

Effects of Sleep Deprivation on Sleepiness, Sleep Intensity, and Subsequent Sleep in the Rat

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Summary: The effects of 24 hr of sleep deprivation on cortical EEG and ventral hippocampus EEG recordings, ventral hippocampus spike rates, sleep stage percentages, and bout length measures were studied in rats. Two groups, differing only in the rate and distance they were forced to walk during deprivation by the water wheel method, were recorded continuously (23 hr per day) for one baseline, one deprivation, and two recovery days. During deprivation, microsleeps, increased hippocampal spike rates, and increased amplitude of the EEG recordings all suggested the intrusion of sleep processes. Nonetheless, there was no evidence to support the idea that these animals were not substantially deprived of sleep. No important differences were found in the recovery data of the two groups, even though one group walked three times as far as the other during deprivation. This supports the idea that, in conjunction with large amounts of sleep deprivation, changes in exercise and energy depletion may have little effect on sleep measures. During recovery, increased hippocampal spike rates and bout lengths, as well as increases in EEG amplitude, were interpreted in terms of increased sleep "intensity." High amplitude NREM sleep rebounded first, followed by rebounds in both paradoxical sleep and low amplitude NREM sleep. This pattern was compared to patterns previously reported for humans, cats, and rats. Finally, the tendency for some measures to fall *below* their baseline levels after an initial rebound was discussed in terms of "sleep inhibition" and servomechanism theory. **Key Words:** Sleep—Sleep deprivation—Sleepiness—Sleep intensity—Hippocampal spikes.

Although sleep deprivation experiments are a major tactic in studying the function of sleep, several ambiguities cloud their interpretation (Naitoh, 1976; Rechtschaffen, 1972; 1979). This study of 24 hr of sleep deprivation in the rat uses several procedures aimed at reducing these ambiguities.

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One major issue in sleep deprivation experiments concerns the effects of the stimuli used to induce deprivation. Sleep deprivation is not itself an experimental manipulation but a response to manipulations designed to prevent sleep, e.g., sleep disrupting stimuli, analeptic drugs, enforcement of continuous motor activity. Subsequent behavioral or physiological effects could result from either the sleep deprivation or other consequences of the experimental stimulation, e.g., exercise or energy depletion. In the present study we deprived rats of sleep by maintaining them on a rotating wheel partly submerged in water (Licklider and Bunch, 1946), so they had to walk at approximately the speed of wheel rotation to avoid the water. We used two speeds of wheel rotation which induced similar durations of sleep deprivation. In turn, this permitted some evaluation of the role of increased exercise and energy depletion, associated with the higher rotation rate, on the subsequent recovery from sleep deprivation.

A second issue concerns the efficacy of the deprivation procedure. Typically, as deprivation progresses, there are EEG signs of incipient sleep processes during behavioral wakefulness. Declines in alpha time (Bjerner, 1949; Armington and Mitnick, 1959) and a slowing of the EEG may appear in humans (Mirsky and Cardon, 1962; Williams et al., 1962; Naitoh et al., 1969) and cats (Feldman, 1961). Even these changes may not fully reflect the intrusions of partial or incipient sleep processes. In our laboratory, studies in cats (Hartse et al., 1979) and rats (Metz and Rechtschaffen, 1976; Metz, 1978) have shown that spike activity in the ventral hippocampus (VHS), which is typically most prevalent during non-rapid eye movement (NREM) sleep, tends to increase just prior to sleep onset before there are prominent changes in the EEG. In the present study, VHS rates were monitored during continuous recordings of the deprivation period in an attempt to measure the intrusions of NREM sleep related processes more sensitively.

A third issue is the measurement of recovery sleep. Increases in sleep time during recovery almost never compensate entirely for the lost sleep. The temporal compensation may not be complete because the recovery sleep is in some sense more "intense." Sleep is defined by changes along certain dimensions (e.g., arousal threshold, EEG amplitude). In the absence of conclusive information on the functionally important dimensions of sleep, it seems reasonable to try to measure sleep intensity by changes along these definitional dimensions. For example, Williams et al. (1964) found increased auditory arousal thresholds during recovery sleep in humans. Frederickson and Rechtschaffen (1978) found increased arousal thresholds for auditory (but not trigeminal nerve) stimulation during recovery sleep in rats. In the present study we used several putative measures of sleep intensity.

EEG amplitude. The separation of NREM sleep into a low amplitude phase and a high amplitude phase has yielded important information in cat (Ursin, 1971) and human (e.g., Berger and Oswald, 1962) sleep deprivation studies, since the two phases respond differently. This differentiation of NREM stages had not previously been made in sleep deprivation studies of the rat. Therefore, we divided rat NREM sleep into S1 (relatively low amplitude) and S2 (relatively high amplitude) sleep.

Typically, stage scoring decisions are based on visual detection of relative EEG amplitude changes within a day. Small but consistent changes in EEG absolute amplitude can go undetected with this method. The present study utilized an automatic scoring system which mimics relativistic visual scoring (Bergmann et al., 1977b), but also provides information on the absolute amplitude changes that occur from day to day.

Ventral hippocampal spikes. VHS rate may reflect not only incipient NREM sleep processes during wakefulness, but also the intensity of NREM sleep itself. Studies of the cat (Hartse et al., 1979) showed that the spike rates declined in the 30 sec of NREM sleep just preceding awakening or paradoxical sleep (PS) periods, before there were apparent changes in the EEG. Accordingly, we monitored VHS rates as possible indicators of NREM sleep intensity.

Sleep bout length. It is reasonable to hypothesize that a more intense sleep might show a decreased vulnerability to interruption by internal and external stimuli as well as to unknown processes which spontaneously produce awakenings. Accordingly we monitored the length of periods of uninterrupted sleep (sleep bout length).

In addition to the refinements in the sleep deprivation experiment described above, the present study provides data from a relatively large number of animals (as sleep deprivation experiments go) on the distribution of sleep stages during recovery sleep. The order in which the different stages recover following total sleep deprivation is thought to have relevance for the issue of which stages are most important. As we shall see later in the discussion, there is considerable variance in the data on this issue.

METHODS

Sixteen adult male Sprague-Dawley albino rats weighing 280–400 g were recorded for a variable number of prebaseline days and for four consecutive experimental days: one baseline, one sleep deprivation, and two recovery days. Food and water were available *ad lib* except during deprivation (see below).

The cortical EEG was recorded unilaterally from two stainless steel screws, one approximately 3 mm lateral to bregma and the other 4 mm lateral to lambda. Electromyogram (EMG) was recorded from silver plates cemented to the skull beneath the temporalis muscle (Rosenberg et al., 1976) in 11 rats and from stainless steel wire loops in the cervical musculature in 5 rats. Bilateral VH recordings were made from teflon-insulated stainless steel wire electrodes (125 μ ; Medwire), twisted together to form a rigid shaft and stripped of insulation for 0.5 mm at the tips. The tips were spaced 1 mm apart and electroplated with platinum to lower resistance. Hippocampus electrodes were inserted stereotactically to coordinates 2.4A, 4.5L, -3.5H (Pellegrino and Cushman, 1967) and fixed with dental cement. The leads from all electrodes were soldered to a miniature connector cemented to the skull. VH placements were histologically verified in all animals with 50 μ cresyl violet-stained frozen sections.

