Normal Adult Human Sleep as a Problem-Solving Process II: Sleep Cycling Data on Naps and Sleep Stage and Sleep Deprivation

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If sleep merely recycled whenever it began, there would no explaining it in the terms I have proposed (articles at http://www.hdbkpersonality.com/academic/index.htm). A daytime nap that was inevitably dominated by much slow-wave sleep would be utterly unfathomable from my point of view. If sleep is actually an adaptive response to events occurring in the interval since last sleep, it follows that the length of that interval should exert an important influence on the progression of sleep stages. An interval much shorter than normal should mean many fewer memories than normal to be assessed and integrated into long-term memory, and this should exhibit itself as less initial time spent in stages 3 and 4 and a consequently shorter latency to the first REM period. As the waking interval lengthens to the normal span, initial sleep should approach normal nocturnal sleep in appearance, meaning there should be more slow-wave sleep, more of a delay in REM onset, and, with this delay, eventually a noticeable shortening of this first REM period, due to it coming before the slow-wave memory-integration process has been completed.

This initial slow-wave memory integration process may be thought of as forming a person's basic adaptation, which is then refined in subsequent sleep cycles. The basic adaptation occurs during the first two spans of slow-wave sleep, which are separated by either a REM period or time spent in stage 2. When a REM period is involved, memories activated during the REM period participate in the midcourse correction; when only a stage 2 interval bridges the two slow-wave spans, adjustments presumably are made on the basis of the factors that inhibited the REM period. The intervening REM or stage 2 period is seen as being curtailed in length because it comes before the basic adaptation is complete, a circumstance that brings about a strong "critical" reaction.

The Early Morning Nap

In a study conducted by Webb, Agnew, and Sternthal (1966), subjects were monitored during early morning (0900±15 min) naps subsequent to awakening at home at 0700 from presumably good nights of sleep. They were each allowed to nap for a minimum of 120 minutes. Table 1 displays their mean sleep stage percentages with those corresponding to the first and last 120 min of normal nocturnal sleep. Group A consisted of subjects who had taken part in many previous sleep studies, while Group B, the nonhabituated group, were subjects who had never before participated in sleep studies and who "were given only one habituation session (prior to the morning the recording was made) in which they were allowed to sleep in the laboratory while partially wired."

As can be seen from Table 1, the sleep of both groups closely paralleled that of the last 120 min of nocturnal sleep, with the more striking resemblance coming in the case of Group A. Webb et al. (1966) interpret Group B's results as being indicative of a "first night effect." I concur in this and therefore will discuss each group separately, considering Group A first.

These subjects merely dressed, had breakfast, and reported to the laboratory — that was their 2-hr "day." It is difficult to see how any of this could seriously interfere with the return to nocturnal themes in these naps. The most obvious interpretation of the fact that Group A's sleep resembled that of the last 120 min of nocturnal sleep is that their naps merely continued that sleep, and this is what I believe took place.

TABLE 1 Percent of sleep in each stage during the first and final 120 min of nocturnal sleep of a group of laboratory-acclimated control subjects vs. that for two groups of subjects during a 120-min early morning nap

Stage[a]	1st	Last	Group A Habituated	Group B Nonhabituated
0	0.4	1.0	6.8	2.0
1	6.1	6.7	11.3	12.7
REM	3.3	39.5	38.2	25.8
2	36.4	46.2	39.2	57.2
3	10.7	3.4	4.5	2.3
4	43.1	3.2	0.0	0.0

[a] Stage scoring according to a modified version (Williams et al., 1964) of the Dement-Kleitman system and for REM using the Dement REM scoring manual (Dement, 1962). Table data from Webb, Agnew, and Sternthal (1966).

If it is true that this sleep constituted a return to nocturnal themes, I would expect the latency to first REM period to be relatively short. In fact, a long mean latency would represent a challenge to everything that has been said. The mean latency to first REM for these subjects was 6.8 min, with the shortest actual latency for both groups being less than 1 min. This, I claim, is consistent with my interpretations. I will now turn to the sleep of Group B.

Sleep researchers do not usually bother to record their subjects' sleep their first night in the laboratory. The reason is that this sleep differs significantly in many respects from that on subsequent nights. One of these differences concerns the distribution of EEG stages during sleep, an effect that also should be understandable from the point of view I have developed.

I see acclimation to the laboratory as essentially an attempt by the brain to become habituated to the new sensory signals that come to it continuously during the first night: the new feeling of the bed, the new smells, the new sounds, the continual pokes from the electrode placements. I see this newness as inhibiting concentration, thus making it more difficult for a person to achieve and hold in mind those integrations upon which the confidence to dream depends. Consistent with this view is the finding that subjects generally take longer to fall asleep on their first night and that they usually awaken more frequently during it (Agnew, Webb, and Williams, 1966; Mendels and Hawkins, 1967). Consistent, too, is the pronounced tendency to "miss" the first REM period and to have less REM period time overall (Agnew, Webb, and Williams, 1966; Rechtschaffen and Verdone, 1964).

