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Sleep Deprivation in Healthy Elderly Men and Women: Effects on Mood and on Sleep During Recovery

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Summary: Elderly women had better recovery sleep than elderly men following 36-h sleep deprivation, as evidenced by higher sleep maintenance/efficiency and more slow wave sleep (particularly in the amount of stage 4 sleep). During recovery sleep, both groups showed REM latency reduction (two men and three women had seven sleep-onset REM periods out of a total of 40 recovery nights), decrease in percentage of early REM sleep and increase in whole-night REM sleep time. Total Mood Disturbance scores on the Profile of Mood States increased in both men and women following sleep deprivation (reflecting a decrease in vigor and increase in fatigue and tension). While the increase tended to be greater in women, in both groups self-ratings of mood returned to baseline after 1 night of recovery sleep. These observations underscore the importance of gender in determining late-life sleep structure and suggest that the ability of older women to achieve slow wave sleep and to have long uninterrupted sleep in greater than that of men. **Key Words:** Sleep deprivation—Healthy elderly.

Researchers in sleep and aging generally believe that the ability to have slow wave sleep and long, uninterrupted sleep periods declines with age (1). The issue of ability versus need is not entirely settled, however; and, for this reason, the dearth of published data on the ability of healthy elderly persons to recover from sleep deprivation is surprising. Two recent reports have supplied some information in this area. In a study of 10 elderly subjects (eight women and two men, aged 61–77 years), Carskadon and Dement (2) reported an increase in slow wave sleep and in sleep continuity measures after 38 h of sleep deprivation. These authors concluded that sleep stage response to sleep deprivation was similar in the elderly to responses reported earlier in young adults, that the increase in stage 4 sleep following sleep deprivation persisted to the

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second recovery night, and that the sleep deprivation condition was associated with decreased performance on an auditory vigilance paradigm and with increased sleepiness. The study by Bonnet and Rosa (3) on 12 healthy men aged 55–71 years subjected to 64 h of sleep deprivation noted an increase in percentage of stage 3 sleep during recovery sleep and a significant decrease in REM sleep latency on the 2 consecutive recovery nights. There was little change in stage 4 sleep reported, in contrast to the increase observed by Carskadon and Dement in their largely female sample. Bonnet and Rosa noted that REM latency increased during recovery sleep in a contrast group of 12 young normal subjects. The authors speculated that the decrease in REM latency observed in the elderly normal subjects following sleep deprivation might be related to a "decreased pressure" for slow wave sleep with aging.

While both these reports suggest that sleep continuity and sleep architecture can be improved in the elderly following varying periods of sleep deprivation, differential effects related to gender may also be present. Thus, the predominantly female sample (80%) of Carskadon and Dement (2) showed a robust and persistent increase in stage 4 sleep following 38 h of sleep deprivation, as well as a stable REM latency, while the elderly men in the sample of Bonnet and Rosa (3) showed mainly an increase in stage 3 sleep following 64 h of sleep deprivation, as well as a decrease in REM sleep latency. Previous reports of baseline, or unchallenged, sleep in the healthy elderly (4,5) have noted important gender-related differences in sleep continuity and slow wave sleep, with elderly men showing more impaired sleep maintenance and less slow wave sleep than elderly women.

This present article examines the differential ability of healthy elderly men and women to recover from 36 h of sleep deprivation. We have attempted to replicate the findings of Carskadon and Dement (2) and Bonnet and Rosa (3) that sleep continuity can be improved in the elderly, and to examine further this ability in relation to gender. Our hypothesis was that elderly women would have "better" recovery sleep, i.e., superior sleep maintenance and greater slow wave sleep, than elderly men. An additional focus was to examine the effects of sleep deprivation on mood in the healthy elderly. We initially speculated that men might be more susceptible than women to the mood-disturbing effects of sleep deprivation, since their ability to recover from sleep deprivation was expected to be inferior to that of women (in measures of sleep continuity and slow wave sleep).

