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Sleep Loss in Elderly Volunteers

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Summary: Sleep, performance, and sleepiness were assessed in 10 elderly volunteers (8 women, 2 men; aged 61–77 years) before, during, and after 38 h of sleep loss. Recovery night 1 sleep showed increased total sleep and stages 3 and 4 sleep and decreased stage 1 sleep, wakefulness, brief arousals, and latency to stages 3 and 4 sleep. An increase in stage 4 sleep persisted to the second recovery night. Increased arousal threshold was suggested by a lengthening of respiratory events and a reduction in arousals associated with leg movements. Performance was impaired during sleep loss, associated with an increased tendency to fall asleep. Reported sleepiness increased, except in three subjects who denied sleepiness. Latency to sleep onset declined. All measures returned to basal values after a night of sleep. Sleep in one volunteer failed to respond to sleep loss. With this exception, the response was similar to that reported in younger volunteers, although shorter-lived. **Key Words:** Human—Sleepiness—Performance—Multiple Sleep Latency Test.

Many studies have linked disturbed sleep and aging [see Miles and Dement (1) for review]. Older persons generally have shorter sleep than younger individuals and sleep that is marked by numerous waking intervals. In addition, very brief arousals lasting 2–15 s have recently been shown to be a significant disruptive feature in the sleep of older persons (2). Other disturbing events in the sleep of older persons include disturbed breathing and periodic leg movements in sleep (3,4). Many older persons tend to compensate for disturbed sleep by spending more time in bed—some as long as 12 h a day (5). Such attempts are probably inefficient because the continuity of sleep may be as important to daytime well-being as the amount of sleep (2).

In an attempt to verify this hypothesis, we have begun to test a number of sleep schedules in older persons to determine if sleep continuity can be improved. The first of these schedules—total sleep loss—was done as an initial trial to determine if a significant deprivation would lead to improved sleep consolidation upon recovery.

Many studies of sleep deprivation and its effects in normal young adult males have been reported (6,7). Very few such experiments have been performed in other age groups, thus limiting the ability to generalize the findings. Webb (8) recently reported that sleep deprivation in older (aged 40–50 years) male subjects resulted in marked changes in sleep structure during recovery from 2 nights of sleep loss, with similar findings in older as compared

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with younger (aged 18–22 years) subjects. These sleep data were collected on the morning following 2 nights (50 h) of sleep loss, however, and are difficult to compare with most other sleep loss studies, in which recovery sleep is given at night.

Studies of sleep loss in younger individuals generally report a consistent pattern of response. During the sleep loss vigil, performance measures fall from basal levels, reports of sleepiness and fatigue rise, and the tendency to fall asleep increases. Recovery sleep is usually longer and deeper (more stages 3 and 4) than baseline sleep. In the present study, we examined not only the sleep stage response to sleep loss, but also transient arousals, periodic movements, and sleep-related respiratory disturbances. In addition, we measured the effect of sleep loss on daytime sleepiness and performance.

METHODS

Subjects

Ten volunteers were selected from individuals responding to notices posted in senior recreation centers and social security offices. Subjects were chosen based on general good health and a lack of specific sleep complaints. The group included two men and eight women, aged 61–77 years (mean age 69.3 years). All volunteers gave informed consent according to the guidelines of the Stanford University Committee for the Protection of Human Subjects in Medical Research. No subject reported any history of psychiatric disturbance. All volunteers were given a battery of standard cognitive tests (9). Results indicated no pronounced intellectual deficits in the group, nor was there any evidence of dementia.

Procedures

Two groups of five subjects stayed in the Stanford Summer Sleep Laboratory for 5 consecutive nights and days. An orientation session, including practice with all forms and tests, was held on the evening of the first night. All testing (sleep and performance) was performed in individual rooms. The first night and day of the study were considered adaptation, and the data are not included in this report. Bedtime on the adaptation night was 2300 (5 subjects) or 2340 h (5 subjects), and rising time was 0800 h. Baseline measures were gathered on the second night (bedtime 2200 h; rising time 0800 h) and day (0800–2200 h). Sleep was not permitted on the third night and day. The recovery period included the fourth (REC1) and fifth (REC2) nights and days on which bedtime was 2200 h and rising time was 0800 h. Research assistants continuously accompanied subjects to prevent napping. Meals were served at 0815, 1200, and 1800 h, and snacks were available on request. Alcohol and caffeine were prohibited during the study, and subjects were requested to reduce consumption of these compounds for the week before the study.

Nocturnal sleep monitoring included the following: (a) electroencephalogram (EEG) from referential C_3 or C_4 (10) leads, with referential O_1 or O_2 leads to assist in determining sleep onset; (b) referential electro-oculogram (EOG) from right and left outer canthi; (c) electromyogram (EMG) from mentalis/submentalis leads; (d) nasal airflow using thermistors taped beside each nostril; (e) respiratory effort from abdominal or thoracic mercury-filled capillary strain gauges; (f) EMG from right and/or left anterior tibialis; (g) and lead II electrocardiogram. Recording parameters for EEG were 35 Hz ½ amplitude high-frequency cutoff and 0.3 Hz ½ amplitude low-frequency cutoff (time constant 0.24 s). Paper speed for all sleep recordings was 10 mm/s. Sleep stage scoring of all records was performed by one expert rater (MAC) using a 30-s scoring epoch with the Rechtschaffen and Kales (11) criteria, including a 75-μV amplitude criterion for stages 3 and 4 NREM sleep.

We scored transient arousals when alpha arousals in the EEG lasted >2 and <15 s and were not associated with a sleep stage change in the epoch scoring (2). Respiratory disturbances included apneas or reduced-amplitude breathing (hypopneas) lasting ≥10 s (12). Leg movements were considered periodic if they occurred in a series of three or more with an interval of 4–90 s (13).

Performance on a modified version of the Wilkinson Auditory Vigilance Task (WAVT) (14) was measured at 1015 and 1415 h each day, with additional tests given at 0215 and 0615 h during sleep loss. Each test lasted 45 min and comprised a series of 900 tones presented at 3-s intervals. The majority of tones lasted 0.5 s; a 1-s tone was presented at random within each block of 20 tones (once each minute). Subjects were asked to respond to the longer tones by pressing a button. A 1-min practice test with a higher ratio of target tones occurred immediately before each test. The total number of response failures, response failures associated with sleep in the EEG recording, and total time asleep during the test were tabulated.

