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Psychiatric and EEG Observations on a Case of Prolonged (264 Hours) Wakefulness

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THE FIRST sleep deprivation study on man was carried out in 1896 by Patrick and Gilbert,[1] who kept three young adults awake for 90 hours. Visual hallucinations occurred in one subject. In the ensuing years, a number of studies have been done and a frequent finding has been the development of some kind of psychotic symptomatology. Perceptual illusions, transient hallucinatory episodes, and depersonalization are among the most commonly observed symptoms.[2-5]

With longer durations of sleep deprivation, more severe disturbances have been reported. Among 275 servicemen who had undergone 112 hours of sleep deprivation, Tyler[6] saw a few instances of behavior resembling symptoms of acute schizophrenia. Luby et al[7] reported a subject who developed a florid psychotic picture during the course of 220 hours of wakefulness which was completely reversed following 14 hours of sleep.

The effects of sleep deprivation ordinarily disappear when subjects are allowed to sleep. Even profound changes in test performance undergo up to 90% recovery after only one night of sleep.[8] However, it appears that the reversal may be delayed when sleep deprivation has progressed to full-blown psychosis. Katz and Landis[9] reported on a young man who volunteered to go without sleep for ten days, just to prove that it was an unnecessary habit. In the last few days of his vigil, he had persecutory delusions concerning one of the experimenters which did not entirely vanish in later months. Brauchi and West[10] described the case of a patient whose psychotic symptoms persisted for several months following the acute onset at the time of a seven-day vigil. In spite of psychiatric care which included the administration of chlorpromazine, several days elapsed before the reversal of acute paranoid psychoses apparently precipitated by sleep loss in two patients described by Bliss and co-workers.[11] Somewhat along this line, Koranyi and Lehmann[12] described the re-emergence of the original acute manifestations after 100 hours of sleep deprivation in six chronic schizophrenic patients.

Taking such findings into account, as well as the results of their own studies, West et al[13] have described "the psychosis of sleep deprivation." They suggest that psychotic reaction is "inevitable" if complete sleep deprivation continues for longer than 100 to 120 hours. Speculations regarding the mechanism of these changes center around a presumed disorganization of ego function as a result of fatigue and drowsiness.

A parallel line of research has complicated the problem. It has recently been established that sleep, long conceived of as an essentially unitary state, actually consists of *two* states which differ dramatically in their physiological properties and have almost nothing in common except recumbency and outward quiescence. One of these states is the more recently elucidated "rapid eye movement" (REM) sleep. The other, which occupies the greatest part of behavioral sleep, is the well-known electroencephalogram (EEG)

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defined state, characterized by the presence of slow waves and sleep spindles, which has come to be known as nonrapid eye movement (NREM) sleep. Several recent reviews thoroughly compare and contrast these two states with emphasis upon the properties of REM sleep.[14-16] In view of their marked disparity, it is quite likely that these two kinds of sleep perform entirely different functions, and consequently, it follows that selective elimination of one or the other would have different effects. Thus, it becomes a moot question as to which of the two is mainly responsible for the effects of complete sleep deprivation.

In an early study, Dement,[17] and Dement and Fisher [18] deprived subjects of REM sleep only and found that in the immediate postdeprivation period the amount of REM sleep underwent a marked compensatory rise. They also reported minimal personality changes.

