Performance During Frequent Sleep Disruption

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Summary: Performance on a simple addition task was measured during three schedules of frequent sleep disruption for 2 nights. Five young adults had their sleep briefly disturbed for 2 nights in 3 separate weeks either every 1 min, every 10 min, or at sleep onset after an undisturbed 2.5-h sleep period. Subjects were required to perform a two-number, two-digit addition problem as rapidly as possible on awakening. Main effects were found for sleep disruption condition and time of night, and a significant interaction between the two was also observed. Latency to response was longest for the 10-min condition on night 1, on night 2, however, response latencies were longest in the 1-min condition. Response latencies were fastest in the 2.5-h condition for both nights of disruption. Arousal thresholds were also gathered across both nights. Arousal thresholds were consistently the highest in the 1- and 10-min conditions for both nights of disruption, reaching maximum threshold levels at the end of night 1. Arousal threshold was significantly positively correlated with response latency. Sleep stages (slow-wave sleep (SWS), SWS + REM (SWSR), and total sleep time minus stage 1 sleep) were poor predictors of performance changes across the 2 disruption nights. The data were best explained by sleep continuity theory, which posits that a period of at least 10 min of uninterrupted sleep is required for restoration to take place. Key Words: Sleep fragmentation—Sleep continuity—Arousal threshold—Performance—Sleep deprivation—Sleep apnea.

Patients with sleep apnea experience a number of consequences from their disorder, including excessive daytime somnolence, personality alterations, memory impairment, and nocturnal confusion (1,2). Such symptoms commonly occur after sleep deprivation; therefore, one could posit that patients with sleep apnea are sleep deprived. Total daily sleep time may be tremendously increased in these patients, however, and this is incompatible with a pure sleep deprivation hypothesis.

Recent studies (3,4) indicate that sleep continuity may be more integral to restoration of performance than total sleep time or specific sleep stage amounts. The sleep continuity theory (3,4) states that for sleep to be restorative, the sleep process must continue undisturbed for a period of time >10 min. This theory claims, for example,
that high sensory thresholds following sleep deprivation are instituted to maintain the continuity of sleep to allow sufficient time for effective protein synthesis (5,6). Therefore, specific amounts of sleep stages are not important independent of sleep continuity.

Both clinical and experimental studies have supported the sleep continuity theory. One study (7) showed that although sleep stage amounts do not seem strongly related to the severity of obstructive sleep apnea, the number of arousing events and the periodic placement of these events are highly related to the severity of obstructive sleep apnea. For example, patients with mild obstructive apnea and relatively little excessive daytime sleepiness (EDS) had periods of 20–85 min during the night without any apneas or associated sleep disturbance whereas patients with severe EDS rarely had a period of sleep as long as 10 min without an apnea.

Another experiment modeled the sleep disruption observed in moderate to severe sleep apneics in normal sleepers by awakening young adult subjects from sleep every minute for 2 nights (3). This procedure resulted in increased sleepiness and decreased daytime performance. Post-day performance for subjects awakened from sleep every minute for 2 consecutive nights was as poor as performance from subjects who were totally deprived of sleep for 40–64 h.

In another study (4), normal young adult subjects were either sleep deprived for 64 h, awakened every minute, every 10 min, or at sleep onset after an initial sleep period of 2.5 h during 4 nonconsecutive weeks. This study modeled the frequency and placement of sleep disturbance that occurs in severe and moderate sleep apnea. Subjects performed best in the 2.5-h condition and performed in an intermediate fashion in the 10-min condition. After the subjects were disrupted every minute, performance was again similar to that after total sleep deprivation for 64 h. These studies did not assess the time course of performance loss during sleep fragmentation, however. An analysis of the time changes in performance during sleep disruption is important for obtaining a better understanding of the time course of developing performance loss. In one study (3), as subjects were frequently aroused during the night, they often became confused or disoriented. Subjects could not remember simple tasks and at times reported an inability to understand English (their native language), even though they knew they were being spoken to and were required to produce a verbal response. Nocturnal confusion and decreased performance upon awakening was noted in several experimental studies across a variety of tasks (see ref. 8), and varied with the depth of sleep, phase of the circadian rhythm, and amount of sleep loss. Such effects therefore should be maximized in patients with sleep apnea and in a disruption paradigm in which nocturnal awakenings are frequent.