Introspective sleepiness was measured using two scales given at 30-min intervals when subjects were awake. The Stanford Sleepiness Scale (SSS) is a Likert scale of 7 numbered statements (15), the lowest number associated with greatest alertness. The linear analog scale (16) is a 100-mm line on which one extreme is labeled "very wide awake" and the other "very sleepy." A score of 0 on this scale corresponds to maximum alertness and 100 represents maximum sleepiness. Knowledge of previous ratings was unavailable at each subsequent rating.

The Multiple Sleep Latency Test (MSLT) (17) was administered at 2-h intervals beginning at 0930 h each day and throughout the sleep loss period. Vigorous activity was suspended 15 min before each test, and subjects lay in bed for 5 min before the test performing several calibrations (eyes open, eyes closed, and so forth) to ensure an adequate recording signal of EEG and EOG, which were monitored continuously during the test. After the calibrations, subjects completed the half-hourly sleepiness rating scales, were asked to "lie quietly, keep your eyes closed, and try to fall asleep," the bedroom lights were extinguished, and doors closed. On the adaptation day, the tests lasted 20 min whether or not subjects fell asleep; on subsequent days, tests lasted until three consecutive epochs of sleep were recorded or a maximum of 20 min if this criterion was not met. Sleep latency on this test was the interval between lights out and the first 30-s epoch of sleep (stage 1) or 20 min if no sleep occurred.

Because of the great intersubject variability in sleep data of elderly volunteers (18), we used nonparametric analyses. Data from baseline, sleep loss, REC1, and REC2 were compared using the Wilcoxon matched-pairs signed ranks test. Comparisons of all daytime measures were made between comparable times of day. Correlation coefficients were computed using the Spearman rank-order correlation method. Probability values <0.05 were considered significant throughout.

RESULTS

All volunteers appeared to tolerate the sleep loss procedure quite well. None complained of any physical or psychological difficulties other than tiredness and fatigue, which was particularly evident in the early morning hours of sleep loss. None of the volunteers showed evidence of emotional disturbance during the sleep deprivation, nor did any request to quit the study at any time.

TABLE 1. *Nocturnal sleep*

Variables	BSLN	REC1	REC2
Total sleep time (min)	467 (54)[a]	531 (52)	437 (48)[a]
Stage 1 sleep time (min)	52 (22)[a]	38 (22)	53 (27)[a]
Stage 2 sleep time (min)	248 (46)	260 (39)	220 (39)
Stage 3 sleep time (min)	36 (7)[a]	61 (19)	33 (14)[a]
Stage 4 sleep time (min)	27 (20)[a]	58 (26)	36 (19)[b]
Slow wave (stages 3 and 4) sleep time (min)	63 (25)[a]	119 (31)	70 (24)[a]
Total REM sleep time (min)	104 (30)	114 (31)	94 (27)
REM time 1st third (min)	20 (13)	22 (9)	24 (15)
REM time 2nd third (min)	45 (19)	46 (18)	29 (17)[b]
REM time 3rd third (min)	39 (29)	45 (10)	40 (16)
Sleep-onset latency (min)	16 (15)	6 (8)	12 (6)
Slow wave sleep latency (min)	20 (7)[a]	12 (8)	34 (31)[a]
REM sleep latency (min)	82 (43)	71 (36)	108 (92)
Wake time after sleep onset (min)	100 (52)[a]	51 (47)	133 (57)[a]
Number of arousals >15 s	30 (13)[a]	20 (11)	31 (11)[a]
Number of transient arousals	75 (48)[a]	47 (34)	65 (53)
Transient arousals/h of sleep	10.3 (7.3)[a]	5.6 (4.5)	9.2 (8.3)[a]
Number of respiratory disturbances	28 (32)	40 (45)	25 (31)
Respiratory disturbances/h of sleep	3.9 (4.7)	4.7 (5.2)	3.4 (4.2)
Average duration of respiratory disturbances (s)	16.1 (4.3)[a]	19.6 (5.2)	17.9 (5.7)
Number of leg movements	118 (104)	134 (141)	133 (136)
Number of leg movements with arousal	48 (36)	36 (31)	45 (43)
Leg movements with arousal/h of sleep	6.1 (4.9)[a]	4.2 (3.9)	6.5 (6.7)
Average inter-leg movement interval (s)	37 (6)	34 (6)	32 (5)

Values are means (SD). BSLN, baseline; REC1 and REC2, first and second recovery periods, respectively. Significant differences (Wilcoxon matched-pairs signed ranks test): [a]$p < 0.05$ from REC1; [b]$p < 0.05$ from BSLN and REC1.

Nocturnal sleep

Table 1 lists nocturnal sleep data for baseline, REC1, and REC2 as group means and standard deviations. These data show that sleep during REC1 night differed significantly from baseline and REC2 night. Total sleep time was longer on REC1 night, largely because of a significant increase in slow wave (stages 3 and 4) sleep. Stage 4 sleep remained elevated relative to baseline on REC2 night. Stage 1 sleep was significantly reduced as compared with baseline on REC1 night, while stage 2 and REM sleep times remained relatively unchanged across the three conditions. Analysis of the distribution of REM sleep by thirds of night (calculated from sleep onset to the last epoch of sleep) revealed a reduction of REM in the middle third of sleep on REC2 night.

Nocturnal sleep onset latency was calculated as the duration between lights out and the occurrence of three consecutive epochs of stage 1 sleep or the first epoch of stage 2 sleep. This value was quite low on every night, although there was a nonsignificant trend for faster sleep onset on REC1 night. The latency from sleep onset to the first appearance of slow wave sleep was significantly reduced on REC1 night relative to baseline and REC2. REM sleep latency from sleep onset showed no significant changes.