SUBJECTS AND METHODS

The sample consisted of 20 healthy seniors, 10 men (mean age 70.1 ± 4.6, range 62–77 years) and 10 women (mean age 68.7 ± 6.4, range 59–79 years). All were living in the community and were physically and socially active. Nine men and nine women were Caucasian, while the remaining member of each group was black. Men averaged 12.8 (4.9) years of formal education, and women averaged 13.0 years (4.1). Both men and women had a mean Folstein Mini-Mental State (6) score of 29 and a mean Hamilton Depression (7) rating of 1, thus excluding any significant symptoms of dementia or depression. The mean Hachinski (8) score in the men was 1.3 (0.9), and in the women, 1.2 (0.8).

Subjects were recruited through the Office of Alumni Affairs at the University of Pittsburgh, through local senior citizens' organizations, and through churches and syn-

agogues. A total of 129 subjects were screened to yield the sample of 20. To qualify for inclusion in the study, subjects were required to show no evidence of current or past psychiatric disorder [as determined through the administration of the SADS-L (Schedule for Affective Disorders and Schizophrenia—Lifetime Version)], no sleep-wake complaint, and no evidence on physical and neurological examination of current, active disease. In addition, results of routine laboratory studies (complete blood count, chemistry screen, thyroid function tests, serologic test for syphilis, and urine analysis) were required to be within normal limits. The daytime waking electroencephalogram (EEG) was within normal limits in eight of nine men in whom the EEG was obtained; one man had a mildly abnormal record. Among the 10 women, the EEG was within normal limits in five subjects; in the remaining five women, a mild degree of focal slowing, generally in the temporal regions, was evident. (Because the degree of EEG abnormality was mild in all cases and was without demonstrable clinical significance, the abnormality was not considered to be adequate grounds for exclusion.) There was no family history of neuropsychiatric disorders in the first-degree relatives of 16 subjects. Among the men, one subject reported a history of depression in his sister, and another reported a history of dementia in his sister. Among the women, one subject reported a history of alcohol abuse in two of her brothers and a sister, while a second subject reported a history of alcohol abuse in one brother.

All subjects had been free of psychotropic drugs and alcohol for two weeks at the time of the sleep studies. Two subjects (one man and one woman) were taking a stable dose of Synthroid (0.1 mg), and two other subjects (both men) were taking low doses of a thiazide diuretic. In the men, mean blood pressure at the time of physical examination was 159.5/86.0 mm Hg; in the women, 153.6/81.4 mm Hg. In the men, the average age at death of parents was 75.8 (10.6) years; and in the women, 83.0 (16.0) years (NS). Subjects participated in a 6-consecutive-night protocol: 3 nights of baseline EEG sleep studies (with monitoring for sleep apnea and nocturnal myoclonus on night 1), 1 night of sleep deprivation, and 2 nights of recovery sleep (nights 5 and 6). Subjects completed the Profile of Mood States (POMS) (9) at 9:00 a.m. and 9:00 p.m. on the day preceding and the 2 days following sleep deprivation, to document the effects of sleep deprivation and recovery sleep on mood. (Because we started using the POMS after the protocol was begun, POMS data were collected from six men and eight women.)

Polysomnography was performed during each subject's regular sleeping hours, with time of going to bed and getting up held constant (± 30 min) throughout the protocol. The sleep deprivation period began at the conclusion of night 3 (typically 6:30–7:00 a.m.) and extended to the start of night 5 (typically 9:00–10:00 p.m.), thus lasting ≥ 40 h in some instances, but not <36 h. Subjects were accompanied by relatives during the day following sleep deprivation in order to insure that no naps were taken. Electroencephalographic (C3–A1 + A2 at 50 μV/cm for 0.3–30 Hz), referential electrooculographic (right and left outer canthi), and bipolar submental electromyographic activity were recorded on a Grass 78B polygraph at a paper speed of 10 mm/s. On night 1, airflow was monitored following recommended and standard procedures (10) by using nasal and oral thermistors, and respiratory effort was monitored by a bellows. In accordance with Smallwood and colleagues (11), hypopnea was scored if airflow during sleep decreased to less than one third of baseline ≥ 10 s, and apnea was scored if airflow was absent for ≥ 10 s. The apnea index (AI) or combined apnea/hypopnea index (AHI) was computed as the ratio of apneic events (or sum of apneas and hypopneas) to

net sleep time in hours. Nocturnal myoclonus (PMS) was recorded and scored according to procedures and criteria presented by Coleman (12); myoclonus index was computed as the ratio of PMS to net sleep time in hours.