All recordings were made with the animals in a Plexiglas chamber with a removable floor. Just beneath the floor was a 7.6 cm diameter horizontal cylinder which

could be rotated by a variable speed motor. During deprivation the chamber was filled with water to the middle of the cylinder, the floor was removed, and the cylinder was rotated.

After surgery, animals were placed in a recording room. An LD 12:12 schedule with light onset at 2 p.m. was maintained. After a minimum of 7 days of recovery from surgery, rats were placed in the experimental chamber, and a Microdot wire recording cable was attached to the electrode connector. This junction was sealed with silicone rubber and tape. On the first day in the chamber, trial recordings were obtained, and the rats were adapted to the water wheel. Two training periods of 2 hr each (one in the light and one in the dark) were provided to allow the rat to adjust to the deprivation procedure. On the second day, prebaseline recording was begun. The animals were recorded for 23 hr each day. One hour was used for calibration of the equipment and care of the rat. Prebaseline recordings were continued until the recordings stabilized. Before deprivation could begin, 2 days of recording with similar scoring criteria and sleep percentages had to be obtained. The last of these 2 days was used as the baseline day for statistical comparisons. Deprivation began at light onset and lasted 24 hr. The animals were randomly assigned to one of two groups: a slow group, for which the water wheel was rotated at 1 rpm, and a fast one, for which the wheel was rotated at 3 rpm. At 3 hr intervals during the deprivation the cylinder was stopped, the floor replaced, and the animals were given access to food and water for 10 min. At the end of the deprivation period the floor was replaced and the animals were allowed to recover. Two days of recovery recordings were taken.

Initial amplification and ink recordings of EEG, EMG, and hippocampus activity were made on a Beckman Type R polygraph. The signals were filtered as follows: the EEG was bandpass filtered at 2.5 Hz (12 dB/oct) to 12 Hz (24 dB/oct); the EMG was high pass filtered at 30 Hz (24 dB/oct) with a 24 dB notch filter at 60 Hz; the VH recordings were bandpass filtered at 8 Hz (6 dB/oct) to 40 Hz (6 dB/oct). The amplitudes of the cortical EEG, EMG, and VH EEG recordings were quantified by an integrator which reset when a preset voltage was reached. Thus, the number of resets per epoch (30 sec period) was proportional to accumulated voltage in that epoch.

To detect spikes in the hippocampal recordings, the signals (polygraph output) were first full-wave rectified and then passed to Schmitt triggers which set amplitude criteria for spikes to be counted. Criteria were set for each animal to detect only the highest amplitude spikes. Resets and spikes were totaled for each epoch, transferred to a PDP11-10 computer, and stored on flexible disk. Integrator channels were calibrated daily with a standard 200 μ V, (100 Hz for EMG) sine wave and the Schmitt trigger levels were checked for drift. The voltage required to reset the integrators was varied from animal to animal according to individual amplitudes so that a reasonable range of reset values could be obtained for each measure. For statistical analyses, reset rates were converted to microvolts according to calibration values.

Each 30 sec epoch was scored for wakefulness or sleep stage by a modified version of the computer scoring system developed by Bergmann et al. (1977b). This parametric method uses relative EEG and relative EMG amplitudes of indi-

vidual animals to classify each epoch as either waking (W), moderate amplitude NREM sleep (S1), high amplitude NREM sleep (S2), or PS. The original system has been validated against both visual scoring of the polygraph recordings and visual observation of the rats' behavior (Bergmann et al., 1977a; Bergmann, 1978).

The first step in the scoring is the construction of a frequency histogram of EEG amplitudes for one day's recording of each animal (Fig. 1), i.e., the number of epochs at each EEG amplitude reset value plotted against that value. Typically, there is a high incidence of low reset values corresponding to the low amplitudes of W or PS. Then there is a relatively low incidence of intermediate reset rates generally corresponding to transition periods and beginning S1 sleep. Finally, a second mode (generally less distinct than the first) corresponds to the higher EEG amplitudes of NREM sleep. An arbitrary "range" is computed for the distribution to eliminate capricious extreme values in establishing scoring boundaries. The low end of the range is the lowest EEG amplitude reset value to contain 5% of the day's epochs. The high end of the range is the highest value to contain the same number of epochs. Scoring boundaries T1 and T2 are defined as falling at one-third and two-thirds of this range, respectively. These boundaries on the EEG frequency histogram are used to discriminate W and PS (below T1) from S1 (between T1 and T2) and from S2 (above T2). W and PS are discriminated by EMG levels and the history of the epoch.

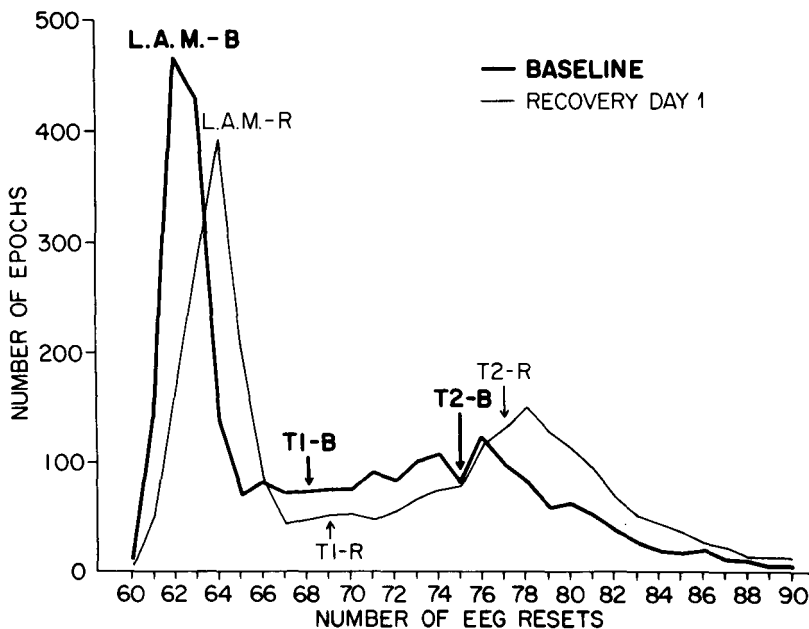


FIG. 1. Frequency histograms of cortical EEG amplitude (resets) from one rat before and after sleep deprivation. T1 and T2 are scoring criteria (see text), L.A.M. stands for "low amplitude mode." Note shift of the recovery histogram to a higher position on the amplitude (reset) axis during recovery.

