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The Alerting Effects of Naps in Sleep-Deprived Subjects

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ABSTRACT

The effect of napping for varying durations after one night of sleep deprivation was examined. Sleep latency tests were used to determine levels of sleepiness/alertness at 2, 4, 6, and 8 hrs following a morning nap of 0, 15, 30, 60, or 120 min duration. Ten normal-sleeping, young adult volunteers spent two consecutive days and the intervening night in the sleep laboratory on each of five weeks. Baseline sleep latencies were recorded the first day, sleep was deprived that night, a nap was taken at 0900 hrs, and sleep latencies were again recorded on the second day. The naps had differential alerting effects related to their duration, but none of the naps returned mean sleep latency for the 8 hrs to its basal levels. Alertness increased with nap duration, reaching its highest level with a 60-min nap; the 120-min nap was no more alerting than the 60-min nap. During the second hour of the 120-min nap, sleep became more fragmented with more shifts to stage 1 sleep or wake. Increased alertness was not strongly related to the sleep stage composition of the naps, the best predictor being minutes of slow wave sleep. Increased alertness was not detected until the second latency test 4 hrs after napping.

DESCRIPTORS: MSLT, Excessive sleepiness, Naps, Sleep deprivation, Restoration of alertness.

Excessive daytime sleepiness (EDS) is a common problem. In otherwise healthy individuals it is associated with rotating sleep-wake schedules (e.g. shift workers), irregular sleep-wake schedules (e.g. hospital staff on call), and rapid travel across time zones (i.e. jet lag) (Nicholson et al., 1985; Seidel, Roth, Roehrs, Zorick, & Dement, 1984; Wittig, Zorick, Roehrs, Sicklesteel, & Roth, 1983). It also is a major and disabling symptom of various sleep-wake disorders (Zorick et al., 1982). A number of factors affect one's level of sleepiness/alertness, including pharmacological agents that stimulate or depress the central nervous system (CNS), pathologies of CNS sleep-wake mechanisms, circadian rhythms in sleepiness/alertness, the integrity of prior sleep (i.e. the presence of brief arousals fragmenting sleep), and the amount of prior sleep and wakefulness (Roth, Roehrs, & Zorick, 1982; Webb & Agnew, 1975). While our understanding of factors producing sleepiness is being clarified, reversal of sleepiness or recovery of alertness (sleepiness and alertness are reciprocals) is not well understood.

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Recovery of alertness compromised by sleep loss has received the most research attention and typically has been studied using performance and mood measures. In general, studies during or after sleep loss have shown that recovery naps reverse the disrupted performance and mood. But how specifically the recovery relates to (1) the nap duration, (2) the severity of the sleep loss, (3) the circadian phase timing of the nap, (4) the sleep stage composition of the nap, and (5) the "sleep inertia" experienced subsequent to the nap has not been clarified (Naitoh, 1981).

Another measure of sleepiness/alertness has been developed recently and may be helpful in addressing the above questions. The Multiple Sleep Latency Test (MSLT) measures the latency to polygraphically defined sleep in 20-min opportunities given at 2-hr intervals across the day (Carskadon & Dement, 1977). The MSLT is a reliable measure which is sensitive to sleep loss, sleep restriction, sleep extension, and the effects of CNS depressant drugs in normals (Carskadon & Dement, 1979; Carskadon & Dement, 1982; Roehrs, Tietz, Zorick, & Roth, 1984). Clinically, it differentiates normals from patients with EDS and among EDS patients with different disorders (Zorick et al., 1982).

This study examined the recovery of alertness after sleep loss as a function of nap duration using

the MSLT. In addition to determining the alerting effects of naps of different lengths, the time course of those alerting effects was measured over 8 hrs of continued wakefulness.

Methods

Subjects

The subjects were 10 (5 men and 5 women) normal-sleeping volunteers, aged 18–32 yrs ($\bar{X}=23.6$ yrs), recruited from local colleges. All subjects reported consistent bedtimes (no greater than 1.5 hrs variation), nocturnal sleep latencies of less than 30 min, total sleep times of 6.5–8.6 hrs nightly, daily naps less than twice weekly, and avoidance of cigarette smoking and drug use. The subjects were screened for good health with the Cornell Medical Index. Finally, each subject was screened with a MSLT. There was no evidence of sleep onset rapid eye movement (REM) periods suggestive of narcolepsy, and for each subject, the mean sleep latency across the four nap tests was greater than 10 min. The subjects signed an informed consent and were paid for their participation in the study.

