Sleep, 17(5):438–443

Fundamental Research

Experimental Sleep Fragmentation

Timothy Roehrs, Lori Merlotti, Nancie Petrucelli, Edward Stepanski and Thomas Roth

Henry Ford Hospital, Sleep Disorders and Research Center, Detroit, Michigan, U.S.A.

Summary: Thirty-six healthy young men and women (age range 21–35 years) were studied in an experimental model of sleep fragmentation. On 2 nights sleep was disrupted by presenting tones to produce brief electroencephalogram (EEG) arousals (without shortening sleep time) and daytime function was assessed the following day with the Multiple Sleep Latency Test and a divided attention performance test. The fragmentation of sleep produced significant disruption of nocturnal sleep and reduced daytime alertness. Adaptation in EEG-defined arousals occurred from the 1st to the 2nd night of fragmentation. Threshold (measured indirectly) characteristics of EEG-defined arousals were somewhat different than those of previous studies requiring behavioral awakening. The percent of tone series producing arousal, number of tones necessary for arousal and duration of the arousal all reflected heightened thresholds in stage 3/4 and rapid eye movement (REM) sleep compared to stage 1 and stage 2 sleep. In the last 3 hours of sleep versus the first 3 hours, arousals occurred less frequently, required more tones to produce, resulted in shorter durations and in fewer sleep stage changes, except for REM sleep where the converse was the case. **Key Words:** Sleep fragmentation—Brief arousals—Daytime sleepiness/alertness—Multiple Sleep Latency Test.

The functional significance of sleep fragmentation (SF), an interruption of the normal continuity of sleep with frequent (as often as 1/minute) and transient (3–10 seconds) electroencephalogram (EEG) arousals, has become clear. The arousing stimulus differs in various sleep disorders and it can be identified in some conditions (i.e. apnea, leg movements, pain), whereas in others (i.e. sleep of elderly) it is idiopathic. Generally, the arousals produce fragmented rather than shortened sleep, and the fragmented sleep is associated with impairment of daytime function (1–4).

Several experimental models of SF in healthy normals have been developed. In one series of studies, increased daytime sleepiness and reduced performance was found after sleep was disrupted by tones presented via earphones at rates of 6 arousals per hour or less (5–8). In another set of studies, sleep was fragmented with tones presented via loudspeakers and sleepiness was increased with arousal rates of 1 per minute, but not 1 per 4 minutes (9,10). These studies have defined arousal mostly based on behavioral responses (i.e. verbal report, movement or taking a deep breath).

A different experimental model of SF in normals also uses tones presented via earphones as the arousing stimulus, but establishes arousal using EEG criteria alone. These studies have produced EEG arousals without shortening total sleep time per se, which has led to increased sleepiness the following day (11). Another study using the same methods showed that SF reduced, in a rate-dependent manner, the restorative capacity of a 60-minute nap following 1 night of sleep deprivation (12). However, in all of these studies adaptation to the arousing stimulus develops very rapidly and is a limitation of these experimental models of SF.

The present study used a more complex method of tone presentation with several stimulus characteristics of the tones varied in an effort to reduce adaptation. The study also provided an opportunity to assess the threshold characteristics of EEG-defined brief arousals. This paper presents the SF data on baseline from a larger study that evaluated the effects of benzodiazepines on SF.

METHODS

Subjects

Thirty-six healthy young (age range 21–35 years) men and women with normal nocturnal sleep and daytime alertness were studied. The protocol was reviewed and approved by the Institutional Human Rights Review

Accepted for publication April 1994.
Address correspondence and reprint requests to Timothy Roehrs, Ph.D., Henry Ford Hospital, 2921 West Grand Blvd., Detroit, MI 48202, U.S.A.

TABLE 1. *Tone presentation methods*

Tone wave form:	Sine waves			Complex waves
No. tones:	2	2	4	4
Tone duration (sec)	2	2	5	5
Tone frequency		988 ± 110 Hz		
Decibel level		90 dB		
Intertone interval (sec)	10	2	2	2

Committee. Each subject signed an informed consent and was paid for participation.

Screening

Subjects were in good health and drug free. They gave a medical and drug use history and underwent a physical exam, a routine audiometric exam, standard blood and urine laboratory analyses, and a urine drug screen within 2 weeks of the beginning of the experiment. Subjects with a past history of psychiatric disorders, drug abuse or alcoholism, a history of seizures or serious head injuries, a current medical disorder, regular use of central nervous system- (CNS-) acting drugs, or known hypersensitivity to benzodiazepines or other CNS depressants were excluded. Subjects were also excluded if they had an abnormal electrocardiogram, blood pressure or laboratory test results.