Intrasleep disturbances on the 3 nights were assessed by several variables. The amount of wakefulness within the sleep period (wake time after sleep onset) was significantly lower on REC1 night as compared with baseline and REC2, and this was reflected by a reduction in the number of arousals >15 s. Arousals briefer than 15 s—transient arousals—were significantly reduced on REC1 night relative to baseline, but not as compared with REC2

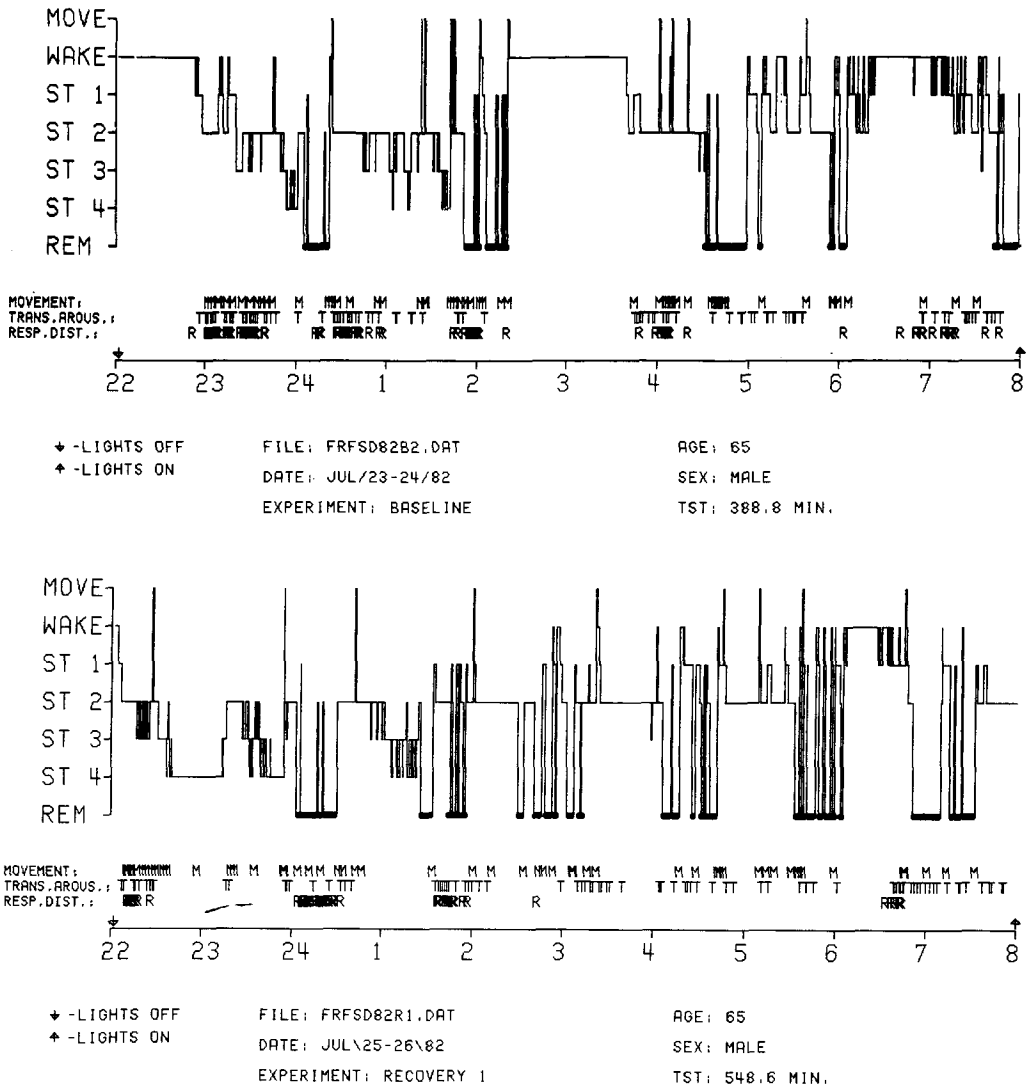

FIG. 1. All-night sleep profiles of baseline **(top)** and first recovery period **(bottom)** in a 65-year-old male volunteer selected at random as typical of the group. Graphs are based on scoring of the predominant stage in 30-s epochs. MOVE, movement time (tracing obscured during sleep); WAKE, wakefulness; ST1, ST2, ST3, ST4 = NREM sleep stages 1–4; REM, REM sleep. Discrete events, shown as letters beneath the graphs, indicate body movements during sleep (Movement, M), transient ($> 2 < 15$ s) arousals during sleep (Trans. Arous., T), and respiratory disturbances (apnea or hypopnea) during sleep (Resp. Dist., R). The time scale beneath each graph indicates the clock time through the night. TST, total sleep time.

night. When transient arousals were adjusted for time asleep (transient arousals/h of sleep), REC1 differed significantly from baseline as well as REC2.

The average number of sleep-related respiratory disturbances increased slightly on REC1 night, although this result was not significant and reflected to a large extent an increase in one subject from 17 respiratory events on baseline to 156 on REC1 night. When respiratory disturbances were adjusted for sleep time (respiratory disturbances/h of sleep), no significant differences among conditions were found. The average duration of respiratory events, on

