

Sleep Fragmentation in the Elderly: Relationship to Daytime Sleep Tendency¹

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CARSKADON, M. A., E. D. BROWN AND W. C. DEMENT. *Sleep fragmentation in the elderly: Relationship to daytime sleep tendency.* NEUROBIOL. AGING 3(4) 321-327, 1982.—Sleep in the elderly is known to be disturbed, and many elderly persons also complain of daytime sleepiness. The present study assessed sleep and waking behavior in 12 male (aged 63 to 86) and 12 female (ages 63 to 82) subjects. Sleep stages, respiration, and movement were recorded at night, and daytime sleep tendency was measured using the Multiple Sleep Latency Test during a single 24-hour period. Daytime sleepiness did not correlate with total sleep time or any sleep stage, but was significantly correlated with measures of sleep fragmentation. The latter included transient arousals, a measure of <15-sec awakenings, and sleep-related respiration disturbance. These findings suggest that fragmented nocturnal sleep is a significant cause of reduced daytime well-being in elderly individuals. The continuity of both sleep and wakefulness appears to be disrupted with age. Experimental strategies for achieving a rational sleep hygiene are discussed.

Sleep stages Sleep fragmentation Aging

A NUMBER of reports suggest that elderly persons are less likely to stay awake throughout the day than are younger individuals, i.e., that the elderly tend to take a greater number of naps [15, 26, 27, 32, 35]. This increased tendency to sleep during the day has been attributed to a return to the polycyclicality of infant sleep [23], to "sheer boredom" [32], or to "an attempt to compensate for impaired sleep" [21]. The latter explanation alludes to the findings of many studies—both questionnaire surveys and sleep laboratory evaluations—that have linked disturbed sleep and aging. It is generally accepted, for example, that the nocturnal sleep of elderly individuals is characterized by a number of changes from the sleep of younger adults: decreased amount of sleep, increased number and length of intrasleep arousals, increased time in stage 1 sleep, and decreases in slow wave sleep stages [19]. These changes are usually assumed to indicate a deterioration rather than a maturation of sleep processes.

Few studies have attempted to examine directly the relationships between nocturnal sleep disturbance and daytime well being in older humans. At least one underlying causal factor—disturbed breathing during sleep—has been associated with both disturbed nocturnal sleep in the elderly and an increase in daytime sleepiness [11]. In the present analysis, we have examined other features of nocturnal sleep in older subjects that may contribute to changes in daytime somnolence.

Before describing this study, however, we will introduce our concept and measurement of daytime sleepiness. We define sleepiness operationally as the tendency to fall asleep. At certain times and under certain circumstances a high sleep tendency is appropriate. Thus, for example, one ex-

pects and desires to be sleepy at bedtime and through the night. Conversely, one generally wishes to be alert—with a low sleep tendency—in the daytime.

A precise measure of the tendency to fall asleep is possible using standard sleep laboratory techniques. The measure, called the Multiple Sleep Latency Test, simply involves determining the time it takes to fall asleep five or six times across a single day. Table 1 lists standard features of the Multiple Sleep Latency Test. It should be noted that the standard testing interval is two hours, and the maximum length of each test is 20 minutes. Furthermore, tests are ended at the unambiguous onset of sleep—thus they are not naps. The setting for the Multiple Sleep Latency Test is a bed in a quiet, darkened room.

We feel that this test is a direct measure of the underlying or physiological tendency to fall asleep, which differs from the manifest—that is the behavioral or subjective—sleep tendency. In other words, a person may fall asleep very quickly on the Multiple Sleep Latency Test (high physiological sleep tendency) yet appear and feel relatively alert when not in the testing situation. Many factors can apparently mask a high physiological sleep tendency. Activity is the most obvious; motivation, discomfort, stimulation, anxiety, and so forth are also important. On the other hand, factors that are generally associated with sleepiness—a boring lecture, a warm room, a heavy meal, alcohol, a long automobile drive—may not actually cause sleepiness, but may simply permit the behavioral manifestation of a high physiological sleep tendency.

