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Electroencephalographic and Autonomic Activity During and After Prolonged Sleep Deprivation

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Simultaneous recordings of EEG, EOG, EMG, HR, GSR, respiration, skin temperature, and plethysmogram were obtained from a 17-year-old boy following 236, 246, and 264 hr. of wakefulness, during 3 recovery nights, 1 week, 6 weeks, and 7 months after end of deprivation. The EEG indicated dominant slow activity with minimum alpha during deprivation. Opening and closing of eyes had little effect on the EEG and stimuli did not produce alpha enhancement. Prolonged sleep loss caused a chronic shift to increased activity of the autonomic nervous system but with diminished responsiveness to external stimuli. During the early period of the first recovery sleep, increased responsiveness to stimuli was seen in all autonomic variables except GSR. Specific GSR's did not appear until the second recovery night. There was an increase in REM sleep during the first 3 recovery nights.

THE INCREASED INTEREST in sleep in recent years has included increased attention to the effects of loss of sleep. While most of the work in this area has been concerned with the effect of sleep loss on various indices of performance, there have been some reports of physiologic changes after varying periods of continuous wakefulness.[1, 2, 5, 9, 13] With respect to EEG, a decrease in alpha activity after periods of wakefulness has been a consistent finding. The interpretation of this finding has differed, however, with one group maintaining that the decrease in alpha is a reflection of increased acti-

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Supported by National Science Foundation Grant GB 922 to the San Diego State College Foundation, by the Bureau of Medicine and Surgery, Department of the Navy, under research task MR 005-12-2304, and by N.I.M.H. Grant MH 08185 to Stanford University. The opinions and assertions contained herein are the private ones of the writers, and are not to be construed as official, or as reflecting the views of the Navy Department or the Naval service at large.

The authors wish to express their appreciation to the subject, Randy Gardner, and his two co-investigators, Joe Marciano, Jr., and Bruce McAllister, for their diligence and cooperation. The sustained efforts of Marion Austin, Kay Van Kirk and Chuck Hasley, HN, USN, of the Psychophysiology Laboratory staff, and the editorial assistance of Ardie Lubin are also gratefully acknowledged.

Received for publication Dec. 16, 1964.

vation,[9, 13] and a second group interpreting the alpha decrement as an indication of a drift toward sleep and decreased activation.[1, 2]

The psychophysiologic findings have not been as consistent as the EEG data, but the interpretation of the data has again been in terms of increased or decreased activation. In a study of 3 subjects after a waking period of 60 hr., Malmo and Surwillo[9] found a reliable increase in palmar conductance, respiration, and heart rate—which they interpreted as indications of autonomic activation consistent with the decrease in alpha activity. Malmo and Surwillo's measures were obtained during a tracking task with heat being administered for errors.

Ax and Luby[2] studied the physiologic changes in base level and the response to pain stimuli in 5 subjects after 123 hr. of wakefulness. In contrast to the findings of Malmo and Surwillo, Ax and Luby state that their results strongly support the hypothesis that prolonged wakefulness produces a marked decline in arousal or activation level.

Scholander[12] and Burch and Greiner[5] have reported conflicting skin resistance data. Scholander found that after 24 hr. of wakefulness the experimental group showed a higher mean level and less habituation of the GSR to white noise than the control group. Burch and Greiner,[5] on the other hand, noted a decreased responsiveness of the specific GSR to electric shock with continued sleep deprivation.

The present paper presents EEG and autonomic data from a 17-year-old boy who went 264 hr. without sleep. Resting levels and response to stimuli were obtained during the 264 hr. presleep vigil, during recovery sleep, and during awake and sleep records obtained after the sleep recovery period.

