Performance and Sleepiness as a Function of Frequency and Placement of Sleep Disruption

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ABSTRACT

Eight normal young adult sleepers spent 4 nonconsecutive weeks in the laboratory. Each week consisted of a baseline night followed by 2 consecutive nights of disrupted sleep, followed by 2 recovery nights. Disruption conditions included: a) brief awakening after each minute of accumulated sleep, b) brief awakening after each 10 min of accumulated sleep, c) 2.5 hrs of normal sleep followed by a brief awakening at each sleep onset, and d) total sleep deprivation. Morning testing revealed that all disruption conditions decreased sleep latency in a morning nap test. Performance after 1-min disruptions approximated that seen after total sleep loss. Performance decrements were less in the 10-min condition and least in the 2.5-hr sleep condition. Performance under baseline and total sleep loss conditions was used to predict performance during the sleep deprivation condition using four sleep stage rules. Total time asleep and total time asleep minus stage 1 predicted performance poorly. Total SWS plus REM predicted performance best but could not differentiate the 10-min and 2.5-hr conditions. Therefore, it was concluded that the data were most parsimoniously explained by the Sleep Continuity Theory—i.e., that periods of uninterrupted sleep in excess of 10 min are required for sleep to be restorative.

DESCRIPTORS: Sleep, Sleep disruption, Sleep deprivation, Sleep Continuity Theory, Restoration, Sleep fragmentation.

While the function of sleep is controversial, it is accepted that total sleep loss reliably results in increased sleepiness, decreased performance ability, and degraded mood. It is also accepted that a period of recovery sleep following sleep deprivation serves to reverse decrements and restore baseline levels of sleepiness, performance, and mood.

Historically, it was hypothesized that the rate of restoration during sleep was directly related to depth of sleep as measured by sensory threshold (Wohlisch, 1957). More recently, investigators have linked restoration to amounts of slow wave sleep or REM sleep. For example, one experimental approach (Rechtschaffen, Gilliland, Bergmann, & Winter, 1983) implied that rats selectively deprived of SWS and REM sleep in a yoked-control design will eventually die while control animals randomly deprived of as much total sleep will not. However, another series of studies (Lubin, Moses, Johnson, & Naitoh, 1974; Johnson, Naitoh, Moses, & Lubin, 1974) examined differential performance effects which might be associated with selective loss of SWS or REM sleep in humans. Unfortunately, these studies were unable to demonstrate differences between conditions depriving SWS or REM sleep and concluded that the major factor determining restoration was total sleep time.

Another recent study (Bonnet, 1985a) has examined the effect of frequent disturbance of sleep on daytime function. Normal young adults had their sleep briefly disturbed after each accumulated minute. The procedure resulted in a minimal loss of total sleep; however, sleepiness, performance, and recovery sleep after two days of disruption closely approximated values seen in total sleep loss studies of a similar duration. Three possible theoretical viewpoints were invoked as a potential explanation of these results:

1) Continuity rather than stage amounts or total sleep times determines restoration—This hypothesis maintains that neither total sleep time nor specific stage of sleep determines restoration. Rather, a period of sleep free from any arousing disturbance is required for restoration. This theory maintains, for example, that SWS is important because it institutes high sensory thresholds which reduce the probability of arousal and therefore increase un-
disturbed sleep. The theory also predicts that recovery sleep after sleep loss will be characterized by even higher sensory thresholds as a further attempt to restore balance in a homeostatic sleep system. If sleep is disturbed before a critical period of time elapses, restoration does not take place.

2) Stage 1 sleep is not sleep—The disruption procedure greatly increased stage 1 sleep while decreasing SWS and REM. If stage 1 does not "count" as a restorative stage, then the total amount of sleep in the disruption study would have been much less. Therefore, total sleep time (i.e. sleep minus stage 1) might still predict little sleep and thus little restoration.