Data were analyzed using a one-way repeated measures analysis of variance (ANOVA) with gender as factor and night as the repeated measure. This allowed us to determine effects due to sleep deprivation, group effects, and night-by-group interactions. In addition, baseline sleep measures (the means of nights 2 and 3) were compared using Dunnett's test with recovery night 1 (night 5) and recovery night 2 (night 6) separately in each group, in order to determine which recovery effects were differentially present in each group and which persisted to the second recovery night.

RESULTS

Sleep continuity

The overall ANOVA indicated significant treatment effects on measures of sleep continuity, including a reduction in sleep latency, an increase in time spent asleep, and an increase in sleep efficiency and sleep maintenance. The ANOVA indicated no significant group effects or night-by-group interactions. However, pairwise contrasts using the Dunnett's test indicated that the increase in time spent asleep persisted into the second recovery night among women, though not among men.

In comparing baseline sleep continuity values to first recovery night, men showed an increase in time spent asleep ($p < 0.01$) and an increase in total recording period ($p < 0.05$). By contrast, among women, all sleep continuity indices (with the exception of number of arousals and minutes of early morning awakening) were significantly increased on the first recovery night, and as previously noted, the increase in time spent asleep persisted into the second recovery night.

Two subjects, both men (aged 62 and 70 years), failed to show an increase in sleep efficiency on the first recovery night, but one of them (aged 70 years) did show a delayed increase in sleep efficiency by the second recovery night.

Sleep architecture

The overall ANOVA indicated a significant treatment effect in measures of slow wave sleep (minutes and percentage of both stages 3 and 4 sleep) from baseline to recovery sleep. Group effects were also noted, with women having more slow wave sleep at baseline and during recovery. An interaction between group and treatment effects was noted in minutes of stage 4 sleep and in total minutes of slow wave sleep. This increase in slow wave sleep was associated with a decrease in both stages 1 and 2 sleep. None of these sleep architecture changes persisted into the second recovery night, as indicated by the Dunnett's test comparison of baseline and night 6 values. In within-sex pairwise comparisons of baseline versus recovery night 1, only women showed a significant increase in percentage of stage 4 sleep and percentage of slow wave sleep. While men showed an increase, this was not significant on the Dunnett's test.

Three of 20 subjects (a 69-year-old woman and two 70-year-old men) did not show an increase in slow wave sleep on the first recovery night, but one subject did show a delayed increase on the second recovery night (the same 70-year-old man with a delayed increase in sleep efficiency).

TABLE 1. *Effects of 36-h sleep deprivation on recovery sleep in elderly men and women*