The original purpose for the prolonged period of wakefulness was a carefully planned research project to study the effects of sleep loss by the S and 2 coinvestigators for the San Diego Science Fair. All 3 boys were seniors in high school at the time. The goal of 264 hr. of wakefulness was chosen in order to add to the originality of the project since, insofar as was known, no one had ever stayed awake under observation for this amount of time (260 hr. was the "record"). The S was continuously observed by one of his 2 coinvestigators and every 6 hr. was administered a series of tests to measure performance and ability to respond to stimuli. After 150 hr. of wakefulness, he was under the care of a local Navy physician, and, during the last 3 days, he was almost continuously in the presence of 1 of the authors (W.D.). No systematic physiologic data were obtained before the first examination in the laboratory at 236 hr. The physician's notes, however, indicated no concern over any of the physical or psychologic findings. During this presleep period, neurologic and psychiatric examinations were also performed. There were no marked psychologic changes, but there were some neurologic changes which disappeared after recovery sleep.[11] No stimulants, not even coffee, were consumed by the subject during the deprivation period.

Procedure

A total of 11 records were obtained at the times and dates listed in Table 1. In the discussion to follow, the records will be divided into the following categories: presleep (Records 1 and 2), recovery sleep (Records 3, 4, and 5) and postrecovery (Records 6, 7, 8, 9, 10, and 11). The initial analysis will consist of a comparison of the presleep and postsleep awake records followed by a comparison of the recovery and postrecovery sleep records.

We used an Offner 12 channel type R Dynograph,* instrumented to record 3 channels of EEG, 2 of EOG, and 1 channel

*Offner Electronics, Inc., Schiller Park, Ill.

TABLE 1. TIMES AT WHICH RECORDS WERE OBTAINED DURING STUDY

Record	*Time*	*Date*	*Condition*	*State*
1	2:00 A.M.	Jan. 7	Presleep, 236 hr.	Awake
2	3:00 P.M.	Jan. 7	Presleep, 249 hr.	Awake
3	6:00 A.M.	Jan. 8	Recovery sleep	Asleep
4	8:00 P.M.	Jan. 9	Recovery sleep	Asleep
5	9:00 P.M.	Jan. 10	Recovery sleep	Asleep
6	9:30 P.M.	Jan. 15	Postrecovery	Asleep
7	3:00 P.M.	Jan. 18	Postrecovery	Awake
8	9:30 P.M.	Feb. 13	Postrecovery	Awake–asleep
9	9:00 A.M.	Feb. 15	Postrecovery	Awake
10	9:30 P.M.	Mar. 12	Postrecovery	Awake
11	2:00 P.M.	July 22	Postrecovery	Awake

each of EMG, plethysmograph, GSR, HR, skin temperature, and respiration. Stimulus onset and duration and voice were indicated by a stimulus-marker channel. Monopolar EEG recordings were made from electrodes in the left frontal, parietal, and occipital areas of the scalp with the reference electrode attached to the right ear lobe. In addition to the dynograph write-out, the EEG was also recorded on a Precision Instrument Model 400 tape recorder.* The EOG electrodes were placed on the outer canthus of each eye with the reference electrodes on the opposite ears. A reflectance photo plethysmograph was attached to the left middle finger. Muscle activity was recorded from an electrode placed on the submental area. The reference electrode was placed 1 in. to the side of the muscle. Tursky electrodes,† filled with Redux electrode paste,‡ placed on the volar pads of the right index and right ring fingers, were used to record skin resistance. A current of 38 μamp. was impressed through the electrodes. Heart rate leads were placed on the midclavicular line in either the fourth or fifth left intercostal space with a reference electrode below the right clavical. The heart rate was obtained with an Offner 9851 cardiotachometer.§ Skin temperature was obtained from the left little finger by means of a glass bead thermister. A strain gage belt placed around the chest recorded respiration.

*Precision Instrument, Palo Alto, Calif.
†Lexington Instrument, Waltham, Mass.
‡Sanborn Co., Waltham, Mass.
§Offner Electronics.

