SLEEP STAGES AND PERFORMANCE

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13. ABSTRACT
The relation of sleep stages to performance is reviewed. Data from 12 subjects who were deprived of sleep for two nights, to study the recuperative effects of slow-wave sleep and REM sleep following sleep deprivation, are presented. There was no significant difference in waking behavior and performance in those subjects denied REM sleep or slow-wave sleep from those subjects allowed all stages of sleep on recovery nights after total sleep loss. The significance of stages of sleep to waking behavior is not yet clear.

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ASPECTS OF HUMAN EFFICIENCY

Diurnal Rhythm and Loss of Sleep

The Proceedings of a conference held at Strasbourg in July, 1970 under the aegis of the NATO Scientific Affairs Division

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Sleep Stages and Performance[1]

Laverne Johnson, Paul Naitoh, Ardie Lubin, Julie Moses

U.S. Navy Medical Neuropsychiatric Research Unit

What type of sleep should one have for effective waking performance? Does each stage of sleep meet a unique need? What are their recuperative values? These remain unanswered questions of sleep research.

The initial observation by Aserinsky and Kleitman (1953) of rapid eye movements (REMs) in infants and the demonstration by Dement and Kleitman (1957) that sleep can be divided into cyclical stages were the major impetus for the current onslaught of sleep research. The association of REM with dream recall (which prompted some to proclaim that the royal road to the unconscious had finally been opened) resulted in the focusing of most attention on REM sleep. The preoccupation with REM sleep has lead Ardie Lubin to comment that many sleep researchers suffer from 'REM tunnel vision'. This diagnosis was prompted by the observation that REM researchers, when given a night of sleep, see only REM sleep. However, Ardie admits to another type of visual problem, namely slow-wave myopia. He, and those who believe that slow-wave sleep (stages 3 and 4) is the most crucial period, have trouble focusing clearly beyond the first third of the night.

Basic for those who believe that there is a need for particular sleep stages are the results obtained from differential sleep stage deprivation. Dement (1960) found that when subjects were prevented from entering REM sleep, there were, with each successive night of deprivation, earlier and more frequent attempts to enter REM sleep. REM rebound, an increase of REM sleep over the baseline amount, normally occurs when REM deprivation stops. Dement and Fisher (1963), Kales, Hoedemaker, Jacobson, and Lichtenstein (1964), Sampson (1965), and Agnew, Webb, and Williams (1967) found a similar build-up in this effort to obtain REM sleep during REM deprivation.

Agnew, Webb, and Williams (1964) found that attempts to enter stage 4 increase with each night of stage 4 deprivation and that, like REM, there is stage 4 rebound when deprivation is ended. For both REM and stage 4 deprivation, the number of arousals necessary to prevent the deprived sleep stage from occuring often triple from the first deprivation night to the fifth deprivation night. These

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results have been interpreted as indicating a need for REM sleep and a need for slow-wave sleep. In our own sleep stage deprivation studies, we have been impressed with each subject's persistent attempts to obtain the denied stage. Like others, we felt we must be frustrating some need. If such needs exist, then do the two types of sleep fill different needs? What will be the consequences to performance if subjects are denied either REM or slow-wave sleep?

Most of the research has been with REM deprived subjects and the dependent variables have been primarily personality or emotionality measures. In his initial study, Dement (1960) reported that five nights of REM deprivation resulted in 'psychological disturbances such as anxiety, irritability, and difficulty in concentrating... but these were not catastrophic' (page 3). Subsequent studies by Snyder (1963), Kales *et al* (1964), Sampson (1965, 1966), Agnew *et al* (1967), Clemes and Dement (1967), and Cartwright, Monroe, and Palmer (1967) have not found similar results. REM deprivation in human subjects does *not* lead to specific psychopathological changes that are common to most subjects.[2]

Only Wilse Webb and his colleagues (Agnew *et al*, 1967) have compared the effects of REM deprivation to slow-wave sleep deprivation. In addition to psychological variables, they included performance measures. No significant REM or stage 4 deprivation effects were found with paced addition, strength of grip, or pursuit rotor tasks. With respect to personality changes, it was the overall impression of Agnew *et al* (1967) that the subjects deprived of stage 4 sleep displayed hypochondriacal and depressive reactions, while the subjects deprived of REM sleep showed signs of confusion, suspicion, withdrawal, and were less integrated and interpersonally effective.

Most of us believe that sleep has a restorative or recuperative function. In our study of the possible functions of sleep stages, we specifically asked the question, what are the relative recuperative values of REM sleep and slow-wave sleep after total sleep deprivation? We knew that we could expect impairment after two nights of total sleep loss (64 hours) and that we could objectively measure this decrement with performance tests. We also knew that if the subject had one full night of recovery sleep, there would be a significant restoration of ability to perform and a change toward predeprivation efficiency. After two nights of recovery sleep, we knew that most subjects would be back to their predeprivation levels. What we did not know was whether similar recuperation would occur if we denied these sleep deprived subjects either REM sleep or slow-wave sleep during the first two nights of recovery sleep.