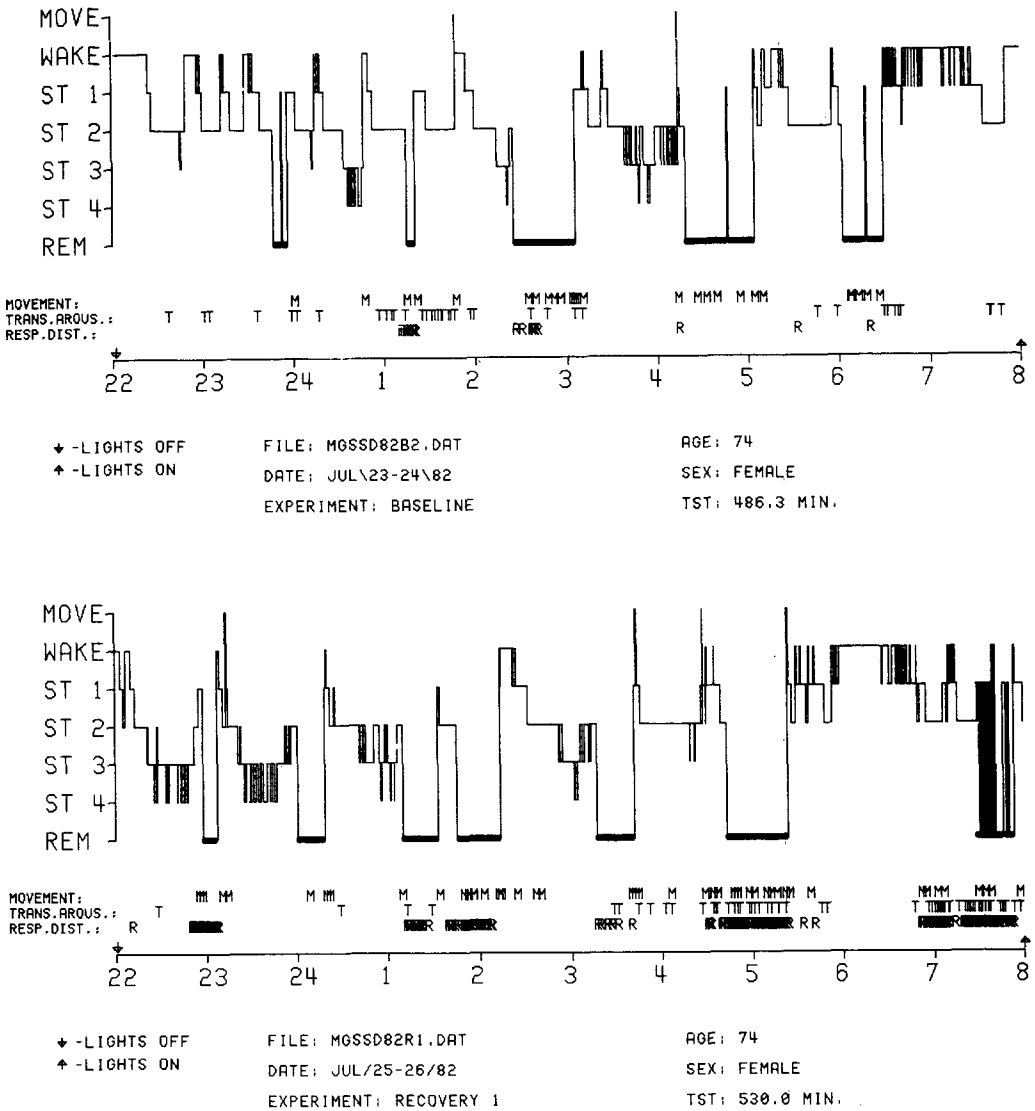

FIG. 2. All-night sleep profiles of baseline **(top)** and first recovery period (REC1) **(bottom)** in a 74-year-old female volunteer who showed a marked increase in respiration disturbances during REC1 night sleep. Note clustering of respiratory disturbances during REM sleep episodes. Labels and abbreviations as in Fig. 1.

the other hand, showed a small but consistent and significant increase on REC1 night (19.6 s) compared with baseline (16.1 s).

Periodic leg movements during sleep increased slightly but nonsignificantly on both recovery nights. Taking into account only those leg movements associated with EEG arousal, there was a slight decrease on REC1 night. When these arousal-associated leg movements were adjusted for sleep time (leg movements with arousal/h of sleep), there were significantly fewer during REC1 as compared with baseline. The average interval of periodic leg movements was unaffected by recovery from sleep loss.

