Sleep Extension in Sleepy and Alert Normals

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Summary: Twenty-four healthy, young (21-35 years old) men with no complaints of daytime sleepiness, no habitual napping, and polysomnographically verified normal nocturnal sleep extended their time in bed (TIB) to 10 h for 6 consecutive nights to assess the effects of sleep extension on daytime sleepiness and performance. Twelve subjects had basal average daily sleep latencies of \( \leq 6 \) min on the Multiple Sleep Latency Test and 12 had latencies of \( \geq 16 \) min before TIB was extended. The sleep extension improved daytime sleepiness differentially in the two groups. The degree of improvement was greater in the sleepy subjects than the alert subjects and the pattern of improvement differed between the groups. Sleepy subjects showed an immediate and uniform increase in alertness, while alert subjects did not show improvements until late in the extension. However, sleepy subjects never achieved the baseline level of sleepiness/alertness seen in the alert subjects. Key Words: Multiple Sleep Latency Test—Sleep extension—Daytime sleepiness—Sleepy and alert subjects.

Survey and laboratory studies indicate that when given the opportunity (i.e., on weekends or in the laboratory without time cues), adults without sleep complaints and with objectively verified normal sleep will sleep beyond their habitual sleep times (1,2). Further, when sleep is experimentally restricted for a night, the amount of sleep obtained on a subsequent night is extended (3). Thus, Webb and Agnew (3) hypothesized that many healthy adults have an accumulated sleep debt that is a result of chronically insufficient sleep relative to sleep need. However, such data show merely that people can sleep more if given the opportunity. If sleep debt is a truly useful construct, some daytime benefits should be associated with additional sleep and the extent of the benefit should be associated with the degree of the sleep debt. Thus, one critical test of a hypothesized sleep debt is to assess the daytime effects of sleep extension.

Some studies of sleep extension suggest it is detrimental. These studies show increases in subjective measures of daytime sleepiness and reduced performance efficiency on vigilance and complex psychomotor tasks in healthy young adults who extended bedtimes from the usual 8 to 10 or 11 h for 1-2 nights (4-7). However, studies

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that have used the Multiple Sleep Latency Test (MSLT) to assess the effects of sleep extension found improvements in daytime sleepiness/alertness after bedtime was increased to 10 h for 4 consecutive nights (8,9). To determine whether or not the apparent detrimental effects of sleep extension in earlier studies were the result of "oversleeping," a recent study assessed the differential effect of sleep extension after restricted versus normal sleep (10). Bedtime was increased to 11 h for 1 night following either a nominal 8.5-h or a restricted 6.5-h bedtime schedule on 1 previous night. Again, mean sleep latency was increased (i.e., sleepiness was reduced) and performance measures were improved following extended sleep. These effects were observed regardless of the amount of sleep the night prior to the sleep extension.

The failure to find differential benefits of sleep extension in the above study may be due to the acute nature of the sleepiness in the restricted 6.5-h bedtime schedule. It has been shown that a modest restriction of sleep time will accumulate over successive nights to increase one's basal level of sleepiness (11). A recent report described the basal daytime sleepiness of a large sample of healthy young adults with no sleep-wake complaints (12). Average daily sleep latency within the sample ranged from 2 to 20 min on the MSLT with ~25% of subjects having unusually short or long sleep latencies.

The present study assessed whether or not differences in basal level of sleepiness may interact with the effect of extending sleep time. Subjects were selected for their unusual sleepiness (MSLT average daily sleep latency of ≤6 min) or alertness (MSLT average daily sleep latency of ≥16 min) and their bedtime was extended to 10 h for 6 consecutive nights. No control groups were employed in this study because a specific prediction regarding a differential benefit of sleep extension was being made: Sleepy subjects would benefit and alert subjects would show little or no benefit. Further, several studies have shown there is little change in MSLT scores over consecutive days with a constant 8-h sleep schedule each night (8,13).

**METHODS**

**Subjects**

The subjects were 24 healthy, nonsmoking men aged 21–35 years with no complaints of daytime sleepiness, no habitual napping, and normal nocturnal sleep, all determined by the screening described below. They signed informed consents and were paid for participation.