It is not clear how much habituation time Group B had. Apparently it was only 2 hr. Thus it would be understandable if the subjects did show some evidence of a first night effect. In comparing the sleep of Groups A and B, we find that Group B showed less REM time and a large increase in stage 2. In addition, their mean latency to first REM period was 38.9 min, in contrast to 6.8 min for Group A. All of this, I contend, is consistent with my distraction hypothesis.

The morning nap is therefore in this instance entirely in line with what one would be led to expect from the point of view of my interpretive framework. This was, however, something of a special case. Let us turn now to the characteristics of the afternoon and early evening naps, neither of which can be explained as a simple continuance of previous nocturnal sleep.

The Afternoon and Early Evening Naps

Maron, Rechtschaffen, and Wolpert (1964) studied the sleep stage distributions of the 1:30 pm afternoon and 7:30 pm early evening naps. Their findings expressed as means are summarized in Table 2 and shown in comparison with the mean characteristics of the first 120 min of nocturnal sleep.

TABLE 2 Sleep stage characteristics of early and late afternoon naps vs. those during the first 120 min of normal nocturnal sleep

	1:30 pm Nap	7:30 pm Nap	1st 120 Min of Nocturnal Sleep[a]
% sleep in REM periods	14.6	3.9	3.3
% sleep in stages 3 and 4	19.3	41.3	53.8
Mean latency to first REM	64.2 min	84.0 min	117.8 min
Mean time asleep	106.4 min	98.9 min	120.0 min

[a] Data from Webb et al., 1966.

Shown in Table 2 is the expected increase in slow-wave sleep as the interval between the nap and previous sleep lengthens. Shown here, too, is a longer REM period (REMP) latency and an apparent inhibition of REM sleep due to an increase in the amount of slow-wave sleep occupying the second NREM period. Maron et al. (1964) say, however, that only 4 of the 9 evening nappers had REM periods, whereas these were found in 8 of the 9 afternoon nappers' sleep. This and the increased REM latency mean that what we are perhaps seeing is not inhibited REM but merely delayed REM, that if the late afternoon nap had been longer, its REM portion would have been much greater than that in a corresponding span of normal nocturnal sleep.

In a study conducted by Webb and Agnew (1967), subjects were allowed to nap for 180 minutes at various times in the afternoon. A portion of their data is reproduced in Table 3 and shown with the mean sleep stage percentages for the first three hours of normal nocturnal sleep. Although this data does not bear directly on the 7:30 pm nap, Table 3 does indicate inhibition of REM more than mere delay.

TABLE 3 REM and slow-wave sleep percentages for 180 minutes of sleep

Stage	21:30 pm Nap	2:00 pm Nap	4:00 pm Nap	Nocturnal Sleep[a]
REM	12.0	15.0	8.0	7.7
Slow-wave	19.0	25.0	26.0	50.1

[a] Data from Webb and Agnew, 1965.

The data pertaining to the morning, afternoon, and early evening naps, therefore, seem to be at least in general accord with the viewpoint I have developed. However, there exists at least one way to test the interpretations that have been made.

I have interpreted the morning nap as primarily a continuance of previous nocturnal sleep. If this is the case, then the existence of such a nap should have little effect on the sleep of the night that follows. In contrast, an afternoon nap that shows a good deal of slow-wave sleep should have dramatic effects on subsequent nocturnal sleep. One expectation is that nocturnal sleep should show a significant decrease in slow-wave sleep, because some slow-wave work in processing the day's events has already been accomplished.

Williams, Karacan, and Finley (1970) have studied the effect of a 2-hr morning or afternoon nap on the nocturnal sleep that follows. They found that a morning nap had "no effect" on subsequent nighttime sleep and that after an afternoon nap "a reduction in stage 4 sleep always occurred that night."

Williams et al. (1970) do not mention the mean length of the first REM period on nights that follow an afternoon nap. Neither do they discuss changes in the latency to this REM period. If, however, the afternoon nap and the sleep of the following night do in fact represent an instance in which slow-wave consideration of the previous day's events has been parceled out into two sleep intervals, I would expect that the first nighttime REM period should have a tendency to come sooner than normal and to have a longer than average length, since more of the basic adaptation has been completed before the REM period takes place.

I know of no study that deals explicitly with the characteristics of the first REM period of nocturnal sleep subsequent to an afternoon nap; nevertheless, I do find a report by Jones and Oswald (1968) to be intriguing. Jones and Oswald monitored the sleep of two men who habitually slept only three hours a night. A portion of their findings for one of the men is reproduced in Table 4.

