

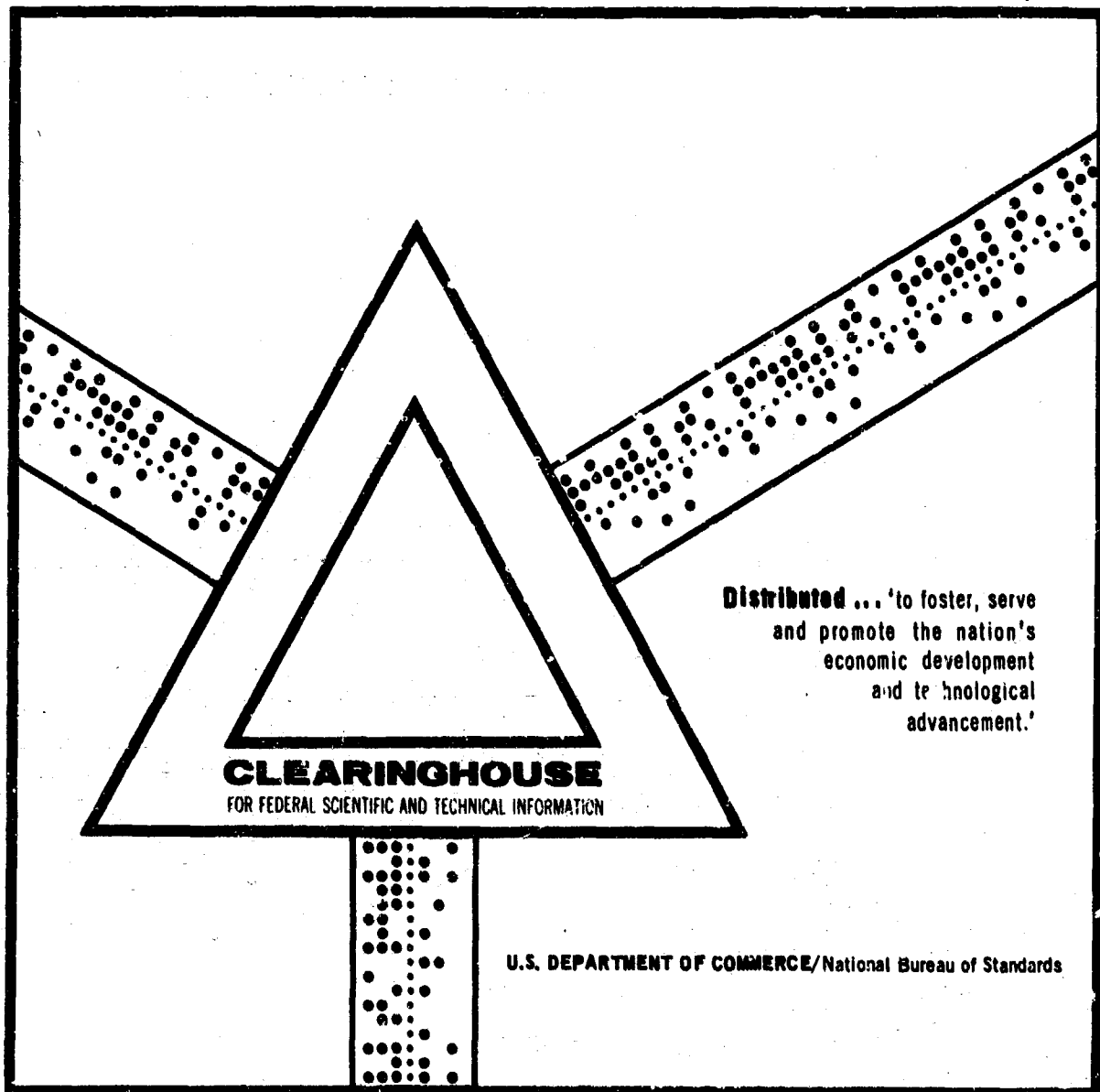
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SLEEP LOSS AND ITS EFFECTS ON PERFORMANCE

Paul Naitoh

**Navy Medical Neuropsychiatric Research Unit
San Diego, California**

August 1969



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Paul Naitoh, Ph.D.

Navy Medical Neuropsychiatric Research Unit
San Diego, California 92152

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SLEEP LOSS AND ITS EFFECTS ON PERFORMANCE

Paul Naitoh, Ph.D.

Abstract

The effects of sleep loss on human task performance were discussed under total, partial, and selective deprivations of sleep. Some of the frequently used psychological tasks in studies of total sleep loss were described in sufficient detail so that experimenters could choose, on the basis of materials presented in this monograph, adequate tasks to fit their experimental objectives. Factors which played critical roles in determining the degree of task sensitivity to total sleep loss were listed. Effects of shortened hours of sleep on human task performance were discussed. Effects of selected sleep deprivation on performance were also briefly commented upon. The commentary of this monograph covered almost all studies conducted on sleep loss under laboratory conditions, including a series of on-going experiments on total and selective sleep deprivations at the Navy Medical Neuropsychiatric Research Unit, San Diego, California 92152. The commentary was followed by a bibliography on sleep deprivation with author and subject indices.