To answer these questions, performance measures on addition, memory, and vigilance tasks were obtained from sixteen Navy enlisted men, age 17 to 21. After four baseline days, twelve subjects were totally sleep deprived for two

[2] Those interested in consequences of sleep stage deprivation in psychiatric patients are referred to Vogel and Traub (1968) and Vogel, Traub, Ben-Horin, and Meyers (1968).

nights. Total sleep deprivation was followed by either (1) uninterrupted recovery sleep for five nights (the control group, $N = 4$; (2) two nights of REM deprivation and then three nights of uninterrupted recovery sleep (this REM deprived group was allowed all non-REM (NREM) sleep, $N = 4$); or (3) two nights of slow-wave sleep deprivation and then three nights of uninterrupted sleep (this slow-wave deprivation group was allowed all stages of sleep but 3 and 4, $N = 4$). The remaining four subjects were allowed approximately eight hours of uninterrupted sleep every night during the two week period. Both uninterrupted and interrupted recovery sleep were limited to the period between 10 p.m. and 6 a.m. A rebound effect for both REM and slow-wave sleep was present for the appropriate groups on the first night of uninterrupted sleep, indicating that the elective sleep stage deprivation was effective.

Performance on the Wilkinson addition test, auditory vigilance, X crossout test, Williams immediate word recall test, and the mental adding test (plus 7), deteriorated significantly during total sleep loss. Fewer signals were detected on the vigilance task, fewer additions were attempted, fewer words were scanned on the crossout test, and fewer words were recalled in the memory test. Accuracy in all tasks tended to decrease as sleep loss increased.

All subjects improved after the first night of recovery sleep. *There was no significant difference in the amount of recovery for the three kinds of recovery sleep.* These results are illustrated in Figures 1 through 6.

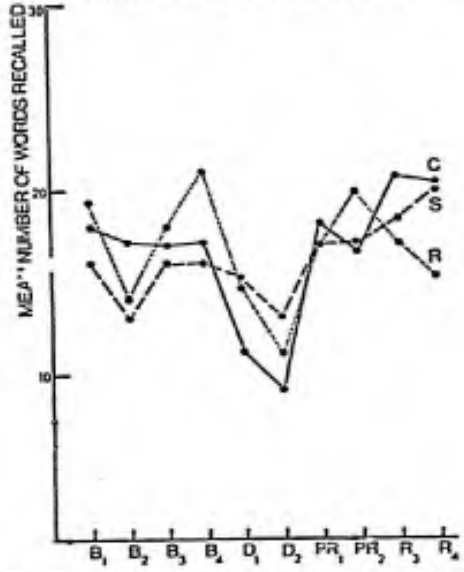

Figure 1. Performance on Williams word memory test during baseline following total sleep deprivation and following partial and total recovery sleep. C was control group, S group was denied slow-wave sleep, R group was denied REM sleep on PR_1 and PR_2 nights. Same time periods and experimental procedures used for Figures 2 through 7.

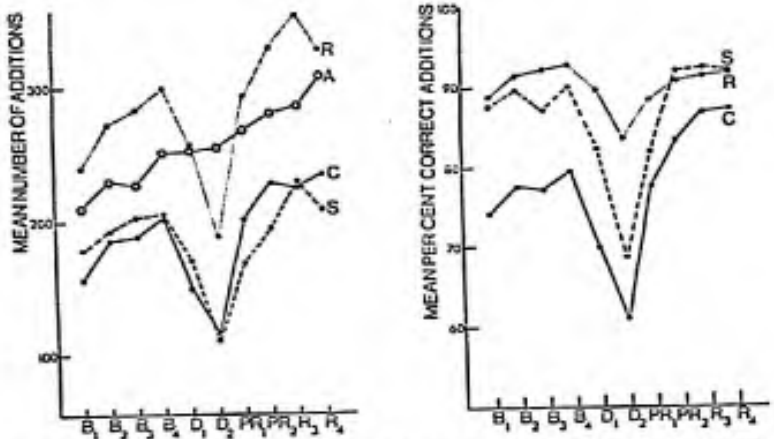

Figure 2. Performance on Wilkinson addition task (60 min). The A group in this and subsequent graphs was a special control group of four subjects who were never deprived of any sleep.

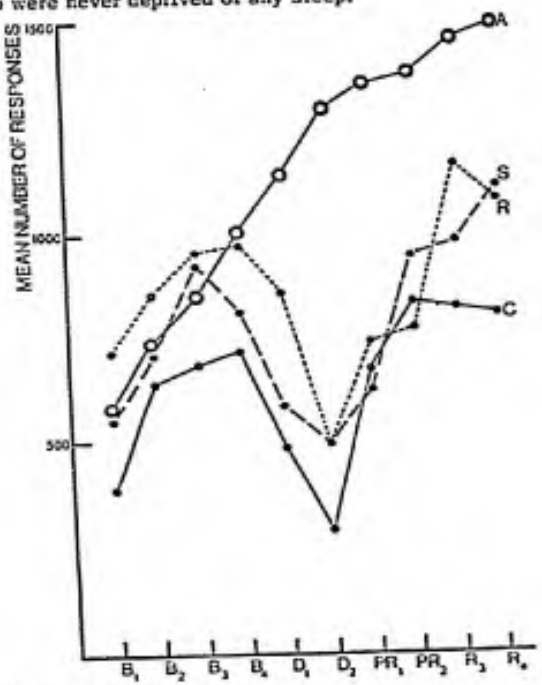

Figure 3. Performance on plus 7 task.