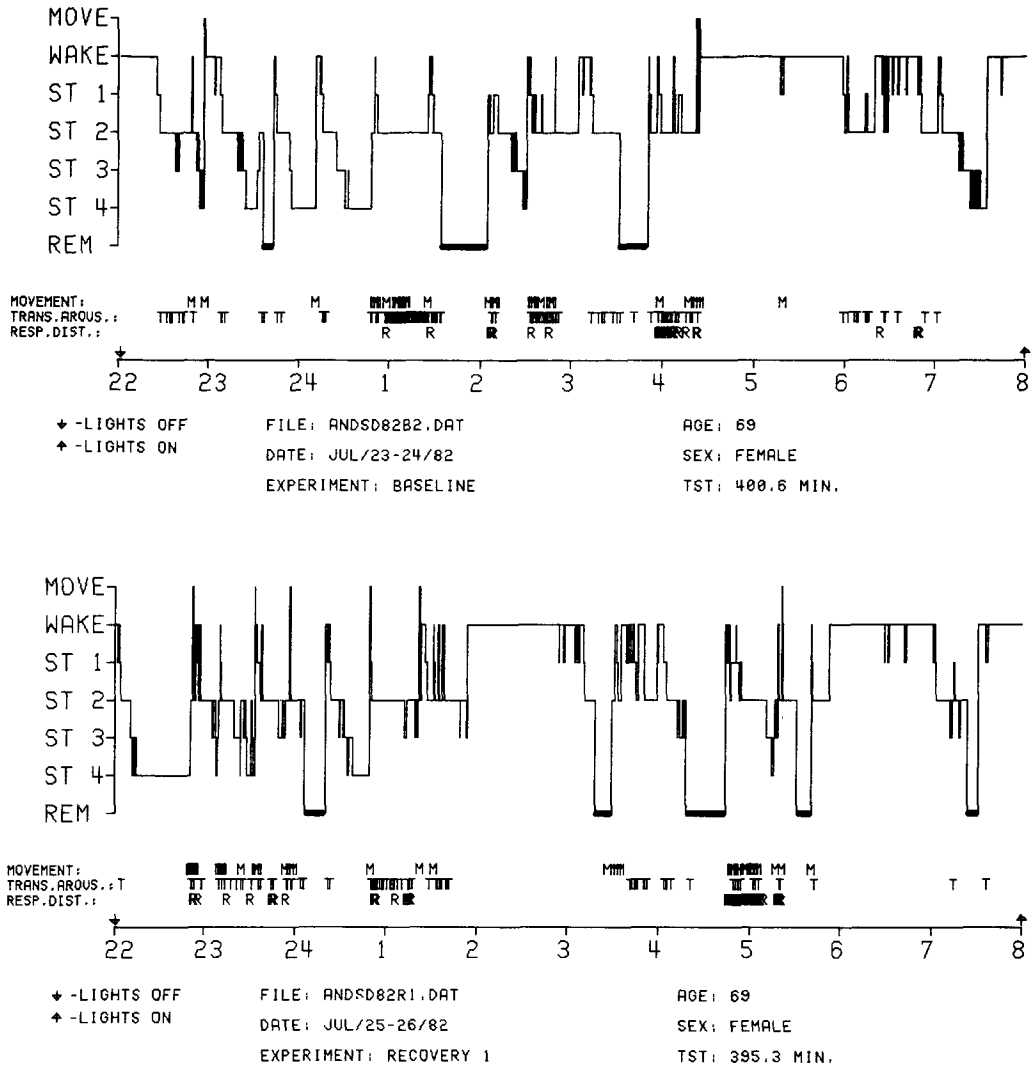

FIG. 3. All-night sleep profiles of baseline **(top)** and first recovery period (REC1) **(bottom)** in a 69-year-old female volunteer with an anomalous response to sleep loss. Although sleep time did not increase, the first hour of sleep on REC1 shows greater consolidation of sleep than baseline. Note clustering of respiratory disturbances during NREM sleep. Labels and abbreviations as in Fig. 1.

Figures 1–3 are sleep profiles from the baseline and REC1 nights in three subjects. Figure 1 illustrates the profiles of a 65-year-old male subject, selected at random as typical of the group. The following baseline versus REC1 night variables were found: total sleep time, 389 vs. 549 min; stage 1 time, 70 vs. 61 min; stage 2 time, 195 vs. 256 min; stage 3 time, 28 vs. 42 min; stage 4 time, 11 vs. 55 min; REM time, 85 vs. 135 min; number of >15-s arousals, 44 vs. 25; transient arousals, 101 vs. 86; respiratory disturbances, 79 vs. 40; and leg movements with arousals, 11 vs. 9. Of particular interest in these profiles is the comparison of the first sleep cycle, demonstrating clearly an increased consolidation of sleep during the early slow wave period on REC1.

Figure 2 illustrates the sleep profiles in the 74-year-old woman in whom respiratory disturbances increased markedly on REC1 night. Baseline versus REC1 night data include: total sleep time, 486 vs. 530 min; stage 1 time, 98 vs. 92 min; stage 2 time, 220 vs. 180 min; stage 3 time, 36 vs. 76 min; stage 4 time, 6 vs. 19 min; REM time, 126 vs. 162 min; number of >15-s arousals, 40 vs. 32; transient arousals, 41 vs. 59; respiratory disturbances, 17 vs. 156; average duration of respiratory disturbances, 14 vs. 20 s; leg movements with arousals, 28 vs. 3. Of particular interest are the first two slow-wave sleep episodes on REC1 night, in which the increased amount and consolidation of slow-wave sleep as compared with baseline are apparent. Also of interest is the clear tendency for respiratory disturbances to cluster coincident with REM episodes.

The profiles in Fig. 3 illustrate the sleep in one subject who was atypical of the group. This 69-year-old female subject was the only volunteer in whom REC1 night sleep was almost indistinguishable from baseline. Comparative baseline versus REC1 night data in this woman include: total sleep time, 400 vs. 395 min; stage 1 time, 38 vs. 33 min; stage 2 time, 224 vs. 205 min; stage 3 time, 30 vs. 34 min; stage 4 time, 51 vs. 54 min; REM time, 57 vs. 68 min; wake time after sleep onset, 156 vs. 177 min; number of >15-s arousals, 32 vs. 42; transient arousals, 147 vs. 94; respiratory disturbances, 25 vs. 46; and leg movements with arousals, 105 vs. 103. In spite of the overall lack of difference in sleep parameters, the profiles illustrate a relative consolidation of the first sleep cycle on REC1 versus baseline in this subject. Two lengthy waking episodes later in the REC1 account in large measure for the sleep disturbance. Also of interest in the profiles of this subject is a tendency for respiratory disturbances to cluster in stage 2 sleep. It should also be noted that this woman's REC2 night also showed little response to sleep loss (e.g., total sleep time, 397 min; stage 1, 38 min; stage 2, 188 min; stage 3, 26 min; stage 4, 73 min; REM, 73 min; wake after sleep onset, 186 min).

Performance

Table 2 summarizes the results of the WAVT. All measures showed a significant impairment of performance during sleep loss as compared with baseline and both recovery days. No significant differences were found when baseline, REC1, and REC2 were compared with one another. The number of missed responses on the WAVT was significantly correlated with the percentage of time spent asleep on this test in seven of the 10 subjects. The mean overall correlation coefficient for the 10 subjects was 0.62 ($p < 0.001$, zero-mu t test). Figure 4 compares the number of missed responses on the WAVT across the four conditions as a function of whether the EEG recording showed that subjects were asleep (usually stage 1) when the target tones were played. In the morning test, non–sleep-related and sleep-related misses during sleep loss were increased as compared with baseline. The afternoon test, on the other hand, showed a significant increase only of sleep-related misses during sleep loss.

Sleepiness

MSLT scores and introspective sleepiness ratings given immediately before these tests are summarized in Table 3. Three of the subjects (2 women, 1 man) denied feeling sleepy throughout the study, giving ratings of 1 on the SSS and no higher than 12 on the analog rating scale even during the sleep loss period. These three subjects account, in large measure, for the relatively high intersubject variability on these two scales. The daily mean scores on both sleepiness rating scales during sleep loss, nonetheless, showed an overall increase from baseline in reported sleepiness. In addition, the recovery day ratings indicated recovery to baseline levels.