FOREWORD

The first version of this monograph was prepared as a paper presented at the Ninth Navy Human Factors Institute, the U.S. Navy Postgraduate School, Monterey, California, in 1967. Since then, the first version has been expanded on the basis of three separately prepared papers: "Electroencephalographic changes after prolonged sleep loss," a paper read at the Western EEG Society meeting in 1968; "Prolonged sleep deprivation and performance," presented at the Annual Conference of the American Psychological Association in 1968; and "Role of sleep research in human factors," a paper given at the Annual Conference on Engineering in Biology and Medicine in 1968.

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I. INTRODUCTION

Upon occasions, man will be forced to maintain his vigilance and skill for 48 hours or more with a total absence, or with reduced amounts, of sleep. The reasons for remaining sleepless by choice are many and varied. Family, or military, emergencies and heavy work-loads necessarily involved in the early phases of many field missions, such as space flight and underwater living, are but a few of the many reasons. Despite sleep deprivations of varying severity, a man would be expected to perform his task efficiently and flawlessly (1, 213, 258, 293).

Surely, a man could stay awake and function for a certain period of time by living on "his adrenalin." However, what would be the cost of such sleep loss on work efficiency and human reliability? What would be the general behavioral consequence of prolonged wakefulness?

So far, only seven men have remained awake for more than 200 hours, and their experiences have been well-documented. These seven men are: Peter Tripp (200 hours sleep loss, studied by West and Williams and their associates), Randy Gardner (268 hours sleep loss, examined by Dement and Johnson), and a group of four subjects: Dave Groplear, Harvey Hall, Rob Smith and Jay Lampert (205 hours sleep loss, observed by Kollar and his colleagues). The psychological observations of these subjects showed a common occurrence of visual and, to much less extent, auditory illusions. Visual misperceptions, such as a moving door frame and swirling steam from the floor, were experienced by some of these seven men and by many other sleep-deprived subjects at one time or another (100, 107, 196, 198, 18). In reporting these visual and auditory illusions, sleep-deprived subjects expressed some doubt generally as to the reality of their perceptual experiences, and they would report seeing an unusual shimmering object in the air with calm amusement. In other instances, however, the sleep-deprived subjects experienced visual and auditory perceptions of an illusory nature which succeeded in convincing the sleepy subjects, at least for a while, of being real and of being threatening as well. For example, in this author's experience, a subject suddenly stopped performing his tracking task in the middle of a task session. Upon immediate questioning, the subject revealed that he had heard distinctly a voice instructing him to "stop performing and rest," although no such instruction had been given. An episode of threatening visual hallucination was described by Penau and his associates (242). One of the four subjects, who had been sleepless for 168 hours, arrived at the psychophysiological test room as scheduled. After a minute or so of tracking a spot of light shown on a cathode ray oscilloscope, the subject watched as the oscilloscope transformed itself gradually into Humpty-Dumpty, and, simultaneously, a gorilla appeared in a corner of the room. The gorilla moved down and merged with Humpty-Dumpty to form a blended picture of both. The form then started to move threateningly toward the subject. The threat which this visual hallucination caused was seen behaviorally in the subject's panicked flight and in his physiological reaction. During the experience of visual hallucination, his heart rate jumped from its usual 90 to 100 beats per minute to 160 beats per minute, and his brain waves showed unusually high (for a man who had stayed awake 168 hours) sustained amplitude alpha activity (Figure 1). The threat caused by "Humpty-Dumpty Gorilla" was, however, transient. In the session following this episode, the man again aborted the session by bolting from the experimental room but once outside he calmed down rapidly. The third run was without episodic fright.

Despite dramatic delusional and confusional states of some of the subjects whose sleep was deprived, the view of Luby and Gottlieb that prolonged sleep deprivation results ultimately in a psychotic state should be regarded as a hypothesis because of the small proportion of sleep-deprived subjects who have developed a full-blown systematized paranoid delusion. Those sleep-deprived subjects who developed florid psychotic behaviors appeared to have had atypical personality and personal histories (146, 195, 294, and 128). Therefore, with Johnson (128), the author has reached the conclusion that no new psychopathology would occur a nouveau after prolonged total sleep deprivation.

In his daily routine, a man obviously would not be required to stay sleepless for 200 hours. He might, for one reason or another, be forced to stay awake for 48 hours or less, but the risk of developing visual and auditory hallucinations and paranoid psychotic behavior severe enough to interrupt the performance schedule under such circumstances would be negligible. It would be equally unlikely in field missions that any crew member would be required to remain fully awake on duty for more than 48 hours. Thus, the problem of induced "sleep loss psychosis" remains a matter of academic interest only.

Nonetheless, there is experimental evidence that short sleep loss can cause visual and auditory misperceptions, disorientation in time, deficiency in short-term memory, and can cause some minor changes in personality. While measured effects of these perceptual, behavioral, and social effects of sleep loss have been small, they could well become of grave consequence in any long-term field operation, such as living in a capsule in the vacuum of space, in a habitat under the hyperbaria of the ocean, or in a tunnel habitat under the ice of Antarctica. Under such living conditions, a crew member has to sustain skilled performance and remain alert. Any life-support system must be checked regularly with much care given to details.