Procedure

The 10 subjects were assigned to two groups of five, with one group being tested each Monday and Tuesday, and the other group each Wednesday and Thursday, for five consecutive weeks. Each subject underwent five different treatments administered in a Latin Square design, one per week, without his/her prior knowledge of the treatment for that week. A treatment consisted of one day of baseline latency tests (baseline day), total sleep deprivation that night, and a second day of latency tests (post-deprivation day) which began with a nap of 0, 15, 30, 60, or 120 min in bed after sleep onset. As a result of the sleep deprivation, all subjects usually achieved sleep onset for the nap within 5 min (in the only 3 exceptions over the 50 naps, the latency was 5–10 min). These five nap conditions comprised the five experimental treatments.

The subjects spent the other 5 days and 6 nights at home to allow sufficient recovery following deprivation. Because of space limitations subjects' sleep was not recorded in the laboratory the night prior to collection of the baseline day sleep latencies. They were instructed to maintain their normal time in bed of 6.5–8.6 hrs nightly, particularly the night prior to the first laboratory day of each condition. Baseline latencies were relatively stable among conditions (see Tables 1 and 2). However, because sleep was not recorded the night prior to the baseline day, conclusions regarding whether basal levels of alertness were achieved must be made with caution.

Subjects reported to the laboratory between 0800–0830 hrs on the baseline day, and electrodes were attached at standard placements for continuous recording of electroencephalogram (EEG) at central and occipital placements, electro-oculogram (EOG), and electromyogram (EMG), in accordance with the standard techniques of Rechtschaffen and Kales (1968). For all latency tests, the subjects were placed in beds in dark, quiet rooms and were instructed to close their eyes and try to fall asleep, during which the EEG, EOG, and EMG were recorded. The latency test was concluded following 20 min of wakefulness or the onset of sleep, which was defined as two consecutive epochs (30 s/epoch) stage 1 sleep or one epoch of any other sleep stage as determined by Rechtschaffen and Kales' (1968) criteria. At the conclusion of the latency test, the subjects got out of bed.

During the baseline day, a latency test was given at 0900 hrs, and then four more tests were given at the same clock times as those of the post-deprivation day for that nap condition in order to control for circadian variation in sleep tendency. The post-deprivation day began at 0900 hrs with naps of varying durations (the five nap conditions), which were followed by four latency tests at 2-hr intervals beginning 2 hrs after the end of the nap. For example, in the 30-min nap condition, the nap ended at 0930 hrs, and latency tests were given 2, 4, 6, and 8 hrs post-nap at 1130, 1330, 1530, and 1730 hrs. In the 120-min nap condition, the nap ended at 1100 hrs, and the latency tests were given at 1300, 1500, 1700, and 1900 hrs.

Cigarette smoking and alcohol and caffeine consumption were not allowed during the two days and the evening prior to coming to the laboratory. On both days, subjects ate light lunches between the first and second latency tests and on the post-deprivation day they went to breakfast at 0700 hrs. All subjects were monitored continuously throughout the days and nights they were in the laboratory to assure no unscheduled sleep.

The latency to sleep was recorded for all latency tests and for the 0900-hrs nap. Each subject's mean latency of the four tests on the baseline day and the post-deprivation day for a particular nap condition was calculated, transformed $\log_{10}(x+1)$, and submitted to a two-factor repeated measures ANOVA. The transformation was necessary to correct for heterogeneity of cell variances (as seen in Table 2). The first factor was the nap condition (0, 15, 30, 60, or 120 min), and the second was the day of latency testing (baseline vs. post-deprivation). Conservative F tests were used following the Greenhouse-Geisser procedure. Post-hoc pairwise comparisons among nap conditions were made using Tukey's HSD test. Similar ANOVAs and post-hoc comparisons were conducted on each of the four latency tests (at 2, 4, 6, and 8 hrs post-nap) separately. Finally, the sleep recordings were scored for sleep stages according to Rechtschaffen and Kales' (1968) criteria.

Results

Effect of Sleep Deprivation on Latency

Sleep deprivation for one night was expected to significantly reduce sleep latency on the nap from basal levels. The mean latency of all five nap conditions at 0900 hrs on the baseline day was 11.4 min, while the mean latency for the nap, recorded also at 0900 hrs after 24 hrs of sleep deprivation, was 1.8 min. There was, as expected, a significant