We have used the Multiple Sleep Latency Test to measure physiological sleep tendency in a number of groups

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TABLE 1
MULTIPLE SLEEP LATENCY TEST

Measures latency to sleep onset
Measurements taken five or six times a day
Tests performed at two-hour intervals
Maximum duration each test = 20 minutes
Subjects lie in bed in quiet, dark room
Instructions: close eyes, lie quietly, try to sleep
Long sleep latencies/low sleep tendency (low sleepiness)
Short sleep latencies/high sleep tendency (high sleepiness)

under several conditions [2-11, 13, 24]. We have found that the single group with the most consistent pattern of alertness across the day is the 10-, 11-, and 12-year-old prepubescent adolescent [9]. Figure 1 illustrates schematically the optimal sleepiness/alertness pattern that seems to characterize this group. These children sleep through the night for about nine hours; when they awake, they are active, vital, and alert all day long. Their physiological sleep tendency is essentially zero throughout the day.

A consistent finding in older persons is a much different pattern of physiological sleep tendency [11]. Figure 2 is a schematic illustration of the typical pattern of sleepiness/alertness in older persons. In the elderly, sleep at night is typically disturbed, and there is a clear tendency for increased physiological sleepiness during the day. Of course, there are individual variations, but the midafternoon bout of increased sleepiness, for example, seems nearly universal.

One additional feature of the Multiple Sleep Latency Test is its sensitivity to changes in the amount of sleep at night. Figure 3 illustrates average Multiple Sleep Latency Test profiles in young adults recorded under several schedules of nocturnal sleep. As this figure shows, daytime latencies to sleep onset decline (sleep tendency increases) with reduced nocturnal sleep.

In the present study, we have used the Multiple Sleep Latency Test in combination with nocturnal sleep recordings to assess nocturnal factors that may affect daytime sleepiness.

METHOD

Subjects

Subjects were twenty-four elderly volunteers who agreed to live in the sleep laboratory and undergo testing for 48 hours. The study was approved by the Stanford University Committee for Protection of Human Subjects; volunteers signed statements of informed consent; and modest compensation was paid to each volunteer. The subjects were recruited from local senior citizen's recreational centers and church groups, and screened by a sleep habits questionnaire and a physical examination. Complaints about nocturnal sleep or daytime sleepiness were not reported by volunteers included in the study. All were in reasonably good health and fully ambulatory. (Details of physical examinations are reported more fully in a previous report [11]). The subjects included 12 women (ages 65 to 82, mean age=73.4) and 12 men (ages 63 to 86, mean age=72.3). All agreed to refrain from ingesting alcohol or caffeinated beverages during the study.

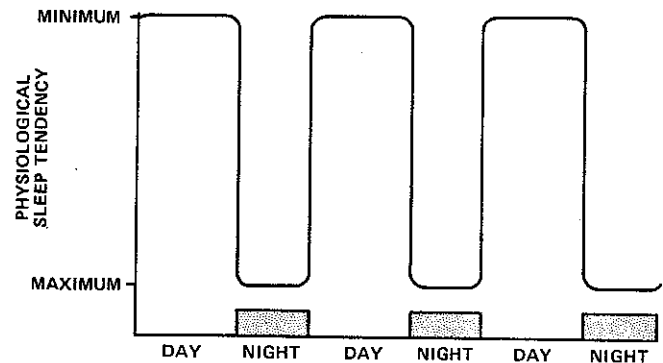


FIG. 1. This schematic illustration shows the ideal synchrony of sleep and wakefulness in prepubertal adolescents. They sleep well and soundly at night and are wide awake all day long.