In many modern occupations, such as airline piloting, ocean fishing, long-distance trucking, and most particularly in medical practice, the effects of small losses of sleep have a serious impact on carrying out professional routines. A man whose work results in an irregular time for meals and for sleep often accumulates a sleep debt (113).

Even hospitalized patients have not been completely freed from the undesirable effects of ubiquitous sleep disturbances and subsequent sleep loss. For example, Kornfield and his associates (163) have reported that the frequent awakening of open-heart surgical patients during a period of intensive post-operative care for the variety of medical checks necessary resulted in interrupted sleep and loss of sleep. Because of this sleep loss, some patients have shown behavior reported to be delirious.

In field operations as well as in long-term duty, the planning of sleep "logistics" must be as carefully considered as the planning for food, clothing, etc., (333). Even the best work environments and the best controlled equipment design would be ineffective in sustaining human skills over an extended period of time without an adequate plan for the logistics of sleep, i.e., providing "human engineered" sleeping quarters and a work-rest schedule which assures the crew sufficient periods for efficient sleep. The invisible, ever accumulating insidious sleep debt can make human subjects suddenly falter either in the most obvious manner, by debilitating sensory illusions, alarming hallucinations, transient paranoid ideations, or in the more subtle manner by decreasing the ability to perform as shown by an increase in errors of omission or by an inability to take or give commands (200, p. 87).

Unfortunately, the importance of sleep logistics has not been fully recognized in planning extended missions or in professional activities, although Trumbull (198) and Adams and Chiles (1) have stressed the importance of

the work-rest schedule. Recently, Alluisi and his colleagues (7) summarized their findings on the unusual work-rest cycle on human performance which indicated clearly the critical role played by sleep. Alluisi *et al* found that the work-rest cycles, other than the one to which man is normally accustomed (namely, 16 hours off duty/8 hours on), disrupted human efficiency and lowered reliability. After 96 hours, they found that keeping crew members on a schedule of 6 hours on duty and 2 hours off (6/2) resulted in severe performance degradation. Why was there such performance deterioration? Part of the reason was lack of sleep. With the 6/2 schedule, crew members averaged less than 4 hours of sleep per 24-hour period. This sleep was far too short to be sustained over a long period. With a work-rest schedule of 4/2, crew members slept 5.5 hours or more per 24-hour period. This extension in hours of sleep permitted some of the highly motivated crew members to maintain their level of performance for a period of at least two weeks. However, the strain of working under even this demanding schedule was clearly shown by a marked deterioration in performance when the crew underwent total sleep loss for three nights (114).

The inability of the Gemini IV crew to adapt to the theoretically and experimentally considered best work-rest schedule, 4/4, further illustrates the general failure of past attempts to modify man's basic 8/16 work-rest rhythm (237, Pp. 172-173; 152, Pp. 175-177; 114).

This monograph was prepared with an attempt to provide sufficiently organized information on sleep loss and human performance to point up empirical facts which appear necessary and to emphasize where theoretical reformulation is mandatory. This monograph started also with the tacit agreement that sleep loss has been a serious issue. There is no elaborate effort made herein to convince readers that sleep deprivation constitutes operational problems. For that conviction, readers are referred to Unit Report No. 68-22, Navy Medical Neuropsychiatric Research Unit, San Diego, California 92152.

II. TOTAL SLEEP LOSS

The various procedures for keeping subjects awake are generally not fully described. A customary procedure, however, is to keep a 24-hour continuous surveillance over the subjects by a team of experimenters. The lack of privacy under this condition is unavoidable. While brief eye closures were usually tolerated, the subjects are quickly awakened when eye closures are associated with head-bobbing or chin resting upon chest. Standing or walking subjects are assumed to be awake, even when their eyes are almost completely closed.

Finding effective techniques to keep subjects awake often test experimenters' ingenuity. For total sleep loss of less than 48 hours, reading, writing, watching TV, listening to the record player, playing cards, working on hobby-crafts, ping-pong playing, and other recreational activities are generally effective in keeping subjects awake. For much longer periods of sleep deprivation, a long walk and an intermittent exposure to cold are found to be relatively effective. However, with loss of sleep longer than 100 hours, only continuous proddings by the experimenters could keep the subjects "awake."

Effects of Total Sleep Loss on Performance

The first study on acute total sleep loss was conducted by Patrick and Gilbert (243) of the University of Iowa in 1896. Physiological and psychological observations, such as reaction time, pain threshold, visual acuity, and memory for nonsense syllables were made on three subjects who stayed awake for 90 hours. The observed degradations in test performance were not reliable. Since this study, many papers have been published on the effects of total sleep loss on human performance but most of the early studies failed to discover predictable and consistently detrimental effects of loss of total sleep on performance. Indeed, the only reliable change, as a result of total sleep loss, was subjective in attitude (111). Such lack of total sleep loss effects on performance was surprising, considering how sleepy the subjects were outside the testing room.

