Eur Arch Psychiatr Neurol Sci (1987) 236:323–328

European Archives of Psychiatry and Neurological Sciences

Quantitative Analysis of the Effects of Slow Wave Sleep Deprivation During the First 3 h of Sleep on Subsequent EEG Power Density

Derk Jan Dijk[1,2], **Domien G. M. Beersma**[1], **Serge Daan**[2], **Gerda M. Bloem**[1,2], **and Rutger H. Van den Hoofdakker**[1]

[1]Department of Biological Psychiatry, University of Groningen, Oostersingel 59, NL-9713 EZ Groningen, The Netherlands
[2]Department of Zoology, University of Groningen

Summary. The relation between EEG power density during slow wave sleep (SWS) deprivation and power density during subsequent sleep was investigated. Nine young male adults slept in the laboratory for 3 consecutive nights. Sepctral analysis of the EEG on the 2nd (baseline) night revealed an exponential decline in mean EEG power density (0.25–15.0 Hz) over successive nonrapid eye movement – rapid eye movement sleep cycles. During the first 3 h of the 3rd night the subjects were deprived of SWS by means of acoustic stimuli, which did not induce wakefulness. During SWS deprivation an attenuation of EEG power densities was observed in the delta frequencies, as well as in the theta band. In the hours of sleep following SWS deprivation both the power densities in the frequency range from 1 to 7 Hz and the amount of SWS were enhanced, relative to the same period of the baseline night. Both the amount of EEG energy accumulating subsequent to SWS deprivation and its time course could be predicted accurately from the EEG energy deficit caused by SWS deprivation. The data show that the level of integral EEG power density during a certain period after sleep onset depends on the amount of EEG energy accumulated during the preceding sleep rather than on the time elapsed since sleep onset. In terms of the two-process model of sleep regulation (Borbély 1982; Daan et al. 1984) this finding indicates that EEG power density reflects the rate of decay of the regulating variable, S, rather than S itself, as was originally postulated.

Key words: Sleep – Slow wave sleep deprivation – EEG spectral analysis

Introduction

The temporal distribution of stages 3 + 4 (slow wave sleep, SWS) of human nonrapid eye movement (NREM) sleep is well established. Maximum amounts of SWS are found in the beginning of a sleep period and a steep decrease in the hourly percentage of SWS is observed thereafter (Dement and Kleitman 1957; Williams et al. 1964b; Webb and Agnew 1971). The total amount of SWS can easily be influenced by manipulating the duration of prior wakefulness. Numerous reports have shown increased amounts of SWS after increased duration of waking (Berger and Oswald 1962; Williams et al. 1964a; Moses et al. 1975; Nakazawa et al. 1978; Karacan et al. 1970a; Berger et al. 1971), whereas after extended sleep (Feinberg et al. 1980, 1982), or daytime naps (Karacan et al. 1970b; Feinberg et al. 1985) a decrease in SWS was observed. All these data are compatible with the hypothesis that the amount of SWS at the beginning of sleep is determined by the prior history of waking and sleeping. The time course of SWS during sleep is not affected by these manipulations. Furthermore, this time course is independent of the time of sleep onset relative to the phase of the body temperature rhythm (Åkerstedt and Gilberg 1981; Czeisler et al. 1980) and is observed under conditions of both entrainment and freerun of the circadian system (Weitzman et al. 1980; Zulley 1979). These data indicate that the time course of SWS is not determined by circadian processes but is a consequence of sleep itself. To resolve the question whether it is the time passed since sleep onset or the events within sleep which determine the time course of SWS, sleep has to be experimentally disturbed without inducing wakefulness. Agnew and Webb (1968) reported an increase in the amount of stage 4 in the last hours of sleep subsequent to depriving subjects of stage 4 during the previous part of sleep. These data suggest that the decrease in SWS over a sleep episode is dependent on the amount of SWS permitted to be present in the beginning of sleep rather than the time passed since sleep onset. For a quantitative description of this dependency the classical visual scoring of the sleep EEG is inadequate. The application of all night spectral analysis revealed that changes in EEG over a sleep period and changes induced by sleep deprivation are incompletely reflected in traditional sleep stages. Under baseline conditions for instance, a progressive decrease over the night in EEG power density within stage 2 has been observed (Borbély et al. 1981). After sleep deprivation, apart from an increase in SWS, an enhancement of power density during SWS and stages 1 and 2 was reported. Even during rapid eye movement (REM) sleep the power densities of the lower frequencies were enhanced (Borbély et al. 1981). From these and related findings (Feinberg et al. 1982; Sinha et al. 1972) it has been concluded that the classical division of NREM sleep into stages is merely an arbitrary subdivision of a continuous (NREM) sleep process which can be quantified by spectral analysis.

