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Effect of Sleep Disruption on Sleep, Performance, and Mood

Michael H. Bonnet

Jerry L. Pettis Memorial Veterans Administration Hospital, Loma Linda, California, U.S.A.

Summary: Eleven young adult subjects were briefly awakened after each minute of electroencephalographic-defined sleep for 2 consecutive nights after undisturbed laboratory adaptation and baseline nights. Two undisturbed recovery nights followed disruption nights. On disruption nights, subjects were awakened with an audiometer and signaled the awakening by subjective rating of sleep state or button push response. The disruption procedure resulted in severely fragmented sleep with only very small amounts of slow-wave and REM sleep. Total sleep time was reduced by approximately 1 h on each night. Arousal threshold increased 56 dB across the disruption nights. Following disruption, subjects performed more poorly and rated themselves sleepier than on baseline. The level of decline was similar to that seen after periods of total sleep loss of 40–64 h. Recovery sleep was also similar to that seen after total sleep loss. It was concluded that periodic disruption of sleep, perhaps by destroying sleep continuity, quickly results in impaired function. These data may help explain function loss in severe sleep apneics. **Key Words:** Sleep deprivation—Sleep disruption—Sleep fragmentation—Sleep apnea.

Many recent studies have shown a significant incidence of daytime sleepiness in the population. This sleepiness in great part has been attributed to sleep loss accompanying arousals associated with apnea or periodic leg movements (1). It is known that total sleep deprivation results in performance loss on many tasks including reaction time, short term memory and additions (2) and in increased daytime somnolence (3). Unfortunately, the effects of partial sleep deprivation and sleep stage deprivation are less clear. In general, partial sleep loss studies have not found significant performance loss or increased sleepiness until total sleep time has been reduced to 5 h/night for 4 days or more (4). A study of chronic primary insomniacs who had been unable to sleep more than 5 h/night for a median of 20 years, however, was unable to document any performance differences between those insomniacs and a matched group of normal controls (5).

Regardless, it is very questionable whether severe sleep apneics could be considered a model case of even partial sleep deprivation. If one calculates total time spent asleep in an apneic who sleeps 10 h/day and who arouses for 5 s at the end of each of 60 apneas/h of sleep, one concludes that this apneic has slept for (55/60 min × 600 min =) 9 h 10

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Address correspondence and reprint requests to Dr. Bonnet at Veterans Administration Hospital (151), 11201 Benton Street, Loma Linda, California 92354, U.S.A.

min. It is difficult to conceive of chronic sleep deprivation developing when more than 9 h is spent asleep each night.

It is known that individuals with sleep apnea are often selectively deprived of slow-wave sleep (SWS) or REM sleep. However, it is not possible to account for their performance loss or sleepiness based on selective sleep stage deprivation because sleep stage deprivation studies have generally concluded that total sleep time rather than sleep stage distribution is the primary correlate of daytime performance (6,7).

One major explanation of the somnolence and performance loss that accompanies significant sleep apnea is that the periodic arousals that accompany the apneas disrupt sleep and make it nonrestorative. It is known that sleep is an efficient restorative system that can reverse the effects of 40 h of sleep loss within an approximate 4-h consolidated period (8). However, current restorative theories posit that sleep is a favored time for protein synthesis (9,10). It is known that protein synthesis is a time-linked process occurring on the order of minutes (11,12). It is possible that disruptions occurring at a rate more rapid than that of protein synthesis could significantly interfere with that process and make sleep nonrestorative.

Except for sonic boom and noise studies, there are few studies of sleep disruption. The sonic boom/noise studies typically examined the effects of a small number of loud noise events on sleep structure (13) and therefore are not good models of sleep disruption at a frequency typical of apnea or periodic leg movements. Three studies bear more directly on the current issue. Sharpless and Jasper (14) presented tones to sleeping cats and found that after approximately 30 presentations they could find no change in ongoing electroencephalogram (EEG) after tone presentation. Townsend et al. (15) studied the effects of 80–90 dB tones each 22 s on sleep in young adult males and found essentially no change in sleep variables. However, this may have been because of rapid habituation and the fact that the initial nights of tone presentation were not analyzed separately. More recently Phillipson et al. (16) played tape-recorded noises to dogs for 30 s of each 3 min period. Unfortunately, continuous recordings were not made to ensure arousal after each presentation or document such. Regardless, decreased sleep latencies and impaired arousal responses to hypercapnia and hypoxia were found after 3 nights of disruption/deprivation. These studies are perhaps not overly convincing in describing the potential effects of periodic disruption on sleep because they all differ from the apnea model in one central respect—participants in these studies could (and clearly did) habituate to the disrupting tones so that, after a while, their sleep was less disturbed. A sleep apneic, responding to increasingly abnormal blood gas values with a homeostatic response, can never habituate in the true sense of the word because arousal and muscle tension increase in the neck are necessary to resume airflow.