In the original system, to score PS, one of the two preceding epochs had to have been scored as S1, S2, or PS; i.e., PS could not occur immediately after more than one epoch of W. This rule had been adopted for normal rats because the integrated EMG can occasionally drop to very low levels for a few epochs in behaviorally awake animals just prior to sleep onset. (Similar rules are sometimes used implicitly in visual scoring procedures.) Even with this rule, however, PS was overscored during deprivation when, perhaps as a result of fatigue or "attempts" to achieve microsleeps, rats could have very low EMGs combined with low EEG levels for several epochs at a time following microsleeps. Some of these epochs represent wakefulness, as indicated by small, intermittent movements which do not contribute substantially to the total integrated EMG. Others may represent a drowsy or semi-sleep state. In either case, such epochs are not unambiguously PS. Recent unpublished work in our laboratory indicates that such epochs lack the strong hippocampal theta characteristic of rat PS. To protect against overscoring PS during deprivation, the original criteria were made more stringent by requiring that three of the 10 previous epochs had to be scored as sleep (S1, S2, or PS) before an epoch could be scored as PS. This modification virtually eliminated the scoring of PS during deprivation, but it had no significant effect on the scoring of baseline data.

The deprivation day was scored using the previous baseline day's scoring boundaries. The recovery days were scored on the basis of their own boundaries for reasons which will become clear in the Discussion section. T1 and T2 were used not only as scoring boundaries, but also as possible indicators of a shift in the EEG histogram. The low amplitude mode (LAM; see Fig. 1) was also used in this way. The high amplitude peak was usually too shallow to choose a meaningful mode.

Only one of the two hippocampus recordings for each animal was included in the analyses. To be included in the study, a hippocampus recording had to be free of movement artifact and have higher spike rates in epochs of S1 and S2 than in W and PS. If both hippocampus recordings met these criteria, as most did, the recording that contained spikes with the largest signal-to-noise ratio, as determined by a "blind" judge, was used.

For the analyses of some of the variables, each day was divided into four blocks: the first three were each 6 hr long; the last was 5 hr because the 1 hr used for care of the animals and equipment calibration was not scored. Time blocks 1 and 2 were in the light and 3 and 4 were in the dark. Originally, the data were scored and inspected on an hour-by-hour basis. They were collapsed into the four daily blocks for ease of statistical analysis and data presentation after inspection revealed that the major systematic changes which resulted from deprivation were captured by the four block analysis. Percent of total time spent in each stage, mean VHS rate per epoch per stage, ventral hippocampus EEG amplitude (VHA) per epoch per stage, and mean bout length (periods of sleep uninterrupted by more than one epoch of W) were calculated for each time block. Since there was very little sleep on deprivation days, sleep stage data and sleep specific variables (e.g., mean VHS rate in PS; bout lengths) were analyzed only for baseline and recovery days. Wake related variables (Mean VHS rate and VHA rate in W) were analyzed for the deprivation day as well.

Sleep stage data were analyzed multivariately to take into consideration the inherent correlations among the percentages of time in each stage. To do this, a log transformation of the sleep stage data (expressed as percents) was performed. A three-way ANOVA with repeated measures was employed for significance testing. Animals were nested in the group factor (slow versus fast) and crossed with time (blocks 1 through 4) and day factors (baseline and two recovery days). The other variables measured in time blocks were analyzed univariately with the same design (with the deprivation day included for the wake related variables). If there was a significant overall interaction of time and day factors, then *post hoc* tests on these variables consisted of a comparison of a block of time in recovery with the parallel block of time in baseline.

Two other sets of variables were analyzed in units of a day. First, the EEG histogram variables (T1, T2, and the LAM) were considered in units of a day because they were determined by an entire day's recording. These variables were each univariately analyzed in a two-way ANOVA (repeated measures) with subjects nested in the group factor (slow versus fast) and crossed with the day factor (baseline and two recovery days). The deprivation day was included in the LAM tests. Second, the proportion of the time that epochs of all four stages were followed by another epoch of the same stage (stage continuity) was computed in units of a day, for convenience. A transformation to stabilize the variances of proportions ($2 \times \arcsin \sqrt{X}$) was performed, and the data were then analyzed multivariately with the same design used for the EEG histogram variables. When there was a significant day main effect, *post hoc* tests on these sets of variables always consisted of comparisons of a recovery day to the baseline day.

RESULTS

Observations of Deprivation Period

The rats adapted well to the deprivation procedure. After pretraining in the deprivation apparatus, the rats very rarely fell into the water. Although food and water were available at intervals throughout the deprivation, the rats did not drink and rarely ate during the deprivation day. The quality of the recordings was maintained even while rats were on the water wheel. Movement artifact in the recordings was not a problem.

Effects of Wheel Speed

Total sleep of both the slow and fast wheel groups was markedly reduced during the deprivation period. The slow speed group had somewhat more sleep (8.8% of total time) than the fast group (1.9%), but the difference was not statistically significant ($p < 0.11$). The two groups did not differ significantly or substantially during recovery on total sleep, sleep stage percentages, VHS rates, cortical EEG amplitudes, or sleep bout lengths.

The groups showed different patterns of statistically significant baseline versus recovery differences on two parameters. One was the recovery pattern of hippocampal EEG amplitude in S1 (VHA in S1). Both groups showed elevations of hippocampal EEG amplitude in all blocks of recovery day 1 relative to their own

baselines. However, the fast group showed a significant rise only for block 3, whereas the slow group showed a significant rise only for block 1. On recovery day 2 both groups showed below baseline values. However, only the last three blocks of the fast group were significantly different from baseline levels. The second group difference was in the recovery pattern of VHA in S2. Although VHA in S2 was elevated on recovery day 1 (as a whole) for both groups, only the fast group increased significantly. Considering the overall similarity of the two groups, even on these variables, and considering the huge number of comparisons made between them, we did not think that the group differences in VHA during recovery were a sufficient basis for complicating the presentation of the results by reporting the two groups separately. Therefore, the two groups were combined in all subsequent analyses.

Sleep Intrusions During Deprivation

The sleep during deprivation consisted of short bursts of elevated amplitude EEG activity commonly known as "microsleeps." As reported by Levitt (1967), these microsleeps occurred when the rats were riding, not walking, on the cylinder. There was a significant linear component to the increase in sleep as deprivation progressed ($p < 0.01$).

In addition to the microsleeps, there were signs of incipient sleep processes intruding into wakefulness during deprivation. Waking EEG amplitude increased, as indicated by a significant increase in the LAM of the EEG histogram (6.1% increase, $p < 0.01$). Also, waking spike rates (VHS in W) were significantly increased during blocks 2, 3, and 4, and VH EEG amplitude (VHA in W) was elevated significantly in blocks 1, 3, and 4 (Table 1).

To examine whether the sleep or sleep related events which occurred during deprivation had a functional role in reducing recovery sleep, the reduction in total sleep time and the increments in VHS in W, VHA in W, and the LAM relative to baseline values were correlated with the increments in total sleep percent and S2 percent on recovery day 1 relative to baseline values. None of these correlations approached significance. Thus, there was no support for the hypothesis that sleepiness during deprivation functionally reduces the sleep need.

TABLE 1. Mean percent change from baseline of sleep and hippocampal measures during deprivation

	Deprivation day			
	Block 1	Block 2	Block 3	Block 4
Total sleep ^a	-97.1	-93.5	-73.3	-86.2
VHS in W ^b	+27.0	+48.7 ^d	+61.4 ^d	+55.0 ^d
VHA in W ^b	+4.1 ^c	+3.6	+9.0 ^c	+4.3 ^c

^a Significance testing not done here.