On a sleep habits questionnaire, subjects reported consistent (±2 hours) bedtimes and risetimes with sleep latencies of less than 30 minutes and total sleep times of 7–8 hours nightly. On their screening nocturnal polysomnogram, subjects had no evidence of a primary sleep disorder and had a sleep efficiency of ≥85% and a mean sleep latency the following day on the Multiple Sleep Latency Test (MSLT) ≥8 minutes. Bedtime on the screening night was maintained at 7 hours (2330–0630 hours) so that experimental sleep times could be equated to screening total sleep times (see procedures below for further explanation). On the screening MSLT day, subjects also practiced the performance tests. Subjects who did not achieve stable performance on the practice sessions or who had a sleep schedule that would change over the experimental period were excluded.

Procedures

Following the screening, each subject entered the sleep laboratory for 2 consecutive nights and days. They reported at 2200 hours and bedtime was scheduled at 2330 hours each of the nights. On the 2 nights (FR1 and FR2) subjects received placebo (2300 hours), their sleep was fragmented, and the following day MSLT and performance was assessed.

To fragment sleep, tones generated by a tone generator (Yamaha FB-01) and controlled by an Apple IIe computer were presented through miniature earphones. The tone generation program is outlined in Table 1 and was designed to vary tone duration, frequency, intertone interval and tone complexity in an effort to reduce adaptation. Tones were presented in series with an average interseries interval of 2 minutes (1 minute 36 seconds to 2 minutes 24 seconds) and a maximum of 12 tones per series. The first eight tones were pure sine waves, whereas the last four tones were randomly chosen from a set of five complex sounds (such as a trombone or a race car engine roar). The first four tones were 2 seconds long and all successive tones were 5 seconds long. Tone frequency varied from 878 to 1,098 Hz with an average of 988 Hz. All tones, both sine wave and complex tones, were 90 ± 3 dB. Each tone was individually calibrated and set to 90 dB by adjusting the dB output with a "velocity value". The first intertone interval was 10 seconds and all subsequent intervals were 2 seconds. Each tone series was terminated by the technician upon signs of EEG arousal [according to an earlier version of published criteria (13)] or after a total of 12 tones were presented. No tone series was initiated until 15 seconds of sleep had elapsed following an arousal. On both fragmentation nights, at the beginning of the sleep period the SF procedures were initiated after 10 consecutive minutes of sleep (stage 2, 3/4 or REM) were achieved. When the SF procedures increased wakefulness during the 7-hour sleep period, sleep time was extended (the SF procedure was continued) to equate experimental sleep times to the screening baseline sleep times. This was done so that possible changes in daytime function could be attributed to SF and not to shortened sleep times.

All recordings were made according to the standards of Rechtschaffen and Kales (14) using Grass model 78-D or Nihon Kohden (models 4312 and 4212) polygraphs. The Grass polygraph was calibrated with a pen deflection of 50 μV = 7.5 mm for the EEG and electrooculogram (EOG), and 50 μV = 10.0 mm for the electromyogram (EMG). The ½ amp low-frequency filter was set at 0.3 with a sensitivity at 5 for the EEG and EOG and 10 with a sensitivity of 1 for the EMG. The ½ amp high-frequency filter was set at 90. The Nihon Kohden machines were calibrated at 50 μV = 10.0 mm for the EEG and EOG and 50 μV = 16.5 mm for the EMG. The ½ amp low-frequency filters were set at 0.3 with the sensitivity at 5 for the EEG and EOG and 0.003 with a sensitivity of 1 for the EMG. The ½ amp high-frequency filters were set at 70. All electrode impedances were less than 10,000 ohms and paper speed was 10 mm/second.

The MSLT was conducted at 0900, 1100, 1300, 1500 and 1700 hours following the standard procedures (15). For the MSLT, subjects went to bed in a darkened room and were instructed to try to fall asleep, while EOGs, submental EMG and EEGs, always including

an Oz placement, were recorded. The recording was terminated after 1 minute of unambiguous stage 1 sleep, the first signs of stage 2 or REM sleep, or 20 minutes of continuous wake according to standard sleep stage criteria (14). Sleep latency was defined as time to the first nonwake epoch (15).