The current study sought to model the sleep disrupting effects of apnea in normal young adults by performing standardized awakenings after each minute of sleep for 2 consecutive nights.

METHODS

Eleven young adult subjects between the ages of 18 and 32 were chosen to participate. Subjects were normal sleepers who rarely took naps as determined by sleep questionnaire. All subjects scored within the normal range on the depression scale of the Minnesota Multiphasic Personality Inventory. Subjects participated for 5 nights after nonscored adaptation. Laboratory nights included baseline, 2 nights of periodic sleep disruption, and 2

TABLE 1. *Sleep stages before, during, and after sleep disruption*

Sleep variable	BL	D1	D2	R1	R2	F	Differences
Latency 1 (min)	5.4	8.2	4.8	5.4	5.8	1.25	
Stage W (%)	3.1	24	28	0.6	1.8	25.1[a]	R1 < R2 = BL < D1 = D2
Stage 1 (%)	6.4	31	28	3.1	5.7	54.1[a]	R1 < R2 = BL < D1 = D2
Stage 2 (%)	43	31	31	40	44	15.5[a]	D1 = D2 < BL = R1 = R2
Stage 3 (%)	8.8	1.1	1.2	11	6.9	62.0[a]	D1 = D2 < BL = R2 < R1
Stage 4 (%)	14	0.7	0.2	20	13	39.2[a]	D1 = D2 < BL = R2 < R1
REM (%)	19	3.7	1.8	22	22	55.0[a]	D1 = D2 < BL = R1 = R2
Stage changes	132	479	504	128	115	123.6[a]	D1 = D2 > BL = R1 = R2
Time asleep (min)	389	334	316	415	391	12.2[a]	D1 = D2 < BL = R1 = R2

Abbreviations: BL, baseline; D, disruption night; R, recovery night.
[a]$p < 0.05$, Newman-Keuls pairwise comparisons.

recovery nights. Subjects were allowed to sleep during their habitual times with the exception that total time in bed was increased by 30 min on disruption nights in an attempt to equalize total sleep time of baseline and disruption nights. Subjects completed the Clyde Mood Scale and Stanford Sleepiness Scale each evening and morning. Morning performance tests included Wilkinson addition (30 min), which was scored for number of problems attempted; simple reaction time (which was divided into two 5-min sections and transformed to response speed—1/reaction time—for analysis and retransformed for the text and tables); and Digit Symbol Substitution (5 min–eight subjects), which was scored for number correct.

On disruption nights, subjects were awakened after each minute of sleep via audiometer (five subjects) using the method of constant stimuli with a 1,000 Hz tone or via intercom. Four audiometer subjects also had approximately eight presentations of 500 and 2,000 Hz tones each night to check for habituation. Subjects verbally acknowledged wakefulness by either performing a simple task such as responding to the technician with the preceding letter of the alphabet (one subject); responding with a number between 1 and 7 on a subjective sleep rating scale (1, wide awake; 2, awake but drowsy; 3, almost asleep; 4, unsure of sleep or waking; 5, just fallen asleep; 6, asleep; 7, very deeply asleep) (five subjects); or pushing a response button located within distant reach on a bedside desk (five subjects). Following their response, subjects were allowed to return immediately to sleep. After a few trials, subjects could follow any of these procedures with only a few seconds of wakefulness. One subject, who consciously decided that it was easier to stay awake than to be awakened each minute, was dropped from the study.

