Winer, 1971) revealed a significant nonrandom variation over time for power densities between 0.25 and 8.0 Hz and in the highest frequency band analyzed (14.25–15.0 Hz) (see Table 2 for details). In the lower frequency bands, power densities increased with increasing duration of prior wakefulness. In the 9- to 14-Hz bins, no significant changes were observed. In the 15-Hz band, power densities decreased with increasing duration of prior wakefulness.

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TABLE 2. Summary of the Statistics Describing the Changes in Power Functions with Increasing Duration of Prior Wakefulness

Frequency (Hz)	<i>F</i> (6, 30)	<i>p</i> <	<i>A</i>	<i>r</i>	<i>t</i> 0	PVA
1	5.54	0.001	105.5	0.218	55.19	46.0
2	8.88	0.001	131.9	0.129	86.48	58.6
3	14.05	0.001	134.2	0.115	15.49	70.0
4	13.61	0.001	121.3	0.140	-77.56	67.7
5	13.40	0.001	119.3	0.139	-137.91	65.7
6	13.24	0.001	116.4	0.142	-169.40	64.6
7	10.04	0.001	110.5	0.158	-166.27	59.5
8	3.15	0.05	111.7	0.152	-213.57	24.3
9	1.29	NS				
10	1.16	NS				
11	1.97	NS				
12	0.96	NS				
13	0.60	NS				
14	0.40	NS				
15	4.45	0.01				
Integrated (0.25-15.0)	8.89	0.001	117.0	0.158	35.66	58.0

Note. Frequency bins are as follows: 1, 0.25-1.0 Hz; 2, 1.25-2.0 Hz; etc. *A*, asymptote (%); *r*, buildup rate (log_e units/hr); *t*0, minutes; PVA, percentage of total variance accounted for by the function presented.

In those frequency bands in which a significant variation over time was observed, there was no indication of a nonmonotonic trend. Power densities in these frequency bands were, however, not equally affected by the duration of prior wakefulness. The differences between power densities at 0400 hr and 1000 hr (the naps with 20 and 2 hr prior wakefulness, respectively) were calculated (Fig. 3). This difference gradually decreased from the 2-Hz band to the 8-Hz band. The difference between power density at 0400 hr and 1000 hr in the 1-Hz band was nearly the same as the difference in the 4-Hz band. Figure 2 shows that the increase in power densities in the lower frequency bands was not linear. For a quantitative description of this increase, a saturating exponential function, $P_t = A * (1 - e^{-r*(t-t_0)})$, was fitted to the data, using the least-squares criterion. P_t represents power density at time t ; A represents the asymptote; r is the inverse of the time constant; t is the duration of prior wakefulness; and t_0 allows power density not to be zero at $t = 0$. Since all values were expressed as a fraction of the value in the first 30 min of baseline night sleep, these baseline values were not included in the fitting procedure. In the lower frequencies, 24% to 70% of the variance could be explained by fitting the three free parameters (Table 2). The highest rate of increase was observed in the 1-Hz band. The buildup rates obtained for the frequencies from 2 to 8 Hz were somewhat smaller and not much different from each other. Going from 2 to 8 Hz, t_0 decreased steeply. Finally, the time course of the integral power density (0.25-15.0 Hz) was quantified. Also, integral power

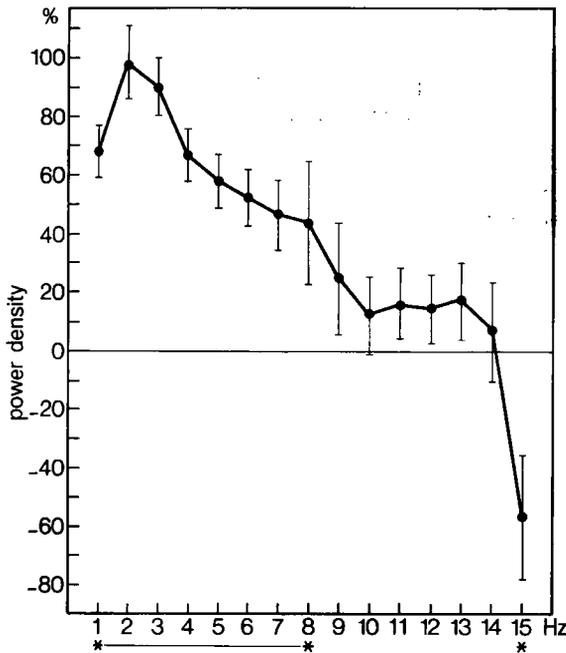


FIGURE 3. Differences in EEG power densities during the first 30 min of sleep starting at 0400 hr and 1000 hr (i.e., power density at 0400 hr minus power density at 1000 hr).

density increased monotonically with increasing duration of prior wakefulness in a saturating exponential way (see Table 2).