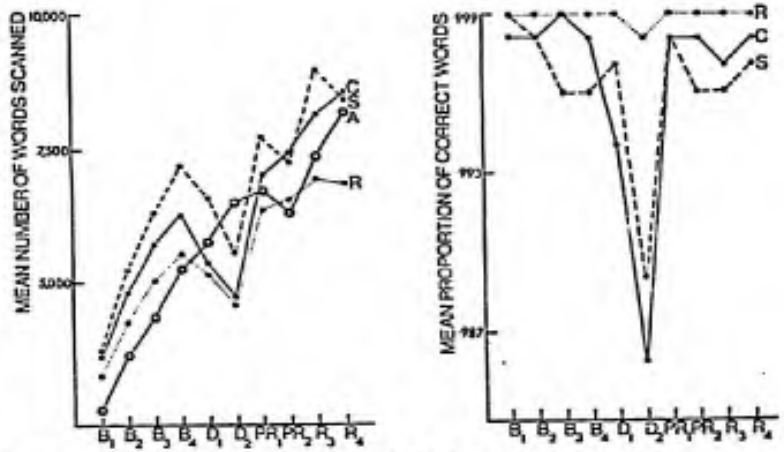

Figure 4. Performance on X crossout (60 min task).

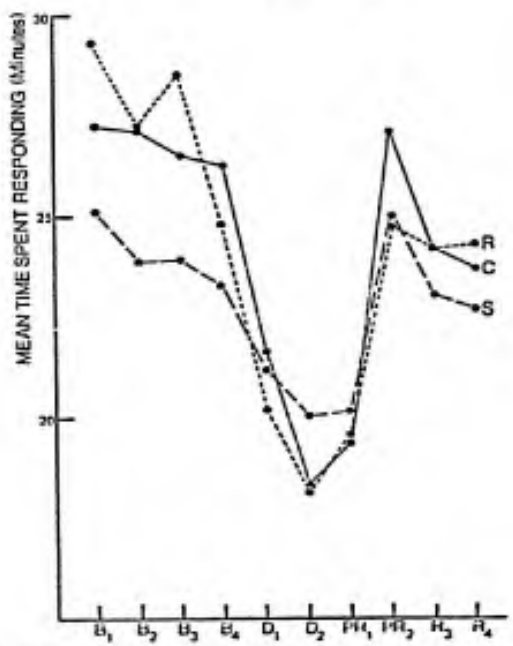

Figure 5. Performance on continuous counting task.

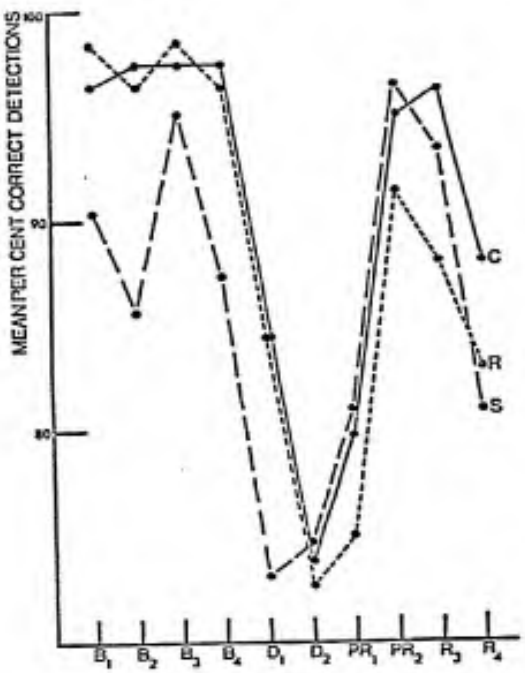

Figure 6. Performance on auditory vigilance.

Similar results were obtained on mood scales (Figure 7). After two nights without sleep, our subjects, not unexpectedly, were more fatigued, depressed, and hostile, and less energetic, happy, and friendly.

As with the performance tasks, the return to the predeprivation psychological states was the same for all subjects regardless of the type of recovery sleep.

The neurological changes found by others (Ross, 1965; Kollar, Namerow, Pasnau, & Naitoh, 1968) consisting of fine hand tremor, horizontal nystagmus, ptosis, and decrease in strength of flexion of the neck, were present in our sleep deprived subjects (Sassin, 1970). All neurological changes were readily reversed by one or two nights of eight hour sleep, whether or not sleep on those nights was totally undisturbed or deficient in either REM or slow-wave stages of sleep.

While the autonomic variables did not show consistent changes during total deprivation, a measure of cortical activity, the surface-negative slow potential (CNV) disappeared with sleep loss. Using a 4.5 second period between the warning stimulus S_1 and the imperative stimulus S_2, the CNV was studied

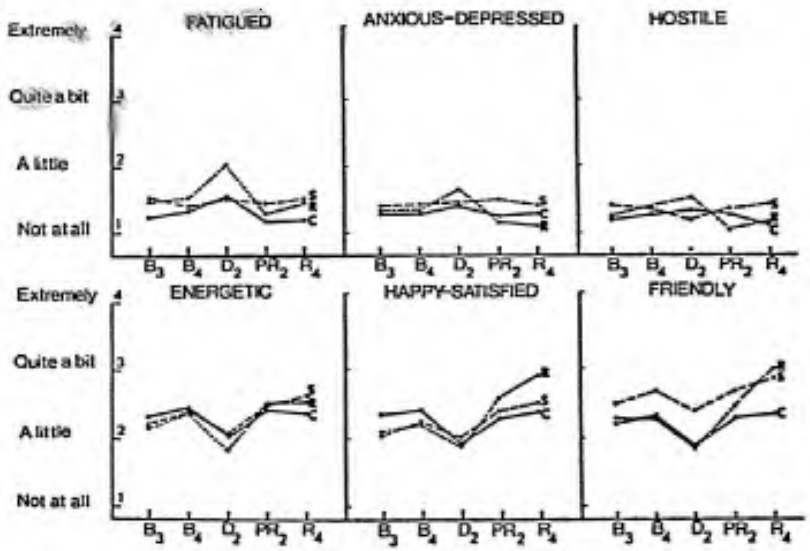

Figure 7. Changes in mood scale.