The present study assessed performance of subjects immediately on their awakening from sleep across 2 nights of sleep disruption following either every minute of sleep, every 10 min of sleep, or immediately after sleep onset following a 2.5-h undisrupted sleep period. Four main questions were addressed: (a) How rapidly does nocturnal performance loss occur across the three disruption conditions? (b) At what time(s) of the night do these disruptions exert their most detrimental effects? (c) How related is performance to abrupt awakening and the depth of sleep? (d) Which explanation—total sleep time (TST), total sleep time minus stage 1 (TST-1), sleep stage amounts [slow-wave sleep (SWS) and SWS plus REM (SWSR)], or sleep continuity theory—is most predictive of the trends in performance across the two disruption nights?
METHODS

Subjects
Five young adults between the ages of 18 and 28 were chosen to participate for 4 nonconsecutive weeks over 2 months. Potential subjects were required to be normal sleepers (range 6–8 h a night) who rarely took daytime naps, as determined by an initial sleep questionnaire. Subjects were also given a selected medical history, the Brief Michigan Alcoholism Screening Test, the Brief Drug Abuse Screening Test, and the depression scale of the MMPI. All subjects admitted to the study scored within the normal ranges on all of these scales.

Procedure
Subjects participated for 4 nonconsecutive weeks (21 nights). Five nights of each week were spent in the laboratory, and subjects were allowed 9 days off between each experimental session. On the first night of each experimental week, subjects were allowed to sleep undisturbed (baseline). Nights 2 and 3 were disruption nights, and nights 4 and 5 were recovery nights. Each night, subjects arrived at the sleep laboratory 1 h before their preestablished bedtimes for a standard EEG hookup. On disruption nights, total bedtime was increased by 30 min to attenuate sleep loss that accrued as a consequence of the disruption procedure. During 1 week, subjects were totally deprived of sleep for 64 h. No nocturnal performance data were collected from this week, however, because response latencies measured during total sleep deprivation from fully awake subjects may not be directly comparable to response latencies gathered from subjects who are awakened from sleep. Response latencies gathered from subjects awakened abruptly from sleep are longer when compared with response latencies from subjects who are fully awake, even after sleep deprivation (8).

Disruption conditions
During nights 2 and 3 (disruption nights 1 and 2) in 3 separate weeks, subjects were awakened from sleep according to the following three schedules: (a) every minute after the first K-complex, sleep spindle, or rapid eye movement (1-min condition); (b) every 10 min after the first K-complex, sleep spindle, or rapid eye movement (10-min condition); or (c) undisturbed sleep for 2.5 h before being repeatedly awakened immediately at sleep onset for the remainder of each respective disruption night (2.5-h condition). The order of conditions was randomized, and subjects did not know beforehand which condition they were assigned for any particular week.

Awakenings
Subjects were awakened on disruption nights with a Beltone Model 109 screening audiometer through an earphone insert taped to their preferred ear. Tones were initiated at approximate sleep threshold (40–60 dB) and were increased in 10-dB steps until the subject awoke (when subjects gave a verbal response that they were “awake”). No tones exceeded 110 dB. When subjects did not awaken at 110 dB, the technician called their names over the intercom until an awakening response occurred, and this was recorded as 120 dB (this occurred for only one subject, nine times on the second disruption night in the 1-min condition). After the awakening response was achieved, the intensity level of the tone was decreased 10 dB for the beginning of the next trial. If the subject did not awaken, tone intensity was increased in 20-dB increments until the subject awoke.
Immediately on awakening, subjects were asked to solve mentally and report verbally solutions to a random two-digit/two-number addition problem. Addition problems were presented either at each awakening (2 Ss) or at alternate awakenings. Inspection of the data from subjects who answered addition problems after every awakening and from subjects who answered addition problems on alternate awakenings revealed no differences in performance across the night. Thus, data from both groups were combined for analysis.

Response latencies to the addition problems were recorded by a response button which the technician used to mark when each question was asked and when the subject responded. The technician also marked on the record whether the answer to the addition problem was correct or incorrect.

Response latency was defined as the total amount of time elapsed between the ending of the question and the verbal response of the subject. The latency was recorded as the time lapse between the time the question was initially asked and the time an answer was given. If subjects were confused as to the question asked, the technician repeated the question until the subject understood. Subjects were allowed to return to sleep if they could not respond within 200 s (the answer was determined to be incorrect and the response latency was coded as 200 s). This event occurred nine times for one subject on night 2 in the 1-min condition. The name of a subject who fell asleep during the procedure was repeated over the intercom until the subject gave a verbal indication of wakefulness. The same question was then repeated for the subject. No subject was ever allowed feedback as to the accuracy of a response.