	Men (n = 10)						Women (n = 10)						Night effect (p)	Group effect (p)	Interaction group × night (p)
	Nights 2 and 3		Night 5		Night 6		Nights 2 and 3		Night 5		Night 6				
Sleep continuity															
Total recording period (min)	442.0	(28.9)	457.7	(29.5)[a]	450.8	(29.2)	429.1	(46.5)	452.1	(64.6)[b]	438.3	(54.0)	0.001	NS	NS
Sleep latency	19.7	(12.4)	9.2	(11.7)	16.0	(14.1)	23.2	(18.6)	6.8	(3.4)[a]	20.3	(26.2)	0.01	NS	NS
Time spent asleep (TSA) (min)	374.9	(48.5)	419.0	(35.8)[b]	394.8	(46.7)	361.8	(42.6)	427.5	(61.7)[b]	391.4	(47.4)[a]	0.0001	NS	NS
Arousals	6.8	(2.3)	5.5	(2.8)	5.7	(3.5)	7.6	(3.1)	5.6	(2.9)	7.4	(3.9)	0.08[c]	NS	NS
Awake (min)	47.4	(38.0)	29.5	(22.9)	40.0	(40.8)	44.2	(35.8)	17.8	(15.1)[a]	26.6	(16.7)	0.01	NS	NS
Awake last 2 h	22.7	(26.2)	15.9	(15.5)	24.5	(34.8)	21.6	(19.8)	8.1	(9.8)	15.5	(10.8)	NS	NS	NS
Sleep efficiency	84.7	(7.8)	91.6	(5.4)	87.6	(8.5)	84.6	(8.0)	94.6	(3.4)[a]	89.6	(7.3)	0.0005	NS	NS
Sleep maintenance	88.9	(9.0)	93.4	(5.1)	90.9	(9.3)	89.4	(7.7)	96.0	(3.3)[a]	93.7	(3.8)	0.005	NS	NS
Sleep architecture															
Stage 1 (min)	20.7	(7.2)	15.6	(10.3)	19.3	(10.3)	18.8	(9.4)	8.7	(7.8)[a]	15.3	(9.4)	0.02	NS	NS
(%)	5.5	(1.7)	3.7	(2.5)	4.9	(2.4)	5.2	(2.5)	2.1	(2.0)[b]	3.8	(2.2)	0.002	NS	NS
Stage 2 (min)	248.4	(46.2)	257.3	(64.1)	249.5	(48.1)	202.1	(26.4)	213.8	(56.7)	210.5	(41.2)	NS	0.03	NS
(%)	66.0	(7.2)	61.1	(14.2)	63.3	(10.2)	55.9	(5.8)	50.2	(7.8)	54.1	(7.4)	0.03	0.01	NS
Stage 3 (min)	25.0	(16.8)	34.1	(17.0)	27.6	(17.1)	40.0	(13.0)	56.7	(25.0)[b]	41.2	(19.1)	0.005	0.03	NS
(%)	6.9	(4.7)	8.1	(3.8)	7.1	(4.3)	11.3	(4.0)	13.6	(6.1)	10.9	(6.0)	0.099[c]	0.03	NS
Stage 4 (min)	6.2	(10.0)	17.9	(29.7)	11.9	(21.4)	33.5	(24.3)	74.2	(52.1)[b]	41.8	(37.3)	0.001	0.01	0.05
(%)	1.7	(2.7)	4.5	(8.0)	3.1	(5.6)	9.0	(6.1)	16.6	(11.3)[b]	10.2	(8.6)	0.005	0.01	NS
Stages 3 and 4 (min)	31.1	(24.1)	52.0	(40.7)	39.5	(30.9)	73.5	(25.6)	130.9	(56.7)[b]	83.0	(44.5)	0.0001	0.002	0.05
(%)	8.5	(6.7)	12.6	(10.6)	10.1	(8.1)	20.3	(5.9)	30.1	(11.3)[b]	21.0	(10.6)	0.001	0.002	NS
REM measures															
REM periods	3.7	(1.0)	4.6	(1.0)[a]	4.2	(1.0)	3.1	(0.7)	4.6	(1.0)[b]	3.9	(0.7)[a]	0.0001	NS	NS
REM time (RT:min)	74.0	(11.7)	93.8	(20.8)[a]	86.5	(27.6)	66.6	(22.6)	74.0	(22.6)	82.6	(17.7)	0.01	NS	NS
REM time (%)	19.8	(2.8)	22.5	(5.6)	21.7	(6.1)	18.4	(5.6)	17.5	(5.2)	21.0	(3.2)	NS	NS	NS
REM activity	114.3	(53.5)	122.7	(42.7)	131.4	(73.2)	103.9	(48.3)	90.4	(51.2)	122.4	(65.7)	NS	NS	NS
REM density	1.50	(0.48)	1.30	(0.34)	1.43	(0.42)	1.53	(0.46)	1.17	(0.36)[b]	1.42	(0.57)	0.0005	NS	NS
REM latency	62.5	(24.4)	31.6	(18.8)	39.2	(25.0)	75.0	(37.6)	72.4	(53.6)	42.0	(20.8)[a]	0.02	0.06[c]	NS
REM period 1															
REM time (RT1)	21.2	(10.3)	14.4	(9.3)	24.4	(16.4)	22.7	(11.2)	16.8	(12.2)	23.5	(13.4)	0.05	NS	NS
REM activity	30.3	(21.2)	20.9	(19.3)	34.7	(36.6)	30.2	(14.7)	18.1	(14.1)	33.3	(23.9)	0.02	NS	NS
REM density	1.37	(0.50)	1.27	(0.47)	1.25	(0.39)	1.34	(0.32)	1.08	(0.28)	1.39	(0.63)	0.097[c]	NS	NS
RT1/RT (%)	29.1	(15.8)	15.9	(10.6)[a]	27.0	(10.8)	36.0	(14.5)	22.3	(13.6)[a]	28.6	(16.5)	0.005	NS	NS
RT1/TSA (%)	5.9	(3.2)	3.4	(2.1)	6.1	(3.9)	6.4	(3.1)	4.3	(3.5)	6.1	(3.6)	0.02	NS	NS