under baseline, total sleep deprivation, and recovery conditions in eight subjects. The S_1 (warning stimulus) was a click; the S_2 (imperative stimulus) was a set of photo-flashes. During baseline, all subjects developed the CNV between S_1 and S_2. One night of sleep loss decreased the CNV, and two nights of sleep loss abolished the CNV. Figure 8 illustrates the response from a single subject, and Figure 9 the average response for all eight subjects. Only those CNV trials which occurred when the background EEG indicated the subject was awake and when the reaction time was less than a second, were used in this analysis. Our results were not due to sleep but rather to sleep loss. Figure 10 indicates that for the analyzed CNV trials, there was no significant difference in reaction time between the baseline and the second day of sleep deprivation. Thus, a fast reaction is possible even in the absence of the cortical expectancy wave. The type of recovery sleep, again, was not a factor in the return of the CNV.

To date, the search for observable changes in the awake state uniquely related to selective deprivation of specific sleep stages has been generally unrewarding. Perhaps following total sleep loss, sleep of any type is sufficient for

recovery and recuperation is not the unique function of any stage of sleep under this condition.[3]

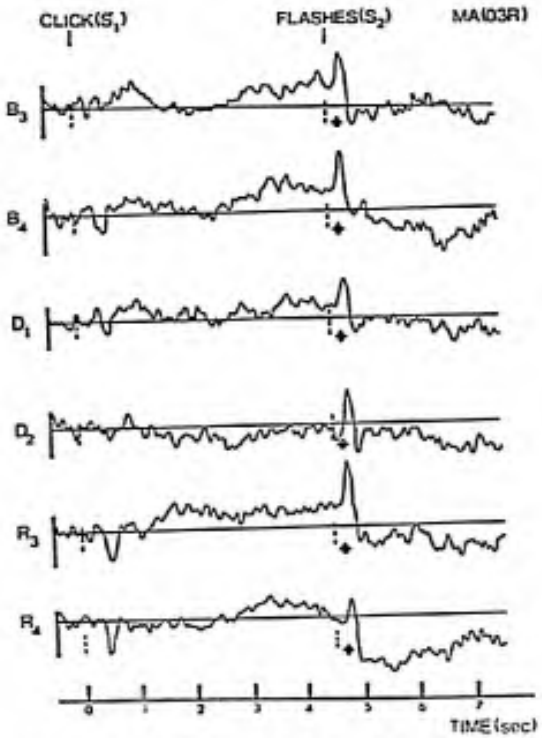

Figure 8. Disappearance of CNV for single subject during total sleep deprivation ($D_1 - D_2$) with return following recovery sleep ($R_3 - R_4$). Average reaction time for each CNV is shown by arrow.

[3] In a study undertaken since the Strasbourg Symposium, we have reversed our design. In the current study, selective stage deprivation precedes total sleep loss. We are now depriving one group of subjects of all slow-wave sleep for three nights and another group of REM sleep for three nights. Both groups are then deprived of all sleep for one night. For the 4 subjects deprived of slow-wave sleep and the 4 deprived of REM, there are still no differences between the two groups following the three nights of stage deprivation. But following the single night of total sleep loss, the subjects deprived of slow-wave sleep show significantly more impairment on the Wilkinson Addition Test and are more fatigued and depressed than the subjects deprived of REM sleep before total sleep loss. If these results prove reliable, it would appear that subjects without slow-wave sleep are less able to withstand the extra stress of total sleep loss.

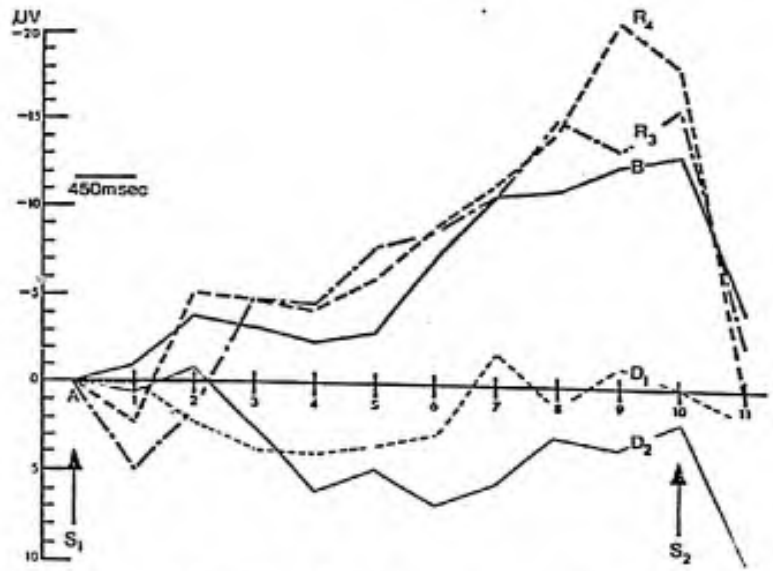

Figure 9. Average of the CNVs of 8 subjects. The CNVs were adjusted so that all had same starting point at the time of S_1. (From Naitoh, Johnson, & Lubin, 1971 (in press))

Work in our laboratory has indicated that approaches other than those concentrating on the time spent in specific sleep stages may be more productive. The recent finding that the maximum release of growth hormone occurs during the first one to two hours of sleep and appears to be correlated with slow-wave sleep is intriguing and points toward a new area of study (Sassin, Parker, Mace, Gotlin, Johnson, & Rossman, 1969). Figure 11 indicates that growth hormone was secreted during slow-wave sleep, that there was no secretion during the same period when sleep was denied, and that growth hormone was secreted when sleep was permitted the next morning. This study clearly indicated the association of growth hormone with sleep and also indicated that growth hormone secretion did not have an independent circadian cycle. The demonstration that growth hormone did not have an independent circadian rhythm was important because of an earlier experience with cortisol (Weitzman, Schaumberg, & Finkbein, 1966). Initial reports that early morning cortisol secretion was linked to REM sleep were shown later to have been in error. Cortisol was found to have an independent rhythm irrespective of the presence of REM sleep or of all sleep for that matter (Rolfwarg, Sachar, Curti, Finkelstein, Ellman, Fishman, & Hellman, 1970).

