Recovery of Performance During Sleep Following Sleep Deprivation

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ABSTRACT

Very few studies have systematically examined recovery of performance after sleep deprivation. In the present study, 12 young adult males were sleep deprived for periods of 40 and 64 hrs. Each period was preceded by baseline nights of sleep and followed by two recovery nights of sleep. Immediate recall and reaction time were tested at 2300, 0145, 0400, 0615, and 0830 during baseline, deprivation, and recovery nights. Performance efficiency showed a progressive decline after 2 hrs of recovery sleep following both periods of deprivation. Return to baseline was apparent after 4 hrs of sleep following 40 hrs awake and after 8 hrs of sleep following 64 hrs awake. These results suggested that, in terms of behavioral efficiency, an equal amount of sleep is not required to compensate for sleep lost.

DESCRIPTORS: Total sleep deprivation, Recovery sleep, Performance, Body temperature, Awakening.

A substantial body of data is available demonstrating that sleep loss reduces efficiency on a variety of tasks (see Johnson & Naitoh, Note 1). Few efforts have been made, however, to systematically assess recovery of performance following sleep deprivation. Specifically, little is known about the amount of sleep required for recovery or the degree to which recovery of performance relates to the amount of prior wakefulness. The present study examined these issues.

Several studies of sleep deprivation have reported recovery of performance after a single (usually 8-hr) night of sleep following anywhere from 40 to 110 hrs of continuous wakefulness (e.g. Fenz & Craig, 1972; Lubin, Hord, Tracy, & Johnson, 1976; Webb & Agnew, 1973; Williams, Geiseking, & Lubin, 1966; Williams, Kearney, & Lubin, 1965; Williams, Lubin, & Goodnow, 1959). Taken together, these experiments suggest that an equal amount of sleep is not required to recover from sleep lost. However, sleep deprivation itself was the main concern of these studies and, therefore, recovery was given minimal attention.

There are only three studies with a primary focus on performance after recovery sleep, but each of these studies suffers from methodological difficulties. Moreover, none have adequately described the time course of recovery in terms of the amount of accrued sleep. Wilkinson (1963) reported that significant performance decrements were still evident after one night of recovery sleep. These results, however, may be inconclusive because recovery sleep began before the subjects' habitual bedtime. The resulting disruption of the circadian rhythm may not have allowed optimal conditions for recovery (Taub & Berger, 1973). Morgan, Coates, Brown, and Alluisi (Note 2) also placed recovery sleep at non-habitual times. This manipulation, as well as failure to control for circadian variations in performance (Johnson & Naitoh, Note 1), may have led to the incongruous 75% recovery reported after only 2 hrs of recovery sleep as compared to 56% recovery after 3 hrs of sleep. Additionally, baseline
sleep was not of similar length to recovery sleep in their experiment. Performance after awakening from sleep has been demonstrated to be less efficient than performance during normal waking hours (e.g. Langdon & Hartman, Note 3; Wilkinson & Stretton, 1971) and during intervals of wakefulness at the same circadian time as sleep (Ekstrand, Barrett, West, & Maier, 1977; Rutenfranz, Aschoff, & Mann, 1972; Stones, 1973, 1977). Therefore, the extent of recovery cannot be properly evaluated since there is no way of knowing how subjects performed following the same amounts of normal sleep.

Aschoff, Giedke, Poppel, and Wever (1972) examined recovery after 40 and 64 hrs of sleep deprivation as part of a larger project assessing the effects of sleep interruption and sleep deprivation on circadian rhythms. In addition to testing after a full night of recovery sleep following both periods of sleep deprivation, performance was assessed after awakening from sleep in the middle of the recovery night following 40 hrs awake. The poorest performance of the entire experiment was evident at 0300 and 0600 during this recovery night. Return to baseline was seen after a full night of recovery sleep following both periods of sleep deprivation. Although these data were suggestive of recovery within one night, no inferential statistics were reported and different performance tasks were used in the 40 and 64 hr conditions. Consequently, comparison of recovery between the two periods of sleep deprivation was not possible. In addition, there were no controls for the sleep stage from which subjects were awakened. Recent evidence (Bonnet, 1983; Dinges, Orne, Evans, & Orne, 1981; Stones, 1977) has suggested that arousal from the various sleep stages may have differential effects on waking behavior.