Divided attention performance testing was done at 1130 and 1530 hours. Each subject was trained on the divided attention task before the experiment. The 15-minute task was presented on a monochrome video screen controlled by an Apple IIe computer. The task involved tracking a moving target (an open circle) with a crosshair by using a joystick maneuvered with the preferred hand. At the same time, subjects responded by pressing a button (located on the response pad adjacent to the joystick) to the appearance of a target stimulus (the open circle became white) in the center or on the periphery (a white circle) of the screen. Both reaction times to the central and peripheral stimuli and tracking deviations were recorded.

The following study restrictions were adhered to by all subjects: 1) no alcoholic or caffeinated beverages after 1600 hours on study nights, 2) no napping during the study and 3) no medications without the approval of the investigator.

Each nocturnal polysomnographic recording was coded so that the scorers were unaware of the treatment or night. The records were scored manually in 30-second epochs following the standards of Rechtschaffen and Kales (14) and an interrater reliability of 90% or better was maintained. EEG arousals were scored according to a preliminary version of the recently published criteria (13). An interrater reliability of 88% between three scorers of the EEG arousals was achieved. Measures of threshold were tabulated, including number of tone series producing arousal, number of tones necessary to produce arousal, duration of EEG arousal, duration of EMG arousal and number of tone series producing sleep stage changes. To correct for the different sleep stage amounts on a given night, number of tone series producing arousal and sleep stage changes (the two measures are not mutually exclusive) were converted to percentages of the number of series initiated in a given sleep stage or night. The MSLT recordings were scored following the published MSLT guidelines (15).

One-, two- and three-factor repeated measures design MANOVAs (SAS Institute) were conducted on the arousal, polysomnographic and daytime performance parameters with night (screening vs. the 2 fragmentation nights or the fragmentation nights alone), sleep stage and time of night as factors. Probability levels corrected by the Greenhouse-Geisser procedure were used for all comparisons. When appropriate, post hoc contrasts (SAS Institute) were conducted.

TABLE 2. *Arousal parameters for sleep stages and nights*

	Sleep stage			
	1	2	3/4	REM
% Series with arousal[a]				
FR1[b]	88.0 ± 15.8	85.7 ± 13.5	48.0 ± 34.4	52.3 ± 22.7
FR2	79.1 ± 19.3	74.9 ± 17.3	45.1 ± 32.4	47.5 ± 21.3
No. tones to arouse[a]				
FR1[b]	3.22 ± 1.90	4.52 ± 1.55	7.83 ± 4.74	6.66 ± 1.60
FR2	4.10 ± 2.38	5.71 ± 1.63	7.13 ± 1.84	6.91 ± 1.20
Duration of EEG arousal (seconds)[a]				
FR1	9.86 ± 2.23	8.94 ± 1.29	10.8 ± 2.56	11.1 ± 2.30
FR2	9.44 ± 12.1	9.28 ± 9.9	10.3 ± 16.8	11.2 ± 14.0
Duration of EMG arousal (seconds)[a]				
FR1	8.07 ± 2.66	8.45 ± 1.67	10.9 ± 2.95	9.64 ± 2.55
FR2	9.02 ± 2.36	8.85 ± 1.52	10.8 ± 2.20	9.94 ± 2.26
% Series with sleep stage change[c,d]				
FR1	19.9 ± 19.4	31.8 ± 17.9	65.4 ± 31.1	31.6 ± 19.8
FR2	12.9 ± 17.2	30.0 ± 18.1	48.0 ± 29.2	30.5 ± 16.4

Data are means ± SD.
See text for description of post hoc comparisons.
[a] $p < 0.001$, main effects of sleep stage.
[b] $p < 0.01$, main effects of night.
[c] $p < 0.01$, main effects of sleep stage.
[d] $p < 0.001$, main effects of night.

RESULTS

Arousal parameters for the 2 nights

Overall on FR1 an average of 115 tone series were presented and arousals occurred on 78% of the series [number of arousals per hour of sleep time, arousal index (AI) = 14]. On FR2 there were 118 tone series presented with a 68% rate of arousal (AI = 12). Thus, on average, sleep was disturbed every 4 minutes on FR1 and every 5 minutes on FR2.