While our studies have shown a relation between slow-wave sleep and growth hormone, the nature of this relation is still unclear and is being investigated.

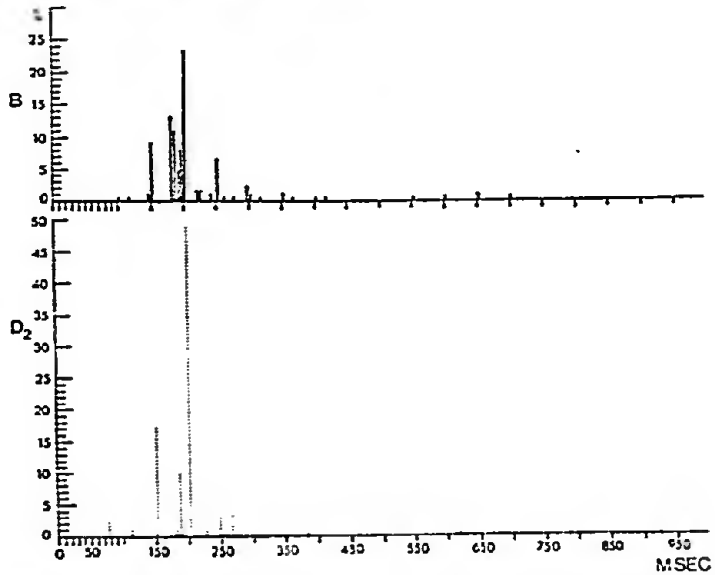

Figure 10. Percentage distribution of reaction time to S_2 (turning off of photoflashes) during baseline (B) and following two nights of total sleep deprivation (D_2).

Looking in another direction, spectral and cross-spectral analyses of the EEG during sleep suggest that perhaps we have been too quick to view stages of sleep as discrete unique states. Those familiar with sleep recordings must be impressed with the awake-like characteristics of both the autonomic and EEG activities during REM sleep. The clear presence of alpha during REM sleep is still, to us at least, an unexpected occurrence. Spectral analysis (Figure 12) further highlights the similarity between the REM and awake states. Pair-wise coherence, a quantity expressing strength of linear relation between activity at various frequencies, indicates yet again the similarity in EEG activity between REM and awake. These coherence data are also presented in Figure 12.

In contrast, NREM sleep apears to be different from both awake and REM. Sleep spindles (sigma activity, 12–14 Hz) are unique to NREM sleep. Figure 13 indicates that stages 2, 3, and 4 appear to be very similar with respect to both spectral profiles and pair-wise coherences. Perhaps sleep should be classified as either spindle sleep or REM sleep. In support of this position is a study by Larson and Walter (1970) in which our sleep data was subjected to quadratic function discriminating analysis. The clear separation of stages 2, 3, and 4 into one grouping with awake, REM, and 1 into a second grouping orthogonal to NREM sleep was found.

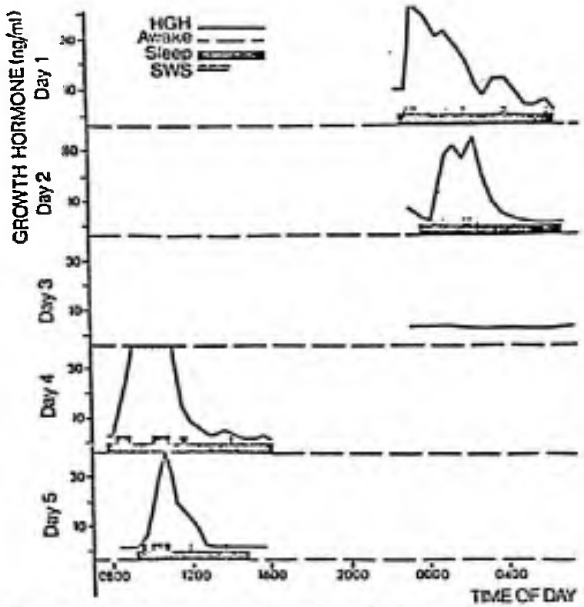

Figure 11. Illustration of reversal of sleep-waking cycle and shift in growth hormone secretion. Secretion of growth hormone is seen with nighttime sleep onset on day 1 and day 2, no secretion during sleep deprivation necessary for reversal to day sleep on day 3, and secretion with morning sleep onset on day 4 and day 5. (From Sassin *et al*, 1969)

These findings suggest that the neurophysiological activities during REM sleep, and perhaps the functions that these activities initiate, are also present during waking. Certainly slow-wave sleep stages 3 and 4 are not uniquely different from stage 2. Thus, when we deprive subjects of stage REM or slow-wave sleep, we may be only partially denying them the functions served by these states. REM 'needs' may be acquired during similar awake neurophysiological activity, and stage 2 may easily provide the necessary activity if slow-wave sleep is denied. The increasing number of reports of well-functioning nonclinical subjects without slow-wave sleep indicates that slow-wave sleep may be desirable, for an as yet unknown reason, but it is not essential. Patients on phenelzine, a monoamine oxidase inhibitor, have been without REM sleep for months and appear to function well (Wyatt, Fram, & Snyder, 1970).