In sum, there have been few sleep deprivation studies with a primary focus on recovery of performance and none has provided a comprehensive assessment of recovery after differing amounts of sleep loss or recovery sleep. In order to test the possibility of recovery within a single night of sleep, the present study examined performance following 40 and 64 hrs of sleep loss with controls for time of day and the use of appropriate baseline sleep. The time course of recovery was assessed by performance testing at repeated awakenings across the night to compare the effects of 40 and 64 hrs of wakefulness within the recovery night.

Method

Subjects

Twelve males were selected from a pool of 250 respondents to advertisements in local newspapers. Screening of applicants was based upon their written responses to a Selected Medical History, Sleep Behavior Questionnaire, Brief Michigan Alcohol Screening Test, and Brief Drug Abuse Screening Test. Subjects were considered for further study if they reported an habitual 7.5 to 8.5 hrs sleep routine, falling between the hours of 2300 and 0830 on at least five days per week, for at least six months prior to the experiment. Individuals on this schedule were selected so that their sleep-wakefulness cycle would conform with the experimental regimen. This was required because it has been shown that experimentally altered sleep schedules (Taub & Berger, 1973), or the use of habitually irregular sleepers (Taub, 1978), may increase intersubject variability in performance. Criteria for exclusion from the study were: irregular sleep habits, habitual daytime naps, problems with falling asleep or staying asleep, frequent drug or alcohol usage, or major medical or psychiatric disorders. All subjects were drug-free for at least two weeks prior to the study and refrained from alcohol or caffeine consumption during the experiment. The subjects accepted into the study reported a mean of 8.1 hrs sleep (SD=.4), between 2300 and 0830, at least five days per week. These habits had been maintained for 0.5 to 5 yrs (mean=3) with no reported sleep disturbances. Napping frequency was less than once per week. Subjects ranged in age from 18 to 28 yrs with a mean of 22 yrs. Each participant was paid $210.00 for serving in the experiment.

Performance and Physiological Measures

Performance was assessed with the Williams Word Memory Test of immediate free recall (Williams et al., 1966) and with a simple auditory reaction time measure (Lisper & Kjellberg, 1972). These tasks were selected because of their demonstrated sensitivity to sleep deprivation and because each task reflected different performance capabilities, i.e., cognitive or perceptual-motor functions. Of equal importance was the brevity of each task since awakenings from sleep needed to be as short as possible. In addition to the performance measures, oral body temperature was recorded as an index of physiological activation, and continuous EEG was recorded during sleep to control for the sleep stage from which subjects were awakened.

Williams Word Memory. Stimuli were one or two syllable high frequency nouns. Two separate 15-word lists were presented via tape player at each testing session. A total of 60 lists were used in random order for each subject. To ensure sensory registration, the subject wrote each word as it was presented. A 2-min recall period was allowed after each list during which the subject rewrote, in any order, as many words as possible. The score was the sum of the words recalled from both lists.

Reaction Time. Stimuli were 1000 Hz tones, 300 msec in duration, with rise-decay times of 10 msec. A tape player was used to play 150 stimuli to the subject through headphones at 60dB(SPL) for 10 min. The mean inter-stimulus interval was 4 sec with a variation of ± 2 sec. During the task, the subject maintained an upright, sitting position on the side of his bed, with a pushbutton response switch held in his preferred hand resting on his knee. He was instructed to press the switch as quickly as possible upon hearing each tone. Reaction times were recorded in milliseconds on a digital clock interfaced with
a printer which recorded each response on-line. The score for each testing period was the median reaction time for the entire 10 min. For statistical analysis, these scores were transformed to their reciprocals (Myers, 1979) in order to better approximate a normal distribution. The transformed scores will be called Response Speed.

**Body Temperature.** Sublingual temperature was recorded with an electronic thermometer (Ivac Model 811). The temperature probe was allowed to warm for 10 minutes during the reaction time task to assure a reliable recording.

Sleep EEG. Continuous recordings of left and right EOG (referred to A2), and central (C2, A2) and occipital (Oz, A2) EEG were obtained using gold cup electrodes, applied to the face and scalp, interfaced with a Grass Model 78 polygraph. Sleep stages were identified according to the EEG criteria of Rechtschaffen and Kales (1968).