The association of REM sleep and dreaming [14] plus the deprivation-compensation phenomenon suggested some sort of "need" for REM sleep. Assuming a concomitance of REM sleep and instinctual drive discharge, Fisher and Dement [19] speculated that "dream (REM) deprivation, carried out intensively enough and for a prolonged period of time might bring about a very large dream deficit, a great intensification of the pressure of instinctual drives toward discharge, eventual eruption of the dream cycle into the waking state and the development of hallucinations, delusions and other psychotic symptoms." In addition, this provided a handy explanation for the so-called psychosis of sleep deprivation. Unfortunately for this latter explanation, subsequent experiments in which REM deprivation was carried out for 15 to 16 consecutive days did not produce overt psychotic reactions, although there were signs that such a change was impending.[20] Whether or not selective REM deprivation would eventually produce psychosis, it certainly could not account for psychotic reactions that appeared after as little as four to six days of complete sleep deprivation. Thus, if sleep loss is to be considered the specific cause of the reported psychotic reactions in these cases, the major factor would have to be either NREM sleep loss alone or a combination of the effects that NREM or REM loss might produce singly. There are experiments which could be taken as support for the notion that the critical event in sleep deprivation is the loss of NREM sleep, specifically of the high voltage, slow wave portion called Stage 4. Berger and Oswald [2] deprived subjects of sleep for about 100 hours. Agnew and associates [21] found an increase in Stage 4 time after "depriving" subjects of this phase for two consecutive nights. Both groups of investigators hypothesized some sort of "need" for Stage 4 sleep.

One fact which seems well established is that the increase in REM sleep on recovery nights is proportional to the duration of the immediately preceding REM deprivation. In other words, the effect of REM deprivation is cumulative. Similar information pertaining to Stage 4 deprivation particularly long term deprivation (200 hours or more) is not available. The opportunity to study a case of unusually prolonged wakefulness has enabled us to make some evaluations of the psychological manifestations related to sleep loss, as well as to comment on the changes in the normal sleep cycle following 11 days of total sleep deprivation.

Report of a Case

The subject was a 17-year-old, single, white, male high school senior who undertook the prolonged vigil during the Christmas vacation. His purpose was to study the effects of sleep loss as part of a research project to enter in the local science fair. A goal of 264 hours of continuous 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 long; 260 hours was the "record." [22] Two close friends acted as "co-investigators." They administered tests to the subject and helped him to stay awake. The subject was in the presence of one or both of them at all times, and for the final 90 hours, at least one of the authors was in constant attendance.

The subject is the oldest of four children. The father is a career military officer, and the family has had frequent moves because of this. There is no history of psychiatric disorder in the family. Both parents were concerned about possible ill effects as a result of sleep loss, but felt less anxious when constant medical supervision was offered. The subject has always been a confident, self-assured individual and describes himself as

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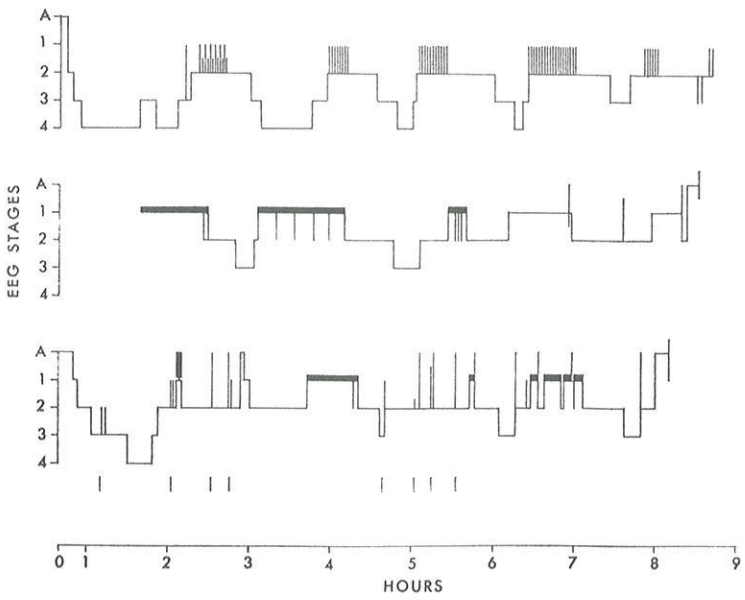

Continuous plots of the EEG stages during the first recovery night, represented by the upper two plots (a table of 14 hours, 40 minutes); and one week postrecovery represented by the bottom plot (about seven hours totally). Stage 1 represents REM sleep. The thick bars above Stage 1 indicate the relatively stable REM periods. Note the instability of the REM periods during the first recovery night, with frequent alternations between REM and awake (*A*), and between REM and Stage 2.

a little on the "egotistical" side. Peer relationships have been adequate despite the many changes in home address. He makes friends easily, and usually has one or two close friends.