In light of the interest of this symposium in diurnal rhythms, it would perhaps be remiss if the temporal patterns of sleep stages, and the relation of disruption of the sleep cycles to performance, were not mentioned.

Capitalizing on the fact that delta activity is the single best discriminator

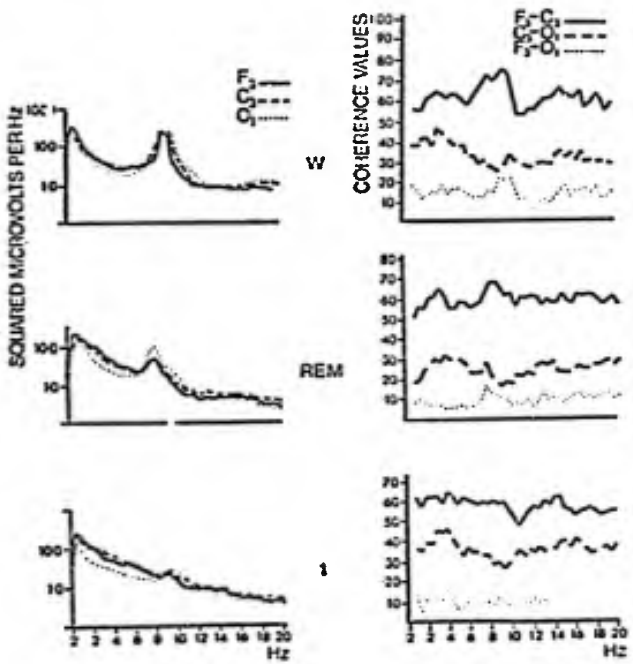

Figure 12. EEG spectra and coherence for awake and sleep stages REM and 1.

between sleep stages (Lubin, Johnson, & Austin, 1969), a sine function, fitted to the delta activity, was used to measure the regularity of sleep (Lubin, Nute, & Martin, 1970).

Figure 14 shows the time course of delta (.2 to 2.0 Hz) intensity for subject 07C on a baseline night. The delta intensities were derived from a spectral analysis of 15-second periods which were then added together for every 2-minute interval.

Sleep stages, scored from the pen write-out, show that W, 1, and REM coincide with the troughs of delta activity, stages 3 and 4 coincide with the peaks, and stage 2 occurs during the ascending and descending limbs of each delta peak.

Subject 07C shows five delta peaks and the amplitude of the peaks decreases as hours of sleep increase. Each of the five peaks has approximately the same shape—a slow buildup of delta intensity followed by a fast drop to stage 2 and then the low delta level of REM sleep. These low flat delta episodes tend to

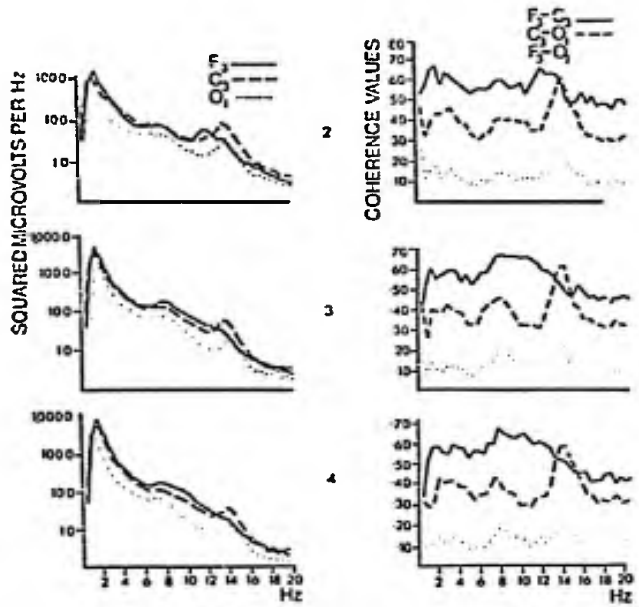

Figure 13. EEG spectra and coherence for sleep stages 2, 3, and 4.

get longer as hours of sleep increase; i.e., the REM episodes increase in duration.

The auto-correlation of delta intensity computed from lag 0 to lag 200 (where each lag equals two minutes) is also shown in Figure 14. Clearly, the data are very regular and not far from sinusoidal in shape.

A sine function plus the corresponding cosine function was fitted to the delta intensities of Figure 14 for periods ranging from 70 minutes to 130 minutes. A maximum squared correlation (squarcor) of .50 was obtained when a period of 87 minutes was used. A simple exponential decay function (c^t where c is a fraction between zero and unity) was used to premultiply the optimal sine function (the one using a period of 87 minutes). Various values of c were used so as to give endpoints that were 1/2, 1/3, 1/4, or 1/5 of the maximum delta peak. The damping ratio of 1/3 gave a maximum squared correlation of .71.