Robinson and Herman (261) kept three subjects awake for 65 hours and tested them on hand grasping, tapping, aiming, reading letters, and mental multiplication. These subjects were tested once a day for 11 to 26 days before sleep deprivation, and for 4 to 5 days after deprivation. The experimenters concluded that insomnia did not affect test results in any marked or consistent manner. Lack of performance decrement was ascribed to an increased amount of effort by the subjects. Lalett (173 and 174) conducted total sleep loss experiments of up to 72 hours to determine the effects on tests of repeating 12 digits backward, code, and analogy. Results of these tests were inconclusive due to the compensatory effort made by the subjects. Robinson and Herman (261) indicated, however, that complex and longer tasks were affected more by sleep loss. Weiskotten (312) stayed awake for 62 hours, during which time he tested himself in learning and re-learning nonsense syllables. His conclusion was that moderate sleep deprivation had little effect on accuracy and the chief deterioration was found in the power of concentration.

Before sleep deprivation studies were able to produce consistent and replicable sets of results, major refinements in the methods of handling performance data were necessary. In the new point of view, the absence of responses, not the excited responses, became a major target of research. In contrast to the earlier hypothesis

which emphasized accuracy of performance, this new approach stressed that the ever-increasing number of absences or pauses in a subject's responses was the major behavioral symptom of sleep deprivation. Each brief episode of "no response" constituted performance decrement due to sleep loss. The absence of an appropriate response, or "lapse," was predicted to increase in frequency and in duration with increasing amounts of sleep loss. This approach recognized that, even after extended sleep deprivation, there remained always a period of time during which the sleep deprived subjects would perform accurately, regardless of how brief and intermittent this lucid period might become with accumulating hours of sleep deprivation. Any performance measures, e.g., accuracy, which could not pin-point these brief periods of lapse, would be unable to uncover the effects of sleep deprivation on performance.

Investigators at Walter Reed Army Institute of Research were instrumental in pointing out the importance of this lack of response (229). Hence, this new approach was labelled "Walter Reed Lapse Hypothesis." The use of "lapse" as a measure of performance degradation was not, however, originated by this group. Patrick and Gilbert (243) described the failure of one of their subjects to memorize the digits because of his inability to focus his attention. They called such failure a "kind of mental lapse." Bills (23) noted that blocks occurred in mental task performances. Bills also found that the frequency and duration of these blocks increased with fatigue and that errors tended to occur at the time of these blocks. He concluded, after detailed studies of these blocks or lapses, that they were involuntary rest periods which delayed the start of fatigue. Warren and Clark (306) applied this concept of blocking to a sleep loss study to determine if mental work could be blocked and, if so, would the block be similar to that caused by fatigue. After 65 hours of total sleep loss, all four subjects in this test showed an increased number of blocks on tasks of alternate addition and subtraction and on color-naming, but they did not show an increased blocking in their performances of the finger tapping task. The errors in mental arithmetic and color-naming had no apparent relationship with hours of sleep deprivation. For both the sleep-deprived and control subjects, however, errors tended to occur at the time of blocking.

Bjerner (24) studied blocks, or the periods of lowered reactive capacity which serve as involuntary rest with recuperative effects from fatigue, in five subjects who stayed awake for two nights. Simultaneously with each subject's performance on a self-paced serial reaction test, he obtained electroencephalograms (EEG's) and electrocardiograms (EKG's). He observed that those responses which had more than twice the average reaction time were accompanied by a transient fall in pulse rate and a decrease in EEG alpha activity. For those responses with a reaction time of 5 seconds or longer, the period was characterized by brief sleep -- EEG alpha disappeared and was replaced by delta, sleep spindles, and K-complex. Carmichael and his associates (111) noted in their summary of the World War II study at Tufts College on fatigue and sleep deprivation that the basic deterioration of performance was not due to a loss of a particular skill but, rather, due to periodic blocking.

In 1956, the Walter Reed group undertook a series of studies on sleep loss and performance designed to evaluate "tests for their sensitivity to sleep loss, and to construct tests that would be resistant to sleep loss" (339). Blending their results from fatigue and total sleep loss led to the formulation of four predictions which have become the core of the Walter Reed Lapse Hypothesis. They are:

- (1) Sleep deprived subjects show brief intermittent lapses. These lapses increase in frequency and duration as hours of sleep loss increase.
- (2) Certain factors in a test situation tend to alert the subject, thus preventing or shortening these

lapses. Some examples of the alerting factors would be (a) the massive sensory stimulation caused by physical exercise, electric shock, loud noise, adrenergic drugs, etc., (b) uncertainty, (c) feedback of information upon the quality of performance, and (d) task change.

(3) Automatic response sequences are relatively resistant to sleep loss.

(4) Many, but not all, tests will be affected by diurnal rhythm.