In the two-process model of sleep regulation (Borbély 1982; Daan et al. 1984; Daan and Beersma 1984) the changes in NREM sleep over a sleep episode and after sleep deprivation are supposed to reflect changes in a hypothetical process, S. In this model the timing of sleep results from an interaction between a homeostatic process (S) and a circadian process (C). The regulating variable S increases during wakefulness in

Offprint requests to: D. J. Dijk

a monotonic saturating way and declines exponentially during sleep. Sleep onset and the termination of sleep occur when S reaches a sleep or wake threshold. These thresholds are modulated by a circadian oscillation. A central supposition in the two-process model is that the level of the variable S is reflected in the mean power density (0.75–15.0 Hz) per NREM-REM cycle. This EEG parameter has been shown to decay exponentially over the first three NREM-REM cycles during undisturbed sleep (Borbély et al. 1981). However, from the experiment by Agnew and Webb (1968) it must be concluded that during experimental disturbance of sleep EEG power density does not reflect the level of S, i.e., the reduced amount of stage 4, during stage 4 deprivation does not indicate a reduced level of S. Furthermore, the observed increase in the amount of stage 4, after stage 4 deprivation indicates that during stage 4 deprivation the decay of S is slowed down. This may lead to the hypothesis that EEG power density is proportional to the rate of change of S rather than to S itself. Under this hypothesis it should be possible to quantitatively predict EEG power density after SWS deprivation from the EEG power density during SWS deprivation. In this article we report an experiment to test this hypothesis, the results of which clearly support the idea. They are discussed in the framework of the two-process model of sleep regulation.

Methods

Nine male subjects (mean age 22.8, range 20–28 years) were paid to participate in the study. They were free of sleep complaints as assessed by a general sleep quality scale. They all gave their written consent and were asked not to use drugs or alcohol and to avoid irregular activities throughout the experiment. The subjects slept in the laboratory for 3 consecutive nights and went to bed at their habitual bedtimes (23:00–00:00 h). For the 1st and 2nd night sleep was restricted to 8 h and 15 min but subjects were allowed to rise earlier if they felt wide awake. For the 3rd (experimental) night the duration of sleep was not restricted.

During the first 3 h of the experimental night the subjects were deprived of SWS by means of acoustic stimulation. Care was taken not to induce wakefulness by adjustment of the loudness of the clicks. Sleep was recorded on all 3 nights. The EEG was derived from C3–A2 and C4–A1. To avoid differences in EEG amplitude between nights due to small differences in electrode placement, electrodes were not removed between the 2nd and 3rd nights. In addition to the EEG, EMG, and EOG signals, a time signal was recorded. Paper recordings were made at a paper speed of 10 mm/s. All signals were on-line analog to digital converted with a sample rate of 64 Hz. Before the analog to digital conversion the EEG signals were low pass filtered at 25 Hz (24 dB/octave). Digitized data were stored on magnetic tape. On both EEG signals spectral analysis was performed on consecutive 4 s epochs, with a fast fourier transform routine (DEC Laboratory subroutine package). By adding the power densities over adjacent frequencies the data were reduced to 1 Hz bin width between 0.25 and 15 Hz, and stored on disk. The EMG was rectified and integrated over 4 s epochs, and also stored on disk. In addition, paper recordings were scored manually according to the criteria of Rechtschaffen and Kales (1968).