Table 1

Mean sleep latency at 0900 hrs for the five nap conditions

Nap Conditions	Mean Latencies (min) (SDs in Parentheses)	
	Baseline Day	Post Deprivation Day
0 min	11.8 (5.8)	1.0 (1.0)
15 min	11.8 (6.2)	1.7 (1.1)
30 min	8.2 (7.0)	1.6 (1.6)
60 min	12.5 (6.4)	2.1 (2.1)
120 min	12.8 (6.7)	2.4 (2.9)
Mean	11.4	1.8

decrease in sleep latency at 0900 hrs following deprivation, $F(1/9)=49.3$, $p<.001$ (see Table 1). There were no significant differences among the five nap conditions in latency at 0900 hrs on the baseline day nor at the onset of the nap (0900 hrs) on the post-deprivation day. This shows that there were no systematic differences in sleepiness among nap conditions. Furthermore, the effects of continuous deprivation lasted throughout the day. An examination of the four latency tests on the post-deprivation day for the 0-min nap condition (in this condition no recovery sleep was allowed) shows that deprivation produced a consistently short latency during the day (see Figure 1). Latency did not differ among the four tests throughout the day.

Effect of Nap Length on Mean Sleep Latency

The mean latencies of the four latency tests for each condition are presented in Table 2. Mean latency increased as a function of nap duration, from a mean latency of 2 min in the 0-min nap condition to a maximum of 6 min in the 60-min nap condition. Analyses of variance (Greenhouse-Geisser corrected probabilities were used throughout) revealed a significant main effect of days and nap duration and a significant interaction (see Table 3 for the abbreviated summary table). Post-hoc comparisons ($p<.05$) showed that mean latency on the

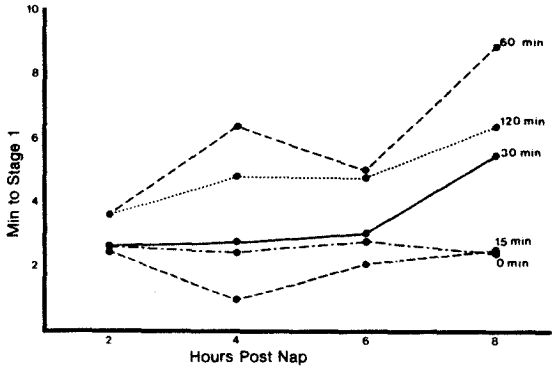

Figure 1. The time course of the alerting effects of the five nap conditions. Mean values for 10 subjects.

Table 2

Mean sleep latency of the four latency tests for the five nap conditions

Nap Conditions	Mean Latencies (min) (SDs in Parentheses)	
	Baseline Day	Post Deprivation Day
0 min	8.9 (4.8)	2.1 (1.2)
15 min	10.7 (5.5)	2.6 (1.7)
30 min	8.5 (5.5)	3.5 (2.8)
60 min	9.1 (4.4)	6.0 (3.5)
120 min	11.1 (4.8)	4.9 (2.3)
Mean	9.7	3.8

Table 3

Analyses of variance summary tables (abbreviated)

Source	*df*	*MS*	*F*	Tail Prob	G-G[a] Prob
Mean Latency of 4 Tests					
Day (D)	1	3.3618	73.70	.000	
Error	9	0.0349			
Nap Dur (N)	4	0.1376	3.94	.009	.028
Error	36	0.0349			
N × D	4	0.0973	7.91	.000	.002
Error	36	0.0123			
Latency 8 hrs Post Nap					
Day (D)	1	2.8594	28.14	.000	
Error	9	0.0101			
Nap Dur (N)	4	0.2449	4.51	.004	.016
Error	36	0.0543			
N × D	4	0.1686	6.05	.000	.002
Error	36	0.0278			

[a]Greenhouse Geisser corrected.

Table 4

Sleep stage composition of the naps for four nap conditions

Nap Conditions	Sleep Stage Durations (min)				Number of Subjects With REM	Total Sleep (min)
	Stage 1	Stage 2	Stage 3-4	REM		
15 min	3.2	6.8	2.9	2.0	2	14.9
30 min	2.4	11.6	11.6	4.6	3	30.2
60 min	3.3	20.9	33.0	2.5	2	59.7
120 min	13.3	42.7	36.0	20.9	7	112.9

post-deprivation day of a particular nap condition never reached the baseline value for that nap condition. Comparisons showed that latencies following experimental naps of 0 and 15 min duration were equal, and in both of these nap conditions, the latencies were significantly shorter than after 60 and 120 min naps. Moreover, the mean latency in the 60-min nap condition was significantly increased over the 30-min nap condition. Interestingly, the mean latency to stage 1 sleep after the 120-min experimental nap was 4.9 min, intermediate to and not significantly different from the 30-min and 60-min nap conditions.