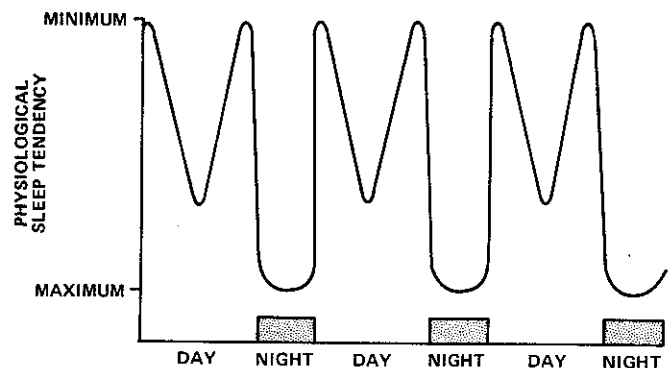


FIG. 2. Physiological sleep tendency in adults is very different from children, as illustrated in this figure. Older adults tend to have fragmented nocturnal sleep and cannot maintain full alertness throughout the day.

Subjects participated in the study in groups of four or five who came to the sleep laboratory for 48 hours. Sleep was recorded for two nights from 2200 to 0800, and sleep tendency was measured by the Multiple Sleep Latency Test on the subsequent days. Sleep recordings on both nights included and standard measures of electroencephalogram (EEG), electro-oculogram (EOG), and electromyogram (EMG) [22]. On one night, respiration was recorded using nasal thermistors and abdominal or thoracic mercury-filled strain gauges. Ear oximetry, using the Hewlett-Packard ear oximeter, was recorded in eight women. Anterior tibialis EMG was recorded in the men and in four women on the same night as respiratory variables. Sleep stages were scored according to standard criteria [22] in 30-second epochs. Respiration during sleep was evaluated for apneas (cessation of airflow >10 seconds) and reduced amplitude breathing (decline of respiration amplitude to <50% lasting >10 seconds with return to full amplitude accompanied by EEG arousal). In addition to the standard sleep stage scoring, sub-epochal transient arousals were also tabulated. Transient arousals

were defined as any clearly visible EEG arousal (usually alpha rhythm) lasting two seconds or longer, but not associated with any stage or state change in the epoch scoring. These brief arousals were sometimes, but not always, associated with a body movement or respiration event.

The following nocturnal sleep parameters were evaluated:

Sleep latency. Minutes from lights out to the first three consecutive epochs of Stage 1 sleep or the first epoch of Stage 2.

Sleep period time. Minutes from sleep onset to the last epoch of sleep, including all intervening wakefulness.

Wakefulness after sleep onset. Minutes of wakefulness within the sleep period.

Wakefulness after final arousal. Minutes of wakefulness occurring after the last epoch of sleep and before the end of the night (0800).

Sleep efficiency index. Total sleep time divided by the total time in bed (2200 to 0800).

Total sleep time. Minutes of all sleep stages.

Stage 1, 2, 3, 4, and REM time. Minutes of each sleep stage scored using standard criteria.

Sleep stage percentages. Percentage of each sleep stage calculated with sleep period time as the denominator.

Number of body movements. Movement events during sleep lasting 0.5 seconds or longer.

Stage 1 shifts. Number of times Stage 1 sleep was entered from another sleep stage.

Wake shifts. Number of times wakefulness was entered from a sleep stage.

Transient arousals. Number of EEG arousal events >2 seconds but not associated with stage or state change.

Transient arousal index. Number of transient arousals per hour of sleep.

Respiration events. Number of apneas and reduced amplitude breathing during sleep.

Respiration disturbance index. Number of apneas and reduced amplitude breathing per hour of sleep.

In the present study, the Multiple Sleep Latency Test was administered six times each day at two-hour intervals from 0930. Subjects were asked to lie quietly in bed, close their eyes, and try to fall asleep. The subjects were tested in individual bedrooms with lights off and the door closed. EEG, EOG, and EMG were monitored continuously for 20 minutes or until three consecutive 30-second epochs of sleep were observed, at which time the test was stopped. The score on the test was the elapsed time from lights out until the first epoch of sleep, or 20 minutes if no sleep occurred. A daily score was obtained from each subject by averaging the six individual test scores. An average daily score of less than five minutes has been seen in patient groups considered to be pathologically sleepy [13, 16, 24, 28].