TABLE 4 Delays (in min) from first EEG sleep spindle to first REM period

Night 1	32
Night 2	35
Night 3	43
Night 4	7
Night 5	29
Night 6	42

What I find, intriguing is the 7-min latency to first REM period on Night 4. On this occasion, the first REM period continued for the unusually long time of 37 min. Jones and Oswald say that both of these men led very active lives, and neither could be convinced to stay in the laboratory setting except to sleep. The possibility of prior napping was therefore very real. Had this particular individual napped for about 30 min to an hour a few hours before reporting, his nap would have contained much slow-wave sleep and possibly even a REM period. This could very easily explain the short latency and the unusually long first REM period. In any case, these are the types of effects I think a late nap should introduce.

Interrupted Sleep

Before considering the REM state derivation data, I wish to discuss one more experimental manipulation of sleep. Webb and Agnew (1969) studied the 8-hr sleep of 6 subjects that was interrupted by a 1-hr period of wakefulness after 4 hr. This experimental program was followed by another in which 4 hr was interjected between the two periods of sleep. The authors in each case then compared the two 4-hr sleep intervals to see if a recycling of sleep had occurred.

A person's basic slow-wave adaptation is largely completed within three hours. After this time, a person begins to primarily rely on the REM state to hone his adaptations. Thus, I would not expect the second sleep period of these subjects to be dominated by slow-wave sleep, since the slow-wave work has already been done. My feeling would be that the second sleep interval should be dominated by REM sleep, just as the second four hours of continuous sleep is.

Webb and Agnew report that for both groups of subjects "almost all of the stage 4 sleep occurred in the first period and little of it in the second period. Conversely, REM sleep was limited in the first period and was considerable in the second period. The overall percentage of sleep stages across the eight hours closely resembled that found across eight hours of uninterrupted sleep."

There is one aspect of these data that merits further discussion. The four hours of wakefulness in the second experiment is a goodly long time. It is difficult to imagine that the slow-wave thought of the previous four hours could remain frozen in place during for this span so that thought processes could immediately take up where they left off. A more reasonable assumption would be that wakefulness would modify them, with the amount of modification being a function of the length of the interval awake. Therefore, I would expect the sleep after 4 hr of wakefulness to not look exactly like its counterpart after a 1-hr period of wakefulness. There should be a bit more slow-wave sleep after the four-hour span. A question exists, however, whether this increase should be visible in terms of such a coarse measure as percentage of total time.

In their preliminary report, Webb and Agnew (1969) speak of "an insignificant but apparent increase in stage 4 during sleep after the four-hour interruption when compared with the one hour interruption." Therefore, even the details of their results meet with what seem to be reasonable expectations based on the view of sleep I am attempting to establish.

REM Period Deprivation via REM Sleep Interruption

REM period deprivation by means of the technique of REM interruption consists of awakening subjects whenever instrumental indications show that a REM period has begun. I will examine the data pertaining to this procedure by pooling information from three experiments, all of which used the same criterion for awakenings (Dement, 1960; Kales et al., 1964; Sampson, 1965). I will then round out this discussion by turning to the subject of REM period prevention as achieved by partial sleep deprivation.

First REM deprivation night

The first effect of REM period interruption is to cause an increased number of nighttime REM attempts. This increase begins with the first deprivation night and grows with each succeeding night of REM-period interrupted sleep. A normal night of continuous sleep usually contains 4 or 5 REM attempts. Dement reports that his subjects made 7 to 22 attempts during their first night of deprivation, with the mean number being 10.9. Kales et al. and Sampson show much the same result. Kales et al.'s two subjects made 13 and 16 REM period attempts, respectively, on their first deprivation night. The mean for Sampson's subjects was 11.2.

I have taken the position that REM periods evoke and organize memories held in long-term storage that then participate in subsequent NREM thought. Another way of saying the same thing is to state that a REM attempt is an indication that NREM thought has come about as far as it can alone. The need to have a REM period so that the work of sleep can continue does provide an explanation for REM prevention leading to an increased number of REM period attempts. This explanation is credible, however, only after certain problems are met.

If a person is truly ready to have a REM period after his basic slow-wave adaptation is complete, and if he must have the REM period in order for his thought to advance beyond this point, it would seem natural to expect that a person's second REM attempt should immediately follow his return to sleep. Further, his third and subsequent REM attempts should also come with equal speed. Thus, our problem is not to explain why awakenings lead to an increase in REM attempts, but rather why this increase the first deprivation night is so small. I believe at least two factors are important in this.

The proposition that if REM sleep is prevented, a person should attempt a REM period immediately upon falling asleep assumes that the act of arousal plays no part in disorganizing NREM thought. It in fact assumes that the integrations that led to the REM attempt remain frozen in place until the opportunity for sleep resumes. This would appear to be highly unlikely. One would think that the act of arousal would tend to disorganize previous thought and that therefore a contributing factor to REM attempt delays would be the time needed merely to assemble these integrations once again.