DISCUSSION

In the present nap study, EEG power density in the lower frequencies increased monotonically with increasing duration of prior wakefulness, whereas the length of the interval between lights-off and the onset of sleep was *not* a monotonic function of the duration of preceding wakefulness. In the afternoon, sleep latencies were lower than in the evening. This dip in sleep latency has also been reported in experiments in which sleep propensity was measured several times in one day by means of the multiple sleep latency test (MSLT; Richardson *et al.*, 1982). Carskadon and Dement (1985) showed that this midafternoon decline in sleep latency does not disappear when food intake is uniformly distributed over the day. It can be argued, however, that the pattern obtained by the MSLT is a consequence of multiple testing within a single day (Richardson *et al.*, 1982). In the present experiment, the interval between two successive naps was at least 3 days. Therefore, it must be concluded that the midafternoon decline in sleep latency is not a consequence of lunch or multiple testing.

The pattern of sleep tendency as measured by the MSLT is essentially bimodal, with a second minimum in sleep latency in the early morning (Richardson *et al.*, 1982;

Carskadon and Dement, 1985). The observed short sleep latencies at 0400 hr in the present experiment may correspond to this second minimum. But, since we did not measure sleep latencies beyond 0400 hr, we cannot be sure whether or not this really represents a minimum. Under conditions of temporal isolation there are two preferred phases for sleep: one located near the minimum of the body temperature rhythm, the other near the maximum of the body temperature rhythm (Zulley and Campbell, 1985). Under entrained conditions, these phases of the body temperature rhythm coincide with the early morning and afternoon, respectively. It has been suggested that this bimodal pattern of sleep propensity is due to an approximately 12-hr rhythm of pressure for deep NREMS (i.e., SWS) (Broughton, 1975).

Classical visual scoring arbitrarily divides NREMS in stages. Borbély *et al.* (1981) showed that in response to sleep deprivation, apart from the increase in SWS, power densities in stage 2 are also affected. Therefore, the classical visual scoring completely reflects the changes induced by manipulating prior wakefulness. This makes it very difficult to quantify the relation between prior wakefulness and NREMS by means of visual scoring. Despite this limitation, visual scoring in the present experiment revealed that the percentage of SWS in the first NREM-REM cycle at different times of day could be adequately described with a monotonic function. After removal of this trend, no significant variation over time remained. One could argue, though, that the (nonsignificant) higher percentage of SWS at 1400 hr as compared to 1800 hr does reflect signs of a high pressure for NREMS. Quantification of NREMS by means of spectral analysis revealed a significant variation over time in power densities between 0.25 and 8.0 Hz. In contrast with sleep latency, however, power densities during NREMS in the first 30 min of sleep were a monotonic function of prior wakefulness. In a recent experiment by Knowles *et al.* (1986), the amount of SWS during the first hour of sleep was shown to be a monotonic function of the waking interval preceding sleep. In their experiment the interval varied from 3 to 28 hr. These data could also be satisfactorily described with a saturating exponential curve. If the "postlunch dip" in sleep latency were due to a high pressure for NREMS, one would expect high power densities in the delta and theta range (0.25–8.0 Hz) in the afternoon to be followed by lower power densities in these frequencies in the evening. In the data presented here, there is no sign of such a time course. In the two subjects in whom each nap was recorded thrice, EEG power density also increased monotonically (data not shown). Thus the absence of high power densities in the afternoon in the group data cannot be attributed to small differences between subjects in the timing of the high pressure for NREMS. Obviously, there is a dissociation between sleep propensity and EEG power density during sleep. In the present experiment, the duration of prior wakefulness and circadian phase are confounded. The dissociation between sleep propensity and EEG power density, however, does indicate that, in contrast to sleep propensity, EEG power density is not influenced by circadian phase. Further evidence that EEG power density is independent of circadian phase was derived from an experiment in which the duration of prior wakefulness was kept constant, but the circadian phase at which sleep was initiated was varied by shifting the circadian system by means of light pulses (Dijk *et al.*, 1987). In this experiment, EEG power density was not affected by varying the circadian phase.