3) Reduced SWS/REM—The disruption procedure effectively eliminated SWS and REM because subjects must be asleep for longer than one minute to reach these states. Therefore, the nonrestorative sleep may have derived from the absence of these states. This hypothesis cannot be easily reconciled with the earlier conclusion that total sleep rather than amounts of REM or SWS determines restoration (Lubin et al., 1974; Johnson et al., 1974). However, those studies did not examine REM plus SWS deprivation.

In the current study, four sleep schedules were examined in an attempt to differentiate among these three hypotheses. The four schedules included a 64-hr total sleep loss condition and a disruption after each minute of sleep condition to replicate the earlier findings (Bonnet, 1985a). The third condition allowed 10 min of sleep before each disruption. The final condition allowed 2.5 hrs of undisturbed sleep followed by immediate disruption after the appearance of a spindle, K-complex, or rapid eye movement for the remainder of the night.

It was hypothesized:

1) If the primary restorative sleep factor was continuity of sleep, then performance would be best in the 2.5-hr sleep condition, which was the only condition which allowed the accumulation of more than 10 min of undisturbed sleep. If 10 min were greater than the critical sleep amount to allow restoration, then performance in the 10-min and 2.5-hr conditions would be similar. If 10 min were less than the critical sleep amount, then performance in the 1-min and 10-min disruption conditions would be similar.

2) That if the primary restorative sleep factor was nonstage 1 sleep, then performance following the 10-min disruption condition would be the best of all conditions. If the primary restorative sleep factor was total sleep time, then performance would be at near baseline levels in all conditions except total sleep loss.

3) The conditions were designed to allow differential amounts of SWS and REM. Essentially no SWS or REM would accumulate in the 1-min disruption. The 2.5-hr condition should allow normal amounts of SWS but greatly curtail REM. The 10-min disruption was chosen because it would significantly decrease both SWS and REM. It was hoped that the large variance in sleep stage amounts and distributions in the baseline, disruption, and deprivation conditions would allow specific performance predictions to be made in the various sleep conditions. These performance predictions were then used to determine which restorative hypothesis predicted best.

Method

Subjects

Eight young adult subjects, aged 18–28 yrs, were chosen to participate for 4 nonconsecutive weeks over a two-month period. 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 for 4 nonconsecutive weeks. After an unscored laboratory adaptation night, subjects spent 5 nights in the laboratory during each of the 4 weeks. The 5 nights were baseline (BL), 2 consecutive disruption or deprivation nights, and 2 recovery nights. Subjects had recording electrodes attached one hour before bedtime each night (except for total sleep loss nights) and at standard sleep recordings (Rechtschaffen & Kales, 1968) could be made. Subjects also completed the Clyde Mood Scale and a simple reaction time task each evening. They 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, 30 min of Wilkinson Vigilance, and had a sleep latency test before the recording electrodes were removed. At this point subjects repeated the Clyde Mood Scale and the reaction time task. Following baseline and recovery nights, subjects were free to leave the laboratory after they had completed testing. Following the total sleep loss and 1-min disruption nights, subjects were encouraged to remain at the laboratory during the day. Those subjects with specific plans (class, jobs, etc.) were allowed to leave the laboratory after the first total sleep loss or 1-min disruption night if they checked in frequently with laboratory personnel. After the second night, subjects were asked to remain in the lab or were accompanied by technicians if they left. The order of conditions was assigned randomly except that each pair of subjects had their total sleep loss nights scheduled to occur on the same week (so that the technician could concentrate on keeping both
subjects awake rather than watching the EEG machine).

The first 3 subjects in the experiment participated in only 3 weeks of the study and were not exposed to the 2.5-hr sleep condition.

**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 Lisper & Kjellberg, 1972). 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 three awakening conditions were as follows: 1-min Condition—Subject awakened 1 min after the appearance of a well-defined spindle, K-complex, or rapid eye movement; 10-min Condition—Subject awakened 10 min after the appearance of a well-defined spindle, K-complex, or rapid eye movement; and 2.5-hr Condition—Subject allowed to sleep undisturbed for 2.5 hrs and then awakened immediately after the appearance of a well-defined spindle, K-complex, or rapid eye movement.