Dement and Kleitman (1957) offer some evidence on this point. As a part of their study of the normal night they made many REM and NREM awakenings for the purpose of sampling mental content. I quote their experience regarding the manner in which subjects returned to sleep after being awakened from REM sleep.

"As a rule, eye movements did not immediately recur upon the return to sleep. In 191 awakenings during eye movement periods in the earlier study there were only

16 instances of' recurrence, Interestingly enough, 12 of the 16 were preceded by 1-5 min of stage 2 sleep without eye movements, and 9 of the 16 were occasions when the subjects were unable to remember dreams and thus only had to say, 'I don't remember,' before going back to sleep. Furthermore, it was noted in the part of this study concerning auditory thresholds, where the subjects had only to clench their jaws three times, that eye movements recurred much more often, about 50% of the time. It seemed that when the subjects had to awaken fully and describe a dream, the return to sleep generally was more like the initial sleep onset and a new cycle was initiated, whereas a brief or 'partial' awakening often allowed the eye movements to continue."

This statement would argue that sleep time mental integrations are fairly easily disrupted. Even clenching one's jaws three times without really awakening was enough to disrupt a REM period for a time in 50% of the cases. This statement would indicate, further, that actual awakenings cause even more disorganization, with the extent of this being roughly proportional to the length of the waking interval.

Of his dream interruption procedure Dement states, "During each awakening the subjects were required to sit up in bed and remain fully awake for several minutes." Kales et al. are less specific; however, they do state that their subjects were "fully awakened" at the onset of each REM period. Sampson states that subjects were "kept awake for a minimum of three-four minutes." Thus disruption of thought was a possible factor in lengthening the interval between dream attempts during these experiments.

Another, I feel, had to do with the criterion used for REM period awakenings. All three experiments waited for rapid eye movements to signal the beginning of a REM period. As a general rule, the stage 1 EEG, the EMG voltage drop, and the onset of REMs are not simultaneous. It is my impression also that REMs often are the last indication to appear and that the interval between their appearance and what is now considered the time when the REM period begins can be more than 1 to 2 minutes in length. Dement states that "each awakening was preceded by a minute or two of dreaming," meaning that each came a minute or two after the first appearance of REMs. Kales et al. claim awakenings usually "within 15–40 sec after the first eye movement burst." Sampson makes no claims; however, approximately the same amount of delay can be safely assumed. If we add one or more minutes to each of these times, it can be seen that these subjects did achieve some REM period time with each of their REM attempts. It would not be true to say of these subjects, therefore, that upon returning to sleep they found themselves at the same conceptual point they were before attempting a REM period. Each small interval of permitted REM thought carried them a tiny bit farther, and, in doing so, I feel, must have contributed to a delay in the onset of the attempt that followed.

The statement that the amount of permitted REM sleep is a factor in lengthening the span between REM attempts carries with it the prediction that if shorter REM periods are allowed, the number of REM attempts during a night should increase. One way to interrupt REM periods sooner would be to awaken subjects when EMG suppressions occurred.

Kales et al. (1964) actually conducted two REM period interruption experiments with the same subjects. The first ran for 6 deprivation nights and based awakenings on the appearance of REMs. The second, however, ran for 10 nights and used EMG

suppression. That the second method led to more frequent awakenings most nights can be seen in Figure 4.