The lapse hypothesis has been effective in focusing attention on the aspects of task performances influenced most by total sleep loss and in explaining some of the performance decrements due to sleep loss. It should be noted, however, that Williams himself (315) recently raised a question dealing with the merit of holding onto and continuing to use the lapse hypothesis. Williams *et al* (335) maintain that hypotheses, other than the lapse hypothesis, would be required to explain the effects of sleep loss on the recall test and on the uncertainty of the task. The lapse hypothesis cannot predict sluggishness in the information processing as revealed by the fact that tasks with a high degree of uncertainty of information were more affected by total sleep loss. The lapse hypothesis also cannot specifically predict the impairment of temporary memory storage due to total sleep loss.

The lapse, in context of total sleep loss research, best summarizes a variety of situations in which there are absences of the motor responses needed to perform the task or there are intermittencies in motor performance. In other words, the concept of lapse would offer us a shorthand language for describing errors of omission, absence of adequate motor responses, and for describing responses with long reaction time.

Specific Tasks Sensitive to Total Sleep Loss

Addition Tasks

An adding task can be tailored to detect total sleep loss by either increasing its complexity, its duration, or both. Williams *et al* (341) found that a completely self-paced 3-minute paper-and-pencil task, consisting of adding pairs of one-digit numbers, resulted in a significant decrease in the number of additions attempted after 28 hours of sleep loss. But the accuracy of addition was not affected to the same degree even after a total sleep loss of 68 hours. Wilkinson (318) asked subjects to add a list of five two-digit numbers for 25 minutes and later he extended the period of this task to one full hour (328). When the performance measure was the number of additions attempted, sleep loss of only 5 hours influenced the performance detrimentally, i.e., the number of summations attempted decreased. An experimenter-paced addition task was reported by Williams and Lubin (340). In this task a pair of digits, which subjects were to add, were tape-recorded and presented at one pair per 2 seconds, one pair per $1\frac{1}{2}$ seconds, one pair per $1\frac{1}{4}$ seconds, and, finally, one pair per 1 second. With an increase in the number of additions to be completed per unit of time, the effect of two nights of sleep loss was reliably demonstrated as a decrease in the number of additions completed. While this "speed loading" of additions made the task sensitive to sleep loss, the same authors observed that "cognitive" loading would also enhance the sensitivity of the task to total sleep loss. In what the authors called a two-step test of addition, subjects were instructed to add a constant of 8 to each sum just obtained without any paper and pencil. A pair of digits was presented every 2 seconds. Under this cognitive loading -- because adding 8 was executed entirely by thinking without external promptings -- the adding task was able to reveal a sleep loss of one night.

Vigilance Tasks

One of the vigilance tasks, which can be used either in visual or auditory mode, is the Continuous Performance

Task (CPT) devised by Rosvold and his group (267) for brain damage study. In the visual mode of the CPT, the subject saw the letters in a random sequence one at a time on a viewing screen. The task was to press the response key whenever the letter X appeared on the screen. In the auditory mode of the CPT, the subject responded to a tape-recorded voice reading a list of letters, including X. Another version of the auditory CPT required the subject to press a response key if, and only if, the letter X was preceded by the letter A. Approximately 10 minutes on either the auditory or visual CPT was sufficient to detect sleep loss of roughly 50 hours by measuring errors of omission, i.e., the failure of the subject to report each occurrence of the letter X or X preceded by A. The auditory and visual CPT were task-paced or experimenter-paced, whereas a paper-and-pencil version of the CPT task was a subject-paced, X-crossout task in which the subject was instructed to cross out the letter X wherever it appeared in a nonsense word with as much speed and accuracy as the subject could command.

Another form of the visual vigilance task is the Pentagon Test which consists of a display panel of five different colored lights in a pentagonal configuration. The center of the pentagon is 5 feet above the floor and 8 feet from the seated subject. The subject was trained to press a response key each time the red light flashed on and to refrain from pressing the key when all other colored lights came on. The rate of light presentation was 1 per second. Thirty light presentations were grouped to make one test set. Eight lights out of one set of 30 lights were red signal lights. Three different kinds of constraints were placed on the manner of red signal light presentation. In the Redundant Pentagon Test (R task), the red light was presented in succession, as red, red, red, for eight times. In the Standard Pentagon Test (S task), the red signal light appeared with as much randomness as the letter X in the CPT of Rosvold. The red light appeared in a fixed sequence with other colored lights mixed in a random fashion. After practice, the subject could learn such sequence with some accuracy. In the Uncertain Test (U task), the red light appeared with as much randomness as possible. Thus, the S task contained more information per stimulus than the R task. The U task contained the most information per stimulus, forcing the subject to be more vigilant and take more time in his processing before his decision to press, or not to press, the response key. The uncertainty factor introduced in the S and U tasks was observed to enhance its sensitivity to the effects of loss of total sleep by measuring the percentage of omission errors made by a subject after two nights of sleep loss (341 and 338).

The auditory vigilance task of Wilkinson (331) involved a detection of an infrequent and irregular burst of sound, given at the rate of 40 signals per hour. The subject heard a $\frac{1}{2}$ -second, non-signal tone, repeated every 2 seconds. Occasionally, one of these tones was slightly shorter in duration than the rest and the subject was to detect that tone. Both signal and non-signal background tones were buried in an intense white noise to render the task more difficult. With normal and non-sleep-deprived subjects, practice resulted in roughly 70 per cent successful detection. With this seemingly simple method, Wilkinson was able to measure the effects of roughly three hours of sleep loss occurring during a single night.