Results

Presleep and Postsleep Awake Records

EEG

As found by others, the prolonged period of wakefulness resulted in a decrease in alpha activity and an increase in slow activity. Examples of EEG activity during deprivation and following recovery sleep are shown in Fig. 1. There was almost no "eyes-closed" alpha after the S was awake 249 hr. but there was alpha during the eyes-closed record after 10 nights of recovery sleep (Record 7). Computer analysis by Burch Period Analyzer[6] indicated there was 31% delta, 34% theta, and 15% alpha on the presleep record. The postsleep record showed 15% delta, 41% theta, and 28% alpha. The awake eyes-closed record taken 7 months postsleep was very similar to the record obtained 10 days after the first recovery sleep.

From inspection of the EEG alone, it would be difficult to determine whether the pattern reflected a drowsy or an activated state of the subject. That the EEG pattern was more similar to a drowsy state than to an activated state was seen in the fact that no increase in alpha appeared before onset of sleep. If the sleep-deprived alpha suppression were indicative of high arousal, alpha should appear at some time in the inter-

val between eyes-open and sleep, as this area is passed through on the route from high arousal to sleep. If alpha suppression indicated drowsiness, i.e., below the alpha level, then no alpha should appear. On the 2 presleep records when the S was allowed to close his eyes and remain undisturbed, his EEG would quickly show high-amplitude slow waves with some spindling. These bursts were never preceded by alpha bursts. When the S was finally allowed to go to sleep after 264 hr. of wakefulness, he rapidly passed through Stages 1, 2, 3, to 4 without the appearance of alpha.

Visual analysis (Fig. 1) did not reveal any clear differences in amplitude and frequency between his presleep eyes-closed and eyes-open record—suggesting the patient was equally drowsy whether his eyes were open or closed. But computer analyses indicated EEG differences not visually detectable, suggesting the S was relatively more alert during eyes-open and during stimulation. Fourier analysis (autocorrelogram and power spectrum) revealed an eyes-closed alpha component which was not present when the eyes were opened. Similarly, while visual analysis did not indicate any presleep EEG change during sustained stimulation, the computer analyses again indicated further reduction of the limited alpha that was present in the eyes-closed resting record. Period analysis revealed that during stimulation the alpha per cent was 8 in contrast to 15 during eyes-closed. The ab-