The power density data were analyzed in two ways. For the baseline night the average integral power density (0.25–15.0 Hz) was calculated for each NREM–REM cycle using Feinberg and Floyd's (1979) definition of a NREM–REM cycle. For the last cycle to be completed the only prerequisite was that REM sleep had occurred. Movement artifacts were removed automatically by elimination of epochs in which EMG values exceeded a predetermined value.

The accumulation of EEG energy was calculated over successive 30-min periods of NREM + REM sleep (stage 1 included) for the first 7 h of sleep, both on baseline and experimental nights, 7 h being the greatest common sleep length. All comparisons between baseline and experimental nights were made within subjects. Differences were tested two-sided using Wilcoxon's matched pairs signed-rank test (Siegel 1956).

Results

Baseline Night

During the baseline night the well-known decrease in SWS over successive NREM–REM cycles was observed. The first 3 cycles contained 50.2 (14.4), 34.7 (21.4), and 12.7 (11.5) min

Table 1. Sleep stages during baseline and experimental nights. The duration of sleep stages in min (mean ± SD) is presented for the intervals 0–3 and 3–7 h after sleep onset for the baseline and experimental nights

	0–3 h		3–7 h	
Stage	Baseline night	Experimental night	Baseline night	Experimental night
0	6.5 ± 14.2	10.5 ± 17.5	1.8 ± 3.3	0.7 ± 1.2
1	6.3 ± 7.9	21.6 ± 13.6**	12.7 ± 10.3	7.8 ± 5.4*
2	76.0 ± 19.2	109.6 ± 21.5**	123.0 ± 25.8	105.6 ± 14.7
3	28.9 ± 12.8	11.1 ± 12.0*	21.11 ± 14.2	27.7 ± 11.1
4	44.0 ± 19.2	3.6 ± 9.4**	10.5 ± 12.8	36.0 ± 25.2*
REM	15.2 ± 10.9	21.1 ± 12.7	65.2 ± 22.6	58.0 ± 13.6
MT	3.2 ± 1.9	2.5 ± 2.4	5.7 ± 4.3	4.2 ± 2.4
SWS	72.9 ± 15.1	14.7 ± 19.5**	31.6 ± 25.5	63.7 ± 19.0**

*$P < 0.05$, **$P < 0.01$; baseline vs experimental night
MT = movement time
SWS = slow wave sleep (stage 3 + 4)

(SD) of SWS. For analysis of the time course of EEG power density the mean power densities (0.25–15.0 Hz) for all NREM–REM cycles of each subject were standardized on the basis of the absolute amount of EEG energy (0.25–15.0 Hz) accumulated over the first 420 min after sleep onset. The ^{e}log values of normalized power density were plotted as a function of time of the corresponding cycle midpoint (Fig. 1).

Linear regression analysis showed a highly significant negative correlation ($P<0.001$). Adding a quadratic term did not significantly improve the correlation (F ratio = 1.63 NS). This shows that the decrease of the mean power density over successive NREM–REM cycles was essentially exponential. The time constant derived from the regression analysis was -0.243 ^{e}log units $\cdot$ h^{-1}.

Experimental Nights

Table 1 contains the mean duration of the sleep stages for both the baseline and experimental nights. Both nights were divided into two intervals. The first interval started at sleep onset and ended 3 h later. The second one started 3 h after sleep onset and ended 7 h after sleep onset.

Comparison between the baseline and experimental nights showed that during SWS deprivation there was indeed very little SWS left. This reduction of SWS was accompanied by a significant increase in stages 1 and 2 but not in stage 0 and stage REM. In the interval following SWS deprivation there was a strong and significant increase in SWS and a significant decrease in stage 1. No other significant changes were observed.