Values are expressed as means ± SD.
[a] $p < 0.05$.
[b] $p < 0.01$ (Dunnett's test for baseline vs. recovery).
[c] $0.05 < p < 0.10$.

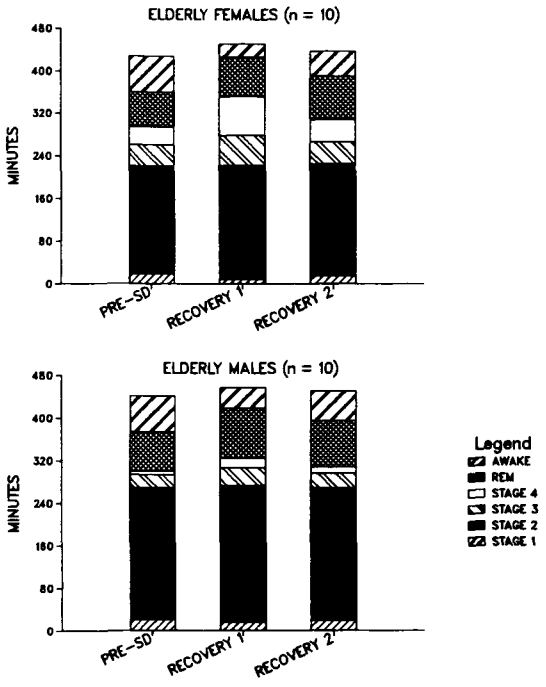

FIG. 1. Sleep/wake architecture (percentage of total recording period) before and after sleep deprivation. Following sleep deprivation, elderly women had better sleep maintenance and more stage 4 sleep than elderly men.

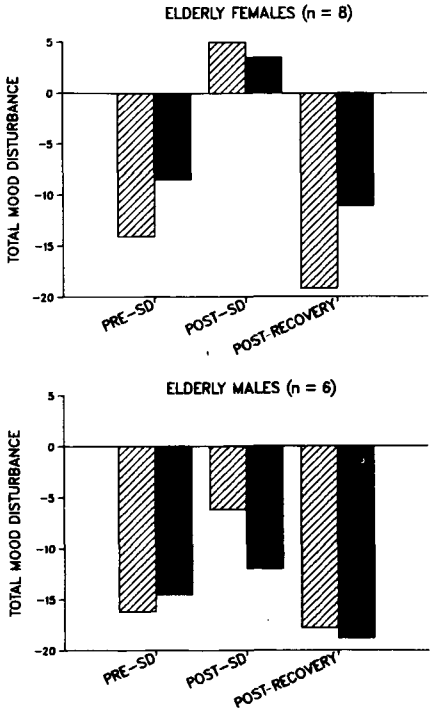

FIG. 2. Both groups registered an increase in Total Mood Disturbance on the Profile of Mood States (POMS) after sleep deprivation, with a trend toward greater disturbance in women; but in both groups self-ratings of mood disturbance returned to baseline after 1 night of recovery sleep. Solid bars, a.m.; hatched bars, p.m.