**Procedure**

Each subject slept in the same, private, sound-attenuated room throughout the study. Sleep was scheduled between 2330 and 0830 on both baseline and recovery nights. With time out for testing during the night, this was allowed for approximately 7.5 to 8 hrs of sleep. On most days, the subjects maintained their usual routine with a reminder to avoid daytime naps, caffeine, and over-exercising. All subjects arrived at 2230 for electrode application and pre-sleep testing.

During deprivation periods, subjects remained at the laboratory (under constant watch) and engaged in leisurely activities such as talking, watching television, reading, taking walks, showering, or playing board games with the technicians. All were requested to avoid over-exertion and lying down. Non-startling procedures were used by the technicians to awaken faltering subjects. Food and non-caffeinatted soft drinks were provided ad-lib. After one night awake, the subjects had the option of leaving the lab for a few hours (e.g. to attend classes if they were students) if they returned before evening. After two nights awake, it was required that the subjects remain at the lab throughout the next day.

**Results**

### Time of Awakening

Across all nights of sleep, mean times for the four awakenings for performance testing were: 0133 (SD=24 min), 0356 (SD=29 min), 0619 (SD=21 min), and 0819 (SD=15 min). Thus, performance as a function of time of night was comparable across all nights of the experiment.

<table>
<thead>
<tr>
<th>Procedures*</th>
<th>Nights</th>
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<tr>
<td>Groups</td>
<td>1 2 3 4 5 6 7 8</td>
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<tr>
<td>1 (N=6)</td>
<td>A A B B B D (40 hrs) R R</td>
</tr>
<tr>
<td>2 (N=6)</td>
<td>A A B B D D (64 hrs) R R</td>
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*A = Adaptation, B = Baseline Sleep, D = Sleep Deprivation, R = Recovery Sleep.
Sleep Stage at Awakening

The scoring of the sleep stage at awakening was based on the last full minute of any stage before the first presentation of the audio signals to arouse the subject. Eighty-nine percent of the total number of awakenings were from stage 2 sleep. During the first three times of awakening (the controlled awakenings), ten percent of the arousals were not from stage 2 sleep. These failures to awaken subjects from stage 2 were a result of time contractions or technical error. To examine the potential effects of sleep stage at arousal, the effects of sleep deprivation and recovery were analyzed both with all observations included and with all non-stage 2 awakenings deleted.

Shifts in Baseline Scores

The five baseline nights (see Table 1) of each group were first examined in a separate analysis of variance to test the possibility of baseline shifts due to repeated testing. No differences were found between the groups on any dependent measure. Within each group, however, baseline performance on both tasks significantly improved between the first and second weeks of the experiment.

Since baseline shifts were found, the baselines for all of the dependent measures used in the main analyses were expressed as weighted averages of the baseline nights surrounding each period of sleep deprivation (see Williams et al., 1966). This was accomplished in the following manner: average scores from each of the five times of night were first computed for the baseline night(s) immediately preceding the 40 and 64 hr conditions of sleep deprivation and recovery, and also for the baseline nights immediately following these conditions. The average scores for each time of night from the baseline nights before each condition and from the baseline nights after each condition, were then averaged together to form one mean baseline, with five testing times, for each condition of sleep deprivation and recovery, and also for the baseline nights immediately following these conditions. The average scores for each time of night from the baseline nights before each condition and from the baseline nights after each condition, were then averaged together to form one mean baseline, with five testing times, for each condition of sleep deprivation-recovery. For example (see Table 1), to form the baseline for the 40-hr condition, observations from each testing time on nights 3, 4, and 5 in group one were first averaged together and then averaged with data from the corresponding times on night 9. In group two, data from nights 9 and 10 were first averaged at each testing time, and then averaged with night 14 at each testing time.