Figure 2 depicts the effects of SWS deprivation on the EEG power densities between 0.25 and 15.0 Hz. During SWS deprivation an attenuation of EEG power densities in the delta range was present, but in the theta band, up to 7 Hz, significant reductions in power densities were also observed. In the higher frequency bands no significant changes were present. During the 4 h subsequent to SWS deprivation a significant increase in power densities in the delta range was present, as compared to the comparable interval of the baseline night but here also changes were not limited to the delta band, as the increase extended up to 8 Hz. For higher frequencies no significant changes were observed.

For an analysis of the time course of this power 'rebound' phenomenon the power densities were integrated over the entire frequency range and over time, resulting in EEG energy. The percentage of EEG energy accumulated since sleep onset relative to EEG energy after 7 h on the baseline night was plotted at 30-min intervals, both for the baseline and experimental night (Fig. 3). Since the absolute power densities of the higher frequency bins are several orders of magnitude lower than those of the lower frequencies (Borbély et al. 1981), changes in integral power density, to a large extent reflect changes in the lower frequency range. The curve (Fig. 3) for the baseline night shows that integral power accumulates fast at the beginning of sleep, whereafter a steady decrease in the rate of accumulation can be seen. This is not surprising since the decrease in the mean power density over successive NREM–REM cycles was shown to be exponential. The modulation of power density over the NREM–REM cycle, disappeared by averaging the accumulation of energy over nine subjects because the timing of NREM–REM cycles, relative to sleep

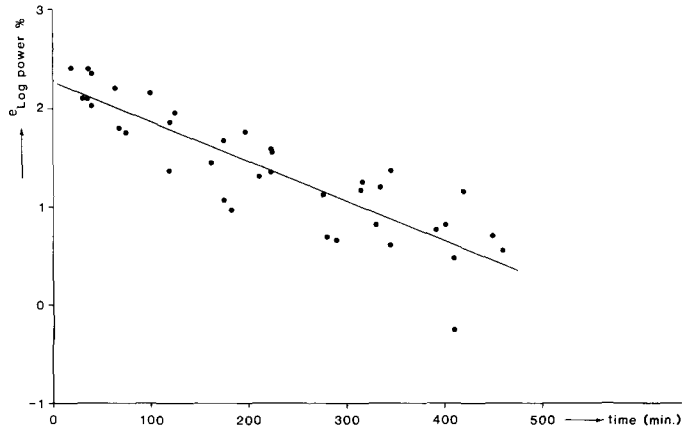

Fig. 1. Correlation between the ^{e}log of normalized mean power density (0.25–15.0 Hz per nonrapid eye movement-rapid eye movement (NREM–REM) cycle and the corresponding cycle midpoint. $n = 38$; $r = -0.868$; $P<0.001$; slope = -0.243 ^{e}log units $\cdot$ h^{-1}