Subjects were awakened on disruption nights with a Beltone Model 109 screening audiometer through an earphone insert earpiece taped into their preferred ear. 1000 Hz tones were presented according to the following rule: Tones were begun at the approximate sleep threshold (40–60 dB) and were increased in 10dB steps until the subject awoke. If the subject awakened to the first tone presentation on the initial trial, tone intensity was decreased 10dB 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 subject did not awaken, intensity was increased in 10dB steps. With this procedure, it was usually possible to awaken subjects within 10–15 seconds. Subjects 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” if he gave a subjective verbal rating of his sleep/wake state (3 subjects, rated on a 1–7 scale) or completed a two-digit, two-number addition problem (5 subjects).

**Results**

**Sleep Variables**

Means for the set of sleep variables selected for analysis can be found in Table 1. The baseline value is the mean of all baseline nights in the study. The mean sleep data presented in the table came from 8 subjects where possible. In general, the sleep data from the 3 subjects participating in only three experimental conditions closely approximated the data from the remaining 5 subjects, and the sleep stage ANOVAs for 8 subjects differed substantially from those for 5 subjects only in that F-values tended to be higher. However, unless otherwise specified, ta-

<table>
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<th>Min Stage 2</th>
<th>Min SWS</th>
<th>Min REM</th>
<th>Sleep Efficiency</th>
<th>Number of Awakenings</th>
<th>Latency</th>
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* R1 - R2 = BL, p < .05; ** R1 - R1 = BL, p < .05; *** R1 - R2 = BL, p < .05; ++ R1 - R1 = BL, p < .05.
bles, figures, and differences noted as significant are based on analyses of the data from the 5 subjects with complete data. The repeated measures analyses of variance for sleep stages included terms for Condition (1-Min, 10-Min, 2.5-Hr. Sleep Deprivation) and Night (BL, Recovery 1, Recovery 2). Significant effects were consistent with those one might expect from sleep loss studies: The initial recovery nights after each deprivation condition had increased SWS and decreased wake time, stage 1, and awakenings. Slow wave sleep returned to normal on the second recovery nights and REM rebounds were apparent. In the present data (Table 1), the sleep stage changes indicated above were significant as study night main effects, but additional significant sleep deprivation condition effects were found only for stage 4 sleep (significantly increased in the 1-min deprivation condition compared to the other conditions). However, it can be seen from the table that expected changes in recovery sleep as a function of deprivation conditions were seen. For example, the 2.5-hr and 10-min conditions resulted in moderate decreases in SWS and in moderate rebounds on Recovery 1. REM sleep could be seen to rebound on Recovery 2 after total sleep loss and 1-min conditions in response to the primary SWS rebound on Recovery 1. The 10-min and 2.5-hr conditions tended to have increased REM on Recovery 1 as well as Recovery 2 because of less SWS rebound on Recovery 1.

Effects of experimental manipulations on sleep during the deprivation nights can also be seen in the data: 1) Total sleep time was reduced in all deprivation conditions, but the total reduction ranged from about 40 min per night in the 10-min condition to about 90 min per night in the 2.5-hr condition to about 120 min per night in the 1-min condition. 2) Awakenings and minutes of stage 1 sleep were increased in a manner analogous to the decreases in Total Sleep Time.