The arousal parameters for sleep stages and nights are presented in Table 2. Two-factor MANOVAs were conducted with sleep stage and night as factors. The percentage of tone series producing arousal declined from FR1 to FR2 ($F_{1,26} = 11.11$, $p < 0.01$). There were also sleep stage differences ($F_{3,78} = 37.60$, $p < 0.001$) but no interaction. The percentage of tone series producing arousal was greater in stages 1 and 2 than in stage 3/4 and REM sleep. The number of tones necessary to produce an arousal also increased from FR1 to FR2 ($F_{1,26} = 6.36$, $p < 0.01$). Again sleep stage differences, but no interaction, were found ($F_{3,78} = 34.49$, $p < 0.001$). Fewer tones were required in stage 1 than in stage 2, stage 3/4 and REM sleep and in stage 2 compared to stage 3/4 and REM. The duration of the EEG arousal did not change from FR1 to FR2, but it did differ among sleep stages ($F_{3,78} = 4.44$, $p < 0.01$). It was shorter in stage 2 than in stage 1, stage 3/4 and REM. The duration of the EMG arousal also did not change from FR1 to FR2, but it did differ among sleep stages ($F_{3,78} = 9.14$, $p < 0.001$). Stage 3/4 EMG arous-

TABLE 3. *Arousal parameters for first and last 3 hours*

	Stage 1		Stage 2		REM sleep	
	First	Last	First	Last	First	Last
% Series with arousal[a]						
FR1	90.6 ± 18.1	79.8 ± 31.8	92.6 ± 11.7	77.9 ± 20.8	53.3 ± 36.7	53.7 ± 23.2
FR2	80.1 ± 30.8	74.6 ± 27.6	79.5 ± 19.2	71.5 ± 20.0	49.8 ± 32.9	67.9 ± 10.8
No. tones to arouse[a,b]						
FR1	2.57 ± 1.90	4.45 ± 3.07	3.20 ± 1.63	5.83 ± 2.10	5.05 ± 2.69	6.92 ± 1.98
FR2	3.65 ± 2.30	4.50 ± 3.23	4.98 ± 1.89	6.31 ± 1.93	6.56 ± 2.60	7.17 ± 1.67
Duration of EEG arousal (seconds)[a]						
FR1	11.0 ± 2.73	9.98 ± 3.43	8.73 ± 1.59	9.56 ± 1.83	9.67 ± 3.81	11.8 ± 2.14
FR2	10.0 ± 3.75	9.21 ± 3.03	9.14 ± 1.46	9.59 ± 2.12	10.8 ± 3.38	11.9 ± 2.53
Duration of EMG arousal (seconds)						
FR1	8.69 ± 3.39	8.36 ± 4.10	8.12 ± 1.82	9.16 ± 1.94	8.08 ± 3.98	9.81 ± 2.82
FR2	9.34 ± 3.79	8.78 ± 3.56	8.58 ± 2.19	8.98 ± 2.70	9.47 ± 4.10	10.5 ± 2.73
% Series with sleep stage change[c]						
FR1	42.0 ± 27.7	38.9 ± 31.2	36.4 ± 23.1	36.0 ± 24.8	41.0 ± 44.4	34.5 ± 17.8
FR2	43.5 ± 36.3	46.1 ± 29.5	32.2 ± 18.1	37.4 ± 20.8	38.5 ± 26.0	34.1 ± 18.2

Data are means ± SD.
See text for description of post hoc comparisons.
[a] $p < 0.05$, interaction of time by sleep stage.
[b] $p < 0.001$, main effects of time of night.
[c] $p < 0.01$, interaction of time by night.

als were longer than stage 1, stage 2 and REM arousals. Finally, sleep stage changes were differentially produced when arousal was initiated from different sleep stages. They occurred with a greater percentage out of stage 3/4 compared to the other stages and with a smaller percentage out of stage 1 compared to the other stages ($F_{3,78} = 21.08$, $p < 0.001$). There also was a greater percentage of stage changes on FR1 than FR2 ($F_{3,78} = 7.26$, $p < 0.01$).