RESULTS

An average of 240.6, 52.2, and 146.2 responses were gathered from subjects during each disruption condition (both disruption nights together) from the 1-min, 10-min, and 2.5-h conditions, respectively. The distribution of awakenings as a function of sleep stage and total sleep times for baseline and disruption night 2, are shown in Table 1. Overall, 90% of the responses occurred after the subjects were awakened from stages 1 and 2. In the 10-min condition, 54% of the awakenings were from stages 1 and 2, whereas 29% of the awakenings were from SWS and 17% of the awakenings were from

<table>
<thead>
<tr>
<th>TABLE 1. Stages from which subjects were awakened</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage</td>
</tr>
<tr>
<td>-------</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>4</td>
</tr>
<tr>
<td>REM</td>
</tr>
<tr>
<td>TST (min)*</td>
</tr>
<tr>
<td>BL = 396</td>
</tr>
<tr>
<td>Total responses</td>
</tr>
</tbody>
</table>

TST, total sleep time.

* Disruption night two values.
REM sleep. Total sleep amounts were scored using standard EEG criteria and are reported elsewhere (4).

Response latencies

All data were analyzed using repeated measures analysis of variance (ANOVA) with terms for disruption condition, night of disruption (first vs. second disruption night), time of night (by thirds), and correct or incorrect solution to the addition problem. Variance for the 3- and 4-way interaction terms were pooled with error to test 2-way interactions. The Greenhouse-Geisser conservative F test was used (9). Data were analyzed with all stages and with only stages 1 and 2. Using both analyses, equivalent results were found. Because most of the awakenings occurred from stages 1 and 2, the data presented are only from these stages.

No effects were found for correct or incorrect solution to the addition problems ($F_{1,4} = 1.33, \text{NS}$). An interaction between sleep disruption condition and night of disruption was significant ($F_{2,32} = 11.9, p < 0.01$). Post-hoc comparisons (Newman-Keuls, $p < 0.05$) revealed that on night 1 response latencies were longest in the 10-min condition (~23 s), intermediate for the 1-min condition (~20 s), and shortest for the 2.5-h condition (~10 s) (Fig. 1). By the middle of night 2, however, response latencies more than doubled in the 1-min condition whereas response latencies increased 15 s on the average in the 10-min condition, and 6 s on the average in the 2.5-h condition. Therefore, by night 2, response latencies were longest in the 1-min condition (~52 s), intermediate for the 10-min condition (~38 s), and again shortest for the 2.5-h condition (~16 s).

To illustrate the magnitude of performance loss in the experimental conditions, a group of five fully awake subjects, who were not sleep deprived, were asked to perform the same addition problems that the experimental groups were asked to solve on awakening from sleep. During their normal waking hours; these subjects were placed in a darkened sleep room and were asked to solve the addition problems when prompted over the intercom. Ten response latencies for each subject were gathered at six points across an 8-h period. The average response latency for these normal waking young adults was 7.0 s. Comparison of hour 1 response latencies to hour 8 response latencies indicated no significant change ($t_{4} = 1.47, \text{NS}$).

![Graph showing response latency curves across disruption nights 1 and 2. Values on the abscissa are clock times.](image)

**FIG. 1.** Response latency curves across disruption nights 1 and 2. Values on the abscissa are clock times.
Sleep stages as predictors of performance loss

To aid in the explanation of the performance decrements in these data, the sleep variables SWS, SWSR, total sleep time (TST), and (TST-1) were used to predict performance loss across the 2 nights of sleep disruption for the 10-min and 2.5-h conditions (no predictions were made for the first third of disruption night 2 for the 2.5-h condition because few observations were made for this time period). Tests for higher order trends (across the last third of night 1 and all three time periods of night 2), using orthogonal polynomial coefficients, revealed no significant departure from linearity across the three conditions. Because no higher order trends were significant, the use of a formula that assumes a linear relationship between performance and sleep stage amounts was used.* For example, a 50% decrease in the amount of SWS would be associated with a 50% decrease in performance (i.e., longer response latencies). Likewise, a 30% decrease in the amount of SWS would be associated with a 30% decrease in performance.