Effects of Sleep Deprivation and Recovery

Before assessing the effects of sleep deprivation and recovery, the counterbalanced conditions (i.e. 40 and 64 hrs) of deprivation and their respective recovery nights, were collapsed across groups resulting in a single group of 12 subjects. Additionally, the first night of the two nights awake during the 64-hr period of sleep deprivation was excluded from analysis because it did not add any information to that gained from the night awake during 40 hrs of deprivation. These procedures resulted in the following eight nights being included in the analyses of sleep deprivation and recovery: the average baseline night for the 40-hr sleep deprivation period (B40), the average baseline night for the 64-hr sleep deprivation period (B64), the night awake during 40 hrs of deprivation (D40), the second night awake during 64 hrs (D64), the first recovery night following 40 or 64 hrs awake (R1-40 and R1-64, respectively), and the second recovery night following 40 or 64 hrs (R2-40 and R2-64, respectively). The data for each dependent measure were subjected to separate 8 (nights, listed above) by 5 (times of night) analyses of variance for repeated measures. These analyses were first calculated with all observations included and then recalculated with observations from non-stage 2 awakenings deleted. Since these deletions resulted in unequal numbers of observations in each cell, a least-squares regression procedure for estimating the sums of squares was applied. The least-squares analyses yielded the same pattern of results as the analyses which included all of the observations. Consequently, observations from all awakenings were used in all reported analyses to provide equal cell frequencies for pairwise comparisons of means.

The critical interaction of nights by time of night was significant for Williams Word Memory (F(4/45) = 3.42, p < .022), Response Speed (F(4/43) = 3.81, p < .013), and Body Temperature (F(4/48) = 4.15, p < .008) thus allowing assessment of the effects of deprivation and recovery with respect to time of night. Mean differences among the baseline, deprivation, and recovery nights were evaluated at each time of night with the Newman-Keuls procedure.

Williams Word Memory. Figure 1 shows the number of words recalled across the baseline nights (B40 and B64), deprivation nights (D40 and D64), and the first recovery nights (R1-40 and R1-64). Performance followed a U-shaped curve during the baseline nights while, during the deprivation nights, ability to recall gradually decreased below baseline with increasing hours of wakefulness. Recall was significantly below baseline at 0830 during D40

1The degrees of freedom for the interaction of nights by time of night were adjusted from the normal degrees of freedom (28 and 308 in each case) to compensate for any bias in the F ratios attributable to heterogeneity of variances and covariances. They were adjusted according to a formula given in Myers (1979, p. 172) which was based on the 40 X 40 variance-covariance matrix of the interaction for each measure.
Figure 1. Immediate recall during the night of 40 hrs sleep deprivation (D40), the second night of 64 hrs sleep deprivation (D64), the first recovery night following 40 hrs (R1-40), the first recovery night following 64 hrs (R1-64), and their respective baseline nights of sleep (B40 and B64).

(p<.05, Newman-Keuls) and at all times except 0130 during D64 (p<.05 in each case). These results verify that sleep deprivation had a detrimental effect on recall abilities and that this effect became worse with greater amounts of wakefulness.

There were continued performance decrements during both of the first recovery nights (R1-40 and R1-64) with the most precipitous decreases in recall occurring at 0130. Further, the extent of the performance decrements across the recovery nights varied with the amount of prior wakefulness. In both cases, however, recall abilities approached baseline by the final awakening. Comparisons among the means at each time of night revealed that recall was significantly below baseline at 2330 and 0130 during R1-40 (p<.01, Newman-Keuls), but had returned to baseline by 0400. During R1-64, ability to recall was significantly poorer than baseline from 2330 through 0615 (p<.01 in each case). Return to baseline was found at 0830. Comparisons between R1-40 and R1-64 showed that speed of responding was significantly lower (p<.01) at 0615 during R1-64, but not at any other time. Comparisons during the second Recovery nights (R2-40 and R2-64) revealed no significant differences from baseline.

Body temperature. Mean scores across baseline (B40 and B64), deprivation (D40 and D64) and the first recovery nights (R1-40 and R1-64) are presented in Figure 3. The U-shaped curve across the baseline nights is similar to the baseline curves for the performance measures. A general elevation of temperature during the deprivation nights, which is similar to Response Speed during the deprivation nights from 2330 through 0615 is again evident.

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Response Speed. Mean scores at each time of night during baseline (B40 and B64), deprivation (D40 and D64), and the first recovery nights (R1-40 and R1-64) are presented in Figure 2. A U-shaped curve of performance is apparent during the baseline nights which is similar to the curves for Williams Word Memory. Responses were generally faster during nights awake as compared to responses following awakening during a normal night of sleep. However, a gradual decline in speed of responding with increasing hours awake is apparent. Pairwise comparisons (Newman-Keuls) revealed that, during D40, responses were significantly faster than baseline from 0130 through 0615 (p<.01 in each case), but did not differ from baseline at 2330 or 0830. During D64, responses were significantly faster than baseline at 0130 (p<.01) and 0400 (p<.05), but did not differ from baseline at any other time. However, comparisons between D40 and D64 indicated that speed of responding at 0400 and 0615 during D64 was significantly slower (p<.01) than at those times during D40.