**Procedures**

Subjects were recruited from an inventory of subjects who had previously been screened with an MSLT for participation in various earlier studies. In a telephone interview the subjects reported their usual nocturnal bedtimes, risetimes, and total sleep times (see Table 1). No exclusions were made based on reported sleep times and bedtimes or risetimes. But subjects with highly irregular sleep schedules (i.e., >2-h variations in bedtimes) were excluded. Subjects also were excluded if they answered yes to questions regarding difficulty sleeping at night, sleepiness during the daytime, and daytime napping. They reported no history of alcohol or drug abuse and no current drug use and drank a weekly average of one to seven alcoholic beverages. Each subject then reported to the sleep center and underwent a medical history and physical examination including blood and urine analyses to verify normal health and the absence of recent drug use.
Subjects then spent 1 8-h night and the following day in the sleep laboratory to reconfirm their level of daytime sleepiness. They reported 1.5 h before their usual bedtime, with 2230 h being the latest arrival time, ensuring a 0000-h bedtime and 0800-h risetime. These limits were established to maintain a minimum of 2 h of wakefulness before the first latency test of the MSLT. After arriving at the laboratory, each subject had electrodes attached for a standard polysomnogram, which included bilateral electrooculogram (EOG), submental and tibialis electromyograms (EMG), central (C3/C4) and occipital (Oz) electroencephalograms (EEG), electrocardiograms (V5), and nasooral airflow recorded with thermistors (14). Subjects then went to bed at the established bedtime, remaining in bed for 8 h while the polysomnogram was collected.

Upon arising in the morning, subjects bathed and were allowed to eat a light breakfast that included a roll and a noncaffeinated beverage of choice. Electrodes were checked and replaced if necessary to prepare for the MSLT. The MSLT was conducted at 1000, 1200, 1400, and 1600 h 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 min of unambiguous stage 1 sleep, the first signs of stage 2 or REM sleep, or 20 min of continuous wake according to standard sleep stage criteria (14).

At 1030 h subjects were trained on a 20-min divided attention task. The task involves tracking, with a joystick, a moving target appearing on a video monitor, while responding on a key to the appearance of a target in the periphery or the center of the video screen. Average tracking deviations and reaction times to peripheral and central targets were recorded. At 1430 h subjects were trained on a 40-min auditory vigilance task. This task consisted of detecting long tones occurring randomly in a continuous series of shorter tones. Reaction times (in four blocks of 10 min), misses, and false-positives were recorded.

After the screening subjects with average sleep latencies on the MSLT of <=6 or >=16 min and whose nocturnal polysomnogram showed no clinical abnormalities were asked to participate. The subjects qualifying underwent 6 consecutive nights of sleep extension (10 h) preceded by a baseline night (8 h). Bedtime was 2330-0730 h on baseline and 2130-0730 h on extension nights. Each subject slept in the laboratory (reporting 30 min before the appropriate bedtime) on the baseline night and on extension nights 1, 3, and 6. The following day MSLT and performance testing were conducted as described previously. On nonlaboratory nights subjects slept at home, maintaining the appropriate sleep schedule and their usual daytime activities.

Sleep was monitored by actigraph on all study nights, including the baseline night and extension nights 1, 3, and 6 when the subjects slept at the laboratory. The actigraph has...
been shown to predict polysomnographically determined sleep or wake with 93% accuracy (16). It also has been shown to be sensitive to the effects of restriction and extension of time in bed (TIB) (17). Actigraphic monitoring of sleep was employed in this phase of the study because the primary purpose of the study was to assess changes in daytime function associated with extended TIB and not to document changes in sleep occurring during extension.

Latency to the first epoch of stage 1 sleep was scored for each latency test according to standard sleep stage scoring criteria (14). Nocturnal sleep efficiency was determined as derived from the actigraph recordings by a previously described algorithm (16). Sleep latency, sleep efficiency, and the previously described measures of performance were submitted to mixed design analyses with group (sleepy or alert) the between-subjects factor and days (baseline, extension 1, 3, and 6) the within-subjects factor using the general linear model analysis (SAS Institute). Conservative probability levels as corrected by the Greenhouse-Geisser procedure are reported for the within-subjects factor. Post hoc comparisons were made following the Duncan procedure.

RESULTS

Self-reported sleep habits

The usual sleep habits of the sleepy and alert subjects are presented in Table 1. The two groups do not differ significantly in any of their self-reported sleep habits, although there are two notable trends in the data. The alert subjects tended to report longer sleep times and later risetimes than the sleepy subjects. Daily variation in sleep schedule was not different between the groups, but then subjects with a >2-h variation were excluded from participation in this study.

Daytime sleepiness/alertness

Average daily sleep latency on baseline and extension days 1, 3, and 6 for the sleepy and alert subjects is plotted in Fig. 1. Sleep latency increased significantly from baseline through extension day 6 in both groups ($F = 8.57, p < 0.001$). The groups differed initially in average daily sleep latency by design, but those significant group differences remained over the whole extension period ($F = 78.70, p < 0.001$). Post hoc comparisons ($p < 0.01$) between groups showed differences on baseline and each extension day (1, 3, and 6). Sleepy subjects never attained the baseline level of the alert subjects. However, the increase in sleep latency in sleepy subjects never reached an asymptote (see Fig. 1).