TABLE 2. *Wilkinson Auditory Vigilance Task*

Variables	BSLN	SL	REC1	REC2
1015 h test				
Responses missed	5.5 (11.1)[a]	11.6 (11.4)	4.9 (6.9)[a]	3.4 (3.6)[a]
Sleep-related misses	0[a]	3.8 (4.5)	0[a]	0[a]
Time asleep (s)	0[a]	249 (334)	1 (3)[a]	0[a]
1415 h test				
Responses missed	7.4 (10.4)[a]	12.6 (11.1)	5.2 (6.0)[a]	4.2 (5.6)[a]
Sleep-related misses	0.7 (1.5)[a]	4.3 (4.2)	0.1 (0.3)[a]	0.3 (0.7)[a]
Time asleep (s)	54 (123)[a]	312 (303)	10 (18)[a]	7 (16)[a]
0215 h test				
Responses missed	—	8.1 (11.0)	—	—
Sleep-related misses	—	1.7 (3.8)	—	—
Time asleep (s)	—	140 (215)	—	—
0615 h test				
Responses missed	—	15.3 (11.3)	—	—
Sleep-related misses	—	3.9 (4.4)	—	—
Time asleep (s)	—	241 (280)	—	—

Values are means (SD). SL, sleep loss. See Table 1 for other abbreviations.
[a]Significant ($p < 0.05$) difference from SL (Wilcoxon matched-pairs signed ranks test).

All comparisons with sleep loss on the MSLT demonstrated significantly reduced scores. The baseline versus REC1 day comparisons demonstrated no significant differences in sleep latency test scores. REC2 sleep latency test scores showed significantly reduced sleep latency at 0930 h as compared with baseline and increased sleep latency at 1130 h and in the overall daily mean.

Relationships among sleepiness, sleep latency, and performance

Table 4 shows Spearman rank-order correlation coefficients between the various measures used to assess wakefulness. A consistently high correlation between the two introspective rating scales was found for all subjects, with the exception of three whose SSS rating never varied from fully alert. The correlation coefficients comparing ratings on these scales and the sleep latency test scores showed a significant association in six of seven subjects on the SSS and six of 10 subjects on the analog ratings.

Correlation coefficients were also computed for the number of responses missed on the WAVT and percentage of time asleep on the WAVT in relation to SSS and analog sleepiness ratings given immediately before the 10 tests (two tests each on baseline and recovery days, four tests during sleep loss) and the sleep latency test that immediately preceded the WAVT. The SSS rating was significantly predictive of WAVT scores in only one subject and of percentage of time asleep on the WAVT in only two subjects. The analog rating was somewhat more predictive, with significant correlations in three of 10 subjects for both WAVT measures. The MSLT scores showed greatest correlation to the WAVT measures, with a significant relationship to the number of responses missed in half the subjects and to percentage of time asleep on the WAVT in eight subjects.

DISCUSSION

The REC1 night response to sleep loss in these elderly volunteers was similar to that reported in younger subjects (16,19–25). In the seniors, however, only one variable—stage 4 time—showed a continued response on REC2 night. A significant reduction of REM

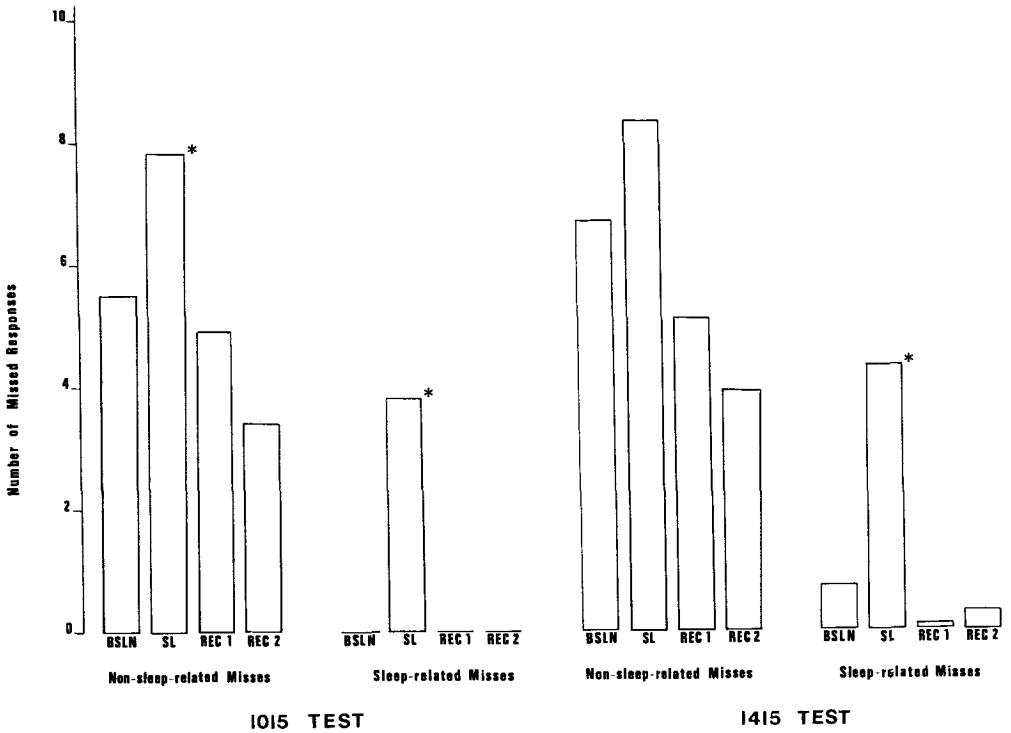

FIG. 4. Average number of missed responses on the Wilkinson Auditory Vigilance Task (WAVT) given at 1015 and 1415 h. Sleep-related misses included those occasions on which the electroencephalogram showed stage 1 sleep when the target tone was presented and the subjects failed to respond. On the WAVT given at 1015 h, sleep-related and non–sleep-related misses were significantly increased during sleep loss (SL) versus baseline (BSLN); at 1415 h, however, only sleep-related misses increased significantly during SL. REC1 and REC2, first and second recovery periods, respectively.

sleep in the second third of the night was also evident on REC2. The relative lack of response on REC2 night is in contrast with several studies in which stage 1 (16,20–22,24) and wakefulness (20) remained lower than baseline; latencies to stage 1, 2, and 3 remained lower (20); and REM sleep (which in general shows no change on the REC1 night) was significantly increased on REC2 night (16,19,23). It should be noted, however, that the sleep loss period was >1 night in three of these studies (19,21,23).