In order to include the important respiration event data, we have analyzed data from the night on which respiration was recorded and sleep latency tests from the subsequent day. Thus the data included the first 24 hours in 11 subjects and the second 24 hours in 13 subjects. Examination of the data showed that only one parameter (Stage 3 sleep time) was consistently different between the respiration and non-respiration nights. When data from the first and second nights were compared, only the REM sleep measures showed a consistent "first-night effect."

The Pearson product-moment computation was used to obtain correlation coefficients. Intragroup comparisons were obtained using *t*-tests. A 0.05 two-tail significance level was used throughout.

TABLE 2
SLEEP CHARACTERISTICS IN 24 ELDERLY SUBJECTS

Parameter	Mean	Standard Deviation	Range
Nocturnal Sleep Latency	19	19	2-98
Sleep Period Time	552	39	463-558
Wakefulness After Sleep Onset	126	68	25-265
Wakefulness After Final Arousal	22	30	0-99
Sleep Efficiency Index	0.72	0.12	0.43-0.90
Total Sleep Time	426	75	250-534
Stage 1 Time	100	35	34-169
Stage 2 Time	218	50	121-309
Stage 3 Time	22	16	0-47
Stage 4 Time*	15	19	0-57
REM Time	72	32	11-147
Stage 1%	18.0	5.8	9.1-30.1
Stage 2%	39.4	8.9	23.7-55.0
Stage 3%	5.3	6.7	0-10.2
Stage 4%	2.7	3.5	0-11.4
REM %	13.1	5.8	1.9-25.1
Number of Body Movements	73	50	27-240
Stage 1 Shifts	75	26	24-130
Wake Shifts	41	20	13-88
Transient Arousals	160	104	24-373
Transient Arousal Index	23	15	3-54
Respiration Events	58	69	0-215
Respiration Disturbance Index	8	11	0-35

*Significant male/female difference was present. Mean Stage 4 time for men = 3 minutes; for women = 27 minutes; $p < 0.01$.

RESULTS

Table 2 lists the average nocturnal sleep characteristics of the 24 subjects for the respiration night recording. Although the sleep recording time was constant (2200 to 0800), a wide range of values was seen for virtually all sleep parameters. For example, total sleep time ranged from about four hours to nearly nine hours. Transient arousals ranged from 24 to 273. When transient arousals were equalized for sleep time (transient arousal index), the range was three to 54 per hour. On only one parameter (stage 4 time) was there a statistically significant difference between male and female subjects. Finally, the average daily Multiple Sleep Latency Test score for the group was 12.2 minutes (standard deviation=4.6; range 5.2 to 17.9), with no significant male-female difference.

Table 3 shows the correlation coefficients describing the relationship of daily multiple sleep latency test scores with the nocturnal sleep parameters. Most nocturnal variables were unrelated to sleep latencies across this group of subjects. There was a nonsignificant trend ($p < 0.10$) for sleep latency scores to decrease with increased shifts to wakefulness. The number of transient arousals and respiration events were the only nocturnal variables to achieve statistically significant correlation with Multiple Sleep Latency Test scores. In both cases, increased numbers of events were associated with declining sleep latency test scores (in-