The subject has been fascinated by "extremes" since early adolescence. On previous occasions, he has undertaken difficult projects because "people told me they couldn't be done." The motivation for the sleep deprivation study arose in part from this tendency. The need for mastery and the subsequent enhancement of self-esteem when the mastery has been achieved have been a repetitive phenomenon during the subject's adolescence.

The social milieu in which the experiment was performed was very supportive. The parents of the subject and his two colleagues were all cooperative. The subject received much support and encouragement from his two companions, and as the vigil progressed, local and national news media focused on the event which increased the group's motivation to succeed.

Psychiatric Observations During the Sleep-Loss Period.—The first three days produced no significant behavioral changes. At approximately 90 hours of sleep deprivation, the subject experienced his only illusion while walking down a darkened street during the early morning hours with one of his companions.

The early morning hours were the most difficult for the subject. At first, physical activity was curtailed due to fatigue, except when needed to offset unusually severe drowsiness. *At no time* was any drug or stimulant ingested by the subject, including coffee. The maintenance of wakefulness was essentially the responsibility of the subject's companions, who watched him closely and did what was necessary to avert falling asleep. This, of course, depended ultimately upon the subject's cooperation in the face of boredom and irritation.

Between the fourth and fifth day of wakefulness, the subject began to experience "waking dreams" and "lapses of memory." These were associated with decreased awareness of the environment and probably represented episodes of microsleep. Irritability became pronounced about this time and progressively increased. There was heightened suspiciousness and feelings of resentment, particularly in response to the constant questions about his state of wakefulness. However, this suspiciousness never reached the intensity of paranoid ideation. At no time was there evidence of delusional systems, hallucinations, or delirium.

A psychiatric interview was conducted at 262 hours of wakefulness. The subject was well oriented as to time, place, and person (fleeting disorientation as to time had been noted occasionally throughout the study), associations were logical and coherent, and there was no loss of contact with reality. In the subdued, relaxed atmosphere of the interview (as opposed to the crowd of watchers and glare of TV cameras as the vigil neared its end), the irritability and suspiciousness were completely absent and the subject was able to perceive and comment on these psychological changes, recognizing them as ego-alien. Throughout the vigil and particularly toward the end, lack of movement and stimulation brought on extreme drowsiness with a concomitant deterioration in performance, while movement, stimulation, and novel experiences, on the other hand, were almost always associated with a return of virtually normal function. As an illustration of this, on the night before the final day, the subject wandered about the city in the company of one of the authors and one of his companions. There was little in his behavior during this period (midnight to 5 AM and 230 hours of sleep loss) to suggest that he had been awake longer than his companions. At one point, in an all night

Recovery and Past-Recovery EEG Sleep Data Indicating Amount of Time for Each Stage of Sleep

	Recovery Sleep Sessions (Min/Stage/Night of Sleep, %/Stage/Night in Parentheses)			Post-Recovery Sleep		
Stages	1	2	3	1 Wk	6 Wk	10 Wk
Awake	0	0	11	15	33	16
	(0)	(0)	(2.02)	(3.54)	(8.57)	(4.0)
1	1	3	1	6	36	22
	(0.11)	(0.5)	(0.18)	(1.42)	(9.31)	(6.0)
2	397	258	224	255	183	227
	(45.2)	(41.3)	(41.4)	(60.1)	(47.13)	(58.0)
3	133	109	98	57	46	39
	(15.1)	(17.4)	(17.9)	(13.3)	(11.86)	(10.0)
4	113	67	60	18	34	12
	(12.8)	(10.7)	(11.0)	(4.24)	(8.85)	(3.0)
REM	236	188	152	73	56	75
	(26.8)	(30.1)	(27.8)	(17.4)	(13.64)	(19.0)
Total sleep (min)	880	625	543	424	388	391

restaurant, the three companions engaged in competition on a complicated pinball machine with the subject holding his own in every way.