There was appreciable within-subject variability across the night in the arousal parameters. Table 3 presents the arousal parameters on each night in the first 3 hours and the last 3 hours for stages 1, 2 and REM. (Stage 3/4 occurred too infrequently in the last 3 hours for inclusion in these analyses.) For these analyses three-factor MANOVAs were conducted with sleep stage, night and time of night as factors. Main effects of time and its interactions are the focus of these analyses. Percentage of arousals showed no main effects of time, but there was a time by stage interaction ($F_{2,52} = 4.97$, $p < 0.01$). From the first to the last 3 hours, percentage of arousals increased in REM sleep, while decreasing in stages 1 and 2. The number of tones necessary to produce an arousal increased from the first to the last 3 hours (main effect: $F_{1,16} = 18.27$, $p < 0.001$), but less so for REM than the other sleep stages (interaction: $F_{2,32} = 3.61$, $p < 0.04$). The duration of the EEG arousal increased in REM sleep, but decreased in stage 1 from the first to the last 3 hours (interaction: $F_{2,32} = 4.52$, $p < 0.02$). There were no time effects or interactions on the duration of the EMG arousal. Finally, the percentage of sleep stage changes decreased from the first to the last 3 hours for all sleep stages on FR1 but not on FR2 (interaction: $F_{1,26} = 6.95$, $p < 0.01$).

Sleep fragmentation effects on nocturnal sleep

The sleep parameters on screening and fragmentation nights (FR1 and FR2) are presented in Table 4. One-factor MANOVAs were conducted to compare nights. The total sleep time on the screening night was 391 ± 41.0 minutes, on FR1 it was 379 ± 28.4 minutes, and on FR2 it was 390.2 ± 22.5 minutes. There were no differences among nights in total sleep time. Recall that bedtime was extended when the SF increased wakefulness.

Sleep-staging parameters were altered by SF. The entries to stage 1 sleep were increased from the screening night level on both FR1 and FR2 ($F_{2,70} = 12.22$, $p < 0.001$), as was the percent stage 1 sleep ($F_{2,70} = 10.26$, $p < 0.001$). Percent stage 2 sleep decreased from the screening level on FR1 ($F_{2,70} = 17.41$, $p < 0.001$), but returned to the screening level on FR2. Percent stage 3/4 sleep was reduced from the screening level on both FR1 and FR2 ($F_{2,70} = 44.36$, $p < 0.001$). Percent REM sleep was reduced from screening on FR1 ($F_{2,70} = 4.77$, $p < 0.01$) and returned to screening levels on FR2. The latency to stage 2 sleep was hastened on FR2 relative to the screening night ($F_{2,70} = 8.95$, $p < 0.01$).

Sleep fragmentation effects on daytime function

The effects of the SF and the resultant sleep disturbance on daytime function are illustrated in Fig. 1.

TABLE 4. *Sleep parameters on screening and fragmentation nights*

	Night		
	Screening	FR1	FR2
Sleep efficiency (%)	89.3 ± 7.2	89.0 ± 7.7	92.5 ± 5.5
Latency stage 2 sleep	22.7 ± 16.8	22.2 ± 21.6	15.7 ± 13.8**
Ent stage 1 sleep	18.0 ± 10.7	32.7 ± 15.3**	30.7 ± 13.3**
No. awakenings	12.6 ± 10.5	16.4 ± 12.9	14.6 ± 12.1
Wake during sleep	23.8 ± 26.9	25.5 ± 23.0	17.1 ± 16.8
% Stage 1	10.8 ± 5.1	18.0 ± 8.1**	15.3 ± 8.7**
% Stage 2	51.9 ± 6.8	61.1 ± 6.9**	57.0 ± 10.4
% Stages 3/4	18.9 ± 7.1	5.5 ± 5.5**	9.5 ± 8.1**
% Stage REM	18.4 ± 6.1	15.4 ± 4.9*	18.2 ± 5.5
Latency to REM sleep	98.7 ± 46.7	106.3 ± 47.4	92.2 ± 40.1

Data are means ± SD.
Differs from screening: * $p < 0.01$, ** $p < 0.001$.

Screening was compared to the 2 fragmentation days with one-factor MANOVAs. Mean sleep latency on the MSLT was reduced significantly ($F_{2,70} = 17.26$, $p < 0.001$) from 14.3 ± 3.6 minutes on screening to 9.8 ± 5.1 minutes after FR1 and to 9.5 ± 5.0 minutes after FR2. The mean latencies on FR1 and FR2 each differed from the screening day, but did not differ from each other. Effects of SF on the divided attention measures (tracking deviation, central and peripheral reaction times) did not reach statistical significance.

DISCUSSION

The results of this study replicate previous studies showing that SF in the absence of sleep loss per se is associated with reduced daytime function (5,6,12). Sleep disrupted by brief EEG arousals on the average of 1 every 4 or 5 minutes leads to a modest reduction (30%) in MSLT sleep latency the following day, meaning sleepiness was increased. With similar levels of SF, a similar increase in sleepiness was found in the previous studies (5,6).