Reaction Time Tasks

A five-choice test of serial reaction was used by Wilkinson in many of his studies (328). One instrumented version of the five-choice serial reaction test -- a modification of the method of Leonard (181) -- consisted of a stimulus panel and response panel. On the stimulus panel were five lights arranged in a pentagon. At one time, one of the five light discs would be lighted. The subject responded by tapping a corresponding

disc on the response panel with a special stylus. As soon as the subject responded, the stimulus panel light was extinguished, but another disc on the panel came on, indicating the beginning of the next trial. No restriction was imposed on speed of response. In this self-paced task, a measure of the gap -- the period of no response -- was found to be very sensitive to total sleep loss. If the duration of the task was longer than 30 minutes, the task was able to detect 30 hours of sleep loss.

Bjerner (24) also used a serial reaction task to show sensitivity to total sleep loss.

Memory Tasks

By using an immediate recall test, Williams, Gieseking, and Lubin (335) and Williams and Williams (343) demonstrated what was interpreted as a deficiency in memory-trace formation after 31 hours of total sleep loss. The Word Memory Test consisted of tape-recorded lists of words which were selected from the AA category in the Thorndike-Lorge Word List. In this test, each word was pronounced, spelled, and then was followed by a 10-second interval of silence during which time the subject wrote the word down. After the entire list of 25 words had been presented, the subject corrected any spelling errors or omission errors with the assistance of the experimenter. Immediately after making his corrections, the subject wrote down all the words he could recall in any sequence on a blank piece of paper. Under these conditions of minimal rehearsal and immediate recall, Word Memory Test measured a short-term memory.

Although memory impairments after total loss of sleep were reported often (243, 313, and 82), no detailed study of the effects of sleep loss on memory processes, i.e., sensory registration, formation of the memory-trace, its storage, and the retrieval of the trace, was made until the work of Williams, Gieseking, and Lubin (335).

In contrast to short-term memory impairment, Williams and his group (335) found no definite impairment of long-term memory after 34 hours of total sleep loss. A test for evaluating long-term memory impairment involved recognition of 25 previously shown identification photographs out of 75 such pictures obtained from a 1944 Army Yearbook.

Tracking Tasks

Pursuit motor tracking tasks were used by Pepler (247) while Malmo and Surwillo (203) used the auditory compensatory intensity form of tracking task. In the latter study, the subject was trained to turn a control knob until a 1,000 Hz tone could not be heard in the earphones. Each task trial was 5 minutes long, followed by a 1-minute rest period. With a deterioration of tracking performance, heater coils on the legs became warm to provide an added incentive for the subject to better his performance as severe deterioration in his performance could result in painfully intense heat (204). Ten task trials were performed in each one hour experimental session. Tracking performances were not impaired after 60 hours of total sleep loss.

The resistance of tracking tasks to sleep loss effects is not puzzling as the subjects are usually over-trained in routines of tracking tasks, receive immediate feedback of their response, and the task duration is very short. These conditions of over-learning, knowledge of the result, and shortness of the task duration make tracking tasks very resistant to total sleep loss effects. Figure 2. shows the tracking errors of four subjects on a unidimensional compensatory tracking task. The subjects stood one at a time, in a semi-darkened quiet room, and, from a distance of 3 feet, viewed a target circle of 1 cm. in diameter displayed on a 5 inch oscilloscope. Shown also on the scope was a 2 mm. diameter light spot which was driven vertically over a 6 cm. course by a

sinusoidal signal from a low frequency function generator. The subjects were trained to keep the spot of light at the center of the target circle by manipulation of a hand-held thumb-wheel. With compensatory movements of the thumb-wheel, the oscilloscope showed only the voltage difference between the tracking signal and tracking response, i.e., a moment-to-moment display of the tracking errors. The task difficulty was easily changed by varying the frequency of the tracking signal from, for example, 0.2 Hz to 0.3 Hz. Subjects were not penalized for excessive tracking errors, and the quality of tracking performance was left entirely to the subject. The upper section of Figure 2. defines the tracking errors by showing the actual tracking record of subject H. H. This record shows a lapse or inability of roughly 20 seconds duration in tracking the signal on channel 2. During this period of lapse, the tracking errors increased, as they should, to 100 per cent as shown in channel 3. The lower section of Figure 2. illustrates two facets of the total sleep loss effects on tracking performance. Firstly, there is increased tracking error after total sleep loss of 100 hours or more. This was in spite of the built-in ruggedness of the tracking task. Secondly, the tracking errors consisted of two kinds: 1) a complete absence of the tracking (i.e., lapse), and 2) deteriorated, poor tracking. As long as the subjects were able to respond, they performed well, indicating that the skill of tracking was not affected. Records marked as R1 and R2 in Figure 2. were taken after one and two nights of recovery sleep and show some effects of residual sleep loss on performance.