In contrast to nights awake, responses were generally slower than baseline during the first recovery nights (R1-40 and R1-64). Similar to recall scores, the extent of the performance decrement across the recovery nights varied with the amount of sleep deprivation. Pairwise comparisons revealed that, during R1-40, responses were significantly slower than baseline only at 0130 (p<.05). During R1-64, Response Speed did not differ from baseline during pre-sleep testing (2330), but fell significantly below baseline at 0130 (p<.01) and remained slower than baseline at 0400 (p<.05), 0615 (p<.01), and 0830 (p<.05). Comparisons between R1-40 and R1-64 showed that speed of responding was significantly slower (p<.01) at 0615 during R1-64, but not at any other time. Comparisons during the second Recovery nights (R2-40 and R2-64) revealed no significant differences from baseline.

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Figure 3. Body temperature during the night of 40 hrs sleep deprivation (D40), the second night of 64 hrs sleep deprivation (D64), the first recovery night following 40 hrs (R1-40), the first recovery night following 64 hrs (R1-64), and their respective baseline nights of sleep (B40 and B64).

nights, is also apparent. Pairwise comparisons revealed that temperature did not differ from baseline at 2330 or 0830 during D40, but it was significantly above baseline from 0130 through 0615 ($p<.01$ in each case). During D64, temperature was significantly below baseline at 2330 ($p<.05$), and then significantly above baseline at 0130 and 0400 ($p<.01$ in each case). No significant difference was found at 0830. During the first and second recovery nights following either 40 or 64 hrs awake, no significant differences from baseline were found at any time.

Discussion

The Effects of Sleep Deprivation

As expected from previous research, performance efficiency in both immediate recall and reaction time declined with increasing time awake. While the course of performance was similar for both tasks, the overall level of performance relative to baseline was quite different for the two tasks. During 40 hrs of deprivation, Williams Word Memory scores were significantly below baseline by the end of the deprivation night. During 64 hrs of deprivation, the memory scores were below baseline throughout the second deprivation night. In contrast, speed of responding was generally faster than baseline during the deprivation nights. However, cumulative effects of sleep deprivation were apparent in the progressive decline in response speed from the first night of deprivation to the second night of deprivation.

The fact that speed of responding was faster than baseline during sleep deprivation is contrary to previous findings. Other studies (e.g. Glenville, Broughton, Wing, & Wilkinson, 1978; Williams et al., 1959) have typically found reaction time to be slower than baseline during prolonged wakefulness. The uncharacteristic results in the present experiment may reflect important methodological differences between this experiment and the earlier investigations. The earlier studies tested performance under sleep deprivation during the daytime and compared these scores to baseline performance also during daylight hours. Because the present study was concerned with recovery sleep, performance testing occurred at night and baseline testing occurred after awakening from sleep. Thus, baseline conditions in the present study were quite different from other studies. Sleep prior to performance testing may have lowered baseline values in comparison to values collected during the sleep deprivation periods (see Ekstrand et al., 1977; Rutenfranz et al., 1972; Stones, 1973, 1977). This difference might have been most apparent in response speed because the task was performed immediately after awakening while the recall task was performed after 10 min of wakefulness.

The Effects of Recovery Sleep

The hypothesis that recovery of performance would be found within a single, 8-hr night of sleep following both 40 and 64 hrs of sleep deprivation was generally supported by the results of this experiment. Following 40 hrs awake, return to baseline performance was found for both response speed and Williams Word Memory after approximately 4 hrs of sleep. Following 64 hrs awake, return to baseline was found after 8 hrs of sleep for word memory. Response speed scores were still below baseline after 8 hrs of sleep. However, no significant differences from baseline were found for either performance measure during the second night of recovery sleep following 64 hrs awake. In addition, no significant differences in response speed were found when the first recovery night after 40 hrs (R1-40) was compared at 0830 to the first recovery night after 64 hrs (R1-64). Therefore, it was concluded that the residual performance decrement in response speed was minimal and that recovery of performance was probably complete after the first night of sleep following both periods of deprivation.