^b For omnibus F and p values, see Table 2.

^c $p < 0.05$ on Tukey's test, compared to baseline.

^d $p < 0.01$ on Tukey's test, compared to baseline.

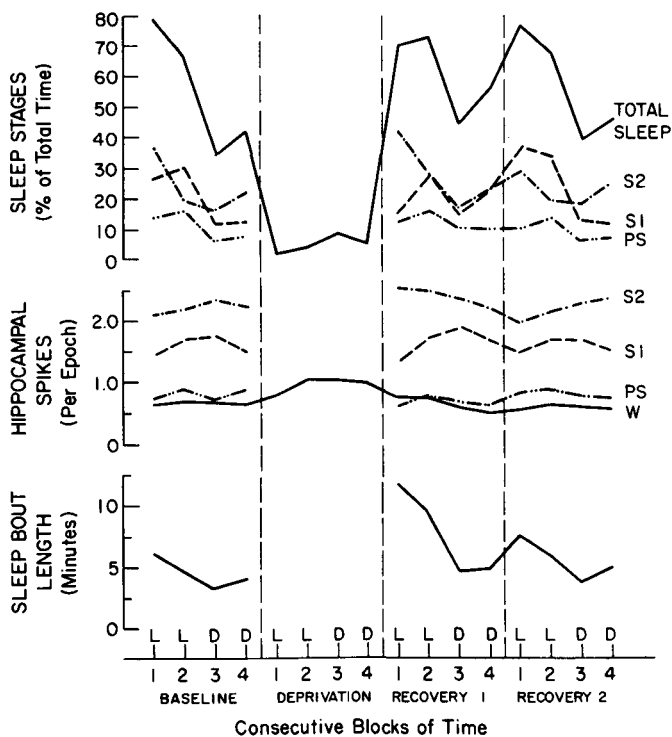


FIG. 2. Mean sleep stage percentages, hippocampal spikes rates, and sleep bout lengths for both fast and slow speed groups combined. Blocks 1, 2, and 3 are 6 hr long; block 4 is 5 hr.

General Observations on Recovery

The first 6 hr of recovery were characterized by a reduction rather than an increase in total sleep relative to the first 6 hr block (block 1) of baseline (Fig. 2 and Table 2). Although the recovery began at light onset, when nondeprived rats are most likely to sleep, at the start of recovery rats took a mean of 52.2 min to achieve a sustained (3 min or more) state of sleep compared to a sleep latency of only 7.5 min in baseline ($p < 0.01$). The rats spent a great deal of time grooming, eating, and drinking during this early recovery period.

Interspersed through the first hour of recovery were periods when both the EEG and EMG were elevated, indicating a NREM-like brain state with eating behavior. On the average, a rat had a total of 10 min of such activity. These periods were not analyzed with the rest of the data.

Putative Measures of Sleep Intensity

Several of the putative measures of sleep intensity showed significant changes during recovery. However, the amount and temporal pattern of change varied among measures.

Although VHS rates had increased markedly during W in the deprivation con-

TABLE 2. Mean percent change from baseline of sleep and hippocampal measures by blocks

	Recovery day 1				Recovery day 2			
	Block 1	Block 2	Block 3	Block 4	Block 1	Block 2	Block 3	Block 4
Total sleep ^a	-9.4 ^d	+12.3 ^d	+31.7 ^d	+32.0 ^d	-0.9	+3.5	+13.6	+9.4
% Stage S1 ^a	-39.3 ^d	-7.3	+57.4 ^d	+78.8 ^d	+42.4 ^d	+11.4	+14.6	-0.7
% Stage S2 ^a	+14.7	+49.3 ^d	-5.8	+4.0	-20.3 ^d	+3.2	+17.7	+18.4 ^c
% Stage PS ^a	-4.9	+4.2	+83.1 ^d	+37.9 ^d	-18.0	-10.8	+12.9	-0.7
VHS in W ^b	+15.6	+3.2	-15.9	-24.6	-11.7	-9.7	-10.8	-10.1
VHS in S1 ^b	-6.9	+2.9	+6.9	+15.4	+3.4	+1.2	-2.3	-1.3
VHS in S2 ^b	+19.4 ^d	+15.0 ^c	+2.6	+1.3	-4.5	-1.9	-0.4	+6.6
VHS in PS ^b	-17.9	-8.3	-4.6	-24.2	+10.7	-0.9	+7.8	-13.1
VHA in W ^b	+3.9	+2.4	+2.0	-1.9	-1.8	-5.5 ^d	-4.7 ^d	-5.4 ^d
VHA in S1 ^b	+3.6 ^e	+3.8 ^e	+4.1 ^e	+2.8 ^e	-1.6 ^e	-2.9 ^e	-3.8 ^e	-3.7 ^e
VHA in S2 ^b	+10.3 ^d	+4.5 ^d	+2.3 ^c	+0.1	+1.2	-2.8 ^d	-2.6 ^d	-2.9 ^d
VHA in PS ^b	+5.2 ^d	+4.7 ^d	+8.2 ^d	+4.0 ^d	-3.1	-3.1	+0.6	-0.3
Mean bout length ^b	+90.9 ^d	+95.3 ^d	+42.0	+18.2	+24.2	+20.1	+11.2	+20.3

^a Sleep percentages tested multivariately (MANOVA $F = 11.2$, $df = 24/283.8$, $p < 0.0001$; univariate F values: Total sleep: $F = 9.5$, $df = 6/84$, $p < 0.0001$; S1: $F = 40.2$, $df = 6/84$, $p < 0.0001$; S2: $F = 11.7$, $df = 6/84$, $p < 0.0001$; PS: $F = 5.6$, $df = 6/84$, $p < 0.0001$), protected t -tests used (see Bock, 1974, p. 266).

^b VHS, VHA, and MBL tested univariately (VHS in W: $F = 3.2$, $df = 9/126$, $p < 0.0015$; VHS in S2: $F = 4.3$, $df = 6/84$, $p < 0.0009$; VHA in S2: $F = 10.4$, $df = 6/84$, $p < 0.0001$; VHA in PS: $F = 3.1$, $df = 6/84$, $p < 0.0087$; MBL: $F = 6.9$, $df = 6/84$, $p < 0.0001$), Tukey's test used.

^c $p < 0.05$, as compared to same period in baseline.

^d $p < 0.01$, as compared to same period in baseline.

^e This variable revealed significant changes only in units of a day ($F = 30.0$, $df = 2/28$, $p < 0.01$; recovery day 1 increase: $p < 0.01$; recovery day 2 decrease: $p < 0.01$).

dition, they increased significantly over comparable baseline periods only during S2 (VHS in S2) for the first two blocks of recovery day 1 (Fig. 2 and Table 2). Given that there were 32 tests of significance of spike rate changes during recovery (four stages \times eight blocks), it is possible that the significant increases during the first two recovery blocks in S2 could have resulted by chance. This appears unlikely, since there were other indications of particularly strong intensification of sleep at these times (see below). Apart from the increments in VHS in S2, however, there was little to suggest that VHS rates sensitively reflected sleep intensification during recovery. Of the 32 comparisons, VHS rates were above baseline values in only 14 instances. Of the 16 comparisons on recovery day 1, spike rates were above baseline in only nine instances.