The pattern of increase in average daily sleep latency differed between groups as indicated by a significant groups-by-extension interaction ($F = 2.94, p < 0.04$). The slope of the function relating sleep latency to days of extension was 0.22 for the sleepy group and 0.10 for the alert group. The exact nature of the differential group increase in sleep latency was assessed by examining the simple effects of sleep extension within the sleepy group and within the alert group. Sleepy subjects showed a significant increase in latency ($F = 9.30, p < 0.002$) as found on the omnibus analysis. Post hoc tests among days revealed no difference from baseline to extension day 1, but differences between baseline and extension day 3 ($p < 0.01$) and 6 ($p < 0.007$). As well, extension day 1 differed from extension day 3 and 6 ($p < 0.01$ and 0.04, respectively). The alert subjects also showed a significant increase in latency ($F = 4.17, p < 0.02$). But the post hoc tests indicated no baseline to extension day 1 or 3 differences. Dif-
FIG. 1. Mean sleep latency (±SD) for sleepy (solid circles) and alert subjects (open circles) on the baseline day and extension days 1, 3, and 6. On baseline the time in bed (TIB) was 8 h and on extension days TIB was 10 h. Analyses yielded group effects, p < 0.001; extension effects, p < 0.001; and group-by-extension interaction, p < 0.04. Sleepy subjects (●) showed immediate extension effects and alert subjects (○) showed effects later.

ferences occurred only between baseline and extension day 6 (p < 0.05). Similarly, extension day 1 did not differ from extension day 3 and only a day 1 to 6 difference was found (p < 0.001).

Test-by-test analyses of group and sleep extension effects produced results quite similar to those for average daily sleep latency with one notable exception. On the 1000-h test, group differences ($F = 68.68, p < 0.001$) and an extension effect ($F = 3.57, p < 0.02$) were observed. In addition, a significant group-by-extension interaction was found ($F = 5.69, p < 0.001$). The interaction is illustrated in Fig. 2. To explore the nature of the interaction, analyses of simple extension effects were conducted within the alert and sleepy groups. For alert subjects there were significant extension effects ($F = 3.93, p < 0.03$), but sleep latency initially declined with the sleep extension before finally increasing. Post hoc comparisons revealed a difference between baseline and extension day 3 (p < 0.02) and between extension days 1 vs. 6 and 3 vs. 6 (p < 0.04 and 0.02, respectively). In contrast, in sleepy subjects there were significant extension effects ($F = 6.56, p < 0.01$) and sleep latency increased over the 6 extension days. There were differences between baseline and extension days 1, 3, and 6 (p < 0.05, 0.01, and 0.001, respectively). Extension days did not differ one to the other.

Results of analyses of the other latency tests were quite similar to the data for average daily sleep latency. At 1200 h group differences were found ($F = 52.87, p < 0.001$), but the extension effect was not significant. Again on the 1400-h test, both group ($F = 41.67, p < 0.001$) and extension ($F = 2.89, p < 0.04$) differences were found. Finally, for 1600 h, both group ($F = 46.76, p < 0.001$) and extension ($F = 3.01, p < 0.04$) effects were found. No group-by-extension interactions were found on the test-by-test analyses for the 1200-, 1400-, and 1600-h tests.
FIG. 2. Sleep latency (means ± SD) on the first sleep latency test at 1000 h for sleepy (solid triangles) and alert subjects (open triangles). Sleep latency is plotted for baseline when time in bed (TIB) was 8 h and for extension days 1, 3, and 6 when TIB was 10 h. Analyses yielded group effects, $p < 0.001$; extension effects, $p < 0.02$; and group-by-extension interaction, $p < 0.001$. Alert subjects ($\Delta$) showed an initial decline and then an increase and sleepy subjects ($\triangle$) showed a uniform increase.

Performance

The divided attention performance measures for each group are presented in Table 2. The data analyses indicated that central and peripheral reaction times were improved over the sleep extension period ($F = 4.95, p < 0.001; F = 4.02, p < 0.02$, respectively). The post hoc comparisons indicated baseline versus extension day 6 ($p < 0.001, 0.02$, respectively) differences on both measures and an extension day 1 vs. 6 difference in central reaction time ($p < 0.05$). No group differences were found on these two measures, but a group-by-extension interaction ($F = 2.91, p < 0.05$) was found on the central reaction time measure. Group differences were detected on extension day 1 ($p < 0.01$), but not baseline or extension days 3 and 6. On the measure of tracking (average deviations), no extension or interaction effects were found. However, group differences in average tracking were detected ($F = 4.60, p < 0.04$). Z scores, the combination of all three measures, showed an overall group difference ($F = 4.11, p < 0.05$) and a trend for extension effects.