Sleep, performance, and mood data were analyzed by analysis of variance and checked by sign test. Significant results ($p < 0.05$) were followed by pairwise comparisons using the Newman-Keuls procedure ($p < 0.05$). Threshold data were analyzed within subject by *t* test.

RESULTS

No differences were seen in the data from subjects who performed the three different types of response at awakening. Therefore, combined sleep data can be seen in Table 1. Significant condition differences were found for every variable except for sleep latency. The sleep disruption procedure resulted in increases in wakefulness and stage 1 with reduction in stage 2 and essential elimination of SWS and REM sleep. Despite sleep extension, total sleep time on the disruption nights was approximately 1 h less than on the baseline night.

TABLE 2. *Sleep stages before and after sleep deprivation as compared with sleep disruption*

Sleep variable (%)	Sleep deprivation[a] (36 h)			Sleep deprivation[a] (64 h)			Sleep disruption		
	BL	R1	Diff	BL	R1	Diff	BL	R1	Diff
Stage W	2.3	2.1	0.2	2.2	0.4	1.8	3.1	0.6	2.5
Stage 1	7.4	6.5	0.9	7.4	4.1	3.3	6.4	3.1	3.3
SWS	23.7	27.8	4.1	23.9	36.4	12.5	22.8	31.0	8.2
REM	23.1	24.8	1.7	22.8	22.7	−0.1	19.0	22.0	3.0

Abbreviations: BL, baseline; Diff, difference; R1, recovery night; SWS, slow-wave sleep.
[a]Rosa et al. (8).

On the first recovery night, wakefulness (stage W) and stage 1 were decreased as compared with baseline whereas stage 3 and 4 were increased. REM sleep was nonsignificantly increased in both recovery nights compared with baseline.

Data from a recent study of 1 and 2 nights of sleep deprivation in another group of young adults in our laboratory (17) are presented in Table 2 to serve as a comparison to sleep data in the present study. It can be seen that in terms of the reduction of stage W and stage 1 that the disruption results were very similar to those seen after 2 nights of total sleep deprivation. In terms of SWS, the increase after 2 nights of sleep disruption fell midway between the increases in SWS seen after 1 and 2 nights of total sleep loss.

Performance and mood data can be found in Tables 3 and 4. Table 3 presents performance and mood data for baseline (average of baseline and final recovery night) and following 1 and 2 nights of sleep disruption. In terms of ANOVA and/or sign test (binomial distribution), performance was significantly worse on simple reaction time (first half of test and entire) following the 2nd night of disruption, addition problems attempted following both disruption nights, and number of correct digit symbols substituted (sign test only) following the 2nd disruption night. In terms of mood, subjects reported being significantly more sleepy (Clyde Mood Scale) after the 2nd disruption night.

For comparison purposes, performance and mood data from four studies (8,18–20) of 64 h of sleep deprivation are presented in Table 4. Looking at performance differences in deprivation and the present disruption study, it can be seen that the decrease in response speed was slightly greater after disruption than after deprivation whereas the reduction in addition problems after disruption was about that seen after 1 night of total sleep loss. In terms of mood, sleepiness after sleep disruption was similar to that seen after 1 night of sleep deprivation.

Cumulative effects of sleep disruption on arousal threshold and sleep rating

Figure 1 is a plot of arousal threshold averaged across each hour of each disruption night and across subjects (five subjects). The large number of observations allowed statistical analysis within each subject. For each subject, values from the first time period of the 1st disruption night were compared with corresponding values from the final hour of the 1st night and with the 1st hour of the 2nd disruption night by *t* test. Data from the final hour of night 1 were compared with the final hour of night 2, and data from the 1st hour of night 2 were compared with the final hour of night 2. All comparisons were significant ($p < 0.05$) for all subjects and indicated increasing thresholds throughout both disruption