A clear adaptation to the effects of the SF procedures, despite the more complex tone presentation methods, was seen in this study. Two factors, alone or in combination, which cannot be dissociated in this study, could account for the adaptation. The first is simple sensory habituation, and the second a reduced sensitivity due to increasing sleepiness as a result of the SF itself. The arousal rate went from 78% to 68% on FR1 to FR2, which then reduced the arousal index from 14 to 12. The sleep parameters also reflected a slight adaptation from the 1st to the 2nd night of SF in that all sleep stage changes showed a return to baseline, and stage REM and stage 2 no longer differed from baseline. Within each SF night there also were changes indicative of an adaptation. In the last 3 hours of sleep compared with the first 3 hours, the arousals were less frequent, required more tones to produce and resulted in shorter durations and fewer sleep stage changes.

Although adaptation to the SF procedures occurred, the daytime effects of the SF did not diminish from day 1 to day 2, which may reflect the accumulated effects of 2 nights of reduced sleep quality. Studies have shown that the daytime effects of small reductions of sleep time can accumulate over successive nights (16). These results would then suggest that reductions in sleep quality will also accumulate over successive nights.

Arousal threshold as defined by decibel level was not measured in this study because the decibel level of the stimulus remained constant throughout; but, by assessing variables such as percent of tones series producing arousal and number of tones necessary for arousal, the threshold characteristics of EEG-defined arousal could be evaluated. The first finding of interest

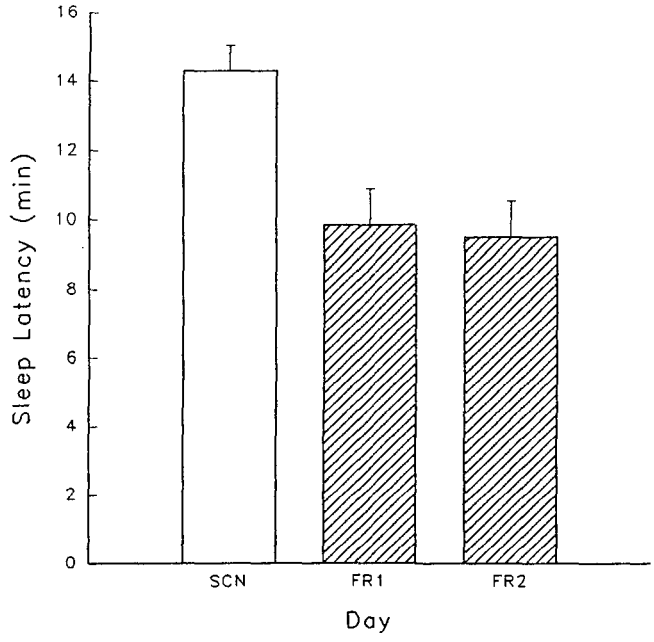

FIG. 1. Mean (± SEM) sleep latency (minutes) on an MSLT after the screening (SCN), the first (FR1) and the second (FR2) night of sleep fragmentation.

is that although threshold overall increased from the 1st to the 2nd night of SF, sleep stage-specific threshold was not altered differentially from FR1 to FR2. As sleepiness accumulated from night 1 to night 2, sleep stages were not differentially protected from arousal.

As implied in the discussion above, differential thresholds from sleep stages were found. For the measures percent arousals, number of tones and duration of arousal, thresholds from REM and stage 3/4 sleep were similar and differed from stage 2. Using EEG definitions of arousal, this pattern of results is similar to that reported by Williams et al. (17). It differs from the typical finding in auditory threshold studies in which awakening and a behavioral response are required to establish the threshold (18–20). In these studies, the threshold in stage 2 and REM sleep typically is similar and differs from that of stage 3/4, where the threshold is higher. The present study results do not confirm that finding with respect to EEG-defined arousal. EEG arousal thresholds did not differ between REM and stage 3/4 sleep, whereas the awakening thresholds do.

This finding must be interpreted with caution, however, as REM sleep occurs for the most part in the later hours of the sleep period, and stage 3/4 sleep occurs in the early hours. Thus, within a night there probably was a greater level of sleepiness in the later hours of sleep than in the early hours, due to the prior SF. The comparability in REM and stage 3/4 sleep arousal thresholds may then be an artifact of a differential level of sleepiness and a sensory habituation. The present finding is similar, however, to that of Williams et al., where care was taken to ensure that adaptation was minimized (17).