A variety of bodily sensations were noted by the subject. The most prominent was a varying, and at times overwhelming, sense of fatigue and a feeling of heaviness and burning of the eyelids. Physical examinations were done repeatedly during the vigil and were essentially negative except for the development of certain minor neurological signs. These included bilateral ptosis, finger tremor, nystagmus, and hyperesthesias of the foot and hyperactive reflexes. A complete description of the neurological examinations has been published elsewhere.[23]

After the subject awoke from his first recovery sleep, there was a marked reversal of fatigue and drowsiness, as well as a marked resurgence of vitality. In addition, all the neurologic findings were reversible with remission beginning after the first recovery night and being complete a week later.

Sleep EEG Recordings.—Continuous sleep recordings were taken during the first three recovery sleep periods and then on three subsequent nights, seven days, six weeks, and ten weeks, respectively, after the termination of the prolonged vigil. In addition, EEG and autonomic data were recorded at 236 and 246 hours of wakefulness and have been described in detail in a separate report.[24] In this presentation, we will be concerned mainly with the variations in amount of REM and NREM sleep, and the latter was divided into EEG Stages 1 through 4, according to specific criteria.[25]

The first recovery sleep period began at 6 AM and was spontaneously terminated by the arousal of the subject 14 hours and 40 minutes later. The subject was asleep within two minutes after getting into bed, and there was a normal, but rapid, progression of NREM stages from 1 to 4, the latter being reached eight minutes after sleep onset. The first Stage 4, seen after 264 hours of wakefulness, was no different in terms of wave form and amplitude than the Stage 4 sleep seen two months later, although it appeared that the arousal threshold during the former was elevated. The initial Stage 4 period lasted 43 minutes. It was interrupted by what appeared to be an abortive REM period 52 minutes after sleep onset. This was followed by 12 minutes of Stage 3 during which there were "saw-tooth" waves and one or two questionable eye movements. The first clear REM period occurred 90 minutes after sleep onset and lasted 10½ minutes.

During the course of the first recovery sleep period, Stage 4 accounted for a total of 113 minutes or 12.8% of total sleep time. All Stage 4 during this night occurred within the first six hours of sleep, about 59 minutes of the total 113 occurring in the initial NREM period, the remainder occurring in successively smaller amounts in the next three NREM periods.

There were ten clear-cut REM periods during the first recovery sleep period. However, during such a period, there was a very marked instability of the EEG with frequent alternations between low voltage and spindle-plus-"K"-complex patterns, the latter considered to define NREM sleep. In view of this, REM sleep was scored as a maximum and a minimum. The latter was the total amount of Stage 1 episodes *within which* there were rapid eye movements. Thirty seconds were subtracted for isolated spindles or "K" complexes, or both, and where more than one occurred, all the time from the beginning to the end of a series of these wave forms was carefully subtracted. By this method, minimum REM sleep accounted for 237 minutes, or 27% of the total sleep period. If the brief intervals of spindling patterns were not subtracted from the body of the REM periods, REM sleep accounted for 303 minutes, or 34.6% of the total sleep time. Since the appearance of a sleep spindle in the EEG during a REM period in this unusual situation does not necessarily mean that REM sleep was interrupted, it is assumed that the true amount of

ount of Time for Each Stage of Sleep

	Post-Recovery Sleep	
Wk	6 Wk	10 Wk
5	33	16
.54)	(8.57)	(4.0)
6	36	22
.42)	(9.31)	(6.0)
5	183	227
0.1)	(47.13)	(58.0)
7	46	39
3.3)	(11.86)	(10.0)
8	34	12
.24)	(8.85)	(3.0)
8	56	75
.7.4)	(13.64)	(19.0)
24	388	391

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REM sleep lies between these two extremes, probably closer to the smaller value. The electromyogram (EMG) from the submental muscle was of little help in differentiating REM sleep because it was silent nearly all the time, including the NREM phases as well. The figure is a plot of the EEG stages during the first recovery sleep and, for comparison, of the all night recordings taken one week later.