TABLE 3. *Mood and performance following baseline and disruption nights*

	F	Significant difference	Binomial distribution on D2	Means BL	Means D1	Means D2
Clyde mood						
p.m.						
Sleepy	2.01		7/9	52.4	52.1	58.8
Dizzy	0.94		6/11	48.0	49.7	48.1
Unhappy	4.47[a]	D2 < D1 = BL	10/11[b]	37.5	38.0	35.4
Clear thinking	7.31[a]	BL > D1 = D2	10/11[b]	48.5	46.6	43.3
Friendly	1.11			49.4	48.2	47.8
Aggressive	2.06			41.4	41.6	39.6
a.m.						
Sleepy	8.13[a]	D2 < D1 = BL	10/11[b]	48.8	52.3	58.7
Dizzy	0.63		4/11	47.8	49.4	48.4
Unhappy	0.42		7/10	36.9	35.9	36.2
Clear thinking	1.06		8/10	46.7	45.1	43.9
Friendly	0.04		4/8	44.6	44.4	45.1
Aggressive	1.84		6/9	40.7	38.9	39.9
Stanford Sleepiness Scale						
p.m.	0.98		6/8	2.5	2.5	3.0
a.m.	0.67		8/10	2.4	2.5	2.8
Performance						
RT1 a.m.	5.25[a]	BL > D2	10/11[b]	0.221	0.234	0.249
RT2 a.m.	1.26		8/11	0.237	0.248	0.255
RTT a.m.	3.21	BL > D2	9/11[b]	0.231	0.241	0.253
ADDS a.m.	3.28[a]	BL > D2 = D1	9/11[b]	110	97	95
DSST a.m.	1.37	BL > D2	7/8[b]	173	160	155

Abbreviations: ADDS, addition problems; BL, baseline; D, disruption night; DSST, Digit Symbol Substitution Test; RT, reaction time; RTT, RT total.

[a] $p < 0.05$, Newman-Keuls pairwise comparisons.

[b] $p < 0.05$.

TABLE 4. *Performance and mood during sleep deprivation and sleep disruption*

	Deprivation				Disruption			
Task	BL	D1	D2	Diff (D2-BL)	BL	D1	D2	Diff (D2-BL)
RT1	0.260	0.229	0.264	0.004[c]	0.221	0.234	0.249	0.028
RT2	0.253	0.244	0.299	0.046[c]	0.237	0.248	0.255	0.018
RTT	0.256	0.234	0.270	0.014[c]	0.231	0.241	0.253	0.022
ADDS	131	115	88	43[b]	110	97	95	15
Clyde Mood Scale								
Sleepy p.m.	50.8	52.1	58.2	7.4[a]	52.4	52.1	58.8	6.4
Sleepy a.m.	51.1	60.3	68.0	16.9[a]	48.8	52.3	58.7	9.9
Stanford Sleepiness Scale								
p.m.	2.1	2.2	3.4	1.3[c]	2.5	2.5	3.0	0.5
a.m.	2.5	3.6	4.2	1.7[c]	2.4	2.5	2.8	0.4

Abbreviations: ADDS, addition problems; BL, baseline; D, disruption night; RT, reaction time; RTT, RT total.

[a] Vaccarino et al. (20).

[b] Webb and Agnew (19) and Donnell (18)—data averaged.

[c] Rosa et al. (8).

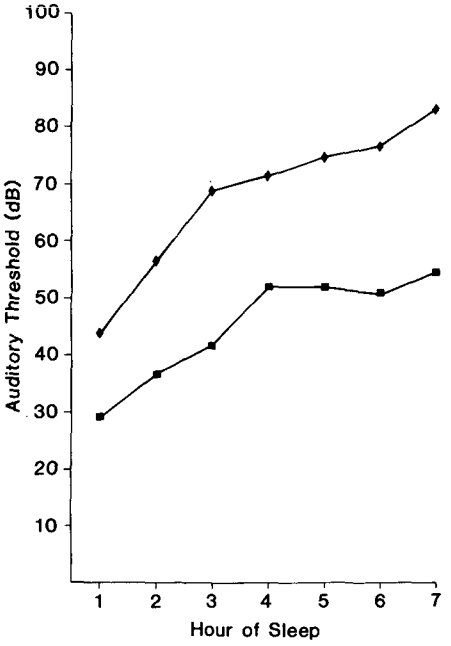

FIG. 1. Auditory arousal thresholds during each hour of sleep during 2 nights (boxes, night 1; diamonds, night 2) of periodic arousal after each minute of sleep.

nights. The combined probability of any of the four data comparisons (across subjects) happening by chance was less than 1 in a trillion.