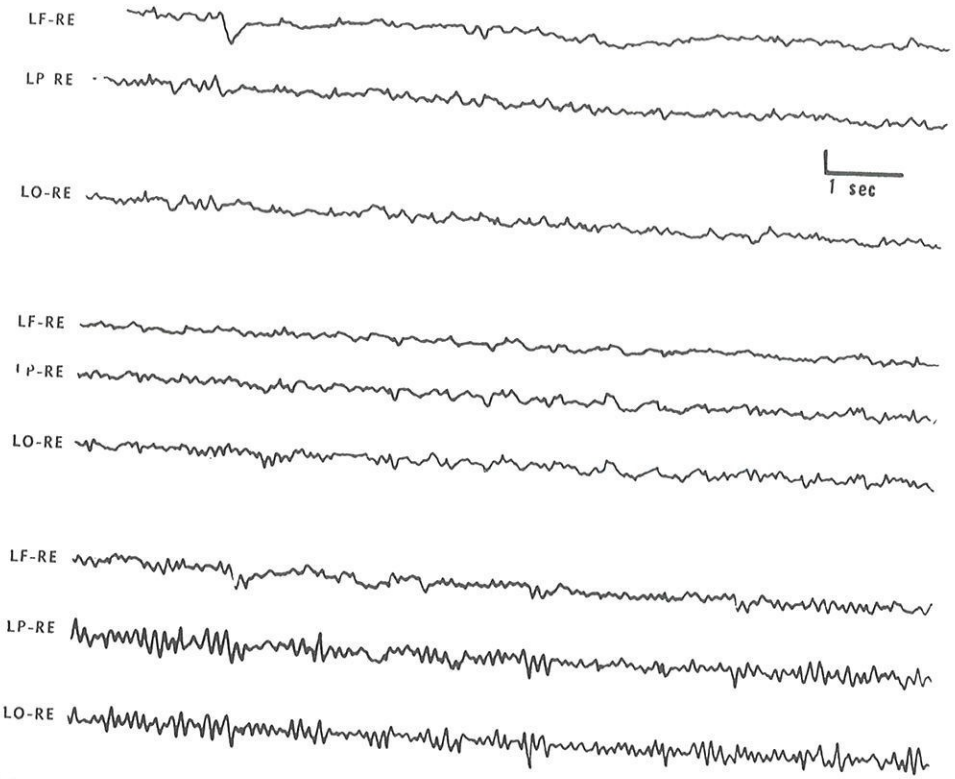

FIG. 1. EEG activity during and after sleep deprivation. *Top:* eyes open after 249 hr.; *center:* eyes closed after 249 hr. awake; *bottom:* eyes closed postsleep.

Lead placements: LF–RE, left frontal to right ear; LP–RE, left parietal to right ear; LO–RE, left occipital to right ear.

sence of visually detectable EEG response (alpha enhancement) to the first stimuli probably reflects the physiologic unresponsiveness seen so clearly in the autonomic variables. Alpha enhancement to stimuli was seen during drowsy portions of the seventh and subsequent postsleep awake recovery records.

Autonomic Resting Levels

The presleep and postsleep resting levels for skin resistance, heart rate, skin temperature, and respiration are presented in Table 2. The data in Table 2 represent the mean scores from a 1-min. sample of stabilized, artifact- and stimulus-free activity. Skin temperature was not recorded on the Day 196 postsleep record. The autonomic variables seem to reflect an activation pattern during the presleep vigil, with perhaps the exception of respiration rate. Skin temperature and heart rate show highest activation and, although a quantified measure of vasoconstriction was not possible, the morphology of the plethysmogram indicated marked vasoconstriction.

Response to Stimuli

During each record a series of 1000 cps tones (each having a duration of 1–3 sec.) at 25 db above hearing threshold were presented over a loud speaker. To assure that the S was awake and heard the tones, he was instructed to tap on a microphone with his left index finger immediately after each tone. The presence or absence of a response to other stimuli, particularly to that of calling the S's name, was also noted on the "pre" and "post" records. The responses to these stimuli were similar to those to the tones.

Responses to the first tone presented on each of the 7 records are presented in Table 3. Autonomic response to the first tone during prolonged wakefulness was depressed for all variables except respiration. There was no skin temperature response on either presleep record regardless of the type of stimuli, and a small GSR (1 K ohm) was evoked to tone only on the first record—after 236 hr. of wakefulness. There were also fewer nonspecific GSR's (11) on the pre-

TABLE 2. AWAKE AUTONOMIC RESTING LEVELS

	Presleep (hr. awake)		*Postsleep (days)*				
Measurement	*236*	*249*	*10*	*36*	*38*	*64*	*196*
Skin resistance (K ohms)	27	46	50	58	71	138	121
Heart rate (beats min.)	83	94	70	73	77	71	72
Skin temperature (degrees C)	26	22	32	36	33	36	—
Respiration rate (breaths min.)	18	18	15	18	16	19	21

TABLE 3. AUTONOMIC RESPONSE TO TONE STIMULUS

	Presleep (hr. awake)		*Postsleep (days)*				
Measurement	*236*	*249*	*10*	*36*	*38*	*64*	*196*
GSR (K ohms)	1.0	0.0	34	5	7	9	27
Heart rate (beats min.)	+4.0	+5.0	+21	+6	+5	+2	+18
Skin temperature (degrees C)	0.0	0.0	− .20	− .05	− .10	0	—
Respiration (breaths min.)	+4.0	+1.5	+ 4.2	+2.5	+ .0	+2.4	+ 5
Plethysmograph (no. beats change)	2.0	4.0	6	5	6	7	8

sleep records when compared to the nonspecific GSR's (34) for a similar time period on the postsleep records.