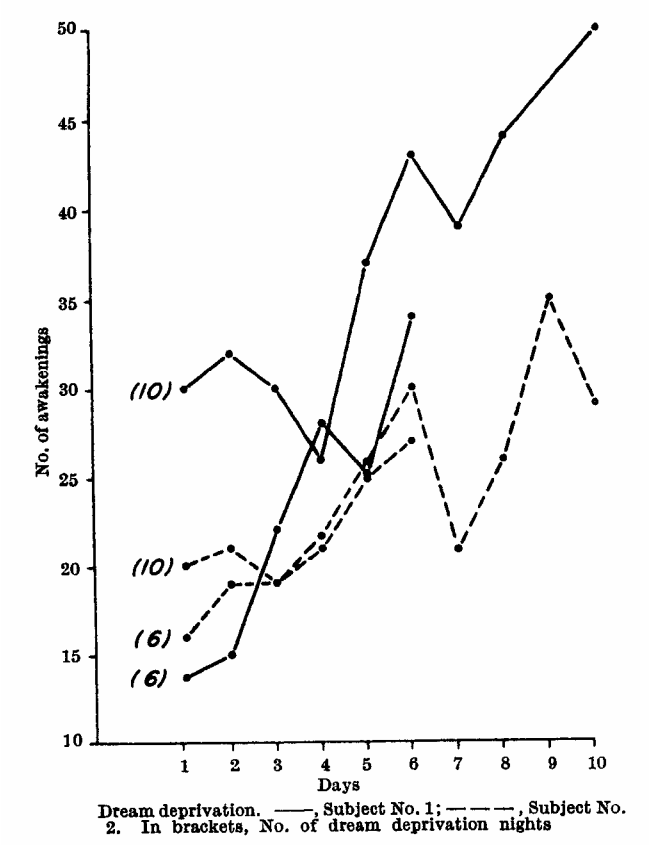


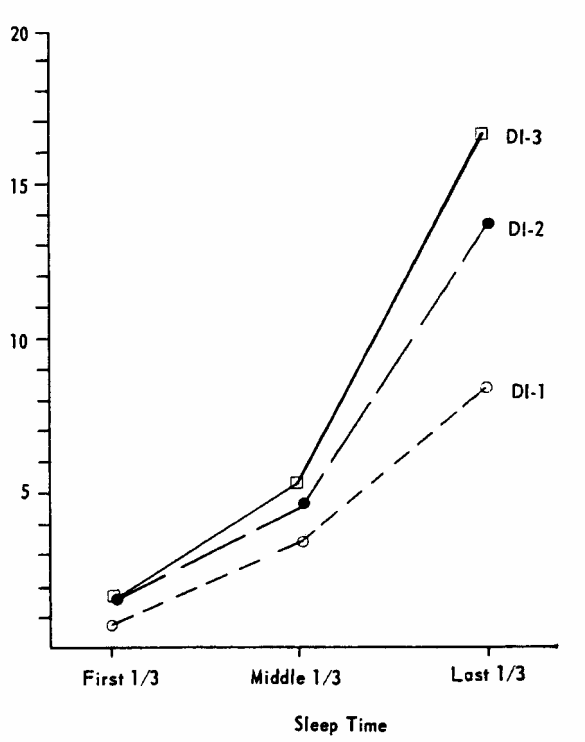
Figure 4. REM period attempts increase the first deprivation night when REM periods are interrupted sooner. Figure from Kales et al. (1964).

Fundamental to everything that has been said is the assumption that the work of sleep is blocked from progressing by REM period deprivation, that a person's thought remains stagnated near the point of his basic slow-wave adaptation. Implicit in this is the prediction that such stagnation should frequently show up in the manifest content of the dreamlets that are permitted during a deprivation night. That is, these reports should have a tendency to be more similar to one another than dreams of the same night usually are.

Fisher (1965) has written of a dream deprivation experiment in which Rechtschaffen elicited mental activity reports with each dream-interruption awakening. He states that "a high degree of continuity of manifest dream content was found to be present." He goes on to say, "When less frequent awakenings are made and REMPs are allowed to follow a more nearly normal course, such a high degree of continuity is rarely observed. In this experiment, the dream reports which showed this high degree of continuity were obtained from awakenings after three minutes of REM. The sequence illustrated not only a

continuity of content, but also the subject's mounting frustration, hostility, and paranoia about not being permitted to finish his task. The fact that such sequences occur for some subjects on some nights, an observation which Dement and I have also made, implies, according to Rechtschaffen, that there is not only a need for REMPs, but a need to complete specific dream experiences as well."

Having possibly explained the number of awakenings on the first deprivation night, I shall consider their distribution. The only information I have concerning this comes from Sampson (1965). Figure 5 is his data; DI-1, -2, and -3 signify the first, second, and third deprivation nights of his dream interruption experiment.



Mean number of awakenings for *DI-1, DI-2,* and *DI-3* in the first, third, middle third, and last third of sleep based on means of six subjects.

Figure 5. REM attempts are initially inhibited despite repeated REM period interruptions.

I would expect an usual inhibition of REM the first part of the first night of REM interruption, because of the need to first complete the basic slow-wave adaptation before the readiness to have sizeable REM periods exists. Subsequently (i.e., after about 3 hr), however, REM attempts should increase as efforts to continue honing the adaptation become frustrated by the inability to activate related memories and integrate them into the adaptation. Figure 5 shows an obvious initial inhibition of REM the first deprivation (DI-1) night followed by an increased number of REM attempts coming after the time when slow-wave work has normally taken adaptive efforts as far as it can without further REM help.

The Second and Subsequent REM Deprivation Nights

In the introductory article in this series ("The Role of Sleep in Psychological Development: Introduction"), it was noted that during a particular waking interval there is a shift in adaptive activity from the frontal areas of the brain to areas presumably containing related memories held in long-term storage (Shadmehr and Holcomb, 1997). It was conjectured that pertinent memories were activated at this time and made ready for integration with the new memories of the day during slow-wave sleep. Prior to the second deprivation night, there would be a full day of memories to become integrated, which would lead to the conjecture that about the same amount of time would be needed for slow-wave integrational efforts before the readiness for a REM period would exist. This of course would be true for each deprivation night. So generally speaking, for each deprivation night, one would expect an initial inhibition in REM attempts until NREM integrational activities reached a dead end. Figure 5 does show this. But shown here also is a tendency for the number of REM attempts to subsequently increase with each deprivation night.