Finally, the arousal threshold studies generally also find time-of-night differences within sleep stages. Later in the night, threshold in all sleep stages is reduced relative to early or middle of the night (19,20). The pattern of time-of-night by sleep-stage data of the present study was somewhat different. For the measures percent arousals, number of tones and duration of arousal, threshold from REM sleep increased, whereas that for stage 2 and stage 1 decreased in the last 3 hours compared with the first 3 hours. Interestingly, that pattern did not change as a function of nights (i.e. there were no triple interactions for these measures). The increased threshold in stage 2 and stage 1 from the first to the last hours of the night may reflect the effects of some sleep loss as a result of the SF during the first hours of sleep that is a within-night adaptation and a sensory habituation as well. However, why threshold then did not also increase in REM sleep is not clear.

REFERENCES

1. Roth T, Hartse KM, Zorick F, Conway W. Multiple naps and the evaluation of daytime sleepiness in patients with upper airway sleep apnea. *Sleep* 1980;3:425–39.
2. Rosenthal L, Roehrs T, Sicklesteel J, Zorick F, Wittig R, Roth T. Periodic leg movements during sleep. *Sleep* 1984;7:326–30.
3. Carskadon MA, Brown E, Dement WC. Sleep fragmentation in the elderly: relationship to daytime sleep tendency. *Neurobiol Aging* 1982;3:321–7.
4. Stepanski E, Lamphere J, Badia P, Zorick F, Roth T. Sleep fragmentation and daytime sleepiness. *Sleep* 1984;7:18–26.
5. Bonnet MH. Effect of sleep disruption on sleep, performance, and mood. *Sleep* 1985;8:11–9.
6. Bonnet MH. Performance and sleepiness following moderate sleep disruption and slow wave sleep deprivation. *Physiol Behav* 1986;37:915–8.
7. Bonnet MH. Sleep restoration as a function of periodic awakening, movement, or electroencephalographic change. *Sleep* 1987; 10:364–73.
8. Downey R, Bonnet MH. Performance during frequent sleep disruption. *Sleep* 1987;10:354–63.
9. Magee J, Harsh J, Badia P. Effects of experimentally-induced sleep fragmentation on sleep and sleepiness. *Psychophysiology* 1987;24:528–34.
10. Badia P, Harsh J, Balkin T, O'Rourke DO, Burton S. Behavioral control of respiration in sleep and sleepiness due to signal-induced sleep fragmentation. *Psychophysiology* 1985;22:517–24.
11. Stepanski E, Lamphere J, Roehrs T, Zorick F, Roth T. Experimental sleep fragmentation and sleepiness in normal subjects. *Int J Neurosci* 1987;33:207–14.
12. Levine B, Roehrs T, Stepanski E, Zorick F, Roth T. Fragmenting sleep diminishes its recuperative value. *Sleep* 1987;10:590–9.
13. American Sleep Disorders Association Atlas Task Force. EEG arousals: scoring rules and examples. *Sleep* 1992;15:173–84.
14. Rechtschaffen A, Kales A. *A manual of standardized terminology, techniques and scoring system for sleep stages of human sleep.* Los Angeles: Brain Information Service/Brain Research Institute, University of California at Los Angeles, 1968.
15. Carskadon MA, Dement WC, Mitler MM, Roth T, Westbrook PR, Keenan S. Guidelines for the Multiple Sleep Latency Test (MSLT): a standard measure of sleepiness. *Sleep* 1986;9:519–24.
16. Carskadon MA, Dement WC. Cumulative effects of sleep restriction on daytime sleepiness. *Psychophysiology* 1981;18:107–13.
17. Williams HL, Hammack JT, Daly RL, Dement WC, Lubin A. Responses to auditory stimulation, sleep loss and EEG stages of sleep. *Electroencephalogr Clin Neurophysiol* 1964;16:269–79.
18. Bonnet MH, Johnson LC, Webb W. The reliability of arousal threshold during sleep. *Psychophysiology* 1978;15:412–6.
19. Rechtschaffen A, Hauri P, Zeitlin M. Auditory awakening thresholds in REM and NREM sleep stages. *Percept Mot Skills* 1966;22:927–42.
20. Watson R, Rechtschaffen A. Auditory awakening thresholds and dream recall in NREM sleep. *Percept Mot Skills* 1969;29:635–44.