To determine what part of the increase in arousal threshold was due to sleep fragmentation and what part was due to simple habituation to the arousing stimulus, four subjects had occasional (approximately 1/h of sleep) arousals during which the frequency of the arousing tone was switched to 500 or 2,000 Hz. From the 1st hour of night 1 to the last hour of night 2, the four subjects had an average increase in arousal threshold to the 1,000 Hz (standard) tone of 56 dB. The average threshold increase to the 500 and 2,000 Hz tones was 37 dB. The average threshold increase during the first 16 presentations of the 1,000 Hz tone on night 1 was 9 dB. The 37 dB increase was both significantly less than the 56 dB increase ($t = 2.51$, 3 df, $p < 0.05$ one-tail) and greater than 9 dB ($t = 3.05$, 3 df, $p < 0.025$ one-tail).

Figure 2 is a plot of subjective rating of sleep at the time of awakening. Analogous within subject *t* tests to those computed with threshold values were computed. The *t*-values for ratings were lower, and each subject had at least one *t*-value that was nonsignificant with between 16 and 37 df. As a result, average *t*-values were computed. Those average values ($t = 8.42$ for beginning to end of night 1; $t = 9.19$ for beginning of night 1 to beginning of night 2; $t = 4.15$ for beginning of night 2 to end of night 2; and $t = 2.4$ for end of night 1 to end of night 2) were all significant with df for the number of observations entered into each test. With 4 df (from the five subjects), all *t*-values except the comparison of the end of night 1 with the end of night 2 were significant ($p < 0.025$). Again, ratings of sleep increased during each night of disruption and leveled off by the end of the 2nd night.

DISCUSSION

The present experiment has shown that periodic, brief disruption of sleep results in a significantly altered distribution of sleep stages, the appearance of subjective daytime

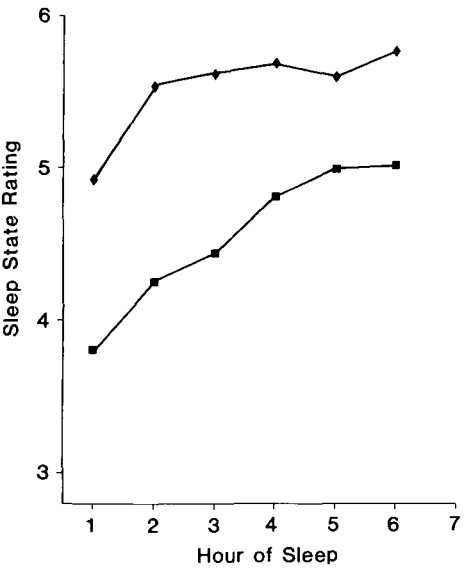

FIG. 2. Subjective ratings of sleep state (see Methods for definers) during each hour of sleep during 2 nights (boxes, night 1; diamonds, night 2) of periodic arousal after each minute of sleep. The subjective ratings were: 1, wide awake; 2, awake but drowsy; 3, almost asleep; 4, unsure; 5, just fallen asleep; 6, asleep; 7, very deeply asleep.

sleepiness and objective performance decrement, and characteristic rebounds (2) when sleep is permitted.

The sleep disruption procedure resulted in severely altered sleep. Wakefulness and stage 1 increased dramatically and SWS and REM sleep were virtually eliminated. Although the present study clearly does not model the pathophysiology seen in sleep apnea syndrome, it is worthy to note that similarly disrupted sleep (i.e., stage changes of similar direction and magnitude) is often seen in sleep apnea (1).

Subjects quickly adapted to the awakening procedure and usually returned to sleep rapidly after being awakened. Highly significant decreases in sensitivity to the auditory arousing stimulus were demonstrated. Because such threshold shifts might be due either to simple habituation (14,15) or to actual decreased sensitivity resulting from nonrestorative sleep, trials with novel frequency tones were interpolated for four subjects. Significant threshold increases (66% of the magnitude of threshold increase to the standard 1,000 Hz tone) were found to the novel stimuli as well as the standard tone. This implies that a majority of the threshold increase across the disruption nights was related to decreased sensitivity to external stimulation.