REM sleep measures

The overall ANOVA showed an increase in REM time, but not in REM percentage, during recovery sleep, without any significant group effects or group-by-night interactions. At the same time, however, REM density showed a significant decrease on the first recovery night, without significant group effects. REM sleep latency also showed a robust decrease during recovery sleep. The Dunnett's test indicated a significant reduction in REM latency among the women from baseline to second recovery night. Similarly, among the women, the Dunnett's test indicated a significant reduction in REM density from baseline to first recovery night.

A further analysis of REM latency distributions indicated that three of the women and two of the men had sleep-onset REM periods [(SOREMPs) REM latencies <15 min] during one or both recovery nights. A total of seven SOREMPs occurred, out of a possible total of 40 (the number of recovery nights). None of the subjects had had a SOREMP before sleep deprivation. Two subjects (one man and one woman) had a SOREMP on *both* recovery nights. Among the seven SOREMPs, three occurred on the first recovery night.

With respect to the intranight temporal distribution of REM sleep, the overall ANOVA indicated a significant treatment effect, with reduction in first REM period duration, particularly on the first recovery night, but without group effect or night-by-group interactions. The duration of the first REM period can also be expressed as early REM percentage, and was similarly reduced during the first night of recovery sleep in both men and women. We calculated "early REM percentage" in two ways: as the ratio of first REMP duration to total REM time (RT1/RT), and as the ratio of first REMP duration to time spent asleep (RT1/TSA).

A further comparison of the five SOREMP-positive subjects with SOREMP-negative subjects indicated no significant differences in age, POMS Total Mood Disturbance scores before or after sleep deprivation, or family history. However, SOREMP-positive subjects had significantly higher percent of stage 3 sleep at baseline than did SOREMP-negative subjects (13.3 vs. 7.7%, $p < 0.02$). Baseline REM latency was not significantly longer in SOREMP-positive subjects: 80.4 vs. 64.8 min. By contrast, SOREMP-positive subjects tended to have more REM time on the first recovery night than SOREMP-negative subjects: 100.4 versus 78.4 min, $p < 0.07$. The two groups did not differ in first REM period duration (early REM percentage) during baseline or recovery sleep.

Effects on mood

Both groups tolerated the 36-h sleep deprivation procedure well (Fig. 2). As a group, women tended to show higher Total Mood Disturbance scores on the POMS than did men, on both the morning and the evening following sleep deprivation (sex effect, $p < 0.09$; treatment effect, $p < 0.001$; night-by-sex interaction, $p < 0.07$). The change in POMS Total Mood Disturbance scores (for both groups) reflected primarily a decrease in self-ratings for vigor ($p < 0.0001$) but an increase in self-ratings on items measuring fatigue ($p < 0.0001$) and tension ($p < 0.02$). Following 1 night of recovery sleep, *both* groups' mood disturbance scores returned to baseline values.

Effects on sleep apnea and nocturnal myoclonus

The extent of sleep-disordered breathing was minimal, with a median AHI of 1.2 (range 0–17.7) in the men and 0.4 in the women (range 0–15.7). Two men and one woman had an AHI of ≥ 5, and only one man had an AHI of >10. The AHI did not change significantly following sleep deprivation, but median duration of apnea became longer in the men, going from 15.8 (range 13.9–27) to 17.0 (range 16–29) s, $p < 0.05$ (Wilcoxon signed-rank test).

Men had nonsignificantly greater myoclonus indices (MI) than women: median values of 19.4 (range 0–179) versus 8.1 (range 2.0–47.6). MI was not affected by sleep deprivation in either group.