Vigilance performance showed no group differences in reaction time or number of errors (misses and false-positives). Extension effects on reaction time were observed in the last 10 min of the task ($F = 2.90, p < 0.05$), with the post hoc analyses showing a baseline versus extension day 6 difference ($p < 0.03$). No interactions were found.

Nocturnal sleep efficiency

No overall group or extension effects in sleep efficiency were detected, but a significant interaction was found ($F = 3.74, p < 0.03$). The nocturnal sleep efficiencies are presented in Table 3. The sleepy group had a higher sleep efficiency than the alert group on the baseline (95.4 vs. 90.3%). This difference in sleep efficiency diminished after the
TABLE 2. Divided attention performance

<table>
<thead>
<tr>
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<th>Baseline</th>
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<th>6</th>
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<tr>
<td><strong>Central RT</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleepy</td>
<td>25.25</td>
<td>24.83</td>
<td>24.08</td>
<td>24.18</td>
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<tr>
<td>(3.36)</td>
<td>(3.43)</td>
<td>(3.16)</td>
<td>(3.76)</td>
<td></td>
</tr>
<tr>
<td>Alert</td>
<td>25.33</td>
<td>23.42</td>
<td>23.83</td>
<td>24.11</td>
</tr>
<tr>
<td>(4.12)</td>
<td>(2.43)</td>
<td>(3.16)</td>
<td>(3.76)</td>
<td></td>
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<tr>
<td><strong>Peripheral RT</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
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<td>23.58</td>
<td>22.75</td>
<td>23.09</td>
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<tr>
<td>(4.58)</td>
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<tr>
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<tr>
<td>(3.96)</td>
<td>(3.73)</td>
<td>(3.07)</td>
<td>(4.08)</td>
<td></td>
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<td><strong>Avg. tracking deviation</strong></td>
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<tr>
<td>Sleepy</td>
<td>95.75</td>
<td>96.92</td>
<td>88.25</td>
<td>89.10</td>
</tr>
<tr>
<td>(8.56)</td>
<td>(9.17)</td>
<td>(9.16)</td>
<td>(7.70)</td>
<td></td>
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<tr>
<td>Alert</td>
<td>78.58</td>
<td>78.00</td>
<td>78.42</td>
<td>75.22</td>
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<tr>
<td>(8.56)</td>
<td>(9.17)</td>
<td>(9.15)</td>
<td>(6.08)</td>
<td></td>
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<tr>
<td><strong>Div. atten. Z score</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleepy</td>
<td>1.06</td>
<td>0.99</td>
<td>-0.01</td>
<td>0.60</td>
</tr>
<tr>
<td>(2.47)</td>
<td>(1.84)</td>
<td>(1.83)</td>
<td>(2.81)</td>
<td></td>
</tr>
<tr>
<td>Alert</td>
<td>0.07</td>
<td>-0.95</td>
<td>-0.96</td>
<td>-0.96</td>
</tr>
<tr>
<td>(2.28)</td>
<td>(1.79)</td>
<td>(1.83)</td>
<td>(1.91)</td>
<td></td>
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Data are means (SD). Measures are expressed in arbitrary computer-generated units. For all measures lower scores indicate improved performance. RT, reaction time.

a Extension effects, p ≤ 0.02.

b Group effects, p ≤ 0.05.

first extension night and the groups were similar for the duration of the extension period.

DISCUSSION

This study found that extending TIB to 10 h for 6 consecutive nights improved daytime sleepiness/alertness differentially in sleepy versus alert subjects. As predicted, the alert subjects showed very small improvements in daytime sleepiness/alertness as compared to much larger improvements in sleepy subjects. The differential improvement may in part be due to a ceiling effect on the MSLT in the alert subjects. However, the differential benefits of sleep extension in the two groups were evident not only in the degree of improvement, but also in the time course. Specifically, the sleepy subjects showed greater as well as earlier improvements in daytime sleepiness/alertness. Further, the present results parallel findings regarding sleep extension in other studies