Figures 3. through 10. show results of the sleep deprivation study now in progress under Laverne C. Johnson and Ardie Lubin of the Navy Medical Neuropsychiatric Research Unit, San Diego, California. These figures describe the most commonly used tasks which have been tailored for their sensitivity to total sleep loss. In the figures, the x axis is identified by the days of the experiment. The task sessions were held at approximately the same time each day, and labelled as baseline data (baseline day 1, B1 through B4, sleep deprivation data (after sleep loss of one night, D1; two nights, D2), and recovery day data (PR1, PR2, R3, and R4). The type of sleep the subjects were allowed after two nights without sleep differed. If the subjects were assigned to Group S, their first and second recovery sleep, after 54 hours of total sleep loss, was interrupted by the experimenter so that they received no slow wave sleep (the sleep stages III and IV). Subjects in the R Group were deprived of REM sleep. The C Group subjects enjoyed uninterrupted sleep after total sleep loss. Setting aside the rationale for such experimental sleep interruption for a moment, the reader's attention is directed to the effects of loss of total sleep on the performances of the subjects.

Figure 3. shows the preliminary results of a paper-and-pencil test devised and designed by Williams and his group (335) primarily for testing the subject's ability to retain 30 commonly used words over a brief period of time. Each test was administered in exactly the same manner as the Word Memory Test of Williams. The results showed that significant decreases in words recalled after a total sleep loss of one night and two nights were observed. Even though the number of subjects was small ($N = 10$), the effects of sleep loss on short-term memory were significantly detrimental.

Figure 4. shows the results of a task performance requiring addition of five two-digit numbers for one hour. The subjects were told to compute as many additions as possible without sacrificing accuracy in adding. Results revealed that both the number and accuracy of additions declined significantly with total sleep loss. The A Group, in which subjects were not sleep deprived, continued to improve its performance. This suggested the presence of the practice effect.

Figures 5, 6, and 7. illustrate the performance results of mental addition task, Plus 7. This task was

performed for 50 consecutive minutes. During the task, the subject reclined comfortably in bed with electrodes attached to him which monitored brain waves (EEG's) and autonomic variables while simultaneously recording his adding task performance. The subject's eyes were kept closed throughout the session. Near at hand was a response panel which could be operated by pressing one of 10 keys. On baseline day 1, the task began with the subject adding 1 to 7 while, at the same time, pressing response panel keys 1 and 7, respectively. To the response panel total of 8, he was then instructed to add mentally another 7 for a newly computed figure of 15. Throughout the test, the subject was instructed to continue adding, pressing the response keys, and mentally computing in this manner. If the subject lost his place in his mental addition, he could either start over again from the beginning or could go back to the last sum he could remember. Figure 5. is a polygraphic record of subject, J.Y., 5 minutes after the start of the Plus 7 task performance on his last baseline day, #4. The two EEG's (Cx-A2 and O1-A2) show clear alpha waves. The four autonomic variables, SP (skin potential), HR (heart rate), FP (finger pulse), and Resp (respiration), are shown on the four polygraph channels immediately below the EEG's. The result of the continuous mental addition is recorded on the last channel labelled TASK. By measuring and interpreting the heights of the pulses recorded on the TASK channel, the manner in which the subject performed his mental additions could be determined. In Figure 5., within a period of 30 seconds, six additions were attempted: $403 + 7$, $410 + 7$, $417 + 7$, and so on. Figure 6. illustrates the task performance for the same subject but after two nights of total sleep loss. During this performance he finished only three additions, $407 + 7$, $417 + 7$, and $421 + 7$, within the 30-second period. His performance reveals a considerable amount of irregularity, i.e., lapses, in the pace of his addition. Associated with these lapses were discernible gaps between digital key responses to 414 and 4 -- 21, poor alpha rhythms in the EEG's, high amplitude and poorly regulated finger pulses, and shallow, slow respiration patterns.

Figure 7. shows, on the y axis, the number of key responses which reflect roughly the number of mental additions performed by the subjects. All three groups which underwent sleep deprivation show significant decrement in the number of responses. A strong practice effect is evident in the data from Group A.

Results of an X-crossout task are shown in Figure 8. The purpose of this paper-and-pencil test was to measure the number of times the subject could cross out the letter X imbedded in the nonsense words. This one hour test consisted of a booklet of 11,000 10-letter nonsense words, 168 words to a page, and 24 words to a column. Twelve of the 24 words in each column contained the letter X. The location of X within each column was random. A different booklet was used every day. The total number of nonsense words detected in 60 minutes and the percentage of correct cancellations of the letter X were reduced after total sleep loss. In other words, all subjects in the C, S, and R Groups scanned significantly fewer nonsense words and made more errors by missing the X after total sleep loss.

Figures 9. and 10. show two aspects of the same auditory vigilance task. The purpose of this task was to determine the subject's ability to detect an auditory stimulus while engaged in a continuous counting task. Preparation of the subject was almost the same as that for the Plus 7 Task, except that the subject was required to count from 9 back to 1 by pressing the corresponding keys of the 10-key panel continually throughout a 50-minute period. While he was counting, a click was given through an amplifier. The subject was to indicate he had heard the signal by pressing the zero key on the panel once. He then resumed counting. There was a total of 30 clicks, given at irregular intervals, during the 50-minute session. The score for the continuous counting was the total number of minutes spent in counting without a gap, or lapse, of 2.8 seconds or more. The auditory

vigilance test was scored on the percentage of clicks the subject detected. Total sleep loss lowered the subject's ability to perform this task significantly.