The proportions of sleep loss for the 10-min, and 2.5-h condition were derived as follows. The amount of sleep stage accrued in the 10-min or 2.5-h conditions for the time period of interest was subtracted from the amount of that same sleep stage accrued by the same time period on baseline nights.

The amount of sleep stage accrued in the 1-min condition was subtracted from the amount of that same sleep stage, as above, accrued for the same time period, as above, on baseline nights. The amount of sleep loss obtained for the 10-min or 2.5-h condition was then divided by the amount of sleep loss obtained in the 1-min condition. This value was then multiplied by the difference between response latencies in the 1-min condition and baseline performance. The resulting value was then added to baseline performance values to yield a predicted performance value for the particular time period of interest for sleep stages (SWS, SWSR, TST, TST-1).

Actual performance values were then subtracted from predicted performance values. The inverse of the difference scores (predicted−actual) allowed a range of −1.0 to +1.0 (in cases in which the difference between actual and predicted was <1, coefficients of 1 were assigned for that particular time period). Negative coefficients indicated that actual performance was worse than predicted; positive coefficients indicated that actual performance was better than predicted. The closer the coefficient is to 1, the more accurate the predictor. Using this method, applied to individual scores, the relative accuracy of sleep stages in predicting performance changes in the 10-min and 2.5-h conditions was assessed. The medians and SD for all five subjects are shown in Table 2.

As Table 2 indicates, sleep stages were poor predictors of performance across the 2 nights of sleep disruption. Sleep stages consistently predicted that performance should be worse than actual performance in the 2.5-h condition (12 of 12 cases, binomial sign test, p < 0.001) and consistently predicted that performance should be better than performance actually was in the 10-min condition (15 of 16 cases, binomial sign test, p

*Px = BLP + (RL1 − BLP)X SSA(BL) − SSA(10 min or 2.5 h/SSA(BL) − SSA(1 min). Px, predicted performance level for one of the four time periods of interest (i.e., N1, lights out 0500 h; N2, lights out − 0100, 0100−0300, and 0300−0500). BLP, baseline performance for the 10-min or 2.5-h condition (median of the first 10 observations of night 1 in the 10-min or 2.5-h condition). RL1 average response latency for the time period of interest in the 1-min condition. SSA, sleep stage amount (either SWS, SWSR, TST, or TST-1). Conditions from which these sleep stage amounts are calculated are indicated in parentheses.

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TABLE 2. Accuracy of sleep stages as predictors of performance loss

<table>
<thead>
<tr>
<th>Condition</th>
<th>SWS</th>
<th>SWSR</th>
<th>TST-1</th>
<th>TST</th>
</tr>
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<tbody>
<tr>
<td>10 min</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N1-5:00</td>
<td>-.15 (.23)</td>
<td>-.06 (.17)</td>
<td>-.04 (.51)</td>
<td>-.26 (.30)</td>
</tr>
<tr>
<td>N2-1:00</td>
<td>-.04 (.13)</td>
<td>-.03 (.21)</td>
<td>-.04 (.06)</td>
<td>-.03 (.21)</td>
</tr>
<tr>
<td>N2-3:00</td>
<td>-.02 (.45)</td>
<td>-.03 (.07)</td>
<td>-.02 (.14)</td>
<td>-.01 (.05)</td>
</tr>
<tr>
<td>N2-5:00</td>
<td>.09 (.45)</td>
<td>-.05 (.16)</td>
<td>-.03 (.08)</td>
<td>-.02 (.07)</td>
</tr>
<tr>
<td>2.5 h</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N1-5:00</td>
<td>.13 (.44)</td>
<td>.30 (.36)</td>
<td>.13 (.41)</td>
<td>.13 (.18)</td>
</tr>
<tr>
<td>N2-1:00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N2-3:00</td>
<td>.02 (.09)</td>
<td>.14 (.43)</td>
<td>.03 (.18)</td>
<td>.06 (.07)</td>
</tr>
<tr>
<td>N2-5:00</td>
<td>.02 (.12)</td>
<td>.05 (.42)</td>
<td>.07 (.07)</td>
<td>.09 (.06)</td>
</tr>
</tbody>
</table>

SWS, slow-wave sleep; SWSR, SWS plus REM, TST-1, total sleep time minus stage 1.
Median scores; SD in parentheses.

< 0.001). No particular sleep variable was the best predictor either within or between conditions.