TABLE 3. Nocturnal sleep efficiency

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
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<th>3</th>
<th>6</th>
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</thead>
<tbody>
<tr>
<td><strong>Sleepy subjects</strong></td>
<td>95.4 (2.75)a</td>
<td>90.4 (3.96)</td>
<td>88.4 (5.35)</td>
<td>88.6 (3.38)</td>
</tr>
<tr>
<td><strong>Alert subjects</strong></td>
<td>90.3 (5.94)</td>
<td>89.4 (5.01)</td>
<td>90.2 (3.75)</td>
<td>87.7 (4.67)</td>
</tr>
</tbody>
</table>

Data are means (SD). Measure is minutes of sleep relative to minutes of bedtime determined by actigraph.

a Group-by-night interaction, p < 0.03; groups differed on baseline.
using the MSLT to assess daytime function (8,9). They further support the results of Carskadon et al. (10) in which sleep extension led to improved daytime function with or without a prior acute (1 night) restriction of sleep.

The performance results of the present study did not support the hypothesis of a differential improvement in sleepy versus alert subjects. Improvements were found on the divided attention and vigilance tasks over the 6 days of the study. However, the pattern and extent of improvement did not differ between sleepy and alert subjects. The one extension-by-group interaction that was found (on divided attention reaction time) should be interpreted as being spurious; the group difference occurred only on extension day 1. Given that the improvements in performance were uniform and that a nonextension group was not included in this study, the improvements in performance seen in each group could be due to practice effects. Practice effects could also have masked differential improvements between the groups and thus explain the absence of an interaction.

An interesting result of this study is that in the alert group the initial 3 nights of sleep extension produced a decreased sleep latency on the first latency test, 2 h after arising. On the other three latency tests (4, 6, and 8 h after arising) in the alert subjects, there typically was no change or a small improvement in sleep latency. The performance evaluations in the earlier Taub et al. (4–7) studies were generally conducted during the first 2 h after arising (starting 30 min after arising). Further, the sleep extension in those studies was acute, 1–2 nights. Consequently, the improvement in function that occurred after 3 nights of sleep extension in the alert subjects of this study would not have been observed because of the acute nature of the earlier studies. Thus, the use of unusually alert subjects combined with an acute sleep extension could account for the Taub et al. findings of detrimental effects associated with sleep extension.

The reason for this initial sleepiness (i.e., within the first 2 h after arising) in alert subjects is unclear at this time. A number of authors have described a "sleep inertia," a temporary loss of performance efficiency after awakening from sleep (18). Usually, it is thought that sleep inertia is short-lasting, enduring for 15–30 min. However, some studies have found sleep inertia for as long as 2 h. But those findings are under conditions of sleep deprivation and not sleep extension, and further not in alert subjects. Most importantly, the sleepy subjects of the present study did not show an increased sleepiness on the first latency test. It is unlikely that sleep inertia is the cause of the initial sleepiness of the alert subjects in the present study.

A circadian dysrhythmia resulting from the change in sleep schedule is another possible explanation. The question remains, however, as to why the alert subjects and not the sleepy subjects are sensitive to the dysrhythmia. It could be that the initial level of sleepiness in sleepy subjects is masking circadian effects, while those effects are not being masked in the alert subjects. In other words, there is some threshold level of alertness that must be attained before the circadian dysrhythmic effects can be detected. In sleepy subjects the circadian dysrhythmia is not important relative to the existing sleep debt, while in alert subjects the circadian dysrhythmia, in the absence of a chronic sleep debt, produces its effect. Notice that the alert subjects appear to adapt to the circadian insult as indicated by the increase in latency on the first latency test after night 3 of extension (see Fig. 2).

Another interesting finding of this study is that the sleepy subjects over the 6 nights of sleep extension never reached the baseline level of the alert subjects. Average daily sleep latency of the sleepy subjects after sleep extension did reach a level comparable
to the mean of average daily sleep latency found in a large sample of healthy normal young adults (12). In that sense one could say that the daytime sleepiness of these subjects was normalized. However, it remains to be determined whether additional sleep extension could have produced levels of alertness similar to those of the alert subjects.

Other variables, of course, may limit the extent to which sleep extension could increase the MSLT score of the sleepy subjects. The difference between the sleepy and alert subjects after the 6 nights of sleep extension could reflect trait differences in the ability to fall asleep and/or sleep need. Patients with insomnia have been shown to have unusually long latencies on the MSLT compared to a nonselected group of age-matched normals (19). Furthermore, the latencies of the insomnia patients were unrelated to their total sleep times, while the latencies (which were in the normal range) of the normals were related to sleep times. Studies are presently being conducted to further assess the differences between alert and sleepy adult normals without sleep complaints.

REFERENCES