The instability of the REM periods was evident on the second and third sleep recordings, but not during the fourth, which was separated from the third by an interval of four nights.

The table shows the absolute amounts and percentages of REM sleep (minimum values) and each of the NREM sleep stages during the recovery and postrecovery recordings. Since a month elapsed between each of the last three all-night recordings, and they are not substantially different, we have regarded them as baselines. There was no opportunity to observe the subject prior to the vigil.

The total REM sleep time appears to show a make-up effect not unlike what would have been expected if the recovery nights had followed 11 nights of selective REM deprivation. There is also a marked rise in Stage 4 time. The great length of the initial sleep periods must be taken into account when considering the percentages.

Comment

Observations of a subject during the course of 264 hours of continuous wakefulness revealed the development of many behavioral changes that were more or less characteristic of sleep deprivation, but a psychotic state did not emerge, nor was there any notable occurrence of individual psychotic symptoms. This suggests that the psychosis of sleep deprivation is not a general phenomenon inevitably brought on by prolonged wakefulness, and that other factors should be considered in explaining frequent development of psychosis observed by others.

In the first place, the subject in this study was a stable, healthy person without prior history of psychopathology. A Rorschach examination and Minnesota Multiphasic Personality Inventory (MMPI), conducted by us a month after the sleep deprivation, showed no deviation from normality. In other reports where florid psychosis developed, some predisposition of the subject seemed evident. For example, the subject reported by Katz and Landis [9] was already in the grip of a peculiar obsession. The subject reported by Luby et al [7] who developed a full-blown psychosis after 220 hours of wakefulness had a traumatic childhood with a history of pyromania and a poor work history. The subject reported by Brauchi and West [10] had been hospitalized four times, and the psychosis following the prolonged vigil was the second psychotic episode in his life. The patients reported by Bliss and co-workers [11] experienced the original sleep loss after the development of an acute paranoid state. Thus, our subject may have been unique in terms of his basically healthy personality; and while he experienced some of the ego-disorganizing effects of sleep deprivation, he was apparently able to cope with them more successfully than previous subjects.

Our subject also had the benefit of constant support and encouragement from his two friends, from his family, from local and national news media, from the medical staff at the San Diego Naval facility, and from the present investigators. He was never alone, literally or figuratively. The degree of emotional support experienced from the environment was very likely a factor in his resistance to severe disturbance.

Finally, the factor of age should be mentioned. As Kleitman [4] has often pointed out, the most important activity in resisting the effects of prolonged wakefulness is vigorous movement. Accordingly, sleep deprivation is not merely the elimination of sleep, but includes the *addition* of a substantial amount of muscular activity which of necessity fills in the gap. It seems likely that a young, fit, 17-year-old person could endure this marked increase in muscular activity and utilize it more effectively than an older person. Such things as joint pains, muscular aches, etc, which tend to reduce mobility with the consequent susceptibility to drowsiness were not experienced by the subject.

If the psychotic symptomatology is not specifically caused by sleep deprivation, there is still a possibility of specificity with regard to such changes as impaired performance, etc. We feel that an explanation for these changes lies in a capacity for individuals to be behaviorally awake and yet virtually asleep—in other words, behaviorally awake in the presence of markedly impaired cortical

function. The evidence for this conjecture is twofold: (A) Williams and his colleagues [8] as well as others,[26] have reported "microsleeps" during which there is frequently a substitution of θ-waves for α-rhythm associated with failure to respond in sleep deprived subjects; and (B) there are several situations in which dissociations between behavior and cortical function occur. One of these is sleepwalking. The episodes generally begin in the middle of Stage 4 and the Stage 4 EEG patterns tend to persist through the sleepwalking behavior.[27] We suggest that many changes, which are generally consequent to sleep loss, particularly in the perceptual and performance spheres, are actually the behavioral concomitants of the drowsy state.