Mean sleep bout length increased significantly and dramatically by over 90% in the first two blocks of recovery day 1 compared to the first two blocks of baseline (Fig. 2 and Table 2). Although none of the remaining block values differed significantly from levels during comparable baseline periods, mean sleep bout length remained elevated throughout the two recovery days.

A measure related to bout length, sleep continuity (the proportion of epoch successions in which one epoch of a state was immediately followed by an epoch of the same state), showed a pattern similar to that of bout length. The continuity of each of the sleep stages was elevated above baseline values for recovery day 1. The continuities of S2 and PS receded to near baseline levels on recovery day 2, but the continuity of S1 remained elevated (Table 3).

TABLE 3. Mean percent change of EEG histogram variables and measures of continuity (units of a day)

	Recovery day 1	Recovery day 2
EEG histogram ^a		
T1	+2.6 ^b	-3.5 ^d
T2	+2.7 ^b	-4.2 ^d
LAM	+2.7	-1.0
Stage continuity ^f		
W-W	+1.6	+0.6
S1-S1	+6.5 ^c	+9.4 ^e
S2-S2	+12.5 ^e	+0.8
PS-PS	+11.9 ^e	+0.5

^a EEG histogram variables tested univariately (T1: $F = 18.3$, $df = 2/28$, $p < 0.001$; T2: $F = 20.4$, $df = 2/28$, $p < 0.001$; LAM: $F = 8.0$, $df = 3/42$, $p < 0.001$), Tukey's test used.

^b $p < 0.05$, as compared to baseline day.

^c $p < 0.02$, as compared to baseline day.

^d $p < 0.01$, as compared to baseline day.

^e $p < 0.002$, as compared to baseline day.

^f Transitions tested multivariately (MANOVA $F = 19.3$, $df = 8/50$, $p < 0.0001$; univariate F values: S1-S1: $F = 8.3$, $df = 2/28$, $p < 0.0015$; S2-S2: $F = 80.9$, $df = 2/28$, $p < 0.0001$; PS-PS: $F = 21.6$, $df = 2/28$, $p < 0.0001$), protected t -tests used (see Bock, 1974, p. 266).

EEG measures of sleep intensity showed a somewhat remarkable pattern. There were strong tendencies for EEG amplitude to increase above baseline levels on the first recovery day, as might be expected, and then to decrease below baseline levels on the second recovery day. Changes in cortical EEG amplitude are reflected in changes in the histogram of EEG resets (see Fig. 1), with a greater incidence of high reset epochs indicating increases in EEG amplitude. In turn, such changes in histogram distribution are reflected in the T1 and T2 values used as scoring criteria, with higher values indicating an increase in the incidence of epochs with high EEG. Although it is not used as a formal scoring parameter, changes in the low amplitude mode (LAM) of the histogram reflect changes in EEG amplitude in a similar manner. Thus, changes in LAM, T1, and T2 reflect EEG amplitude changes in three different areas of the histogram. The extent of these changes is shown in Table 3. All three measures showed an increase in cortical EEG amplitude on the first recovery day, but only the T1 and T2 changes were statistically significant. Although the percentage increases above baseline were small, significance was achieved because of the great consistency of the change across almost all animals. (A shift of the EEG reset histogram from baseline to recovery day 1 in one animal is illustrated in Fig. 1.) All three of the measures reversed on recovery day 2 to show decreases to *below* the baseline mean, again with only T1 and T2 measures reaching significance.

A similar pattern of results was evident in the amplitude of the EEG recorded from the ventral hippocampus (VHA). The changes in VHA in S1 were statistically significant only when considered in units of a day. There was a significant increase in this variable on recovery day 1 and a significant *decrease* on recovery day 2. Of the remaining 12 block-by-block comparisons between VHA on baseline and recovery day 1 (four blocks \times three stages), 11 showed elevations on the recovery day, with seven of these at a statistically significant level (Table 2). Of the 12 comparisons between baseline and recovery day 2, 10 showed *decreases* to below the baseline level on the recovery day, with six of these reaching statistical significance.

Sleep Stage Recovery Patterns

During recovery, total sleep time did not change greatly from baseline levels, but the percent of time spent in the various stages of sleep changed dramatically. Total sleep time was decreased in the first block of recovery day 1, as mentioned earlier, but was elevated for the rest of this day. However, the increases in total sleep time in blocks 2, 3, and 4 were modest compared to the changes any particular stage might show (Fig. 2 and Table 2). Percent S1 was significantly decreased in the first block of recovery day 1, but was significantly increased in the third and fourth blocks of this day as well as in the first block of recovery day 2. Percent S2 was significantly elevated in the second block of recovery day 1 but relatively unchanged in the next two blocks, significantly decreased in the first block of recovery day 2, and significantly increased in the last block of this day. Percent PS was relatively unchanged until the significant increases of the third and fourth blocks of recovery day 1. There was no significant change of PS percent on recovery day 2.

Significance testing on the changes in sleep stage percentages considered in units of a day was not done. For purposes of comparison with other published work, the changes in percentage of total time (in units of a day), are given: Total sleep time was 55.8 on the baseline day, increased to 61.7 on recovery day 1, and decreased to 58.4 on recovery day 2. S1 was 20.5 on the baseline day, increased to 21.1 on recovery day 1, and increased again to 24.7 on recovery day 2. S2 was 23.8 on the baseline day, increased to 27.7 on recovery day 1, and decreased to 23.7 on recovery day 2. PS was 11.0 on the baseline day, increased to 12.9 on recovery day 1, and decreased to 10.0 on recovery day 2.

DISCUSSION

Sleep Deprivation, Exercise, and Energy Depletion

Manipulations which reduce sleep can affect other variables which may influence subsequent sleep independent of the sleep loss. In the current study, the water wheel not only reduced sleep but also enforced exercise.¹ Some studies have shown that exercise potentiates human NREM sleep stages 3 and 4, whereas other studies have not (see Horne and Porter, 1976, or Moses et al., 1977, for references). In cats, treadmill exercise for 2 hr potentiated slow wave sleep and delayed PS, but sleep deprivation by the hand method for 2 hr did not (Hobson, 1968). The delaying of PS by exercise was also reported by Matsumoto et al. (1968) in rats. Since we found an early potentiation of S2 and delayed potentiation of PS during recovery, the confounding of exercise with sleep deprivation presents interpretive obstacles.