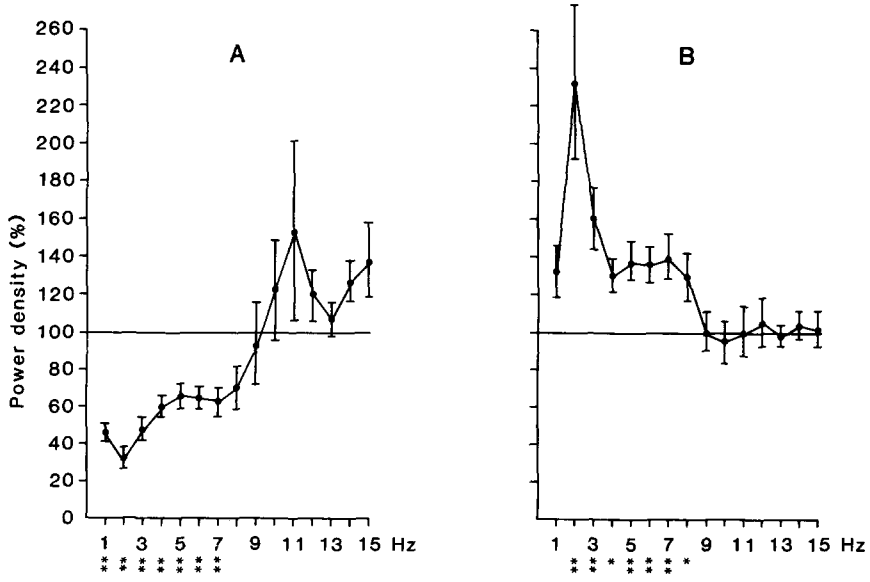

Fig. 2A, B. Mean power densities ± SEM/1 Hz bin width between 0.25 and 15.0 Hz: during SWS deprivation (**A**) and during 4 h after termination of SWS deprivation (**B**). All values are expressed as percentage of power density in the corresponding frequency bin and time interval of the baseline night and are plotted at the upper boundary of each bin. $^{*}P<0.05$; $^{**}P<0.01$; experimental vs baseline night

onset, varied between subjects. In contrast, during SWS deprivation the accumulation of EEG energy was virtually linear. Immediately after termination of SWS suppression an increase in the rate of EEG energy accumulation was observed, followed by a steady decrease.

The question arises whether the EEG energy accumulation after termination of SWS deprivation can be predicted from the amount of energy accumulated during the first 3 h of the experimental night. Figure 3 suggests that the time course of energy accumulation after termination of SWS deprivation was similar to the time course of accumulation during the baseline night starting at that timepoint at which the amount of EEG energy accumulated was equal to the amount accumulated during the first 3 h of the experimental night. This leads to the hypothesis that the EEG energy accumulated during 3 h of SWS suppression would predict the EEG energy for the following hours of undisturbed sleep. To test this hypothesis the power density was integrated over those hours of the baseline night following the timepoint at which the amount of energy accumulated was equal to the amount accumulated during the 3 h of SWS deprivation. By this method a prediction for each subject was generated about the amount and time course of the energy accumulation during the first 4 h following SWS deprivation.

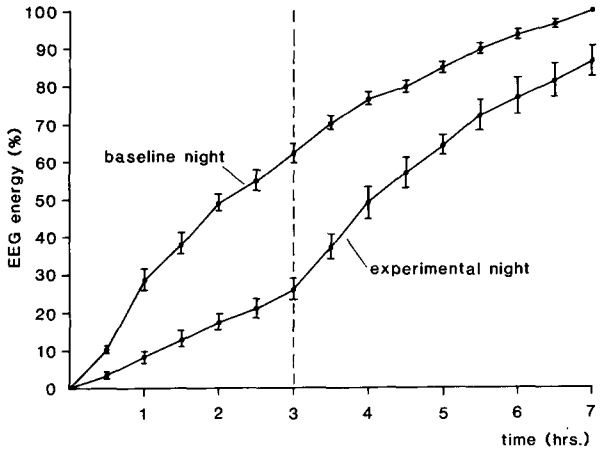

Fig. 3. Accumulation of EEG energy ± SEM during the baseline and experimental night. 100% = energy accumulated during the first 7 h of the baseline night. *Dashed vertical line* indicates the end of SWS deprivation during the experimental night

Figure 4 shows that on average this prediction was very close to the observed accumulation. At the end of the 4-h interval the mean deviation between the observed and the predicted values relative to the observed amount of energy, i.e., ((observed-predicted)/observed) × 100 was −1.1% ± 15.6% (SD). In contrast, the mean deviation between energy accumulated during 4 h after SWS deprivation and energy accumulated during the same 4-h interval (3–7 h after sleep onset) of the baseline night was 33.9% ± 20.7% (SD), whereas the mean deviation between energy accumulated during the 4 h after SWS deprivation and energy accumulated during the first 4 h of the baseline night was −33.2% ± 33.2% (SD).

Discussion

The exponential decrease in mean EEG power density over successive NREM–REM cycles as reported by Borbély et al. (1981), was confirmed in this study and is in agreement with the reported exponential decline of the EEG amplitude (Feinberg 1974). Moreover the time constant of the exponential decline calculated from the data presented here ($0.243 - {}^{e}\log$ units $\cdot$ h^{-1}) is very close to the time constant derived from Borbély's data ($0.238 - {}^{e}\log$ units $\cdot$ h^{-1}) on subjects of about the same age. It has been shown that the changes in EEG power during sleep are independent of electrode placement (Bos et al. 1977). Hence the exponential decay in EEG power density over the sleep period seems to be a fundamental and robust characteristic of undisturbed human sleep.