Heart rate and plethysmogram responses to the tone stimuli were present during the presleep records, but they were never so pronounced as the responses to the tone presented 10 days postsleep. Intense and unexpected stimuli, such as the clapping of hands directly over the S's face, provoked a heart rate increase of 18 beats, irregular respiration, and vasoconstriction, but it produced only a small change in skin resistance (5 K ohms) and no skin-temperature response.

In contrast to the unresponsiveness seen during presleep, there was marked responsiveness to the tones given 10 days postsleep. The presleep and postsleep response to the first tone are presented in Fig. 2. The EEG changes from the presleep to the postsleep period are also illustrated in Fig. 2. The response to an identical tone stimulus 36, 38, and 64 days postsleep was not so pronounced as that on postrecovery Record 7, reflecting habituation to this stimuli. But the S's response to the tone 196 days postsleep was similar to that 10 days postsleep. From Record 6, the S's postrecovery sleep response to the first presentation of other stimuli, however, was always pronounced.

On the 3 recovery-sleep nights there was an increase in both slow wave sleep (especially Stage 4) and REM sleep when compared with the postrecovery sleep records. During the first and second recovery nights, REM periods were unstable and were often interrupted by bursts of Stage 2 activity. Similar alter-

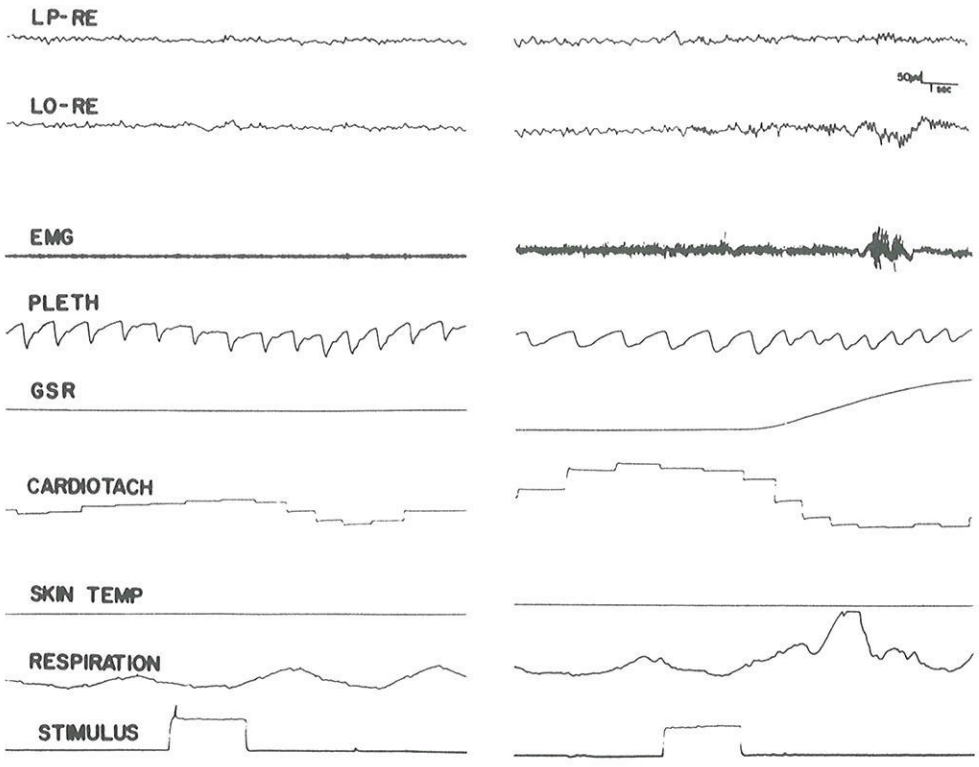

FIG. 2. EEG and autonomic response to tone stimulus pre- and postsleep. At left, 249 hr. awake; at right, 1 week postsleep.