On the other hand, there is evidence that, in conjunction with relatively long durations of sleep deprivation, exercise may not strongly affect sleep measures. The recovery sleep of our fast and slow animals was not substantially different. In humans, two nights of sleep deprivation with bed rest and two nights of sleep deprivation with exercise did not produce different recovery effects (Webb and Agnew, 1973). In a similar study, with one night of sleep deprivation, Moses et al. (1977) also failed to find substantial differences in the recovery effects on sleep stage amounts and several other sleep measures. These authors did report a statistically significant greater increase in percentage of total bed time spent asleep after combined exercise and deprivation than after combined bed rest and deprivation. This result is difficult to interpret. "Total sleep time" was defined as the sum of all the sleep stages except stage 1, which most investigators include as sleep. Since, in baseline, the exercise group appeared to have substantially more stage 1 and wakefulness than the bed rest group, there was more room for this "total sleep" measure to increase in the exercise group. This is an important consideration because time in bed was limited to 8 hr, and sleep amounts were already quite high in baseline. Also, the groups did not differ significantly in the decrease of unambiguous wakefulness (with stage 1 not included as wakefulness) following deprivation. Furthermore, since bed time was restricted, we cannot

¹ Our slow group traveled about 0.2 miles and our fast group about 0.6 miles, quite small compared to the 30 to 40 miles a day rats may voluntarily run on a wheel (Richter, 1967).

know how the two groups would have compared on total time if allowed to sleep *ad lib*.

Since our rats reduced their food and water intake during the sleep deprivation period, sleep recovery patterns could have been influenced by hunger and thirst. As indicated earlier, the first few hours of the recovery period were almost certainly influenced by the large amount of time the rats spent eating, drinking, and grooming when first removed from the water wheel. However, it is unlikely that the prior deprivation of food and water had much effect on sleep parameters during the majority of the recovery period, because it must have been at least partly alleviated by the eating and drinking at the start of recovery. Also, other research has shown minimal effects of one day of food or food and water deprivation on sleep in the rat. Jacobs and McGinty (1971) observed no change in NREM sleep after one day of food deprivation. PS was significantly elevated, but the increment was small, averaging about 2% of total recording time. Borbely (1977) found no significant changes in total NREM or PS sleep after one day of food deprivation. There was less PS at night but more during the day—a shift which could not account for the rebounding of PS during the dark period in our experiment. Borbely found a shortening of sleep bout length following food deprivation, which contrasts with the huge increases in bout length during our recovery periods. Yang and Iwahara (1971) found little change in NREM sleep after one day of food and water deprivation. Their data, by our calculations, show a small rise in PS comparable to the one observed by Jacobs and McGinty. It is therefore conceivable that some of the PS rebound on our recovery day 1 could have resulted from prior food deprivation. However, it seems unlikely that the deprivation would have produced a delay of PS augmentation until the second half (dark period) of the day such as we observed, especially in view of the Borbely finding that PS was shifted toward the light period during food deprivation.

Given that the prior food and water deprivation probably did not have much effect on sleep recovery patterns, we may now ask whether the eating and drinking at the start of recovery day 1 had much effect on subsequent sleep patterns. We have no good data for evaluating a possible postdrinking effect, but the data of Borbely (1977) on sleep patterns during refeeding after 3 days of food deprivation provide some basis of comparison with respect to a postprandial effect. His data show little difference between sleep stage amounts on refeeding and control days, suggesting that sleep stage amounts during our recovery days were not greatly affected by refeeding. Borbely did find increments in sleep episode duration during refeeding, so some part of the increased sleep bout length we observed on recovery days could have been a postprandial effect. However, the postprandial portion of the increased sleep bout length was probably quite small, since our increases in sleep bout length were much larger than those in the Borbely study. Exact comparisons between the two studies are difficult because of differences in scoring and quantitative presentation. Nevertheless, we estimated that the maximal percentage increments in sleep bout duration over control levels were about three times as great during recovery from sleep deprivation than during the refeeding period of the Borbely study.

Since the rats in both our fast and slow group rarely ate during deprivation, and

since the fast group walked three times as far as the slow group, we can infer that the groups were differentially energy depleted. The absence of any substantial differences in the recovery sleep of the groups suggests that energy depletion during 24 hr of sleep deprivation is a relatively unimportant variable.

Feasibility of Deprivation

Dement (1972) has questioned the feasibility of experimental sleep deprivation on the grounds that microsleeps can become so numerous that they eventually fulfill the organism's sleep requirements. These speculations require empirical exposure, since we know of no previous study in which the EEG amplitude of the sleep starved subjects was continuously monitored and quantified. The sleep intrusions during deprivation in the present study support Dement's idea that *total* sleep deprivation is difficult to achieve. Nevertheless, our animals were very substantially sleep deprived. Sleep percents and total daily EEG amplitude were markedly reduced during deprivation. Although waking EEG amplitude increased slightly, it was still largely within the waking range. VHS rates, which are correlated with NREM sleep processes, increased dramatically during deprivation, but the spike rates were still closer to normal waking rates than to normal sleep rates. Finally, there were marked effects of the deprivation procedure on subsequent sleep. Furthermore, our failure to find any substantial negative correlations between measures of sleepiness during deprivation and rebounds during recovery constitutes a lack of support for the hypothesis that sleepiness during deprivation functionally reduces the sleep need. On the whole, the results show that sleep deprivation procedures can be eminently, although not completely, successful.

Putative Sleep Intensity Changes

Sleep time lost during deprivation (sleep debt) is not generally made up during recovery. Few human studies can be evaluated for makeup of sleep debt because recovery periods are frequently terminated artificially. A 17-year-old male who slept *ad lib* after 11 nights of deprivation showed (our computations) 11% makeup of sleep debt in the first 24 hr recovery period, 5% in the second, and 4% in the third (Gulevich et al., 1966). Rats deprived of 5 days of sleep by *d*-amphetamine recovered only 14% of their sleep debt in the subsequent 8 days (Levitt, 1966). Our rats made up 12% of their debt (calculated with sleep on the wheel subtracted) on the first recovery day and 5% on the second.

Sleep *time* debts may be incompletely paid because recovery sleep is in some way especially intense. Williams et al. (1964) found increased auditory arousal thresholds during recovery sleep in humans. Frederickson and Rechtschaffen (1978) found increased arousal thresholds for auditory (but not trigeminal nerve) stimulation during recovery sleep in the rat. In the cat, Ursin (1971) noted that "deep slow wave sleep" showed higher amplitude, slower EEG waves than during baseline. In our study, increases in VHS rates in S2 and in sleep bout lengths during the first 12 hr of the recovery, as well as increases in sleep stage continuity and cortical and hippocampal EEG on the first recovery day, suggest increased intensity.

Our measures of sleep intensity did behave as though they were related to need fulfillment in that they increased during critical stages of recovery. Nevertheless, the relationship between the measures and need fulfillment remains uncertain. For example, one may question why, if the different measures of intensity are related to sleep function, they should show such different amounts and pattern of change during recovery. Perhaps there are different sleep intensities which relate to sleep function in different ways. Longer sleep bouts and stage continuities could reflect the intensification of sleep maintenance mechanisms rather than an extra functional value of the sleep. It makes functional sense that a primary "task" for a sleep deprived organism is to resist any further awakenings. VH spikes could reflect a loss of arousal rather than active sleep processes, which could explain why the greatest changes in VHS rate occurred in wakefulness during deprivation. Increased EEG amplitude could reflect an intensification of only those processes producing high amplitude sleep, which is only one component of normal sleep. These possibilities are not presented because there is strong independent evidence for them, but rather to elaborate the idea that different components of sleep may intensify in different ways.