So for subject 07C, we could explain 70 percent of the variance in delta intensity by assuming that the basic sleep cycle had a duration of 87 minutes, and that delta amplitude decreased to 1/3 of the original value during the night. Only four parameters are needed to describe such a time course (period, damping

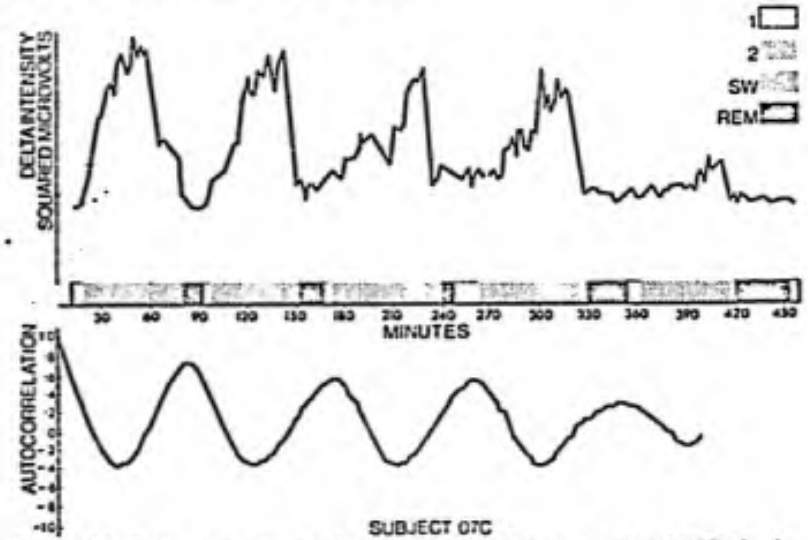

Figure 14. Delta activity as index of sleep rhythmicity—subject 07C. Scale divisions on the ordinate were omitted for delta intensity, as these values were not printed in the computer run for this program. In this analysis, delta intensity values are relative units and not meant for comparison across subjects.

ratio, maximum amplitude, and the time at which the maximum peak occurs). But this subject was one of the best in terms of regularity of sleep. What happens if we take a subject with a major irregularity in his sleep pattern?

Figure 15 shows a plot of the delta intensity for subject 09C on a baseline night. Like subject 07C, the present subject has a drop in delta intensity at about 55 minutes from the start of the record. However, the delta intensity does not fall to the REM level. It falls to that found during stage 2 and quickly returns to a second peak. In other words, the first REM epoch is missed. Also, instead of the delta peak amplitude decreasing regularly with hours of sleep, there is a small peak at about 275 minutes which is followed by a larger peak at 365 minutes.

Figure 15 also shows the auto-correlation function of delta intensity for subject 09C. Clearly the function is very irregular, there is no clear period, and a single sine wave is not an adequate fit. A period of 81 minutes gave a maximum squarcor of .08: A damping ratio of 1/5 gave the maximum squarcor of .14.

For subject 09C, a sine wave, damped or undamped, does not describe the data adequately. From Figure 15, we can see that when the delta peaks occurred, they were of the same approximate shape as for subject 07C—roughly a ramp function. But the missed REM epoch and the reversal in peak size were enough

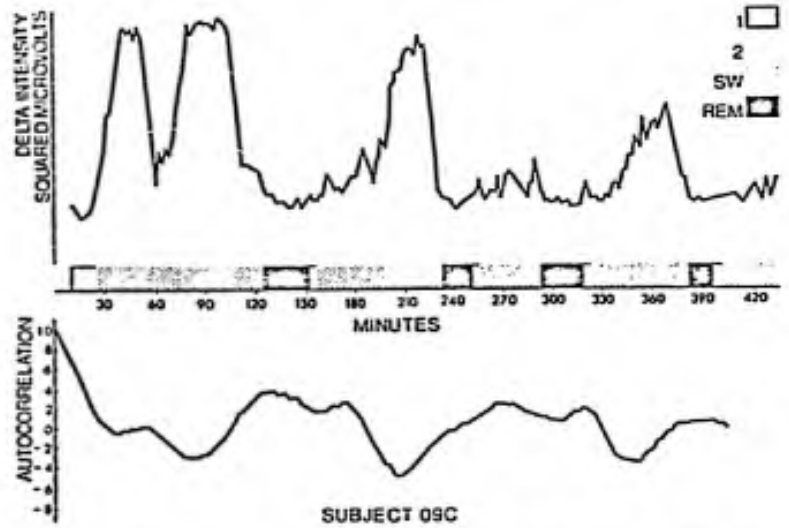

Figure 15. Delta activity as index of sleep rhythmicity—subject 09C. For reasons given in legend of Figure 14, the ordinate for delta intensity has no scale divisions.

to reduce the percentage of explained variance to near zero. Our results to date suggest that REM sleep is the major determinant of sleep rhythmicity. Subjects without slow-wave sleep have regular REM-NREM cycles if REM sleep occurs every 90 – 100 minutes.

In terms of the ten subjects that were studied, subject 07C had the most delta rhythmicity (the damped sinusoid $r^2 = .71$) and subject 09C had almost the least delta rhythmicity (the damped sinusoid $r^2 = .14$).

The average period for ten subjects (using the C3 to A2 channel) was 93 minutes with a standard deviation of 11 minutes. The average damped sinusoid $r^2 = .46$ with a standard deviation of .23. If we weight each optimal period by its damped sinusoid r^2, then the weighted average period was 95 minutes.