Performance and Sleepiness

Performance on most psychomotor tasks is influenced by significant learning effects. In an attempt to control for the systematic effects of learning and habituation in the present experiment, performance data from all weekly baseline nights and Recovery 2 nights were entered into a regression equation (regressed over day in study) for each subject individually. The derived equation estimated systematic performance changes within each subject over the entire 4-week course of the study. Because the ordinal number for each baseline and second recovery night was entered into the regression equation (i.e. study nights 2, 6, 7, 11, 12, 16, 17, and 21), the equation could then be used to predict performance on deprivation and first recovery nights by substitution of the appropriate night numbers. These values were compared with the observed values by ANOVA. It should be noted that these data provide a conservative estimate of baseline performance ability because they assume that all performance variables had returned to baseline levels by the morning following Recovery 2. The repeated measures ANOVA performed on these data had variance partitioned for Experiment (baseline vs. deprivation), Condition (1-Min, 10-Min, 2.5-Hr, Total Sleep Loss), and Night (Disruption 1, Disruption 2, Recovery 1).

Four morning measures—vigilance hit rate, number of correct addition problems completed, median reaction time, and latency to stage 2 sleep (nap)—were analyzed. Results from the four analyses were generally consistent. For all variables, overall performance loss was greatest in the total sleep loss condition and least in the 2.5-hr condition. Performance in the 1-min and 10-min conditions was intermediate. For reaction time and correct additions, performance in the 1-min condition fell midway between sleep deprivation and 10-min performance. Data from the Wilkinson Addition Task are presented in Figure 1. The ANOVA for additions revealed a significant condition × experiment interaction (F(3/36) = 5.24, p < .01, Greenhouse-Geisser df' = 2/15, p < .025). Newman-Keuls pairwise comparisons (used throughout at p = .05 with Greenhouse-Geisser df') revealed that significantly fewer additions were completed in the sleep loss condition as compared to baseline or any other condition. Significantly fewer additions were completed in the 1-min condition as compared to baseline or either other deprivation condition. The 2.5-hr and 10-min conditions did not differ from each other or from baseline.

For reaction time, there was a significant condition × experiment interaction (F(3/36 = 3.25, p < .05, Greenhouse-Geisser df = 3/19, p < .05) and a significant night × experiment interaction (F(2/32) = 4.70, p < .025, Greenhouse-Geisser df = 2/19, p < .025). Reaction time was longer after total sleep loss than in any other condition (.244 s vs. .224, .215, .211, and .213 s respectively in 1-min, 10-min, 2.5-hr, and BL conditions). Similar results were found when slow reaction times (gaps) were ex-

From Table 1, it appears that SWS on R1 in the 2.5-hr condition is at levels comparable to that seen in total sleep loss. This was considered an anomaly because SWS on baseline for the total sleep loss condition weeks was 76 min, a value considerably lower than the normal SWS baseline value.
Figure 1. Mean number of correct addition problems completed following each night of disruption/deprivation (N1, N2) and the first recovery night (R1) as compared to baseline (closed circles) values for the four experimental conditions. The open squares are median scores for the sleep disturbance conditions.

Examined. Reaction time was also longer on Disruption/Deprivation Night 2 than the other nights (.237 s vs. .218, .215, and .214 s on Disruption/Deprivation 1, Recovery 1, and BL).

For hit rate and nap latency, performance in the 1-min disruption condition was similar to that seen after total sleep loss (hit rate data are presented in Figure 2). For hit rate there was a significant condition × experiment interaction ($F(3/36) = 3.26$, $p < .05$, Greenhouse-Geisser $d^f = 3/25$, $p < .05$). Performance in the total sleep loss condition was worse than in the 2.5-hr condition. For nap latency, there was a main effect for the experiment (i.e., nap latencies for all experimental conditions were decreased compared to baseline), $F(1/4) = 34.15$. Nap latency averaged 4.5 min during experimental conditions and 9.7 min on baseline.

Apparent performance differences between additions and hit rate can be best be explained by looking at median values (open squares plotted on both Figure 1 and Figure 2). Examination of the median data shows that addition and hit rate performance were actually very similar in that median performance following the 1-min condition was very close to median performance following total sleep loss for both variables. These mean/median differences are explained by one extremely productive subject whose performance decreased more in the total sleep loss condition and is therefore reflected in the means but not the medians.