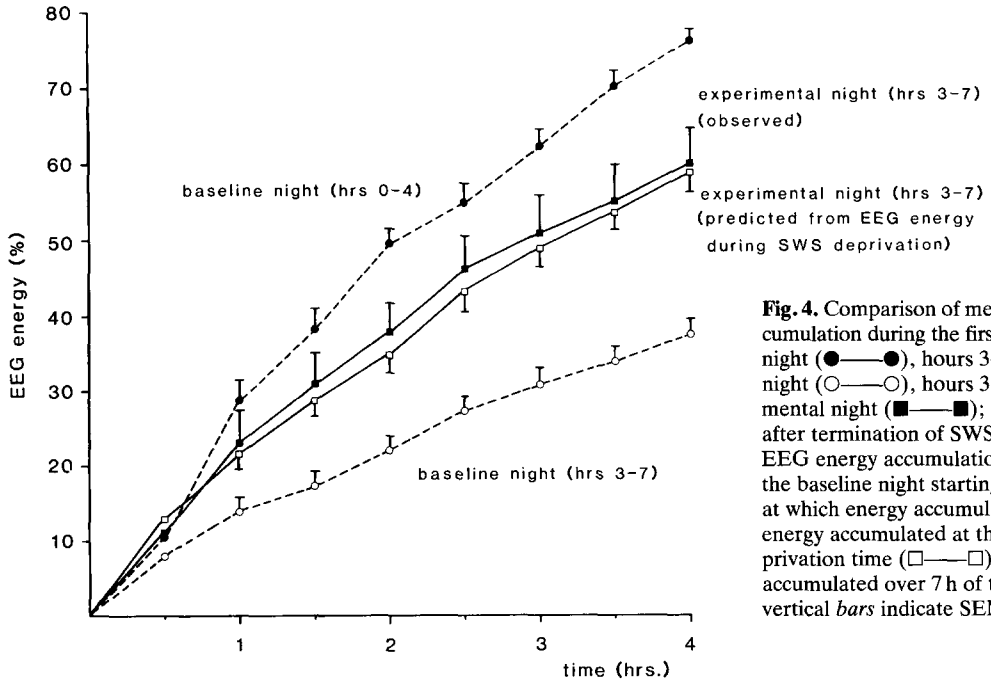

Fig. 4. Comparison of mean EEG energy accumulation during the first 4 h of the baseline night (●——●), hours 3–7 of the baseline night (○——○), hours 3–7 of the experimental night (■——■); i.e., the first 4 h after termination of SWS deprivation, and EEG energy accumulation during the 4 h of the baseline night starting at the timepoint at which energy accumulated was equal to energy accumulated at the end of SWS deprivation time (□——□). 100% = energy accumulated over 7 h of the baseline night, vertical *bars* indicate SEM

SWS deprivation during the first hours of sleep resulted in an increase in SWS in the second part of the sleep period. This finding has previously been reported by Agnew and Webb (1968) and has also been observed in experiments designed for other purposes (Bunnell et al. 1984). Spectral analysis, however, revealed some unexpected effects of SWS deprivation. During SWS deprivation not only were power densities in the delta band reduced but the reduction of power densities extended up to 7Hz. After termination of SWS deprivation an enhancement of EEG power densities was present in the same frequency range. Interestingly, recovery sleep after total sleep deprivation was characterized by enhancement of power densities in the delta and theta range as well and the decrements of power densities over the sleep period were again limited to the same frequencies (Borbély et al. 1981). These findings strongly suggest that a common mechanism underlies the changes seen after SWS deprivation, total sleep deprivation, and the changes in EEG power densities over the sleep period.