Sleep Stage Recovery Patterns

Two issues must first be considered as potentially serious obstacles to the interpretation of sleep stage recovery patterns. One is the possibility that the observed patterns result more from altered circadian rhythms than from stage priorities. Most physiological and performance rhythms in humans persist with only minor changes in amplitude or phase during sleep deprivation of moderate length (Kleitman, 1963; Aschoff et al., 1972, 1975; Froberg et al., 1975). In blinded rats, Richter (1967) demonstrated the persistence of the period length and day-to-day phase relationships of spontaneous wheel running rhythms, which are highly correlated with sleep-wake rhythms, in response to the sleep depriving stimulus of forced swimming. In our study, the sleep stage rhythms were quite similar on baseline and recovery days in spite of the absolute changes in sleep stage amounts. The available data therefore suggest that the observed sleep stage recovery patterns were not grossly affected by deprivation produced alterations of circadian rhythms.

The second issue concerns changes in sleep stage scoring criteria between baseline and recovery. The entire EEG frequency histogram shifted to higher amplitudes on recovery day 1 and to lower amplitudes on recovery day 2. Should we have scored recovery data by criteria computed from baseline data or by criteria computed from recovery data?² Using baseline criteria, cutoffs would have been in unusual positions relative to the peaks and troughs of the recovery day EEG frequency histograms. Using recovery criteria would mean requiring higher absolute amplitudes on recovery day 1 and lower absolute amplitudes on

² This type of scoring problem can arise whenever scoring is based on relative EEG amplitude and experimental manipulation affects overall EEG amplitude, but the problem can go unrecognized without precise quantification of the EEG.

recovery day 2 for scoring NREM stages than during baseline. Ideally, one would determine which scoring method correlated better with behavioral criteria. However, with the relatively subtle amplitude changes of this study, differences in the correlations would have been difficult to detect and would have required an enormous amount of work. Unfortunately, without this information, the choice was more or less arbitrary.

We chose to score the recovery days by their own criteria for two reasons. First, our experience has been that scoring normative records according to their own histograms consistently yields high correlations with behaviorally defined sleep-wakefulness, in spite of variations in absolute EEG amplitude across animals and within single animals over time (Bergmann et al., 1978). Second, it is reasonable to assume that the peaks of the EEG amplitude distribution correspond to the "typical" electrophysiology of the corresponding behavioral state (Bergmann, 1978). Since the peaks shift, the criteria should shift.

To assess the effect of our decision we scored the recovery days of three animals both ways. The average change in daily total sleep percentage would have been about 3% had we scored the recovery days according to baseline criteria. Obviously, method of scoring did not affect these results very much.

Our results showed no significant rebound of any sleep stage during the first 6 hr of recovery when the animals spent much time grooming, eating, and drinking. This result may be relatively specific to recovery from water wheel sleep deprivation, which entails food and water deprivation and wetting of the animal. Even here, however, there was a tendency toward a priority for S2, which increased, albeit insignificantly, whereas PS decreased slightly and S1 decreased significantly. For the remainder of recovery day 1, the results were quite clear. S2 rebounded in block 2, while there was little change in PS or S1. For the remainder of the day, S2 returned to approximately baseline levels, whereas S1 and PS showed massive rebounds. The PS rebound was completed by this time, but the S1 rebound continued into block 1 of recovery day 2. The recovery day 2 pattern of S2 is not easy to interpret; S2 was significantly below baseline in the first 6 hr, but then rose to significantly above baseline in the last 6 hr.

In general, the clearest stage priority pattern was for the rebound of S2 to precede those of S1 and PS. Earlier studies of rat sleep deprivation (Yokoyama et al., 1966; Levitt, 1967; Sternthal, 1973) showed NREM and PS rebounds during the first recovery day, but the day was not divided into small enough time blocks to reveal the sequences of stage recovery observed here, nor were high and low voltage NREM sleep differentiated.

Two conceptual issues are raised by the recovery sequences. First, is there an independent "need" for S1, or does S1 follow passively in the wake of needs for S2, PS, and total sleep? Low voltage NREM sleep is often viewed as a stage which fills no specific function except the need for sleep *per se*; i.e., it occurs when there is a need for sleep but no strong specific need for high voltage NREM sleep or PS. This view is consistent with the reciprocal relationship between S1 and S2 in recovery day 1. With S2 rebounding in the first half of the day, S1 is reduced. With the need for S2 apparently filled in the second half of the day but the need for total sleep still elevated, S1 then rebounds. (Rebounds of PS would not severely limit S1,

because PS constitutes a relatively small percentage of total sleep.) The same results, however, are also consistent with the concept of an independent need for S1 which has a lower sequential priority. Furthermore, the results of recovery day 2 are more consistent with the latter view. In block 1, there was a significant increase in S1 at the apparent expense of S2 and in the face of an apparently normal need for total sleep. That the discharge of an S2 need had been preempted by the need for S1 discharge, rather than simply completed, is suggested by the reappearance of high S2 amounts later in the day.

The second conceptual issue concerns the possible influences of endogenous sleep circadian rhythms on recovery patterns.³ One way to assess this influence would be to change the timing of the deprivation with respect to the light-dark cycle. We have not done this. All we have is a single recovery pattern in relation to one circadian rhythm pattern. Therefore, the attempt to discern the nature of these rhythmic influences from our data is made with caution.

One simple hypothesis is that the rebounds of total sleep and the individual sleep stages might occur at the peaks of their endogenous rhythms, possibly because thresholds of need discharge are lowest at these times. Our data are not consistent with this idea. With maximum rebound defined as the point of greatest increase relative to the same baseline period, neither total sleep, S2, S1, nor PS rebounded maximally at their respective peaks. In fact, there was a trend toward the opposite situation. Only S2 rebounded near its rhythm's peak; PS rebounded at its rhythm's trough, and total sleep and S1 rebounded at relatively low points in their circadian rhythms. On the other hand, the effects of endogenous rhythms may be muted by limits in the maximum amount of time that a given state can occur in any block of time.

We do have some preliminary evidence that changes in the lighting schedule can markedly affect the recovery pattern. Two rats kept in constant light for 1 month prior to and throughout the experiment showed large, early rebounds in PS percent. We think this may be related to the tendency of constant light to dampen or disrupt circadian rhythms (Kawakami et al., 1972; Hagino and Yamaoka, 1974; Mitler et al., 1977).⁴

The priority of S2 sleep in the rat for rebounding early in the recovery from sleep deprivation invites comparison with the early rebounds in humans and monkeys of stages 3 and 4 (defined by relatively large amounts of high amplitude, slow EEG waves and often collectively called "delta sleep"). The results among human studies (Berger and Oswald, 1962; Williams et al., 1964; Gulevich et al., 1966; Kales et al., 1970; Berger et al., 1971; Webb and Agnew, 1973; Moses et al., 1975; Nakazawa et al., 1978) and among monkey sleep deprivation studies (Peg-

³ Berger et al. (1971) dealt with this issue in a human experiment. Two groups were each deprived of one night's sleep. One group began recovery in the morning, when the circadian propensity for PS is near its peak; the other group began recovery at night. Stage 4 rebounded early in both groups. In neither case did PS rebound, although latency to PS was reduced on day recovery and not on night recovery.