Also introduced in this article was the notion that behavioral integrations formed during sleep participate in subsequent waking behavior, which implies that they persist in some form during subsequent waking intervals. After a night of normal sleep, a highly refined set of adaptations persists. After a night of REM dream interruption, however, an unrefined set of adaptations does. This set of adaptations is unrefined in the sense that the behavioral implications of the previous day's events have not been fully incorporated into the person's memory system.

Because successive days in a life tend to involve many of the same challenges, it seems probable that many of the same memories that were activated prior to DI-1 were reactivated prior to DI-2 and DI-3, which would provide possible links between the unfinished adaptive work of the DI-1 night and that of DI-2 and DI-3. This creates the possibility that elements of a person's DI-1 NREM integrations become incorporated into the DI-2 slow-wave adaptation as prefabricated additions, thus providing a basis for an increased number of REM attempts, particularly after the basic adaptation has been formed. In this manner, DI-3 adaptive efforts could have even more REM-ready prefabricated material to work with, leading to even more REM attempts.

The postulated existence of a growing body of prefabricated REM-ready behavioral integrations implies not merely that there should be a progressively greater number of REM attempts on the DI-2 and DI-3 nights after the initial period of REM inhibition, but that the latency to the first REM period should tend to decrease during the second and third DI nights, as well, because of the participation of these additions in the first REM attempts, too. Sampson (1965) states that for the second and third deprivation nights the mean latency to first REM dropped to 66 min from a baseline value of 80 min and that "latencies of less than 60 min were more than twice as frequent as during the baseline condition."

The Recovery Nights

I have postulated the existence of a body of integrated thought that increases in size with every deprivation night. Contained within this supposition are a number of predictions concerning how recovery sleep should proceed. I will turn to these now.

The first prediction is that there should be more REM time than normal the first recovery night but not more REM periods. If one does have a larger than normal amount of REM-ready integrations in mind, then one should be able to have longer REM periods before being called upon to make NREM adjustments, and these adjustments should generally take a less than a normal amount of time. Therefore, no matter how many REM attempts were made on the last deprivation night, I would expect that number to drop to a near-normal value on the first recovery night and that increased REM time should be mainly the result of longer REM periods.

Table 5 shows the expected increase in REM time percentage the first recovery night and a drop to a normal number of REM periods during both of Kales et al.'s (1964) experiments.

TABLE 5 Kales et al.'s experiment: Means for two subjects after 6 and 10 nights of REM period interruption

	No. of Dream Attempts		% Dream Time	
	6Dep. Nts.	10 Dep. Nts.	6 Dep. Nts.	10 Dep. Nts.
Last dep. night	30.5	39.5	—	—
Baseline ave.	4.0	5.5	21.5	20.0
Recovery night 1	4.5	4.5	33.4	38.3
Recovery night 2	4.0	4.5	30.7	36.3

That this has come about through a lengthening of REM periods and a shortening of NREM preparatory intervals is suggested by Table 6.

TABLE 6 Sampson's experiments: Data corresponding to three nights of recovery for six subjects after three nights of dream interruption

	Baseline	Recovery
Median interval between REM periods	88 min	78 min
Mean REM period duration	20.7 min	24.3 min
REM percentage (mean)	22.1	27.5

I have interpreted the initially small number of REM attempts during the deprivation nights as being due to a need to first become reconciled with the events of the immediately preceding day. Such a need would of course also exist during the recovery nights. Therefore, I would expect the REM periods to be initially short and that this inhibition of REM would continue during the first part of each recovery night, with the bulk of the extra REM time coming during the last hours of sleep. Table 7 is from Sampson (1965), and this is precisely what it seems to show. It should be noted that the need to process the new material of each day also helps to explain why REM deprivation recovery extends over several nights.

TABLE 7 Cumulative minutes of REM time for varying lengths of sleep—all subjects combined

	Sleep Time, Minutes			
Condition	60	120	240	360
Baseline mean	1.2	9.2	34.6	74.4
DI recovery night 1	2.8	13.3	49.0	95.4
2	3.2	16.1	48.2	91.8
3	4.5	21.3	56.9	94.5

I feel I have shown my interpretation of sleep to be capable of explaining the dream interruption data, at least to the extent that this has been reported. I would like now to complete this discussion by turning to the question of dream deprivation as achieved through partial sleep deprivation.

Partial Sleep Deprivation

Sampson (1965) conducted two experiments with the same subjects. In his partial sleep deprivation experiment, the subjects were awakened each night after 2-½ hours of sleep. This was done for three consecutive nights and then followed by three or momre recovery nights of uninterrupted sleep. The permitted dreaming in these 2-½-hour periods was only about 10-15 minutes per night, an amount quite close to that allowed during dream interruption. I will now begin to look at this experiment from the viewpoint I have developed to see if the similarities and differences with the dream interruption results can be understood.