DISCUSSION

While healthy elderly adults, like young (12,13) and middle-aged adults (14–17), showed increased sleep continuity and more slow wave sleep on the first recovery night following sleep deprivation, the current data suggest that healthy elderly women show a more robust increase in sleep continuity and slow wave sleep (particularly stage 4 sleep) than do elderly men. Among women the increase in time spent asleep persists

to the second recovery night, but not among men. Three of 20 subjects, however, failed to show the expected increase in either sleep continuity or slow wave sleep: two men and one woman. REM latency decreased in response to sleep deprivation despite the increase in slow wave sleep, with REM latency reduction in women being delayed to the second recovery night. Indeed, five of 20 subjects (20%) had a total of seven SOREMPs on one or both recovery nights, and SOREMP-positive subjects were distinguished by having had higher percentage of stage 3 sleep at baseline and higher REM time during the first recovery night. Sleep apnea and nocturnal myoclonus indices were not significantly affected by 36 h of sleep deprivation, except for a modest increase in duration of apnea episode among elderly men.

Both groups showed increases of POMS Total Mood Disturbance following sleep deprivation, with elderly women tending to score higher than elderly men after 1 night of sleep deprivation. Both groups tolerated the procedure well, and both recovered completely on self-ratings of mood after 1 night of recovery sleep.

These data confirm the earlier reports of an increase in slow wave sleep and improved sleep continuity in the healthy elderly following either 38 (2) or 60 (3) h of sleep deprivation. Similarly, we replicated the observation of Carskadon and Dement (2) of a prolongation in apnea duration, albeit only in men, not in women. Like Bonnet and Rosa (3), but unlike Carskadon and Dement (2), we observed a decrease in REM latency after sleep deprivation, with 20% of subjects having a SOREMP on 7 of 40 (17.5%) recovery nights. SOREMPs are distinctly unusual during the baseline or unchallenged sleep of healthy elderly persons, but are very common among elderly patients with depression (18). Our observation of differentially greater stage 4 sleep in women, both at baseline and during recovery sleep, is consistent with the combined observations of Bonnet and Rosa (3) and Carskadon and Dement (2), and underscores the importance of gender in determining late-life sleep structure. The observation suggests that the ability of women to have slow wave sleep and long uninterrupted sleep is greater than that of men, during recovery from sleep deprivation as well as during baseline sleep.

Previous studies in younger adults (12–17) have suggested that the first recovery night is characterized specifically by a decrease in sleep latency and wake time and by an increase in slow wave sleep, while later recovery nights show an increase in REM sleep beyond baseline levels. REM sleep changes in the elderly appear to be different (or at least more complex) than those seen in younger subjects who undergo sleep deprivation. Thus, in the current sample, REM latency decreased during recovery sleep (while it increased during recovery in the young adult men of Bonnet and Rosa). Similarly, whole-night REM time increased on the first recovery night; there was no delay till the second recovery night, as has been reported in younger adults. However, in contrast to whole-night REM time, the first REM period duration ("early REM percent") did decrease during the first recovery night, compared with baseline. Moreover, whole-night REM density also decreased during the first recovery night.

Vogel has previously suggested that increasing age mediates increasing REM sleep disinhibition (19), a suggestion supported by the observations of Hyashi and Endo (20) and of Reynolds et al. (5) of a shift of REM sleep to earlier times of the night in the healthy elderly. The current data appear to support the concept of an age-mediated increase in REM disinhibition following the stress of sleep deprivation, as evidenced specifically by the decrease in REM latency, the occurrence of SOREMPs, and the

increase in REM time on the first recovery night. That support must be qualified, however, by the decrease in early REM time and whole-night eye-movement density of the first recovery night. Perhaps, as suggested by Borbely (21), the first recovery night increase in slow wave sleep inhibits the robustness of early REM sleep and diminishes phasic REM activity for the night as a whole.

The functional significance of response to SD in the elderly is not well understood. For example, do elderly men and women show differential performance decrements in response to sleep deprivation? Is the apparent increased mood disturbance in elderly women following sleep deprivation related to the observation of greater vulnerability to depression among women generally, or to the higher incidence of sleep complaints in elderly women? While we had originally speculated that men might be more susceptible than women to the mood-disturbing effects of sleep deprivation, our data suggested the opposite effect. If one assumes that sleep parameters are important for mood (and vice versa), then sleep deprivation would be a more "invasive" intervention for women (who sleep better in baseline conditions) than for men and thus would be a more incisive challenge to mood regulation. Such questions merit further investigation by researchers in sleep and aging.