Figure 1 displays the threshold increases but does not sufficiently imply that 40% of the subjects after some periods of sleep of 1 min on the 2nd disruption night could not be awakened with the maximum 120 dB stimulus tone. Again, although this study did not model the pathophysiology associated with sleep apnea, this large and rapidly developing decrease in sensitivity to external stimulation offers a powerful explanation of the decreasing sensitivity of sleep apneics to increasingly abnormal blood gas values. It might also explain the usual observation that severe apneics are "deep" sleepers (1).

The subjective ratings of sleep state obtained from five subjects after they were awakened from sleep lent further support to the auditory threshold data as a measure of decreased sensitivity rather than habituation. Subjects often rated themselves as awake or confused as to state after early awakenings. Subjects placed themselves more firmly asleep as disruption continued. By the middle of the 2nd disruption night subjects had reached the ceiling of the sleep scale and sleepiness ratings stopped increasing. In fact, subjects began

to become confused on awakening. They often could not give ratings. One subject later recounted that at awakening she could hear the technician talking to her but his words did not seem to make sense. Other subjects could not perform simple tasks such as being able to respond with "a" when prompted for the letter that precedes "b." One explanation for this behavior is sleep drunkenness (i.e., arousal from very deep sleep resulting in confusion).

Subjects reported significant daytime sleepiness and showed performance decrement on standard sleep deprivation tasks. When compared with a series of 2-night total sleep deprivation studies, the mood and performance data indicate that the 2 nights of sleep disruption degraded mood and performance more than 1 night of total sleep loss but less than 2 nights of total sleep loss. It is difficult to explain this amount of performance/mood change on the basis of the actual sleep loss that occurred on the sleep disruption nights because that sleep loss was only approximately 1 h on each night. Work by Wilkinson et al. (21) with the addition task found that sleep reduction to less than 3 h/night over 2 nights was necessary to get a significant reduction in performance. If this is true, it implies that the fragmentation itself may somehow interfere with the sleep restorative process. Such interference could work in many ways. (a) Disruption of sleep each 1 min does not allow sufficient time to elapse between awakenings for any significant SWS or REM sleep to occur. Some investigators posit that SWS and/or REM sleep or both are necessary for sleep restoration (22) and that studies that reduce total sleep length (21) or disrupt sleep, as in the current study, decrease those sleep stages and therefore make sleep nonrestorative. However, several elegant studies of selective deprivation in human subjects (6,7,23) have not shown a difference in performance from either stage 4 or REM sleep deprivation. In fact, those studies have concluded that it is total sleep time, not sleep stage distribution, that is most related to performance restoration (6,7). (b) Some restorative theories (9,10) posit that sleep is a time for maximal protein synthesis. Protein synthesis proceeds over a period of minutes (12,24) and is disrupted by exercise. It is possible that sleep fragmentation periodically raises basal metabolic levels, inhibits protein synthesis, and makes sleep nonrestorative. (c) Secretion of growth hormone may be related to the sleep restoration process. Fragmentation may disrupt growth hormone secretion. (d) Stage 1 sleep may not "count" as sleep. Significant amounts of stage 1 were scored during fragmentation. If stage 1 were "not really" sleep, considerably more sleep deprivation than stated actually occurred. In this case total nonstage 1 sleep would have been approximately 3 h in each disruption night, and significant performance loss should have developed. Although future studies will be required to differentiate these possibilities, the behavioral implications of sleep fragmentation, be it in aging, sleep apneics, or other pathophysiology, are clear.

Recovery sleep after sleep disruption closely paralleled recovery sleep after total sleep loss (Table 2). In terms of magnitude of change, stage W and 1 changes were very close to those seen after 2 nights of sleep loss. In terms of SWS rebound, recovery after sleep disruption fell exactly between that found after 1 and 2 nights of total sleep loss.

The presented results serve only to model an arousal pattern that may be somewhat similar to that seen in severe sleep apnea. Clearly, other events associated with apnea, such as oxygen saturation, were not varied. The strength of this approach is that it implies that the significant sleepiness in apnea is directly related to disruption of sleep. However, other apparent differences exist between this study and sleep apneics. Subjects in this study were required to make a verbal or behavioral response more complex than the breathing response required by apneics. Disruption was perhaps more regular in this study than in some sleep apneics. Finally, the study sleep deprivation controls came from several previously published studies by the author and others rather than from a specific control condition.