I have made much mention of something I have called a person's initial slow-wave adaptation and have indicated that dream interruption did not prevent this, but did prevent a subject from going much beyond this point. The 2-½ hours of permitted sleep during the PSD experiment is approximately the amount of time needed for this initial integration to be achieved on a normal night. Therefore, on this basis, I would predict that the DI recovery nights and those of the PSD experiment should be similar. (Note that the large amount of stage 2 deprivation in the PSD experiment should, in my view, have no effect on the data, because all of this was preparatory effort that eventually led nowhere on the DI nights.)

That the recovery nights are in fact similar can be seen from Table 8. A close look at Table 8, however, shows that a small but important difference in the data also exists. On PSD Recovery Night 1, there is no REM increase whatever for the first 6 hr of sleep. Thus we are led to seek the cause of this inhibition.

TABLE 8 Cumulative minutes of REM time for varying lengths of sleep—all subjects combined

	Sleep Time, Min			
Condition	60	120	240	360
Baseline	1.2	9.2	34.6	74.4
PSD recovery 1	1.5	10.3	34.4	72.8
2	6.3	15.9	40.5	95.8
3	1.7	12.7	48.0	100.9
4	0	11.9	44.5	89.7
5	0.3	13.6	39.3	87.2
Mean	2.6	12.9	41.1	88.3
DI recovery 1	2.8	13.3	49.0	95.4
2	3.2	16.1	48.2	91.8
3	4.5	21.3	56.9	94.5
Mean	3.5	16.9	51.4	93.9

I have stated that 2-½ hours is approximately the amount of time needed for a person to work out his basic slow-wave adaptation based on the previous day's events. This statement, however, was made in reference to normal circumstances. It is not altogether clear that 2-½ hours would be enough time subsequent to three nights of partial sleep deprivation. The 4-hr longer days during deprivation would have created an additional slow-wave need, leading to a degree of slow-wave deprivation in addition to REM deprivation. If, during PSD, subjects were actually kept from completing their basic slow-wave adaptations, I would expect that the first recovery night would show an extraordinary amount of slow-wave sleep and an inhibition of REM until this slow-wave adaptation covering all deprivation nights was achieved. Therefore, my prediction would be that the REM inhibition shown in Sampson's sleep recovery data should be linked to an increase in slow-wave sleep.

Sampson, of course, focused his attention on REM period effects. To get a fuller appreciation of what took place during recovery, I will quote from a similar experiment (Webb and Agnew, 1965) in which subjects were allowed only 3 hours of sleep a night for 8 consecutive nights and then one night of recovery sleep.

"The first 3 hours of sleep on the recovery night were similar to the 3 hours' sleep of the experimental (deprivation) nights. Compared with the first 3 hours' sleep on the baseline nights, in the first 3 hours of recovery sleep there was an elevation in the amount of stage 4 sleep, a decrease in the percentage of stage 3, and no significant differences in the percentage of the other stages. During the second 3 hours' sleep of the recovery night, when compared with the second 3 hours of baseline sleep, there was a continued significant percentage elevation of the stage 4 sleep and a significant depression of the stage 1-REM sleep.

"The stage 4 and stage 1-REM characteristics during the recovery night were further examined. It was noted that 83 percent of the total stage 4 sleep obtained during the recovery night occurred during the first 6 hours of sleep. In contrast, 69 percent of the

1-REM sleep occurred after these first 6 hours. Indeed, it was found that the amount of stage 1-REM obtained was almost a direct function of the time slept beyond 6 hours."

These findings would seem to explain the anomalous lack of rebound in Sampson's recovery data.

Total Sleep Deprivation

In my discussion of daytime naps, I indicated the view that the length of a person's waking interval should be an important parameter as far as the distribution of EEG stages in the sleep that follows is concerned. The longer the interval, the more there presumably is to assess, implying that more initial time should be spent in stages 3 and 4.

These notions, which were found useful in explaining the effects of shorter than normal waking intervals, should of course apply to longer than normal intervals, as well. A continuous waking interval of several days should impose the need to try to come to grips with all of the events of these days during the first night of recovery sleep. Thus, I would expect to see much more slow-wave sleep during this first night than normally occurs.

Gulevich, Dement, and Johnson (1966) deprived a subject of sleep for 264 continuous hours. Some of their data are to be found in Table 9, and an increase in slow-wave sleep on the first recovery night is shown.