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REFERENCES

1. Miles LL, Dement WC. Sleep and aging. *Sleep* 1980;3:119–220.
2. Carskadon MA, Dement WC. Sleep loss in elderly volunteers. *Sleep* 1985;8:207–21.
3. Bonnet MH, Rosa RR. Sleep and performance in young adults and older normals and insomniacs during acute sleep loss and recovery. *Sleep Res* 1985;14:69.
4. Williams RL, Karacan I, Hursch CJ. *Electroencephalography (EEG) of human sleep: clinical applications*. New York: John Wiley & Sons, 1974.
5. Reynolds CF, Kupfer DJ, Taska LS, Hoch CC, Sewitch DE, Spiker DJ. Sleep of healthy seniors: a revisit. *Sleep* 1985;8:20–9.
6. Folstein MF, Folstein SE, McHugh PR. "Mini-mental state"—a practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res* 1975;11:189–98.
7. Hamilton M. Development of a rating scale for primary depressive illness. *Br J Soc Clin Psychol* 1967;6:278–96.
8. Rosen WG, Terry RD, Fuld PA, Katzman R, Peck A. Pathological verification of ischemia score in differentiation of dementias. *Ann Neurol* 1980;7:486–8.
9. Covi L, Lipman RS, Frighi L, Rawlings P. Transcultural comparability of outpatient self-ratings: II. The profile of mood states (POMS). *Drugs Exp Clin Res* 1977;1:141–51.
10. Guilleminault C. Sleep and breathing. In: Guilleminault C, ed. *Sleeping and waking disorders: indications and techniques*. Menlo Park, California: Addison-Wesley, 1981:155–82.
11. Smallwood RG, Vitiello MV, Giblin EC, Prinz PN. Sleep apnea: relationship to age, sex and Alzheimer's dementia. *Sleep* 1983;6:16–22.
12. Coleman RM. Periodic movements in sleep (nocturnal myoclonus) and restless legs syndrome. In: Guilleminault C, ed. *Sleeping and waking disorders: indications and techniques*. Menlo Park, California: Addison-Wesley, 1981:265–96.
13. Kollar CJ, Pasnau RO, Rubin RT, Naitoh P, Slater GG, Kales A. Psychological, psychophysiological and biochemical correlates of prolonged sleep deprivation. *Am J Psychiatry* 1969;126:488–97.
14. Johnson LC, MacLeod WL. Sleep and awake behavior during gradual sleep reduction. *Percept Mot Skills* 1973;36:87–97.
15. Webb WB. Sleep stage responses of older and younger subjects to sleep deprivation. *Electroencephalogr Clin Neurophysiol* 1981;52:368–71.
16. Brezinova V, Hort V, Vojtechovsky M. Sleep deprivation with respect to age: an EEG study. *Activitas Neurosa Superior* 1969;11:182–7.

17. Froberg J, Karlsson CG, Levi L, Lidberg L. Circadian variations in performance, psychological ratings, catecholamine excretion, and urine flow during prolonged sleep deprivation. In: Colquhon WP, ed. *Aspects of human efficiency*. London: English Universities Press, 1972:247–60.
18. Reynolds CF, Kupfer DJ, Taska LS. EEG sleep in healthy elderly, depressed, and demented subjects. *Biol Psychiatry* 1985;20:431–42.
19. Vogel GW, Vogel F, McAbee RS, Thurmond AJ. Improvement of depression by REM sleep deprivation. *Arch Gen Psychiatry* 1980;37:247–53.
20. Hayashi Y, Endo S. All night sleep polygraphic recording of healthy aged persons: REM and slow wave sleep. *Sleep* 1982;5:277–83.
21. Borbely A. A two-process model of sleep regulation. *Hum Neurobiol* 1982;1:195–204.