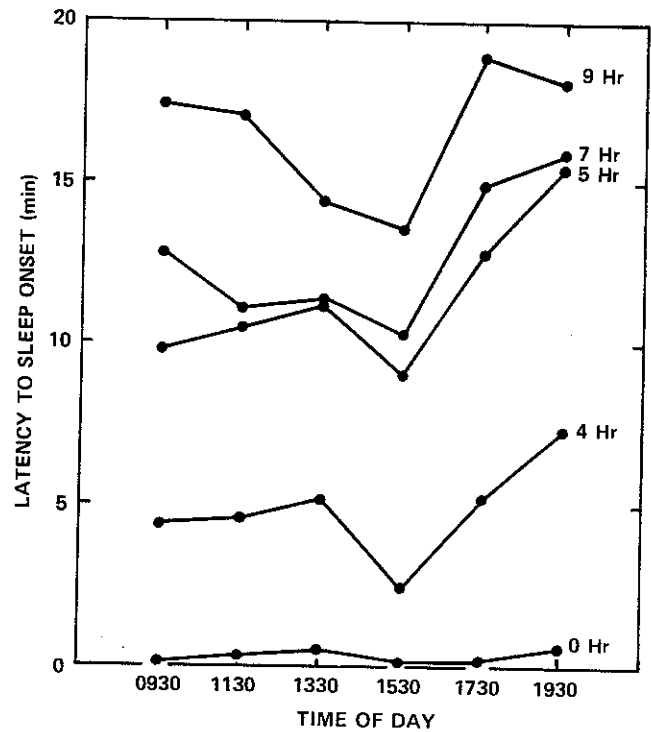
TABLE 3
CORRELATION OF SLEEP PARAMETERS WITH
DAYTIME SLEEP TENDENCY
(DAILY MULTIPLE SLEEP LATENCY TEST SCORE)

Sleep Parameter	Correlation Coefficient	$p <$
Nocturnal Sleep Latency	.198	ns
Sleep Period Time	-.273	ns
Wakefulness After Sleep Onset	-.002	ns
Wakefulness After Final Arousal	.170	ns
Sleep Efficiency Index	-.125	ns
Total Sleep Time	-.140	ns
Stage 1 Time	-.270	ns
Stage 2 Time	-.019	ns
Stage 3 Time	-.025	ns
Stage 4 Time	-.114	ns
REM Time	.212	ns
Stage 1%	-.196	ns
Stage 2%	.082	ns
Stage 3%	-.029	ns
Stage 4%	-.126	ns
REM %	.247	ns
Number of Body Movements	-.199	ns
Stage 1 Shifts	-.309	ns
Wake Shifts	-.386	0.10
Transient Arousals	-.474	0.02
Transient Arousal Index	-.418	0.05
Respiration Events	-.491	0.02
Respiration Disturbance Index	-.407	0.05

creasing sleep tendency). Although there was a significant relationship between the number of transient arousals and the number of respiratory events across subjects ($r=.476$; $p<0.02$), several subjects with few respiratory events showed numerous transient arousals. Figure 4 shows the sleep pattern in one subject in whom transient arousals and respiratory events were closely related. The mean daily sleep latency test score in this subject was 5.8 minutes. Figure 5 illustrates the sleep pattern in another subject who had few apneas and several hundred transient arousals. Mean daily sleep latency test score for this subject was 6.8 minutes.

DISCUSSION

The nocturnal sleep results generally confirmed previously reported findings as summarized by Miles and Dement in their recent review [19]. The present subjects, however, showed greater sleep fragmentation than most studies have reported. For example, a comparison of our subjects in the 70-79 year old range with those examined by Williams and his colleagues [34] showed greater stage 1 shifts and larger amounts of stage 1 sleep in our group [2]. These differences appear to result largely from interlaboratory differences in stage scoring criteria. Specifically, our 30-second epoch stage scoring significantly increased the number of opportunities to record stage changes as compared to the 60-second scoring epoch of the Williams group [34]. Further, the additional recording devices used to monitor respiration, and especially the ear oximeter, may have led to greater sleep disruption in our subjects.



* SECOND DAY EACH CONDITION

FIG. 3. Multiple Sleep Latency Test profiles of young adults with various sleep schedules are illustrated in this figure. Each profile is an average from the second day at the given sleep condition: 9 hours ($N=20$); 7 hours ($N=16$); 5 hours ($N=10$); 4 hours ($N=13$); and no sleep ($N=6$). Several individual subjects were recorded at more than one condition, but no subject was recorded under all five conditions.

