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Performance and Sleepiness Following Moderate Sleep Disruption and Slow Wave Sleep Deprivation

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BONNET, M. H. Performance and sleepiness following moderate sleep disruption and slow wave sleep deprivation. PHYSIOL BEHAV 37(6) 915-918, 1986.—Recent studies have shown that periodically disrupted sleep resulted in significant daytime sleepiness and performance loss in normal young adults. One study suggested that the periodicity of disturbance rather than the total number of sleep disturbances was the primary factor in causing degraded function. However, in that study, increased performance levels could have been associated with increased levels of slow wave sleep. The present study was designed to determine whether the amount of SWS rather than the periodic disruption of sleep accounts for decreased performance of Ss with disrupted sleep. Twelve normal young adults spent two 4-night periods in the laboratory. During one 4-night series, Ss were briefly aroused either following each 10 min of sleep or whenever they entered stage 3 sleep (No SWS condition). During the second series of nights, Ss were briefly aroused after each 10 min of sleep (SWS condition). In the second series, additional arousals were performed after 5-min periods (but not when Ss were in SWS) to equalize the total number of arousals in the SWS condition with those in the No SWS condition. Total experimental arousals were equal in the disruption conditions, and the experimental manipulation was successful in reducing total SWS to infrequent epochs of stage 3 in the No SWS condition while allowing significantly more SWS in the SWS condition. In terms of sleep stages, this difference was balanced by increased stage 2 in the No SWS condition. Despite the differential occurrence of SWS, no performance, mood, or nap latency measure was different in the SWS vs. No SWS conditions, although performance loss of equal magnitude was seen in each condition as a function of sleep disruption. The data imply that it is the frequency and periodicity of sleep disturbance which determines the restorative capability of sleep.

SEVERAL recent studies have shown that systematic disturbance of sleep leads to decreased daytime alertness and performance ability. Periodic sleep fragmentation in normal young adults at a rate of one disturbance per minute of sleep resulted in performance and sleepiness similar to that seen after total sleep loss [1,2]. Significant sleepiness was found in both patients [3] and in normal subjects [1] with sleep which was briefly disturbed at 10-min intervals, even though total sleep time did not differ by more than 40 min from that seen in control conditions.

One study [1] examined performance and sleepiness after three sleep disruption conditions and after total sleep loss in an attempt to explain whether the restorative nature of sleep was more closely related to total minutes of time spent asleep, amounts of sleep stages SWS and/or REM, or pattern of sleep disturbance. Baseline and total sleep loss performance levels were used to predict performance in the sleep disruption conditions (i.e., if total sleep was decreased to half of the baseline level following a disruption schedule, it was predicted that performance following that disruption schedule should also fall midway between baseline and total sleep loss levels). It was found that subjects were significantly sleepier and performed more poorly when their sleep was briefly disturbed each 1 min or each 10 min than would have been predicted by their total time asleep on those nights, even when time spent in stage 1 sleep was not counted. In general, Ss performed better and were less sleepy when allowed 2.5 hr of undisturbed sleep followed by frequent disturbance than when disturbed at 10-min intervals throughout the night, even though total sleep time, stage 2, REM, and sleep efficiency were greater and stage 1 and total awakenings were fewer in the 10-min condition. The only sleep variable normally associated with restoration which was greater in the 2.5-hr sleep condition was SWS (75 vs. 46 min on the second disruption night).

SWS is frequently associated with bodily restoration in the literature for several pervasive reasons, including its proximity to sleep onset, its immediate rebound after sleep loss, and its association with high sensory thresholds and growth hormone secretion [5,6]. As such, it is important to determine whether the results reported by Bonnet [1] are actually related to the effects of sleep disturbance or whether

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they are related to total amounts of SWS in the conditions. The current study sought to produce sleep disruption-associated daytime sequelae while manipulating SWS in a controlled fashion. It was hypothesized that the sleep disruption would produce daytime performance loss but that there would be no difference in daytime performance level in a condition allowing SWS (SWS condition) versus a condition not allowing SWS (No SWS condition).