The increase in EEG power density after SWS deprivation, as compared to the same interval of the baseline night, indicates that the decrease in EEG power density during a sleep period is not dependent on the time elapsed since sleep onset but on the amount of EEG energy accumulated up to that time. The observation that after SWS deprivation the amount of energy accumulated and its time course could be accurately predicted from the energy accumulated during the SWS deprivation and the time course of accumulation during the baseline night has some implications for the relation between process S and EEG power density. Since at the beginning of the baseline night and the experimental night the prior history of sleeping and waking was identical, it must be assumed that the level of S was identical at the beginning of the two nights. So, the reduced power density during the first 3 h of sleep in the experimental night cannot be interpreted as a reduced level of S. Since during the undisturbed part of the experimental night power density was higher as compared to the corresponding part of the baseline night, it must be concluded that the decay of S was slowed down during SWS deprivation. But since EEG energy after SWS deprivation could be predicted accurately from EEG energy during SWS deprivation this EEG parameter must be proportional to the rate of decay of S. Since the decay of S is thought to be exponential and the derivates of exponential functions are exponential, during undisturbed sleep the rate of decay of S will be proportional to the level of S. This is in accordance with the observed exponential decay of EEG power density over successive NREM–REM cycles. So, the present interpretation does not affect theoretical conclusions derived from the hypothesis that power density reflects the level of S. It may however lead to other interpretations of SWS deficiencies, or power deficiencies in subjects with poor sleep continuity, as in depression. In these subjects EEG power density will reflect the rate of decay of S but, due to the many interruptions of sleep, may not be proportional to the level of S (Beersma et al. 1985, 1986; Borbély and Wirz-Justice 1982).

Additional support for the hypothesis that power density reflects the rate of decay of S can be derived from comparison of the amount and time course of SWS in habitual long and short sleepers. The small amount of SWS in long sleepers would mean, as Benoit et al. (1983) suggested, that the rate of decay of S is reduced in these sleepers as compared to short sleepers, which results in a longer sleep period. This hypothesis could also be tested experimentally. If EEG power density does indeed reflect the rate of decay of S, reduction in EEG power density should result in an increase in sleep duration. This hypothesis is presently being tested.

Acknowledgements. This research was supported by BION/STW grant no. 430.162.STW.

References

Agnew HW, Webb WB (1968) The displacement of stages 4 and REM sleep within a full night of sleep. Psychophysiology 5:142–148

Akerstedst T, Gillberg M (1981) The circadian variations of experimentally displaced sleep. Sleep 4:159–169

Beersma DGM, Daan S, Van den Hoofdakker RH (1985) The timing of sleep in depression: theoretical considerations. Psychiatr Res 16:253–262

Beersma DGM, Dijk DJ, Van den Hoofdakker RH, Daan S (1986) Sleep regulation and depression. In: Shagass C, Josiassen RC, Bridger WH, Weiss KJ, Stoff D, Simpson GM (eds) Biological psychiatry, Elsevier, New York, pp 940–942

Benoit O, Foret J, Bouard G (1983) The time course of slow wave sleep and REM sleep in habitual long and short sleepers: effect of prior wakefulness. Hum Neurobiol 2:91–96

Berger RJ, Oswald I (1962) Effects of sleep deprivation on behavior subsequent sleep and dreaming. J Ment Sci 108:457–465

Berger RJ, Walker JM, Scott TD, Magnusson LJ, Pollack SL (1971) Diurnal and nocturnal sleep stage patterns following sleep deprivation. Psychon Sci 23:273–275

Borbély AA, Baumann F, Brandeis D, Strauch I, Lehmann D (1981) Sleep deprivation: effect on sleep stages and EEG power density in man. Electroencephalogr Clin Neurophysiol 51:483–493

Borbély AA (1982) A two process model of sleep regulation. Hum Neurobiol 1:195–204

Borbély AA, Wirz-Justice A (1982) Sleep, sleep deprivation and depression – A hypothesis derived from a model of sleep regulation. Hum Neurobiol 1:205–210