Although the sleep records in this study were scored using the standard (11) amplitude criterion for stages 3 and 4 sleep, a striking change in slow wave sleep was seen on recovery nights. Webb and Dreblow (26,27) have suggested that slow wave sleep in older individuals may be assessed with equal accuracy using frequency criteria alone. The increase of high-amplitude (>75 μV) slow wave sleep following sleep deprivation in elderly subjects suggests to us that the amplitude of slow wave EEG may be a significant factor, perhaps related to arousal threshold. Zepelin and his colleagues (28,29), for example, have shown a reduced auditory arousal threshold during sleep in older subjects, who presumably have lower amplitude EEG. In addition, the data on respiratory disturbances and leg movements reported here suggest that arousal threshold increased during recovery from sleep loss. Thus, for example, the only significant change in sleep-related respiratory variables was a consistent lengthening of the respiratory events, suggesting a reduced sensitivity to respiratory

TABLE 3. *Sleepiness and sleep tendency*

Variables	BSLN	SL	REC1	REC2
Stanford Sleepiness Scale				
0930 h	2.3 (1.1)	3.1 (2.1)	1.8 (0.9)[a]	2.3 (1.1)
1130 h	2.4 (1.3)	2.9 (1.4)	2.0 (0.8)	2.2 (1.3)[b]
1330 h	2.2 (0.8)[b]	2.6 (1.6)	1.6 (0.7)	2.0 (1.0)
1530 h	2.1 (0.9)[a]	3.0 (1.8)	2.1 (1.0)	2.1 (1.1)[a]
1730 h	1.9 (0.9)	2.2 (1.1)	1.7 (0.8)	2.2 (1.3)
1930 h	1.8 (0.8)	2.5 (1.6)	1.9 (1.2)	2.1 (1.3)
Daily mean	2.1 (0.9)[a]	2.8 (1.5)	1.9 (0.8)[a]	2.2 (1.1)[a]
Analog sleepiness scale				
0930 h	29.5 (23.5)[a]	53.0 (32.4)	23.1 (22.5)[a]	33.6 (24.3)[a]
1130 h	30.0 (21.4)	41.8 (30.5)	22.8 (18.3)[a]	31.6 (21.5)
1330 h	30.7 (22.1)	36.5 (29.1)	18.9 (13.9)	24.7 (17.8)
1530 h	31.9 (22.9)[a]	46.5 (31.2)	28.3 (18.4)[a]	23.5 (20.9)[a]
1730 h	20.3 (20.9)	33.0 (25.8)	20.4 (15.9)	26.0 (23.3)
1930 h	23.5 (15.1)	35.5 (30.4)	25.2 (20.9)	24.2 (21.5)[a]
Daily mean	27.7 (19.0)[a]	41.0 (26.7)	23.0 (14.4)[a]	27.2 (20.7)[a]
Sleep latency test				
0930 h	16.8 (5.6)[a]	2.2 (3.8)	13.3 (7.0)[a]	12.9 (5.4)[c]
1130 h	9.8 (5.4)[a]	1.7 (0.9)	11.4 (7.1)[a]	15.0 (5.9)[c]
1330 h	10.6 (8.1)[a]	1.4 (0.9)	6.4 (4.3)[a]	11.4 (6.7)[a]
1530 h	9.7 (5.8)[a]	2.0 (1.2)	7.9 (6.0)[a]	11.6 (6.3)[a]
1730 h	10.6 (5.4)[a]	2.2 (2.1)	9.2 (8.7)[a]	13.5 (6.9)[a]
1930 h	16.0 (4.0)[a]	3.9 (6.0)	13.6 (5.8)[a]	17.2 (6.2)[a]
Daily mean	12.2 (4.4)[a]	2.2 (1.9)	10.3 (4.2)[a]	13.6 (4.5)[c]

Values are means (SD). Abbreviations listed in Tables 1 and 2.
Significant differences (Wilcoxon matched-pairs signed ranks test): [a]$p < 0.05$ from SL; [b]$p < 0.05$ from REC1; [c]$p < 0.05$ from BSLN and SL.

stimuli that can accompany an increased arousal threshold (30). Guilleminault et al. (31) also recently reported increased length of apneas following sleep loss in several elderly subjects. Periodic leg movements associated with an EEG arousal response were also

TABLE 4. *Correlations (Spearman rank-order) among sleepiness, sleep tendency, and performance*

Subject	SSS vs. analog rating	SSS vs. SLT	Analog rating vs. SLT	SSS vs. no. missed WAVT	Analog rating vs. no. missed WAVT	SLT vs. no. missed WAVT	SSS vs. % asleep WAVT	Analog rating vs. % asleep WAVT	SLT vs. % asleep WAVT
1	0.80[a]	−0.38[a]	−0.50[a]	0.73[a]	0.80[a]	−0.57	0.77[a]	0.79[a]	−0.68[a]
2	0.68[a]	−0.27	−0.25	0.20	0.42	−0.72[a]	−0.02	0.38	−0.69[a]
3	0.83[a]	−0.56[a]	−0.52[a]	0.64	0.76[a]	−0.70[a]	0.71[a]	0.81[a]	−0.79[a]
4	0.86[a]	−0.57[a]	−0.54[a]	0.45	0.49	−0.73[a]	0.21	0.15	−0.80[a]
5[b]	—	—	0.02	—	0.08	−0.53	—	0.53	−0.54
6	0.74[a]	−0.59[a]	−0.86[a]	0.51	0.71[a]	−0.81[a]	0.45	0.67[a]	−0.86[a]
7[b]	—	—	−0.04	—	−0.24	−0.66[a]	—	0.00	−0.77[a]
8	0.84[a]	−0.48[a]	−0.42[a]	0.36	0.21	−0.02	0.17	0.24	−0.81[a]
9	0.79[a]	−0.64[a]	−0.68[a]	0.30	0.00	−0.56	0.45	−0.02	−0.73[a]
10[b]	—	—	−0.01	—	0.38	−0.48	—	0.32	−0.63

SSS, Stanford Sleepiness Scale; SLT, sleep latency test; WAVT, Wilkinson Auditory Vigilance Task.
[a]Significant ($p < 0.05$); Spearman rank-order correlation.
[b]Unable to compute correlations in subjects whose SSS ratings did not vary.

reduced during recovery from sleep loss. Finally, the number of transient (<15 s) arousals during sleep also declined significantly during recovery.