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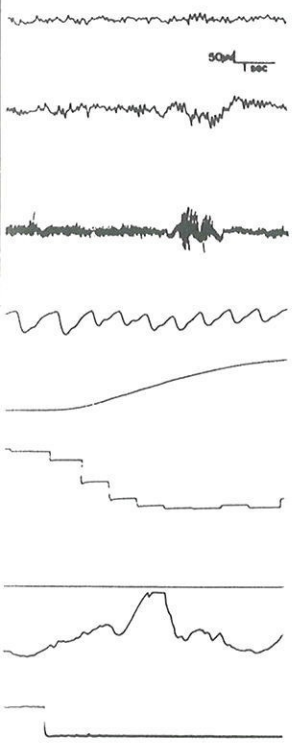

d postsleep. At left, 249

nation between REM and Stage 2 after sleep deprivation has been reported by Williams *et al.*[15] The increase in Stage 4 and REM sleep was seen on the first recovery night. This increase in both slow wave and REM sleep on the first recovery night is in contrast to the findings of Berger and Oswald[3] and Williams *et al.*,[15] who found an increase in slow wave sleep in the first recovery night but no increase in REM time until the second recovery night. The differing degree of sleep deprivation studied probably accounts for these differing results.

During the first recovery night our subject had very little submental muscle activity during the slow wave sleep and it was not possible to use the EMG as an indicator of REM sleep. On the second and subsequent nights of sleep, the usual EMG pattern was present, i.e., muscle activity was more pronounced during slow wave sleep than during REM. Fewer body movements were seen during recovery sleep, especially during Night 1.

The most dramatic autonomic change after the onset of the first recovery sleep was the rapid rise in skin temperature and the marked vasodilation. Within a span of 8 min. the finger skin temperature increased from 26° to 33° C and the amplitude of the plethysmogram pulse tripled. During this same period the HR decreased from 79 to 71 beats/min., but the skin resistance remained constant at 56 K ohms.

Response to Stimuli During Sleep

Several tone stimuli were presented in a random manner during recovery and postrecovery sleep but these stimuli were not fixed in number as the goal was to keep the S asleep. Within the first hour of the first recovery sleep, there was responsiveness in all variables except GSR. Specific GSR's were not seen until Recovery Night 2. After Night 2, response to stimuli was seen in all autonomic variables during all stages of sleep, including REM periods. As the stimuli were not presented in any systematic manner, however, no conclusions are being drawn about autonomic responsiveness with respect to stage of sleep.

Discussion

The opportunity for a thorough study of the subject was somewhat limited by the fact that the wakefulness marathon was well underway before it came to our attention. In particular, the lack of baseline data from the pre-experimental period prevents us from being absolutely certain that permanent changes of some sort did not occur. However, such a possibility must remain conjectural, and at worst could be only very subtle since observations were made for more than 7 months after the period of deprivation, during which time a return to normal physiologic functioning seemed to be well established. The knowledge that such an opportunity occurs so rarely, and the fact that to date no physiologic data are available from such a prolonged period of wakefulness seemed to justify our study of this subject and the presentation of these findings. The question immediately arises, however, whether our findings after 264 hr. of deprivation are greatly different from those that would have been found after 50, 100, or 150 hr. of wakefulness. That is, does sleep deprivation, if greatly extended, have a steadily increasing effect on the subject?