**METHOD**

Twelve young adult subjects between the ages of 18 and 28 were chosen to participate for two consecutive weeks. Subjects were normal sleepers who rarely took naps as determined by a sleep questionnaire. All subjects scored within the normal range on the depression scale of the MMPI.

**Design and Procedure**

Subjects normally participated in the study in pairs. After an unscored laboratory adaptation night, subjects spent four nights, usually Thursday through Sunday, in the laboratory during each week. The four nights were baseline (BL), two consecutive disruption nights, and one recovery night. Subjects had recording electrodes attached 1 hr before bedtime on each night so that standard sleep recordings [7] could be made. Subjects also completed the Clyde Mood Scale and a simple reaction time task each evening. Subjects were put to bed at their normal bedtime (range 2300 to 0100). Each morning subjects were awakened at their normal time of awakening (range 0600 to 0800) and immediately performed 30 min of Wilkinson Addition and 30 min of Wilkinson Vigilance. They then had a sleep latency test followed by a reaction time task, the Clyde Mood Scale, and Stanford Sleepiness Scale.

**Performance Measures**

Subjects performed the same battery of tests each day. They included Wilkinson Addition (30 min and scored for number correct), Wilkinson Vigilance (30 min and scored for Hit Rate), and Simple Reaction Time (the 10-min task described by [4]). Following vigilance each morning, subjects attempted to fall asleep. The nap was terminated at the end of the first epoch of stage 2 sleep or at the end of 15 min, whichever came first.

**Disruption**

The criteria for awakening in the two awakening conditions were as follows:

1. **No SWS condition**. The subject was awakened immediately throughout the night 10 min after the appearance of a well-defined spindle, K-complex, or rapid eye movement. The S was awakened immediately if he went into stage 3 sleep at any time. Total experimental arousals were counted for each disruption night.

2. **SWS condition**. The S was awakened after each 10 min of sleep as in the No SWS condition. However, the S was not awakened prior to the 10 min if he entered SWS. On each night, extra arousals were made after 5 min of sleep if the S was not entering SWS to equalize the total number of arousals with the corresponding No SWS night. Subjects having the SWS condition first (conditions were counterbalanced) were scheduled to have the mean number of arousals that already completed subjects had in the No SWS condition.

Subjects were awakened on disruption nights with a Belton Model 109 screening audiometer through an earphone insert earpiece taped into their preferred ear. One thousand Hz tones were presented according to the following rule: Tones were begun at the approximate sleep threshold (40–60 dB) and were increased in 10 dB steps until the S awoke. If

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**TABLE 1**

**SLEEP AMOUNTS ON EACH STUDY NIGHT**

<table>
<thead>
<tr>
<th>SWS Condition</th>
<th>No SWS Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BL   D1 D2 R</td>
</tr>
<tr>
<td><strong>Total Sleep</strong></td>
<td>426 368 400 438</td>
</tr>
<tr>
<td><strong>Latency to</strong></td>
<td>16   14 10 9.3</td>
</tr>
<tr>
<td><strong>Stage</strong></td>
<td>128 247 266 126</td>
</tr>
<tr>
<td><strong>Changes</strong></td>
<td>4    22 16 2</td>
</tr>
<tr>
<td><strong>%W</strong></td>
<td>4    15 12 4</td>
</tr>
<tr>
<td><strong>%I</strong></td>
<td>48   43 46 43</td>
</tr>
<tr>
<td><strong>%2</strong></td>
<td>8    4 6 8</td>
</tr>
<tr>
<td><strong>%3</strong></td>
<td>12   2 4 16</td>
</tr>
<tr>
<td><strong>%4</strong></td>
<td>20   7 11 24</td>
</tr>
<tr>
<td><strong>%SWS</strong></td>
<td>21   11 13 24</td>
</tr>
<tr>
<td><strong>%R</strong></td>
<td>2    2 2 2</td>
</tr>
<tr>
<td><strong>%M</strong></td>
<td>2    2 2 2</td>
</tr>
</tbody>
</table>