Time Course of the Alerting Effects of the Nap

Latencies among nap conditions at 2, 4, 6, and 8 hrs after the naps were compared with separate ANOVAS (see Table 3 for an abbreviated summary table of the 8 hrs ANOVA). For each analysis (2, 4, 6, and 8 hrs) there was a significant main effect of days. Nap condition differences did not appear immediately. There were no differences in latency among the five nap conditions 2 hrs after the experimental nap. A significant increase in sleep latency for the nap conditions was not recorded until latency test two, 4 hrs after the end of that morning's experimental nap, $F(4/36)=4.94$, $p<.007$ (again Greenhouse-Geisser corrected probabilities were used). Differences in latency among nap conditions increased thereafter with each test (6 hrs test: $F(4/36)=3.05$, $p<.054$; 8 hrs test: $F(4/36)=6.05$, $p<.002$). The time course of the alerting effects of the naps is illustrated in Figure 1. Post-hoc tests showed that the latency onsets at 4, 6, and 8 hrs in the 60-min nap condition were greater than the equivalent latencies in the 0 and 15 min nap conditions; and these three latencies in the 60-min nap condition did not differ significantly from the baseline latencies. Achieving basal levels of alertness did not occur in any other nap condition on any of the tests. The latencies at 4, 6, and 8 hrs after the 120-min nap differed from the 0 and 15 min nap conditions, and were intermediate between and did not differ from the 30 and 60 min nap conditions.

Sleep Stages and Continuity of the Naps

The mean total sleep time for the naps in each of the four conditions (0-min condition received no sleep) was similar to that scheduled: 14.9, 30.2, 59.7, and 112.9 min respectively. Sleep efficiencies (total sleep time over total time in bed) among conditions were 90% or greater (15 min=90%, 30 min=92%, 60 min=96%, 120 min=93%) and did not differ. The total sleep times and the sleep stage composition of the experimental naps are shown in Table 4. There were some noteworthy differences in sleep stage composition among the four naps. The amount of stage 2 sleep increased significantly across the four nap conditions, approximating the geometric progression in nap duration, $F(3/27)=34.39$, $p<.001$; $15=30<60<120$. Slow wave sleep (stage 3-4) increased significantly with increasing nap duration until it reached an asymptote in the 60-min nap condition, $F(3/27)=34.78$, $p<.001$; $15<30<60=120$. Finally, stage 1, $F(3/27)=6.05$, $p<.025$, and rapid-eye movement (REM) sleep, $F(3/27)=6.11$, $p<.020$, were increased only in the 120-min nap condition compared to the three others, and together with stage 2 accounted for most of the extra minutes of sleep for this nap condition compared to the 60-min condition.

The relation of sleep stages to sleepiness/alertness was evaluated by correlating minutes of each sleep stage to the mean sleep latency following the nap. The absolute amount of stage 3-4 in the morning nap was correlated with subsequent sleep latency throughout the day, $r(38)=.38$, $p<.02$—see Figure 2). The amount of stage 2 sleep, while increasing with nap duration, did not correlate, $r(38)=.28$, NS. Stages 1 and REM also did not correlate. While the correlation of stage 3-4 sleep to subsequent sleep latency was significant, it explains only a small amount of the variance in subsequent sleep latency.

The continuity of sleep on the naps was compared to determine how sleep continuity might relate to sleepiness/alertness. Sleep continuity was assessed by counting the number of shifts from other sleep stages (stage 2, 3-4, or REM) to stage 1 sleep or wakefulness. The mean number of shifts to stage 1 or wakefulness was significantly greater for the

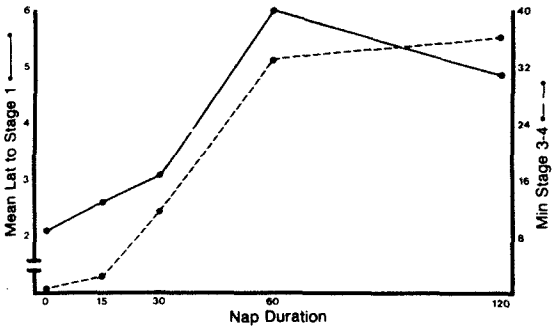

Figure 2. Minutes in stage 3-4 sleep compared to mean sleep latency for the five nap conditions. Mean values for 10 subjects.

120-min nap compared to the 15, 30, and 60 min naps, $F(3/9)=21.5$, $p<.01$. There were on the average 0, 0.2, and 1.1 shifts on the 15, 30, and 60 min naps, but 6.9 shifts on the 120-min nap. The distribution of shifts to stage 1 or wake within the 120-min nap was evaluated further by comparing hour 1 to hour 2. In the first hour there was a mean of 1.5 shifts which is similar to that of the 60-min nap. The second hour of the 120-min nap had significantly more shifts (5.4) than the first hour, $t(18)=3.14$, $p<.01$.