Comparison of our data with those published by others,[1, 4, 9, 13] suggests that our findings in some areas are similar to those reported after shorter periods of deprivation. Armington and Mitnick[1] reported a reduction of alpha after 24 hr. of wakefulness. After 72 hr. of deprivation, these authors reported that further alpha depression had occurred. Malmo and Surwillo[9] also reported early and progressive alpha depression in their study of 60 hr. of wake-

fulness. Alpha depression, thus, appears early during deprivation and is progressive—but at an increasingly slower rate. It is highly probable that the alpha depression seen at 264 hr. would have been similar to that found at 100 hr. The relative unresponsiveness to stimuli seen in our waking subject has not been so clearly noted in other studies. Armington and Mitnick[1] reported EEG changes during tasks and that a sudden noise elicited alpha after 72 hr. of wakefulness. Only by utilization of electronic computers could we detect the effect of opening the eyes and of stimulation upon the EEG trace. In our subject, visually detectable alpha enhancement was not present even to intense stimuli.

With respect to the autonomic variables there are fewer studies with which we can compare our findings and the results of these studies are not consistent.[2, 5, 9, 12] Burch and Greiner[5] report that the number of specific GSR's dropped sharply from Hour 18 to 37 of deprivation. Ax and Luby[2] also found decreasing GSR to a pain stimuli over a period of 27 to 123 hr. of deprivation. In present research at the San Diego Laboratory, a decrease in GSR to stimuli has been noted after 24 hr. of deprivation. Decrease in the GSR, thus, seems to appear quite early in the deprivation period. Whether the unresponsiveness we noted after 264 hr. was more pronounced than those reported by others is, of course, not known. Conflicting findings of an *increase* in GSR responsiveness after deprivation[9, 12] may have been due to the nature of the test environment.

Ax and Luby[2] found changes similar to those of this study for resting heart rate and skin temperature. After 60 hr. of wakefulness, however, they reported inconsistent heart rate and temperature response to stimuli. We found an inconsistent heart rate response to stimuli but no skin temperature response.

As with the EEG, many of our autonomic findings have been noted after shorter periods of deprivation, indicating that the physiologic effects of sleep deprivation appear quite early. It also appears that these changes are progressive but probably at a decreasing rate until a minimum or maximum physiologic level is reached. It would appear that the final effect of extended sleep deprivation is autonomic arousal and CNS depression, but almost complete unresponsiveness of all variables. It would also appear that some variables become unresponsive quite early while others, such as heart rate and vasomotor reactions, continue to be relatively responsive even after prolonged sleep loss.

Here is an apparent paradox. Our sleep-deprived subject is at a high level of arousal judged by autonomic measures, but at a low level of arousal judged by EEG and behavior. There is a divergence of CNS activity and autonomic activity. Thus the question of whether sleep loss arouses or depresses the organism appears to have no single answer. It must be answered in terms of the system being measured and the response to external stimuli may be a better indicator of sleep-loss decrement than the resting levels per se. With less prolonged sleep loss, the nature of test situation and subject motivation have been found to be an important factor in determining level of arousal and responsiveness,[14] but with prolonged sleep loss these external factors appear to have less effect.

Summary

As part of a planned experiment for the San Diego Science Fair, a 17-year-old boy went without sleep for 264 hr. All the behavioral characteristics of sleep deprivation short of psychosis were seen. EEG and autonomic data were obtained after 236, 249, and 264

hr. of wakefulness, during 3 recovery nights, 1 week, 6 weeks, and 7 months after deprivation. Visual and computer analysis of EEG indicated dominant activity in the slower frequencies during presleep records with a return of alpha activity after the first night of recovery sleep. On presleep records, opening and closing of the eyes had little effect, and stimuli did not produce alpha enhancement. The basal autonomic pattern during deprivation was that of activation but also of decreased responsiveness to external stimuli. Sleep onset caused marked responsiveness to stimuli in all autonomic variables except specific GSR. Specific GSR's did not occur until the second recovery night. Both Stage 4 and REM sleep were increased during the 3 recovery nights of sleep and few body movements were seen. The most dramatic autonomic change after sleep onset was the rapid rise in skin temperature and marked vasodilation.

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