*D2 compared to BL.*
the subject awakened to the first tone presentation on the initial trial, tone intensity was decreased 10 dB from that level for the next trial. If the subject did not awaken to the first tone presentation on the previous trial, the tone series was initiated at the intensity level which resulted in the awakening response. If the S did not awaken, intensity was increased in 10 dB steps. With this procedure, it was usually possible to awaken Ss within 10–15 sec. Ss were awakened at the first tone presentation on about 50% of trials and on the second presentation on most of the remaining trials. For purposes of this study, a subject was considered “awake” when he gave a subjective verbal rating of his sleep/wake state [2].

**RESULTS**

An average of 55 arousals was performed in the SWS condition on the first disruption night, and 63 arousals were performed on the second disruption night. Respective values for the No SWS condition were 58 and 60 arousals (differences not significant by paired t-test). Arousal threshold at awakening was examined by comparing threshold values in the two conditions during the first hour of the first disruption night and the last hour of the second disruption night by repeated measures ANOVA. Arousal thresholds increased from night 1 to night 2, F(1,11)=44.8, p<0.0001, although thresholds did not differ at the beginning of disruption night 1 (57 dB in No SWS vs. 52 dB in SWS) or at the end of disruption night 2 (92 dB in No SWS vs. 82 dB in SWS, F(1,11)=2.0 p>0.1, NS) in the SWS vs. No SWS comparison.

**Sleep Variables**

Means for the basic sleep variables for each study night can be found in Table 1. Data analysis for sleep and mood variables was accomplished by first comparing baseline and recovery night values for each week for each variable by within-subject t-test. If significant differences (here, p<0.05) were not found, baseline and recovery values were averaged for further analysis. A significant difference was found only for stage 4 sleep, which rebounded on the recovery night. Therefore, only the baseline stage 4 values were used to compare with the disruption data. At this point repeated measures analyses of variance were performed on the baseline and second disruption night data. The ANOVAs had within-subjects terms for week (1 df), experimental condition (1 df), and interaction (1 df). A significant main effect for experimental condition in this design implied that the variable differed from baseline after both disruption conditions. A significant week by experimental condition interaction implied that the change from baseline to second disruption night was greater in one disruption condition than in the other. Post hoc pairwise comparisons for the significant interactions were performed using the Neuman-Keuls procedure at the 0.05 level.

It can be seen from Table 1 that there were significant reductions in stage 3 percent, stage 4 percent, and total SWS percent in both disruption conditions and that the reductions were significantly greater in the SWS than in the No SWS condition for stage 3 [Interaction F(1,11)=10.9, p<0.01] and SWS [Interaction F(1,11)=10.2, p<0.01]. These changes represent the success of the experimental manipulation in reducing SWS in the No SWS condition. The disruption manipulation resulted in a decrease of about 30 min in total sleep time on the respective second disruption nights; an increase in awakenings, stage changes, and stage 1 sleep; and a decrease in REM as well as SWS parameters. The SWS and No SWS conditions differed only on the SWS parameters and on stage 2 percent [Interaction F(1,11)=7.62, p<0.02], which increased in the No SWS condition and did not change in the SWS condition.
Table 2 presents daytime performance, morning nap latency, and subjective data from each morning of the study. It can be seen from the table that morning performance on correct additions was significantly worse after the second disruption night, F(1,11)=6.99, p<0.01, than on the baseline but that performance did not differ in the comparison between the SWS and No SWS conditions, F(1,11)=0.12. Overall significant performance effects were not found for Vigilance Hit Rate or Reaction Time Median. On the morning nap, latency reductions were found after both disruption conditions, F(1,11)=21.7, p<0.01, but again the disruption conditions did not differ, F(1,11)=0.11, NS.