The multiple awakening paradigm of the present experiment permitted a description of the time course of performance within the recovery night. Examination of performance at each testing time indicated that 40 and 64 hrs of sleep deprivation had differential effects during recovery sleep. While the magnitude and the time of maximum performance decrement were similar for the two deprivation conditions, the time required to return to baseline
levels was twice as long after 64 hrs of deprivation as compared to 40 hrs of deprivation.

The continued decline in performance after 2 hrs of recovery sleep to levels below those reached after as much as 64 hrs of sleep deprivation is in accord with some findings in the literature but not with others. Aschoff et al. (1972) found similar performance decrements after approximately 3 hrs of recovery sleep in a design similar to that of the present experiment. Naitoh (1981) also reported performance decline after 2 hrs of recovery sleep placed at 0400. However, Naitoh found a return to baseline performance after 2 hrs of recovery sleep at noon. Morgan et al. (Note 2) reported an improvement in performance (to 75% of baseline) after 2 hrs of recovery sleep at 2200. The conflict between the results obtained by Naitoh and by Morgan et al. on the one hand, and by Aschoff et al. and the present investigation on the other, may result from the fact that Naitoh and Morgan et al. did not control for two factors which could affect performance. These factors include: 1) the presence or absence of sleep prior to baseline observations, and 2) circadian effects on the sleep process as well as on performance. Control for these factors can only be exercised by assessing performance after periods of baseline sleep, and by placing recovery sleep at its normal circadian time as was accomplished in the present study and in the work of Aschoff et al.

Given these controls, several possible explanations of the performance decrements found during recovery sleep must be considered. It is possible that performance decrements found early in the recovery night were simply a carryover or continuation of the decrements found during sleep deprivation. However, performance continued to decline below deprivation levels during early recovery sleep. The additional decline in efficiency suggests some extra influence on performance that was not present during sleep deprivation. Another possibility may be continuation of a disruption in circadian rhythm initiated during sleep deprivation. During the baseline nights, both body temperature and performance variables exhibited characteristic U-shaped curves (see Colquhoun, 1982) which were a reflection of a common circadian rhythm among the three measures. During the recovery night, however, body temperature quickly reverted to its U-shaped baseline pattern after being disrupted by sleep deprivation. In contrast, the performance measures showed considerable alterations from the baseline pattern during recovery sleep. These alterations suggest that the sleep process itself exerted the additional effects on performance. An important distinction between recovery sleep and normal sleep is the greater depth of recovery sleep as measured by sensory arousal thresholds (Williams, Hammack, Daly, Dement, & Lubin, 1964). It is plausible that deeper recovery sleep amplified performance decrements triggered by sleep deprivation, or magnified the sleep inertia (Lubin et al., 1976) of normal sleep, thus leaving the subject in a more deactivated state upon awakening. Bonnet (1983) and Åkerstedt and Gillberg (1979) have made similar suggestions to account for memory impairments following awakening from sleep.

The pattern of performance during the recovery night may offer insight into the recuperative nature of sleep. Sleep deprivation can be characterized as a period of extended energy depletion, and the concomitant performance decrements are probably a direct function of that energy depletion (see Bonnet, 1980). Since it has been proposed that sleep provides an optimum time for energy repletion and tissue restoration (Adam, 1980; Oswald, 1980), and since performance decrements are reversed by a significant amount of sleep (i.e. 4 or 8 hrs), it follows that recovery sleep is a time of intense energy recuperation. This argument is supported by striking parallels between the biochemical model of energy storage elaborated by Adam (1980) and the course of performance during recovery sleep. From the model it can be predicted that the rate of energy storage will be intensified in response to higher levels of energy expenditure. Similarly, if performance is dependent upon energy expenditure, then the energy recuperation process must be intensified during recovery sleep to more quickly compensate for the energy loss of prolonged wakefulness. The enhanced energy process is probably maximal in the first few hours of sleep in parallel to the maximal period of performance change. Further examination of the time course of recovery, as indexed by arousal thresholds and amounts of stage 4 sleep may reveal additional parallels to performance.

REFERENCES


Åkerstedt, T., & Gillberg, M. Effects of sleep deprivation on memory and sleep latencies in connection with repeated awakenings from sleep. Psychophysiology, 1979, 16, 49–52.

Aschoff, J., Giedke, H., Poppel, E., & Wever, R. The influence of sleep interruption and sleep deprivation on cir-


**REFERENCE NOTES**


(Manuscript received March 20, 1982; accepted for publication July 15, 1982)