An example of the disruption of the sleep cycles is clearly seen in chronic alcoholics withdrawing from alcohol (Figure 16). This patient was experiencing delirium tremens on night 2 and his sleep was composed of only stage 1 and REM (Johnson, Burdick, & Smith, 1970). During the 10-day withdrawal period, the REM-NREM cycle became more evident though slow-wave sleep never appeared. With the stabilizing of the REM-NREM rhythm, there was an improve-

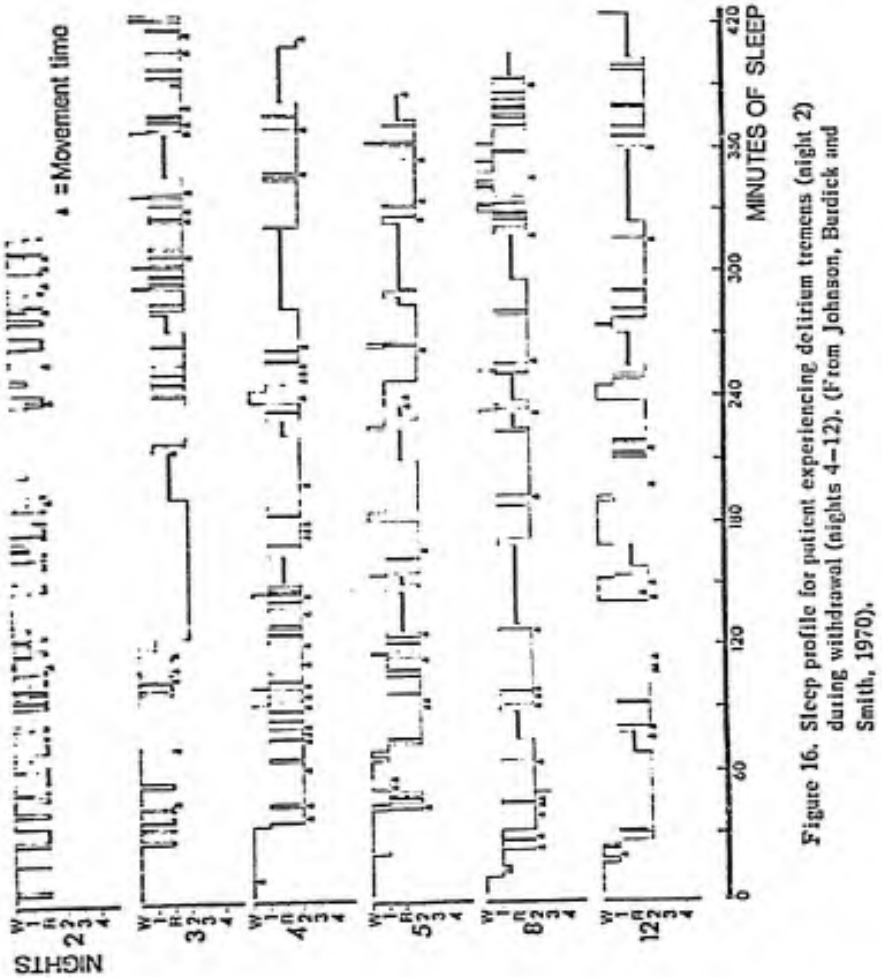

Figure 16. Sleep profile for patient experiencing delirium tremens (night 2) during withdrawal (nights 4–12). (From Johnson, Burdick and Smith, 1970).

ment in performance on cognitive and motor tasks.[4]

In closing, we must candidly admit that the stages of sleep and their relation to performance remain a mystery. The only conclusions that appear warranted from the research to date are (1) the view of sleep stages as unique need states is probably too simple, (2) that research should be directed toward finding correlates other than waking psychological and performance variables (neuroendocrine research is such an area) and (3) that we should not forget that sleep is one part of a sleep-waking cycle and that within sleep there are also clear cycles.[5] The disruption of the sleep-waking cycle and, during sleep, the disruption of the REM-NREM cycle may be more relevant to waking behavior than the total amount of wakefulness or time spent in specific sleep stages.

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[4] A sentinel hypothesis in which REM is seen as providing periodic states of arousal particularly crucial to survival for certain lower animals has been advanced by Snyder (1969). Dewan (1968) has proposed a *P* hypothesis for REM. In this theory, REM serves an information processing role, sifting, evaluating, processing and storing the preceding waking mental activity.

[5] The biochemical mechanisms of sleep are also receiving increased attention, and research in this area has been reviewed by Jouvet (1969).

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DISCUSSION

Schieber: You showed that by means of a discriminant function analysis it was possible to separate the sleep stages into two distinct groups. What are the origin variables you used in this analysis?

Johnson: The variables were those used by Larson and Walter (1970) and were not listed in their article. In our work we have found that delta, sigma (sleep spindles), alpha and theta are most important – beta does not add much. Larson says their determinants will be published in a later paper.

Angibaust: It would be interesting to know whether there was a release of growth hormone at the onset of a sleep induced by drugs during the daytime.

Lewis: In Edinburgh we have been looking at growth hormone in much the same way as Dr. Johnson, and have confirmed all his results and would agree that HGH output is not a circadian rhythm. We have also studied a few shift workers and find that in the period of sleep following the first night of night-shift there is again a peak HGH output associated with slow wave sleep at sleep onset. This then is further evidence that HGH output is related to slow-wave sleep and not to some circadian rhythm.

To pass to another part of Dr. Johnson's presentation, I notice that on recovery day 2 the CNV is no longer negative, but has become positive. Gray Walter has

shown that schizophrenics have positive expectancy waves. Do you think, Dr. Johnson, that in view of this we should go back to Dement's hypothesis about sleep loss and behavioural change?

Johnson: I was pleased to hear of your growth hormone data, but do not believe the positive CNV shift on day 2 is related to schizophrenia. It is an example of finding similar physiological changes in more than one condition or state—another example is the presence of alpha during REM sleep; in this case no one would say that the subject was awake. There is no necessary causal relation between physiological change and condition or state of the subject. Our subjects showed no signs or symptoms of schizophrenia.