It can be seen from Table 2 that the morning subjective report scales (Stanford Sleepiness Scale and Clyde Mood Sleepiness and Clear Thinking Scales) were in agreement with the objective sleepiness and addition data. These three subjective measures indicated decreased function (p<0.02) after disruption but gave no indication of any difference between SWS and No SWS conditions (F values <0.77).

**DISCUSSION**

The current experiment is the third in a series of studies which have examined the effects of periodic disturbance of sleep. Two studies [1,2] have demonstrated that disturbance of sleep at 1-min intervals resulted in nonrestorative sleep (i.e., performance close to total sleep loss levels). One study [1] examined sleep stage distribution and performance in three sleep disruption schedules and found that while total sleep time, total sleep time minus stage 1 sleep, total REM, and total REM plus SWS was greater in a condition with sleep periodically disturbed at 10-min intervals than in a condition which allowed 2.5 hr of sleep before repeated arousal at sleep onset, performance was better when 2.5 hr of sleep was allowed. These data could have been explained by total SWS, which was greater in the 2.5-hr sleep condition.

The present experiment was undertaken to replicate the demonstration of development of significant sleepiness and performance loss in a group of normal young adult subjects with an average of 59 brief disturbances of sleep at 10-min intervals. As in previous studies, it can be seen from Table 1 that the sleep disruption procedure had relatively little impact on total sleep time (a reduction of about 30 min on the second disruption night) but did cause consistent sleep distribution changes. The current study specifically attempted to differentially impact SWS within the disruption paradigm (the No SWS condition) and succeeded in reducing total SWS to 18% of baseline in the No SWS condition versus 55% of baseline in the SWS condition. While significant performance and mood effects were found as a function of the sleep disruption, no significant differences were found in mood or performance when the SWS and No SWS conditions were compared and no strong trends were seen.

These data may be explained either by holding that slow wave sleep changes are not related to daytime function or by the possibility that a 45% decrease in SWS (in the SWS condition) results in functional loss which is not increased by more extreme SWS loss. The latter explanation is unlikely because it has been shown [1,2] that a greater decrease in SWS, when accompanied by an increased rate of sleep disruption, does result in increased functional loss. The parsimonious explanation of these findings is that it is the fragmentation, rather than the SWS decrease, which is related to daytime function. In conjunction with previous studies [1–3], these data further strengthen the contention that sleep parameters, including sleep stage amounts and total amount of sleep, may not be related to restoration of function when periodic sleep disturbance exists. The data do imply, once again, that periods of continuous sleep in excess of 10 min are required for restoration of function during sleep (Sleep Continuity Theory).

The total number of experimentally-produced arousals was relatively small in the current study (about 59 per night) and would compare to an apnea index of about 9 in a patient with sleep fragmented by periodic apneas. It should also be noted that despite relatively few arousals, morning stage 2 nap latencies had declined to about 5 min following the second disruption night. While MSLT values often are obtained later in the day than those reported here, it is common to use 5 min as a criterion value to define pathological sleepiness. Therefore, the reported reduction in nap latency, which was recorded following an hour of morning testing, must be considered clinically as well as statistically significant.

The sleepiness and performance loss reported in the current study appear greater than that reported in patients with mild sleep apnea. The difference can be accounted for by two major factors. First, arousals were closely timed and carefully operationally defined (via verbal report) in the current study so that their impact could be quantified and replicated. In patients with mild sleep apnea, arousal may not be complete. Also, we have shown that patients with mild apnea may have frequent periods of 90 min or longer during sleep without respiratory disturbance [3]. These long periods of consolidated sleep should allow significant restoration to occur. Second, in studies of patients, there is often no clear baseline; and many other medical, educational, and psychological processes may obscure results.

**REFERENCES**