The instability of the REM-period EEG with frequent lapses into spindling was seen in our subject and persisted for the three recovery sleep nights. This phenomenon has been observed to a lesser degree in all other cases of sleep deprivation that have been studied. This may suggest a functional depression of the cortex in sleep-deprived subjects during REM, when we ordinarily expect a period of sustained activation. However, this does not apply to the waking state where EEG activation was quite normal after the first recovery night. We have seen a similar REM-EEG instability in cats after electrically induced grand mal seizures, likewise with no apparent change in activation in the waking EEG.[30]

The results obtained in the present study did not rule out the possibility that REM sleep deprivation alone may eventually lead to psychosis, since other experiments suggest that more than 15 days deprivation would be required.[20] It is also likely that the effects of REM loss and NREM sleep loss are to some extent in opposite directions. Recent studies [28,29] in animals have shown that selective REM deprivation results in neurophysiological changes which are in the direction of heightened excitability, and there is no sign of drowsiness. On the other hand, total sleep loss of short durations in which the major effects may be presumed to be loss of REM sleep *do* result in drowsiness, and certainly an impairment of performance. The greater "need" for slow wave sleep which seems apparent in short-term sleep deprivation experiments appears to be overturned when a larger REM deficit has accumulated, though not entirely. The major sign of a competition between REM and NREM sleep, if such exists, would seem to be the rapid alternation between a spindling and nonspindling EEG during REM periods in the first recovery sleep period.

The rise in amount of Stage 4 sleep was no greater after 264 hours of wakefulness in this experiment than after about 100 hours of wakefulness, as reported by Berger and Oswald.[2] Consequently, the evidence does not support the idea that Stage 4 loss is cumulative. On the other hand, if baseline REM time is somewhere around 70 to 90 minutes, the make-up on the first three days, 236, 188, and 151 minutes respectively, is quite appropriate to an 11 day deprivation. If the recovery sleep periods had been limited to eight hours per day, the rise in REM time would undoubtedly have persisted longer.

A final comment is that we have not ruled out the possibility that psychosis would be inevitable after longer periods of total sleep deprivation, for example, 20 days. However, we cannot suggest a plausible mechanism by which short periods of REM or NREM sleep, or both, would be the specific cause of psychosis in an otherwise healthy individual. We do suggest that the so-called psychosis of sleep deprivation probably results from a variety of factors including intrapsychic, psychosocial, neurochemical, and neurophysiological.

Summary

This study reports psychiatric and sleep EEG observations after 264 hours of sleep deprivation in a normal, 17-year-old, white boy. Our subject developed some behavioral changes frequently reported as characteristic of sleep deprivation, but the so-called psychosis of sleep deprivation did not develop. Several possible reasons for the maintenance of ego functions despite the severe stress of prolonged wakefulness are discussed.

Sleep EEG data from three recovery sleep nights and from three postrecovery nights

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Summary

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are presented. There was a marked increase in REM sleep during the recovery nights comparable to the increases seen in REM deprivation studies. The increase seen in Stage 4 sleep during the recovery nights was no greater than has been observed after 100 hours of wakefulness. These data do not support the idea that the deprivation of Stage 4 sleep is cumulative as is the deprivation of REM sleep.

The data indicate that prolonged sleep deprivation does not necessarily result in psychosis. However, other behavioral changes noted during prolonged wakefulness may be specifically associated with either REM or NREM deprivation, and some possible mechanisms regarding this specificity are outlined.

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Generic and Trade Names of Drugs

Chlorpromazine—*Thorazine.*

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