Bos KHN, Van den Hoofdakker RH, Kappers EJ (1977) An electrode independent function describing the EEG changes during sleep. In: Koella WP, Levin P (eds) Sleep 1976. Karger, Basel, pp 470–473

Bunnell DE, Bevier WC, Horvath SM (1984) Sleep interruption and exercise. Sleep 7:261–271

Czeisler CA, Weitzman ED, Moore-Ede MC, Zimmerman JC, Knauer RS (1980) Human sleep: Its duration and organization depend on its circadian phase. Science 210:1264–1267

Daan S, Beersma DGM (1984) Circadian gating of human sleep and wakefulness. In: Moore-Ede MC, Czeisler CA (eds) Mathematical models of the circadian sleep-wake cycle. Raven Press, New York, pp 129–158

Daan S, Beersma DGM, Borbély AA (1984) Timing of human sleep: recovery process gated by a circadian pacemaker. Am J Physiol 246:R161–R178

Dement W, Kleitman N (1957) Cyclic variations of EEG during sleep and their relation to eye movements, body motility, and dreaming. Electroencephalogr Clin Neurophysiol 9:673–690

Feinberg I (1974) Changes in sleep cycle pattern with age. J Psychiatr Res 10:283–306

Feinberg I, Floyd TC (1979) Systematic trends across the night in human sleep cycles. Psychophysiology 16:283–291

Feinberg I, Fein G, Floyd TC (1980) EEG patterns during and following extended sleep in young adults. Electroencephalogr Clin Neurophysiol 50:467–476

Feinberg I, Fein G, Floyd TC (1982) Computer-detected patterns of electroencephalographic delta activity during and after extended sleep. Science 215:1131–1133

Feinberg I, March JD, Floyd TC, Jimison R, Bossom-Demitrack L, Katz PH (1985) Homeostatic changes during post-nap sleep maintain baseline levels of delta EEG. Electroencephalogr Clin Neurophysiol 61:134–138

Karacan I, Finley WW, Williams RL, Hursch CJ (1970a) Changes in stage 1 REM and stage 4 sleep during naps. Biol Psychiatry 2:261–265

Karacan I, Williams RL, Finley WW, Hursch CJ (1970b) The effects of naps on nocturnal sleep: Influence on the need for stage 1 REM and stage 4 sleep. Biol Psychiatry 2:391–399

Moses JM, Johnson LC, Naitoh P, Lubin A (1975) Sleep stage deprivation and total sleep loss: effects on sleep behavior. Psychophysiology 12:141–146

Nakazawa Y, Kotorii M, Ohishima M, Kotorii T, Hasuzawa H (1978) Changes in sleep pattern after sleep deprivation. Folio Psychiatr Neurol Jpn 32:85–93

Rechtschaffen A, Kales A (eds) (1968) A manual of standardized terminology, techniques and scoring systems for sleep stages of human subjects. US Government Printing Office, Washington, DC

Siegel S (1956) Nonparametric statistics for the behavioral sciences. McGraw-Hill/Kogakusha, Tokyo

Sinha AK, Smythe H, Zarcone VP, Barchas JD, Dement WC (1972) Human sleep-electroencephalogram: a damped oscillatory phenomenon. J Theor Biol 35:387–393

Webb WB, Agnew HW (1971) Stage 4 sleep: influence of time course variables. Science 174:1354–1356

Weitzman FD, Czeisler CA, Zimmerman JC, Ronda JM (1980) Timing of REM and stages 3 + 4 sleep during temporal isolation in man. Sleep 2:391–407

Williams HL, Hammack JT, Daly RL, Dement WC, Lubin A (1964a) Responses to auditory stimulation, sleep loss and the EEG stages of sleep. Electroencephalogr Clin Neurophysiol 16:269–279

Williams RL, Agnew HW, Webb WB (1964b) Sleep patterns in young adults: An EEG study. Electroencephalogr Clin Neurophysiol 17:376–381

Zulley J (1979) Der Einfluss von Zeitgebern auf den Schlaf des Menschen. Rita G Fischer Verlag, Frankfurt

Received March 2, 1987