My predictions, however, embrace more than just an increase in slow-wave sleep. Predicted also is an initial inhibition of REM until a person's slow-wave adaptation is worked out. On a normal night, this reconciliation is largely accomplished during the first two NREM intervals, causing only one obviously inhibited REM period. If, therefore, during recovery sleep a slow-wave reconciliation can be seen to extend over many sleep cycles, I would expect an inhibition of REM during all of the intervening REM periods.

TABLE 9 Time spent in stages 3 and 4 during the first recovery night

	No. of Minutes	% of Total Time
Stage 3 baseline	47	11.7
Stage 3 recovery	133	15.1
Stage 4 baseline	21	5.4
Stage 4 recovery	113	12.8

The top two diagrams of Figure 6 represent a continuous plotting of the EEG stages during the first night of recovery sleep. The slow-wave adaptation during this night evidently extended over the first four cycles of the night and may have even included the fifth. During all of this time, the intervening REM periods were unstable. The authors say that during these there were "frequent alternations between low voltage and spindle-plus-'K'-complex patterns." I have already interpreted such instabilities as patchwork attempts to maintain a REM presentation in the face of a sharp critical reaction to an oversimplified integration. Thus, these results are in accord with my previous interpretations.

This first night of recovery extended for 14 hours and 40 minutes. If it is assumed that this time represented a complete treatment of the material considered in the basic slow-

wave adaptation, then the extraordinary amount of slow-wave sleep would suggest that there should also be more REM dreaming than normal during this first recovery night, and this despite the initial lengthy REM inhibition. That such was the case can be seen from Table 10.

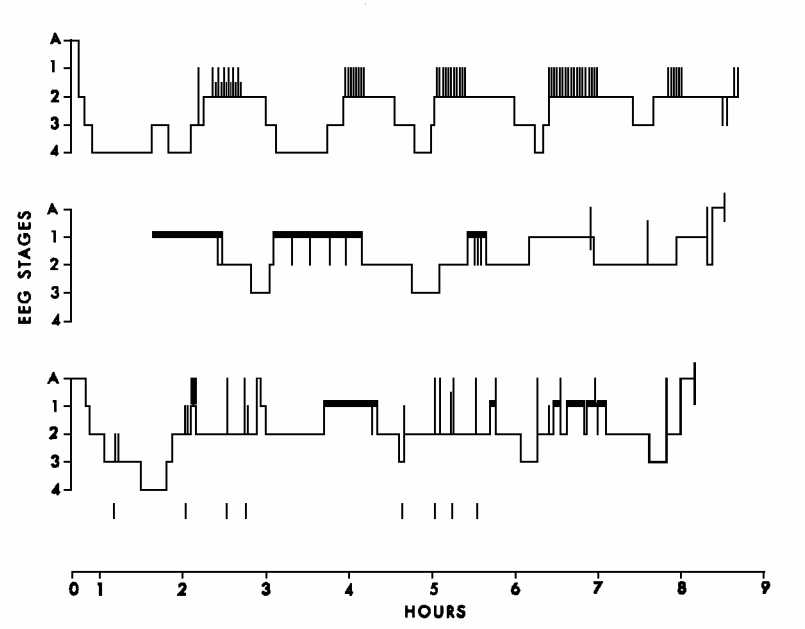


Figure 6. Continuous plots of EEG stages during the first recovery night, represented by the upper two plots (a total of 14 hours, 40 minutes). Bottom plot shows a night of post-recovery sleep occurring one week later (about seven hours in total). Stage 1 represents REM sleep. The thick bars above stage 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. Figure from Gulevich, Dement, and Johnson (1966).

It is inconceivable that a person could bring himself into full contact with all the events of eleven continuous days in merely one night of recovery sleep. A selection process must participate in the slow-wave adaptation of the first recovery night. There would be, it seems to me, a tendency to focus on most recent impressions. These recent happenings could then provide associative linkages to aspects of the past. This, in any case, is one manner in which selection could be accomplished.

TABLE 10 Time spent in REM sleep during the first recovery night

	Number of Minutes	% of Total Time
Baseline REM	68	16.7
Recovery REM	236	26.8

Therefore, during the second and subsequent recovery nights an extraordinary amount of slow-wave sleep should continue to be seen, and, if sleep is allowed to go to completion, there should also be an extraordinary amount of REM. These tendencies are demonstrated in Table 11.

TABLE 11 EEG stage durations during the 2nd and 3rd recovery nights

	Baseline		Rec. Nt. 2		Rec. Nt. 3	
	Minutes	Percent	Minutes	Percent	Minutes	Percent
Stage 4	21	5.4	67	10.7	60	11.0
Stage 3	47	11.7	109	17.4	98	17.9
1-REM	68	16.7	188	30.1	152	27.8

I feel with this and the two previous articles ("The Role of Sleep in Psychological Development: Introduction" and "Normal Adult Human Sleep as a Problem-Solving Process") I have shown my interpretations to be compatible with major aspects of the physiological data pertaining to normal adult human sleep.

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