In our data, the continuity of sleep—as reflected in shifts to wakefulness, transient arousals, and respiration events—appeared to be of greater significance to daytime well being than was total sleep time *per se* across subjects. Thus for example, one of the highest daily sleep latency test scores (17.8 minutes) was seen in a 68-year-old woman who slept only 354 minutes, but who had no respiration events and only 80 transient arousals. One of the lowest daily mean sleep latency test scores (6.3 minutes) was observed in a 79-year-old man who slept nearly 8 hours (479 minutes), but had 163 respiration events and 214 transient arousals. In the latter case, it is not clear that extending nocturnal sleep time would (1) be possible or (2) markedly reduce daytime sleep tendency. Roth and his colleagues [25] have found a similar relationship between sleep disturbance and sleep tendency during the day in middle-aged subjects. The data from the present study do not, however, directly address the issue of total sleep time within subjects. That is, our data from young adult subjects, as noted in the introduction, show that total sleep time is a significant factor in determining daytime sleep tendency. Is this also true in elderly individuals? In a separate study of elderly subjects that included five consecutive days, we found that sleep time and daytime sleepiness tended to correlate within subjects [10]. Thus it appears that both factors—total sleep time and sleep fragmentation—are related to daytime well being in the elderly.

ing in bed as long as 12 hours a day [32]. The effectiveness of this strategy is doubtful, and it may in fact be harmful if it disrupts the normal circadian rhythm of sleep and wakefulness.

If a major problem is the stable consolidation of state over time, what approach might improve sleep and wakefulness in the elderly? One of the first issues that must be addressed is whether the sleep system of older persons is responsive to manipulations that normally potentiate sleep in younger individuals. Thus, for example, studies of acute sleep restriction [14, 18, 30, 31] and sleep deprivation [4, 17, 20, 33] in young adults have shown that slow wave sleep is increased, nocturnal arousals are fewer, stage 1 sleep is reduced, and the NREM-REM cycle remains intact. Webb [29] has recently compared the sleep stage response of middle-aged subjects (ages 40–50 years) to that of young adults on the day following two nights of sleep loss. Although sleep measures were made in the daytime, Webb found that the sleep system of the older subjects was responsive to sleep loss, though to a somewhat lesser degree than the young adults.

Such a "stress test" should be repeated in elderly subjects to determine the responsiveness of the sleep system. If recovery sleep after sleep loss is less fragmented, it is possible that reducing the amount of time in bed—not increasing it—might be a workable approach to improving sleep in the elderly. Thus instead of staying in bed 9, 10, 11 or 12 hours, the older person might sleep "better" if he restricted sleep to six or seven hours at night.

Of course, this approach does not address the issue of daytime alertness. If state consolidation is a problem, however, it is possible that reducing the need to remain awake for the entire day by taking a daytime nap, might improve

waking function overall. We hypothesize, therefore, that limiting nocturnal sleep and introducing a midday nap might improve both sleep and wakefulness by reducing the constraints on both systems. Such an approach would be fairly simple to test.

Other factors that may affect sleep and wakefulness in the elderly person should also be evaluated. For example, exercise, diet, and caffeine and alcohol intake may contribute to the sleep problem or its solution. In addition, compounds that are commonly used to treat medical or psychiatric conditions in older persons may further compromise sleep and wakefulness. Our subjects, for the most part, were not taking medications. Current techniques and approaches to sleep disorders and sleep function could be employed to attain a rational sleep hygiene for the older person.

When fragmented sleep results from other underlying causes, the problem becomes more complex and may be less amenable to such behavioral manipulation. It seems quite likely that disturbed breathing during sleep is causally related to sleep disruption in a significant percentage of elderly persons [1,6]. Other physiologic anomalies, for example movement disorders such as nocturnal myoclonus, may account for sleep disruption in certain of the elderly whose respiration during sleep is normal [12]. These sleep disturbances are almost certainly related to increased daytime sleepiness in the elderly. Furthermore, in the case of sleep-related breathing disturbances, interactions with sleeping pills and other central nervous system depressants may lead to serious, even fatal, consequences. (For further elaboration of this final point, please see the papers by Kripke *et al.* and Guilleminault *et al.*, this issue.)

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