The progression of sleep stages on REC1 night showed a more rapid entry into slow wave sleep as compared with baseline. Webb (8) found a similar pattern in older subjects kept awake for 50 h. Direct comparisons with the Webb data are difficult, however, because of differences in scoring criteria and in the timing of recovery sleep. Nevertheless, whereas Webb's subjects all achieved stage 4 sleep within 24 min of lights out, one of our subjects failed to achieve stage 4 until 90 min after lights out (group average 38 min) on REC1 night. A more direct comparison of our elderly subjects may be made with adolescent subjects studied previously by our group (16), for whom the mean REC1 latency to stage 4 sleep was 12 min, and the longest delay to stage 4 was 19 min. We thus agree with Webb (8) that younger subjects tend to enter slow wave sleep more rapidly than older subjects following prolonged wakefulness.

Performance testing data showed results similar to those of others who have used the WAVT in younger subjects undergoing sleep loss (15,32,33). Performance lapses were greatly increased during sleep loss as compared with baseline. Because we were also monitoring EEG and EOG during the performance task, we were able to determine the relationship of performance lapses to sleep recorded during the test. Perhaps of greatest interest was the afternoon WAVT on which only the sleep-related missed responses showed a sleep loss effect (Fig. 4).

With the exception of three subjects, the introspective sleepiness rating scales showed results similar to those of other sleep loss experiments using the same or similar scales in younger subjects (15,16,20,32). Furthermore, there was a consistently high correlation between the two rating scales, suggesting that the SSS and the analog scale are measuring the same introspected feeling. We found a similar relationship in young adolescent subjects undergoing sleep deprivation (16). The three subjects who denied sleepiness throughout the study are somewhat puzzling. A similar denial of sleepiness in older individuals was evident in another group of subjects whom we awakened several times during a night of sleep (34). It is difficult to determine if these subjects truly do not perceive themselves as sleepy or if they deny feeling sleepy for some other reason, such as a reluctance to admit a weakness.

The sleep loss response on the MSLT was virtually identical to that reported in younger subjects undergoing sleep loss (16,21). Subjects began to fall asleep very quickly during the night of sleep loss and maintained this very sleepy level until permitted a night of sleep, with the exception of one subject who did not fall asleep on the 1930 h test after sleep loss. The recovery of MSLT scores, however, showed a different pattern from younger individuals, in whom sleep latency test scores tend to return to basal levels only gradually across the first recovery day (16,21). MSLT scores in the older subjects showed no significant difference at any time of day on REC1 versus baseline.

The relationship between introspective sleepiness rating scales and MSLT scores was quite good in six of the seven subjects who reported sleepiness during the study. When the MSLT scores of the subjects who denied sleepiness were assessed, two of these individuals showed slightly longer sleep latencies during the sleep loss day (6.0 and 5.6 min) than the overall mean for all subjects (2.2 min). The MSLT results in these two subjects therefore suggest that they were less sleepy during sleep loss; however, each of these subjects achieved very short sleep latencies (<1 min) on one or more of the tests, yet continued to deny sleepiness.

The relationship of the various sleepiness measures to performance on the WAVT showed a variation in the predictive value of these measures for performance scores. The SSS was significantly correlated with number of missed responses in only one of the subjects, a somewhat lesser relationship than was reported by Hoddes et al. (15) and Glenville et al. (32). The analog rating scale correlated significantly with WAVT scores in only three subjects. The MSLT scores appeared to be most closely related to performance, with a significant correlation to number of missed responses in half the subjects. The MSLT scores showed greatest correlation with the percentage of time spent sleeping during the WAVT—both apparently reflecting the tendency to fall asleep. It is clear that the MSLT does not correlate perfectly with performance test scores. We feel that a number of factors—including motivation and the arousing stimuli of a performance testing situation—influence this lack of a one-to-one correspondence. Nevertheless, these data suggest to us that the MSLT has particular validity in predicting vulnerability to falling asleep and is more predictive of test performance than are introspective measures.

We were surprised by the anomalous response of one of the subjects, a 69-year-old woman. Even though this woman showed an increase of introspected sleepiness (baseline SSS = 2.8, sleep loss = 3.2; baseline analog sleepiness = 35, sleep loss = 66), a decrease in performance (baseline number missed = 2.5, sleep loss = 12), and sleep tendency (baseline MSLT = 19.7 min, sleep loss = 1.5 min) during sleep loss, virtually no changes in nocturnal sleep were seen on either recovery night. The increase of stage 4 sleep on REC2 night may reflect a delayed recovery response in this woman, and it is possible that further recovery may have occurred on subsequent nights. In this woman, the MSLT scores also remained well below baseline on REC1 day (mean MSLT score on REC1 = 9.3 min), in contrast to other subjects in whom basal values were achieved after a night of sleep. We found no other abnormalities in this woman. For example, her scores on each of the cognitive measures (9) exceeded age-expected levels. Although all subjects were screened for sleep complaints, it is possible that this woman fits the description of Seidel and Dement (35) of insomniacs with an abnormal response to sleep disturbance. Thus, although this woman had the second lowest basal total sleep time (400 min), she had the highest basal MSLT score (19.7 min). Although ~15% of insomniacs (35) have a similar pattern of reduced sleep and high sleep latencies, the response to total sleep loss has never been tested in this group.

In summary, this study of sleep deprivation in elderly volunteers showed a similar response to that reported in younger subjects. The nocturnal sleep response of older subjects appeared to be shorter-lived, however, and somewhat less intense. Nonetheless, recovery of performance, reported sleepiness, and sleep tendency appeared to be more rapid than in younger individuals. One of the elderly subjects failed to show a normal response to sleep loss in terms of nocturnal sleep, although daytime sleepiness and performance decrements were seen. In general, however, 1 night of sleep deprivation resulted in a consolidation of nocturnal sleep on the initial recovery night in these elderly volunteers. This consolidation of nocturnal sleep and apparent increase in arousal threshold may prolong respiratory events and reduce intercurrent arousals associated with periodic leg movements in elderly individuals.

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