In the two-process model of sleep regulation (Borbély, 1982; Daan *et al.*, 1984), the alternation between sleep and wakefulness results from an interaction of a process measuring sleep debt (Process S) and a process (Process C) that is thought to be generated by a circadian pacemaker. Under normal conditions, Process C sets upper and lower thresholds for sleep debt (Process S). When, after a certain duration of wakefulness, the upper threshold is reached, sleep is initiated and Process S decays until the lower threshold is reached. The upper and lower thresholds covary with the body temperature rhythm. This results in a gating of sleep and wakefulness to the appropriate times of day. Process S is fully determined by the prior history of sleep and wakefulness. In this model, naps can be simulated by assuming that conscious decisions and experimental conditions such as darkness result in a temporary lowering of the upper threshold. The ability of our subjects in the present experiment to fall asleep at all times of day underscores the influence of conscious decisions and environmental conditions on the mechanisms responsible for the initiation of sleep. Since a physiological correlate of the upper threshold remains to be identified, one can only speculate about the mechanism by which conscious decisions affect the initiation of sleep.

Sleep latency can be thought of as reflecting the distance between Process S and the upper threshold. Simulations with standard parameters, however, produce a monotonic decrease in sleep latency and do not result in a midafternoon decline in sleep latency. If one wants to simulate this dip in sleep latency with the two-process model, the upper threshold has to be changed. We suggest that this threshold itself may vary bimodally in the circadian cycle.

Process S is thought to be reflected in the EEG power density during sleep. Over a sleep episode, power densities between 0.25 and 7.0 Hz decrease over consecutive NREM-REM cycles, whereas after sleep deprivation an increase in the same frequency band is observed (Borbély *et al.*, 1981). Reduction of EEG power density during the first 3 hr of sleep by means of acoustic stimuli resulted in an increase of power densities between 0.25 and 7.0 Hz during the second part of the night (Dijk *et al.*, in press). In the present experiment, EEG power densities between 0.25 and 8.0 Hz increased with increasing duration of prior wakefulness. In all these experiments the largest effects were found in the 2-Hz band. The similarities in the effects of these manipulations on power densities strongly suggest that a common mechanism underlies these changes. The decrease in power density in the 15-Hz band with increasing duration of prior wakefulness may be related to the same process, since a decrease in this frequency was also observed after sleep deprivation (Borbély *et al.*, 1981). The changes in power densities in the delta and theta frequencies are all in the same direction; that is, if the value of the hypothetical variable S increases, power densities in the delta and theta band increase, whereas power densities decrease when Process S decreases.

The quantitative relation between power densities and the duration of the period preceding sleep is dependent on the frequencies considered. The time course of power densities in the delta and theta band, and the time course of the integrated power density, can satisfactorily be described by a saturating exponential function. The positive values of t_0 for the first three frequency bins imply, however, that at $t = 0$

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(i.e., when the duration of prior wakefulness is 0) power densities in these frequencies would be negative, which is physiologically impossible. This result points to a systematic deviation from an exponential function, which becomes especially apparent near the extreme of the range of nap times studied. Scheduling naps with preceding waking intervals shorter than those used in the present experiment may lead to necessary refinements of the descriptive function.

The increase rate of Process S originally derived from Borbély's data (Daan *et al.*, 1984) is somewhat different from those presented here (see Table 2). The original time constant was based on integrated power density averaged over an NREM-REM cycle. In the present analysis, power densities were calculated over the first 30 min of sleep. The advantage of this method over the former is that it is not dependent on the length of the NREM-REM cycle. Since power densities in the delta and theta frequencies decrease during sleep, variations in the duration of the first cycle may affect the relation obtained between the duration of wakefulness and power densities during sleep. A preliminary analysis of the relation between the duration of prior wakefulness and integral EEG power density averaged over the first NREM-REM cycle, based on the same data, showed that when average power densities are corrected for the variation in cycle length, the buildup rate obtained is very close to the one originally derived from Borbély's data (Beersma *et al.*, 1987). The difference in buildup rate obtained by the present analysis does not affect the basic assumption that EEG power densities in the delta and theta frequencies are linked to the time course of the hypothetical Process S. But, since the quantitative relation between power density and preceding waking is dependent on the frequency band analyzed and the interval over which power density is averaged, it remains to be seen, by way of simulation, how differences in the buildup rate of Process S affect the quantitative predictions based on integrated EEG power density (Daan *et al.*, 1984). These simulations can only be fruitful when the decay of power density during sleep is also analyzed in a similar way.

Qualitatively, the data in all frequency bands where changes were observed support the hypothesis that these changes are monotonically dependent on prior wakefulness, in spite of the circadian variation in sleep latency.

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