⁴ It is of interest to note that three studies of total sleep deprivation in the cat (Kiyono et al., 1965; Ursin, 1971; Lucas, 1975) reported early PS rebounds during recovery. Possibly this relates to less well-defined circadian sleep-wake rhythms in the cat.

ram et al., 1969a,b; Jacoby et al., 1975) vary according to whether PS rebounds early along with delta sleep, rebounds later, or not at all—with the largest and earliest rebounds of PS tending to occur when sleep deprivation has been greatest. However, all the studies showed an early rebound of stage 3 and/or 4. In some studies, the delta sleep rebounds were at the apparent expense of stages 1 and 2, a possible parallel to the decrease of S1 during the first half of recovery day 1 in our study. In the one cat study which divided NREM sleep according to amount of delta activity, both high delta sleep and PS rebounded early, but low delta NREM sleep did not change from baseline (Ursin, 1971).

As appealing as the analogy between S2 rebounds in the rat and delta rebounds in other species may first appear, the issue is somewhat complicated. We defined S2 by EEG amplitude criteria alone, without considering EEG frequency. In rats, the highest EEG amplitude sleep is not necessarily delta sleep. Rosenberg et al. (1976) have shown that power in the delta frequency band (defined as 2–4 Hz for the rat) during NREM sleep was related to light–dark schedules differently from power in the broad spectrum EEG during NREM sleep. In other words, delta activity and total EEG amplitude can be functionally differentiated. In our study, we do not know how much of the S2 rebound may have been determined specifically by augmentation of delta activity.

In fact, the nature of the stage 3 and 4 rebounds in other species is not perfectly clear either. These stages are scored when EEG slow waves above a criterion amplitude occupy more than a given proportion of an epoch. By itself, a report of increased stage 4 following sleep deprivation does not reveal whether there were more delta waves, whether there was simply an increase in the amplitude of delta waves so that more of them reached criterion, or whether the amplitude of delta waves increased more than the amplitude of other EEG waves. Conceivably, increased sleep intensity might be reflected by increased amplitude in a broad spectrum of EEG frequencies, whereas frequency *per se* is related to other variables such as circadian rhythm. The priority for increase of stages 3 and 4 after deprivation could result from the fact that they are the only stages dependent on high amplitudes. Clearly, what is needed before cross-species comparisons can be made with confidence is a careful analysis of amplitude changes in several frequency bands before and after sleep deprivation.

Negative Rebounds During Recovery

One apparently peculiar phenomenon of our recovery data was the decline of EEG amplitude to below baseline on recovery day 2. Hippocampal EEG amplitude in NREM sleep and the T1 and T2 measures of cortical amplitude were significantly above baseline on recovery day 1 and significantly below baseline on recovery day 2. The LAM of the cortical EEG amplitude histogram showed a similar trend, although the changes were not significant. Such instances of “secondary negative rebound” are not completely isolated. Following 24 hr of total sleep deprivation, humans showed significant increments in total sleep on the first recovery night and then decrements to significantly below baseline values on the second and third recovery nights (Nakazawa et al., 1978).

To explain these data, one of the present authors (AR) hypothesizes a "sleep inhibition," whereby the occurrence of sleep progressively augments processes and/or substances which militate for the termination of sleep and against its recurrence.⁵ The occurrence and duration of sleep would depend on, among other factors, the balance between sleep need and sleep inhibition. Large amounts of sleep inhibition may accumulate during early postdeprivation rebounds. After the sleep need is reduced, the accumulated sleep inhibition could reduce certain sleep parameters to below normal levels. Why some variables would be so affected and others not is unclear. Perhaps the recovery process was not recorded long enough for secondary negative rebounds to appear in some variables.

Sleep inhibition makes adaptive sense; interruptions of extended sleep would prevent neglect of other functions such as eating, drinking, elimination, and vigilance. The concept of sleep inhibition is consistent with the normal alternation of sleep and wakefulness, as well as with the fact that rebounds from prior sleep deprivation are rarely accomplished by a single, uninterrupted, exceedingly long sleep bout. Conceivably, some sleep disorders could eventually be explained by an excessive or insufficient accumulation of sleep inhibition rather than by anomalies of sleep drive or sleep mechanism.

The other two authors (LF and BB) prefer to explain secondary negative rebounds by assuming that sleep parameters are controlled by a system with servomechanism properties, i.e., a closed loop system devised for self-regulation of a constant output (Thaler, 1955). The depriving manipulation grossly perturbs the sleep controlling servomechanism. During "recovery," the servomechanism adjusts the output to regain the previous levels. If the servomechanism is underdamped, a "peak overshoot" is followed by a period of damped oscillation until the sleep parameters return to normal. With circadian influences removed, as they are in Table 2, several variables showed recovery patterns suggesting control by an underdamped servomechanism. Peak overshoots and some periods of damped oscillation can be seen in the recovery curves for S2 percent, PS percent, VHS in S2, VHA in all sleep stages, and even the entire EEG histogram. Of the three types of servomechanism (overdamped, critically damped, and underdamped), the hypothetical sleep controller is most analogous to the underdamped type, which drives the output close to the desired steady state as rapidly as possible. Thus both the positive and negative rebounds may result from a servomechanism rapidly returning output to the steady state region. If sleep mechanisms were organized like servomechanisms, one would not expect an organism to make up all sleep lost during deprivation.

There are complications to this hypothesis for some of the measures, notably the percentages of S1, S2, and PS. For example, if the percentage of S2 is very

⁵ Aserinsky (1969), observing that it was difficult for young adults to extend their sleep beyond 14 hr per day, expressed a similar idea: "Is it possible that . . . as 'fatigue products' build up during the course of sleep, the latter can no longer continue unless there is recuperation afforded by the waking state?" (p. 155). A parallel concept of PS inhibition was used by Hobson et al. (1975) to explain the termination of PS periods, and by Vogel et al. (1977) to explain certain features of sleep in depressed patients.

high, the percentages of the other conditions must be low. Thus, their servomechanisms are not independent and must be either mutually modulated or preempted, making the construction of a straightforward mathematical model considerably more difficult. Secondly, the percentage of W does not appear to follow a simple response curve, leaving the slightly awkward conclusion that W percent is determined by what time is left over from the animal's fulfilling the requirements of its various servomechanisms.

Both models are offered tentatively. We have produced neither direct evidence of some internal sleep inhibition nor a working mathematical model of sleep servomechanisms. Neither model fully explains the data. It is clear, however, that the events surrounding sleep deprivation and recovery from it require explanations beyond a simple hydraulic model whereby the "sleep jug empties" during deprivation and "refills" during recovery.

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