Figure 2. Mean Wilkinson Auditory Vigilance Hit Rate Percent following each night of disruption/deprivation (N1, N2) and the first recovery night (R1) as compared to baseline (closed circles) values for the four experimental conditions. The open boxes are median scores for the sleep disturbance conditions.

Sleep/Performance Interactions

Sleep/performance interactions were explored by examining sleep and performance levels in the two “standard” conditions, baseline and 64 hrs of total sleep loss, and using those changes to obtain performance predictions for the sleep disruption conditions. For each subject, the decrease in performance from baseline to 64 hrs of total sleep loss was first calculated\(^2\) for each of the four performance measures. The difference in performance was then multiplied by the ratio of Total Sleep Time (TST) in the 1-min condition to TST on baseline. This score was added to the respective performance score after total sleep loss to give a predicted performance level at the intermediate level of sleep time for each performance variable in the 1-min condition. The same rule was followed to calculate predicted performance levels based on TST in the 10-min and 2.5-hr conditions. Predictions were constructed based upon four predetermined sleep variables: TST, TST minus min of stage 1 sleep (TST – 1), total slow wave plus REM sleep (SWSR), and total slow wave sleep (SWS). For each subject, actual performance values were compared with predicted performance variables for each of the four

\(^2\)Note: The formula used to calculate predicted performance was $P_{i} = (P_{i\text{BL}} - P_{i\text{BL}(t)}) \times \left(\frac{(SLP_{i})}{(SLP_{i\text{BL}})}\right) + P_{i\text{BL}}$, where $P_{i} = $ predicted performance on task $i$, $P_{i\text{BL}} = $ baseline performance on task $i$, $P_{i\text{BL}(t)} = $ performance on Task $i$ after 64 hrs of total sleep loss, $SLP_{i} = $ amount of sleep in disruption condition $i$, and $SLP_{i\text{BL}} = $ amount of sleep on baseline.
Table 2
Predicted vs. actual performance values

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<th>1-min Condition</th>
<th>10-min Condition</th>
<th>2.5-hr Condition</th>
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<td>Adds Correct (median)</td>
<td>Hit Rate</td>
<td>Nap Latency Overall (min)</td>
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*differs significantly from actual by sign test, p = .05.

Table 3
The proportion of subjects performing better than predicted by sleep

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<th>Experimental Conditions</th>
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* 2.28, p < .025.
** 1.96, p = .05.

The present experiment was designed to examine the effects of five very different types of nocturnal sleep periods on daytime performance. The experimental protocol was an arduous one, both for experimental subjects and for research support personnel. As a result, the total number of subjects is small and statistical analysis misses much of the import of the data. For this reason, the tables and figures have been designed to present data in a manner which is not always justified by the statistics but in a manner which should communicate much more than the statistics.

Table 1 summarizes the effects of the experimental manipulations. At a gross level, it can be seen that the manipulations were successful in es-
tablising several parameters important in differentiating the three hypotheses forming the background for the study. The 1-min and 10-min conditions were directly designed to test sleep continuity (hypothesis 1). As such, the sleep conditions are clearly discrete: 1-min periods of sleep, 10-min periods of sleep, and 2.5-hr periods of sleep. In terms of stage 1 sleep (hypothesis 2), there was a relatively small amount in the 10-min condition, but this increased to almost 100 min in the 2.5-hr condition and to 130 min in the 1-min condition. Finally (hypothesis 3), the 1-min condition essentially eliminated SWS and REM, while the 10-min condition allowed about 50% of normal SWS and 75% of REM, and the 2.5-hr condition allowed about 75% of SWS and 35% of REM.

Daytime performance and sleepiness reflected the differential impact of experimental conditions. The data, particularly the median data (see Figures 1 and 2), show performance in the 1-min condition to be very similar after night 2 to that in the total sleep loss condition. This replicates a previous finding (Bonnet, 1985a). It was consistently (although not always statistically) found in the performance and nap latency tasks that decrements were greater in the 10-min condition as compared to the 2.5-hr condition.