Arousal thresholds
Arousal thresholds were gathered at each awakening across both nights of disruption and across all conditions. Arousal threshold data were analyzed in the same manner as the latency data. Arousal thresholds increased an average of 32 dB from the beginning of night 1 to the end of night 2 in the 1-min and 2.5-h conditions and increased an average of 24 dB in the 10-min condition. The interaction between sleep disruption condition and the time of night was significant ($F_{4,16} = 3.0, p < 0.05$). Multiple comparison testing revealed that the 10-min condition had higher threshold values during the first part of night 1. The 1-min and 10-min condition thresholds were higher than the 2.5-h condition thresholds during the last two-thirds of night 1 and the first two-thirds of night 2. No condition differences for threshold were found for the last third of night 2 (Table 3). In the 1-min condition, arousal thresholds increased significantly across night

TABLE 3. Average arousal thresholds across six time periods of nights 1 and 2

<table>
<thead>
<tr>
<th>Condition</th>
<th>1 min</th>
<th>10 min</th>
<th>2.5 h</th>
<th>Differences$^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Night</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>T1</td>
<td>61.2</td>
<td>65.8</td>
<td>53.3</td>
</tr>
<tr>
<td></td>
<td>T2</td>
<td>73.1</td>
<td>75.3</td>
<td>47.6</td>
</tr>
<tr>
<td></td>
<td>T3</td>
<td>85.1</td>
<td>77.7</td>
<td>65.9</td>
</tr>
<tr>
<td>2</td>
<td>T4</td>
<td>84.1</td>
<td>77.7</td>
<td>64.7</td>
</tr>
<tr>
<td></td>
<td>T5</td>
<td>88.8</td>
<td>85.8</td>
<td>66.8</td>
</tr>
<tr>
<td></td>
<td>T6</td>
<td>93.9</td>
<td>89.5</td>
<td>85.8</td>
</tr>
</tbody>
</table>

1 min, T1 < T2 < T3 = T4 = T5 = T6; 10 min, T1 = T2 = T3 = T4 < T5 < T6; 2.5 h, T1 < T2 < T3 = T4 = T5 < T6. (T1 and T4, 2300–0100h; T2 and T5, 0100–0300h; T3 and T6, 0300–0500 h.)

$^a$ Newman-Keuls, p < 0.05.

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1 but did not increase significantly across night 2. In the 10-min condition, arousal thresholds did not increase significantly until the second third of night 2, when a significant increase occurred. In the 2.5 h-condition, arousal thresholds were significantly higher by the last third of night 1 and leveled off until the last third of night 2, when thresholds were significantly higher.

Pearson product-moment correlations were calculated for each subject between arousal threshold and response latency. These correlations were averaged using Fisher's r to z transformation (10). A significant average positive correlation between arousal thresholds and response latencies was found after comparison with tabled ρ values (r(59) = 0.315, p < 0.05), indicating that as the depth of sleep increased, response latencies increased.

DISCUSSION

This study attempted to answer four questions related to performance during 2 nights of sleep disruption. Question 1 asked how rapidly performance loss occurred across the three disruption conditions.

The detrimental effects of sleep disruption on performance occurred early on night 1 and built exponentially for the 1- and 10-min conditions (Fig. 1). By night 2, a clear delineation between the 1- and 10-min conditions became apparent. Subjects in these conditions were affected severely by sleep disruption and required a long time to answer a simple addition problem (52 s by the middle of night 2 in the 1-min condition). Comparatively, college students took 7 s to answer similar questions while awake (i.e., control group). For the experimental groups on night 1, response latencies were 4 times waking control in the 10-min condition, 3 times waking control in the 1-min condition, and 2 times control values in the 2.5-h condition. By night 2, response latencies on the average were >7 times control values in the 1-min condition, remained at 4 times control in the 10-min condition, and were nearly 2.5 times control in the 2.5-h condition. Performance in the 2.5-h condition was clearly better than any other condition and changed relatively little across disruption nights.

Question two asked at what time(s) of the night these disruptions exerted their most detrimental effects.

Performance declined across the 2 nights in each condition so that all average response latencies on night 2 were greater than all average response latencies on night 1. During the first night, little difference was found between the 1- and 10-min conditions. During the second disruption night, response latencies were much longer in the 1-min condition. From the beginning of night 1, response latencies were shortest for the 2.5-h condition and continued to be the shortest throughout both disruption nights.