In conclusion, periodic sleep fragmentation at a rate of once per minute of sleep for 2 nights resulted in subjective, behavioral, and EEG results that were very similar to those seen after an equal period of total sleep loss. Similar fragmentation as caused by periodic pauses in respiration or other periodic events may account for the daytime sleepiness found in sleep apneics or some elderly individuals.

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REFERENCES

1. Guilleminault C, Dement WC. Sleep apnea syndromes and related sleep disorders. In: Williams RL, Karacan I, eds. Sleep disorders: diagnosis and treatment. New York: John Wiley and Sons, 1978:9–28.
2. Johnson LC, Naitoh P. The operational consequences of sleep deprivation and sleep deficit. AGARD Ms. #193, 1974.
3. Carskadon MA, Dement WC. Effects of total sleep loss on sleep tendency. *Percept Mot Skills* 1979;48:495–506.
4. Carskadon MA, Dement WC. Cumulative effects of sleep restriction on daytime sleepiness. *Psychophysiology* 1981;18:107–13.
5. Bonnet MH. The restoration of performance following sleep deprivation in geriatric normal and insomniac subjects. *Sleep Res* 1984;13:188.
6. Johnson LC, Naitoh P, Moses JM, Lubin A. Interaction of REM deprivation and stage 4 deprivation with total sleep loss: experiment 2. *Psychophysiology* 1974;11:147–59.
7. Lubin A, Moses J, Johnson LC, Naitoh P. The recuperative effects of REM sleep and stage 4 sleep on human performance after complete sleep loss. Experiment 1. *Psychophysiology* 1974;11:133–46.
8. Rosa RR, Bonnet MH, Warm JS. Recovery of performance during sleep following sleep deprivation. *Psychophysiology* 1983;20:152–9.
9. Adam K. Sleep as a restorative process and a theory to explain why. *Prog Brain Res* 1980;53:289–304.
10. Oswald I. Sleep as a restorative process. *Prog Brain Res* 1980;53:279–88.
11. Dintzis HM. Assembly of peptide chains of hemoglobin. *Proc Natl Acad Sci USA* 1961;47:247–61.
12. Goodwin B. *Temporal organization in cells, theory of cellular control processes.* New York: Academic Press, 1963.
13. Lukas JS. Noise and sleep: a literature review and a proposed criterion for assessing effects. *J Acoust Soc Am* 1975;58:1232–42.
14. Sharpless S, Jasper H. Habituation of the arousal reaction. *Brain* 1956;79:655–80.
15. Townsend RE, Johnson LC, Muzet A. Effects of long term exposure to tone pulse noise on human sleep. *Psychophysiology* 1973;10:369–75.
16. Phillipson EA, Bowes G, Sullivan CE, Woolf GM. The influence of sleep fragmentation on arousal and ventilatory responses to respiratory stimuli. *Sleep* 1980;3:281–8.
17. Rosa RR, Bonnet MH. Sleep stages, auditory arousal threshold and body temperature as predictors of behavior upon awakening. *Int J Neurosci* (in press).
18. Donnell JM. Performance decrement as a function of total sleep loss and task duration. *Percept Mot Skills* 1969;29:711–4.
19. Webb WB, Agnew HW. Effects on performance of high and low energy-expenditure during sleep deprivation. *Percept Mot Skills* 1973;37:511–4.
20. Vaccarino P, Rosa RR, Bonnet MH, Kramer M. The effect of 40 and 64 hours of sleep deprivation and recovery on mood. *Sleep Res* 1981;10:269.
21. Wilkinson RT, Edwards RS, Haines E. Performance following a night of reduced sleep. *Psychon Sci* 1966;5:471–2.
22. Oswald I. Sleep as a restorative process: human clues. *Prog Brain Res* 1980;53:279–88.
23. Agnew HW, Webb WB, Williams RL. Comparison of stage 4 and 1-REM sleep deprivation. *Percept Mot Skills* 1967;24:851–8.
24. Fisher LB. The diurnal mitotic rhythm in the human epidermis. *Br J Dermatol* 1968;80:75–80.