When the sleep and performance data are examined together, the individual findings are reinforced. Performance in the 1-min condition was poor and was best predicted by a rule which was based on total slow wave and REM (SWSR) sleep. However, since there was essentially no slow wave or REM sleep in this condition, predictions and observations were essentially at the total sleep loss level. In the 10-min condition, performance was also best predicted by the SWSR predictor. In this condition, subjects had about 60% of total slow wave and REM sleep while having about 90% of their total sleep.

Compared with the 1-min and 10-min conditions, the 2.5-hr condition was an anomaly. On all sleep measures, including total sleep time (78% of normal), minutes of stage 1 (270% of the 10-min condition and 90% of the 1-min condition), and minutes of SWSR (equivalent to the 10-min condition), one would expect performance and sleepiness to range midway between the 1-min and 10-min conditions. In actuality, performance in the 2.5-hr condition tended to be better than in the 10-min condition. Within-subject t-tests (N=5) were performed to compare actual performance in the 10-min and 2.5-hr conditions (even though not indicated by ANOVA). One of the four tests, for correct additions, was significant (t=5.925, p<.01) and indicated that significantly more additions were done following 2 nights of the 2.5-hr sleep condition than 2 nights of the 10-min disruption condition. Similarly, the data from Table 3 reinforce the notion that subjects in the 2.5-hr condition were performing above their predicted levels significantly more frequently than were subjects in the 10-min condition.

The results indicate that subjects performed worse than would be predicted by their TST on additions and vigilance and that their nap latencies were significantly shorter than would be predicted in both the 1-min and 10-min conditions. These data imply that total sleep time is a poor predictor of daytime performance after disrupted sleep. The same results and conclusions apply when stage 1 is subtracted from total sleep (TST -1). Therefore, it is not either decreased sleep time or increased stage 1 sleep which accounts for nonrestorative sleep, and hypothesis 2 must therefore be rejected.

In the present data set, the best predictor of performance was SWSR. This, in itself, provides evidence for the theory that nonrestorative sleep is related to reduced SWS and REM. But, because SWS and REM were approximately equal in the 2.5-hr and 10-min conditions, and performance was significantly better in the 2.5-hr condition, it implies that another underlying mechanism must account for the difference. Hypothesis 3 (reduced SWSR) is therefore weakened.

The data are most parsimoniously explained in terms of sleep continuity (hypothesis 1). Disruption of sleep at a 1-min rate resulted in decrements greater than those at a 10-min rate, which were greater than those at a 2.5-hr rate. The data also indicate that the relative difference in performance between the 1-min rate and the 10-min rate is much larger than between the 10-min rate and the 2.5-hr rate. This suggests a curvilinear recovery function with a major change in slope somewhere between 1 and 10 min of uninterupted sleep.

The need for a consolidated period of sleep is a reasonable one if sleep is viewed as a restorative process which exists over an extended period of time. Indeed, it has been shown that recovery of performance following sleep loss is orderly and follows a 4-8 hr time course (Rosa, Bonnet, & Warm, 1983; Bonnet, 1985b). It is known that protein synthesis, which is favored during sleep and especially during periods of lowest basal metabolic level (Brebria & Altschuler, 1968), proceeds over a period of minutes (Brodsky, 1975; Dietzis, 1961), and that an increase in metabolism can significantly disrupt the protein synthesis process (Fisher, 1968; Mateev, Shetyanov, Anghelova, & Hristov, 1967).