Discussion

These results indicate that napping had alerting effects in sleep-deprived normals which were systematically related to the duration of the nap. However, these alerting effects were not evident on the first latency test 2 hrs after the nap. They appeared on the second test and then increased in subsequent tests across the day. The maximal level of alertness for the 8 hrs of testing was achieved with a 60-min nap, although the mean baseline level of alertness was never fully achieved. Increasing the nap duration to 120 min produced no further increase in alertness. Increased alertness was associated only weakly with increasing amounts of slow wave sleep. The additional sleep of the 120-min nap was highly fragmented which may account for the absence of further alerting effects.

Several interesting questions arise from these data, the first being why the alerting effects of the nap did not appear on the first latency test 2 hrs after the nap. A delayed response to recovery sleep was reported in another study (Carskadon & Dement, 1982). In that study, changes in MSLT scores did not appear until late afternoon following a full night of recovery sleep after two nights of deprivation. These data would suggest that the recovery process requires time, the amount of time possibly being related to the amount of the sleep debt. On the other hand, it has been shown that performance immediately after awakening from sleep is inefficient relative to pre-sleep levels (Naitoh, 1981). While this sleep inertia was felt to endure for 15–30 min after awakening, a recent study indicated that it may last for as long as 2 hrs in recovery from 53 hrs sleep loss (Naitoh, 1981). Another possibility is that the initial expression of alertness resulting from the nap was suppressed due to the interacting effects of circadian sleep tendencies. The normal circadian variation in sleepiness/alertness is characterized by a decline in alertness over the midday hours. In all conditions of this experiment, especially in the 60 and 120 min nap conditions where the greatest alerting effects were later found, the first latency test was conducted over this usual midday decline in alertness. However, the second test also occurred over this decline and some restoration was found on the second test which makes the circadian interaction with recovery a less tenable explanation.

The second issue raised by these data is why the additional 60 min of sleep in the 120-min nap condition produced no further alerting effects compared to the 60-min condition. An initial consideration might be that the additional sleep was not necessary. However, the observation that baseline levels of alertness were never fully achieved would indicate otherwise. A tempting second explanation is that the 120-min condition provided no further slow wave sleep, and hence, no additional recovery effects. The function relating minutes of slow wave sleep to increasing nap duration is remarkably similar to the function relating nap duration to alertness (see Figure 2). However, the correlation coefficient between slow wave sleep and alertness accounts for a very small amount of variance. Previous studies exploring the restorative effects of specific sleep stages have been unable to demonstrate a differential sleep stage effect (Lubin, Moses, Johnson, & Naitoh, 1974; Johnson, Naitoh, Moses, & Lubin, 1974).

A more likely explanation for the absence of additional recovery in the 120-min nap is that the additional sleep was fragmented. There are two possible reasons for the fragmentation. It is established that sleep out of phase with circadian rhythms of sleepiness/alertness, hormones, and body temperature is shortened and fragmented (Akerstedt & Gillberg, 1981). This suggests that the placement of a recovery nap relative to the phase of the circadian rhythm of sleepiness/alertness is important to its ability to restore alertness and the ability to perform efficiently. Thus, for example, one would predict that a nap taken over the usual midday increase in sleepiness would be less fragmented than a nap taken at the usual phase of increased alertness and would thus differentially restore optimal function-

ing. The fragmentation of the 120-min nap may also relate to the increased likelihood of transitions from REM sleep to stage 1 or wakefulness which is often seen in nocturnal sleep. The second hour of the 120-min nap was predominantly REM sleep. Comparisons of 120-min naps (with REM sleep) taken at midday vs. morning may resolve this issue.

The extent to which out-of-phase sleep is fragmented and consequently less restorative may also be related to the severity of the prior sleep debt (the less the debt the more the fragmentation). A 4-hr sleep beginning at 2000 hrs after 8 hrs of sleep the previous night was more disturbed than a 4-hr sleep beginning at 0800 hrs after overnight sleep deprivation (Nicholson et al., 1985). Both 4-hr sleeps were positioned at high points (early morning and early evening) on the circadian rhythm of alertness. However, the 2000-hrs sleep was preceded by 12 hrs of wakefulness and the 0800-hrs sleep by 24 hrs of wakefulness.

These data clearly suggest that understanding recovery of alertness compromised by sleep loss will require consideration of a number of interacting factors including: 1) the duration and sleep stage composition of the nap, 2) the continuity of the sleep obtained on the nap, 3) the circadian phase timing of the nap, 4) the severity of the sleep debt, and possibly 5) a sleep inertia which continues for an extended period after awakening from a recovery nap.

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