Although nonsignificant, a decrease in response latencies in the 10-min condition from the middle until the end of disruption night 2 indicates a possible circadian component. With more subjects, a quadratic trend might reach statistical significance. There was no such indication in the 1-min or 2.5-h condition. In the 1-min and 2.5-h conditions, the rate of sleep disruption, which was 10 times that of the 10-min condition, may have masked an underlying circadian component.

Question 3 asked to what extent performance on abrupt awakening was related to the depth of sleep. Heightened arousal thresholds were expected to be a component contributing to longer response latencies. Such an expectation is consistent with a continuity theory interpretation. As Bonnet (3,4) emphasized, heightened sensory thresholds are instituted by the sleep system to lessen arousability so that restoration

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may progress. Consistent with this proposition, an arousal threshold correlated significantly with performance, indicating that as arousal thresholds increased, performance declined. Therefore, depth of sleep is a component of performance loss and accounts for ~10% of the variance that occurs in response latencies. Arousal thresholds were highest in the 1- and 10-min conditions across both nights of disruption (except for the last third of night 2 when no disruption condition threshold was significantly different from any other), with thresholds in the 1-min condition reaching maximum levels early in the first night.

Comparisons of arousal thresholds within each condition indicated that arousal thresholds leveled off by the end of the first disruption night in the 1- and 10-min conditions. This effect is most likely due to the fact that thresholds reached their maximum levels by the end of the first disruption night. In the 2.5-h condition, however, a different pattern occurred. Thresholds increased at the end of each night but were relatively stable for the middle third of each disruption night. Habituation may help explain why response latencies remained relatively stable across the first part of nights 1 and 2 in the 2.5-h condition (11). Such an explanation was used to explain the lack of responsiveness to frequent tones in a study of behavioral control of respiration in sleep apneics (12) and is common among studies that use frequent awakenings accompanied by frequent behavioral responses (13). Therefore, because high arousal thresholds reflect the pressure to remain asleep, lower arousal thresholds associated with the 2.5-h condition may reflect a lessened pressure to maintain sleep.

Question four asked which explanation—TST, TST-1, SWS and SWSR, or sleep continuity theory—was most predictive of the trends in performance across the two disruption nights.

Total sleep time and TST-1 always predicted that actual performance should be worse than predicted performance in the 10-min condition and always predicted that actual performance should be better than predicted performance in the 2.5-h condition (Table 2). As predictors, TST and TST-1 were among the least accurate predictors for each time period, with the exception of the last third of night 1, when TST was the best. Furthermore, TST-1 (disruption night 2) was greatest in the 10-min condition, intermediate for the 2.5-h condition, and least for the 1-min condition. This cannot explain why the 2.5-h condition yielded response latencies that were significantly better than the 10-min condition at mitigating performance loss. Given such evidence, it seems highly unlikely that TST and TST-1 can account for the significant differences in response latencies across the disruption conditions.

One might also conclude that REM sleep together with SWS and SWSR alone contributed to the performance loss reflected in these data. Such a conclusion might be derived from the fact that little SWSR or SWS alone occurred in the 1-min condition and the most occurred in the 2.5-h condition.

Both of these conclusions are weakened, however, by the relative lack of predictiveness of performance loss for these variables. Neither SWSR or SWS could differentiate between the 10-min and 2.5-h conditions or predict performance loss during either night of sleep disruption. Such a conclusion is further weakened by recent studies that showed little relationship between SWS and restoration. Bonnet (14) found no differences in daytime function when subjects on a 10-min disruption schedule were either allowed or not allowed SWS. Moreover, in a recent review by Spiegel et al. (15), changes in SWS were not accountable for the apparent loss of mental functioning or subjective sleep quality, among other variables, in elderly individuals.
Thus, these data appear to be best explained in a sleep continuity framework. This study adds to a continuing line of evidence, indicating that sleep continuity is the essential component in allowing sleep to be restorative (3,4,7,14). Along with previous work, this study showed performance decrements to be greatest in young adult subjects when awakenings occurred on a disruption schedule of \( \leq 10 \) min.

Therefore, it is not surprising that a moderate or a severe sleep apneic with a history of frequent arousals that interrupt sleep continuity would experience feelings of malaise, nocturnal disorientation, and excessive daytime somnolence. This implies that effective treatments in disorders that fragment sleep are those that can provide periods of consolidated sleep of \( >10 \) min. Future studies should concentrate on broader schedules of nocturnal disturbance and on daytime measures of performance and sleepiness to elucidate further the relationship between sleep disruption, performance, and sleepiness.

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**REFERENCES**