The sleep continuity hypothesis proposes that sleep has evolved as a mechanism to ensure lowered
levels of metabolic activity for periods of sufficient length to allow efficient protein synthesis. Factors which support the inherent importance of consolidated sleep include:

1. The fact that rapid habituation occurs to almost any frequent disrupting stimulus (Bonnet, 1982, 1984; Sharpless & Jasper, 1956) during sleep.
2. Increased arousal thresholds during sleep—otherwise, this seems like a biologic mistake.
3. Placement of consolidated, deep sleep at the beginning of the sleep period to ensure its occurrence.
4. Increased depth of sleep (over and above increased SWS) in children with high metabolic demands (Busby & Pivik, 1983).
5. Increased arousal threshold within sleep stage after sleep deprivation to further decrease the possibility of disruption (Rosa et al., 1983; Williams, Hammack, Daly, Dement, & Lubin, 1964).
6. Preservation of a period of 2–3 hrs of relatively undisturbed sleep even in elderly individuals and severe insomniacs (Bonnet, 1986).
7. Subjective abhorrence to disruption of sleep. Three caveats detract from confirmation of the continuity hypothesis in the present experiment. First is that total slow wave sleep also was least in the 1-min condition, intermediate in the 10-min condition, and most in the 2.5-hr condition. Therefore, SWS as a sleep construct might explain the results. Second, the data seem to indicate that performance was better than expected in the 2.5-hr condition rather than worse than expected in the 10-min condition (see Table 3). Such an outcome might result from an inappropriate experimental assumption, namely, that restoration during sleep is a linear process. In fact, our earlier work (Rosa et al., 1983) has shown that restoration of performance following one night of total sleep loss may occur within 4 hrs of recovery sleep and therefore is probably not linear. Finally, because frequent arousals occurred in the second half of the night in the 2.5-hr condition, one might hypothesize that performance in this condition was a little better as a result of an increased morning arousal level. Subjectively, subjects preferred the 2.5-hr condition to any other and particularly disliked the 1-min condition.

Clearly it is difficult for any single experiment to differentiate several interrelated hypotheses. The current data clearly indicate that total sleep time, either including or excluding stage 1 sleep, is a poor predictor of recovery. The data suggest that continuity of sleep rather than SWS or REM amounts determines restoration. However, these variables are highly correlated. It is hoped that specific research carefully controlling sleep fragmentation and SWS will soon be reported. Certainly, confirmation of the sleep continuity hypothesis and further definition of the just noticeable sleep difference unit, if appropriate, await further research.

REFERENCES


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**Announcements**

**Twenty-Sixth Annual Meeting**

**Society for Psychophysiological Research**

From October 16th through 19th, 1986, the Twenty-Sixth Annual Meeting of the Society for Psychophysiological Research will be held at Le Centre Sheraton Hotel in Montreal, Quebec, Canada.

For information regarding submission of papers, contact: J. Michael Lacroix, Program Chairman, SPR 1986, Department of Psychology, York University, Glendon College, 2275 Bayview Avenue, Toronto, Ontario M4N 3M6, Canada. For registration and transportation information, contact: Joanne Fetzner, Convention Manager, SPR, 2380 Lisa Lane, Madison, WI 53711 (608/271-1500).

**Fourteenth Annual Meeting**

**Psychophysiology Society**

From December 17–19, 1986, the Fourteenth Annual Meeting of the Psychophysiology Society will be held at Charing Cross and Westminster Medical School, Hammersmith, West London. The deadline for submission of abstracts is September 10, 1986.

For further information about guidelines for submission of papers, registration and accommodation details, contact: Dr. J.H. Gruzelier, Charing Cross Hospital and Westminster Medical School, Department of Psychiatry, 24 St. Dunstans Road, London W6 8RP, England.

**Eighteenth International Conference**

**International Society for Chronobiology**

From July 12–17, 1987, the 18th International Conference of the International Society for Chronobiology will be held at the Leeuwenhorst Congress Centre, near Leiden, The Netherlands. Registration information may be obtained from: Mrs. A.J. Goossen-Maartens, Department of Physiology, P.O. Box 9604, 2300 RC Leiden, The Netherlands (telephone (071) 148333, ext. 3625).
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