Factors Influencing Task Sensitivity to Total Sleep Loss

Why are some tasks sensitive to total sleep loss? What factors are responsible for making such tasks sensitive to total sleep loss? Seven factors have been identified but only a few are well-confirmed experimentally. The seven identified factors are:

(1) Duration of task

The longer the task is, the more sensitive it is to total sleep loss. A total sleep loss of 50 hours causes impairment to the subject's performance during a continuous visual vigilance task after only 3 minutes. After 70 hours of total sleep loss, performance will be affected on a task with a duration of 2 minutes (341). Wilkinson (321 and 328) observed that total sleep loss of one night had no appreciable effect on task performances during the first 5 minutes on his five-choice test of serial reaction, vigilance, and addition. However, clear-cut performance deterioration emerged when the performances were evaluated after a 15-minute task session.

(2) Knowledge of results

By immediately feeding back to the subject information on how well he had performed each task at the time of execution, Wilkinson (321) observed that a total sleep loss of 30 hours did not appear to impair performance of the five-choice serial reaction and vigilance test. This immediate feedback of quality of task performance appears to minimize the effects of total sleep loss.

(3) Difficulties of task

Performances of difficult tasks are more sensitive to loss of total sleep. A simple task, such as addition, can be made very difficult by asking the subject to add quickly. Williams and Lubin (340) confirmed the effect of this increase in difficulty. Their results showed that mental addition at a rate of one addition per 2 seconds did not reveal the effects of two nights of sleep loss, but by increasing the speed of addition to one addition to every 1.25 seconds, i.e., a 38 per cent speed increase, sleep loss of two nights was detected.

(4) Task pacing

The self-paced task can resist sleep loss effects much better than the work-paced task. An example of the self-paced task is the five-choice test of serial reaction used by Wilkinson (328). In this task, the subject controlled when and how rapidly he responded to the stimulus. The subject worked at a pace he could command. By saying that the self-paced task could resist the effects of total sleep loss, does not imply that a self-paced task increased the overall human efficiency of the sleep-deprived subject's performance. In the self-paced task, lapses could prevent the completion of a task by causing the subject to lose his place mentally but, as soon as he came out of the lapse, he could again resume the task by picking up his chain of thought. Thus, in self-paced tasks, the effects of total sleep loss could show up in increased amounts of time needed to complete the tasks, although there would be fewer errors of omission and performance accuracy would remain high, i.e., an improved human reliability. Since there are no definitive studies on the mechanisms of self-paced in task performance, no firm estimates on added efficiency and reliability by self-paced can be given.

(5) Proficiency in task performance

Newly acquired skills, such as those involved in driving a car by a person who has just passed his driver's test for the first time, would be more affected by loss of total sleep than those skills which have become almost automatic or second nature.

(6) Task complexity

The more complex the task is, in terms of a complicated chain of mental operations and/or of an orderly execution of complex muscular activities, the more likely it is to be sensitive to loss of total sleep. Wilkinson showed, for example, that a 10-choice card sorting task was affected more than a less complex 4-choice one after 60 hours of total sleep loss (326).

(7) Memory requirements

Any task which needs a short-term memory chain will be affected by loss of total sleep (337). The best dramatic example of sleep deprivation on memory was given by S. I. A. Marshall in his description of paratroops who were dropped in Normandy (200, p. 87).

In summarizing the factors influencing task sensitivity to total sleep loss, it was found that, regardless of the task performed, the long, work-paced, complex task which does not provide information to the subject on how well he is performing can be expected to show higher sensitivity to total sleep loss.

However, there are two major classes of non-task factors which also influence the outcome of total sleep loss on performance. They are:

(1) Psychological factors:

(a) High interest. Ax and his colleagues (12) suggested that interesting tasks resist the effects of loss of total sleep. Wilkinson observed that task performances usually deteriorated to some extent after 20 hours of total sleep loss. The battle game, devised by Wilkinson (326), was so interesting to sailors they could work on the game for one hour without showing the effects of over 80 hours of total sleep loss.

(b) Motivation. While tasks of high interest usually have an inherently high motivational value, a distinction between high interest and motivation should be made. For example, a serial learning task which may have small interest to the subject can become highly motivating if his performances are monitored by his supervisor. Ax and his associates (12) reported that none of the high motivation tasks showed significant performance decrements after a sleep loss of one night, but most of the low motivating tasks, such as unmonitored sustained attention tasks composed of pursuit, reaction time, and clock tests, resulted in poor performances. In general, then, a higher motivation may counteract the effects of total sleep loss, but there is a point beyond which a certain amount of sleep debt and the sheer need for sleep will overwhelm even the subject's willingness to work (341).

(c) Personality. Wilkinson (310) reported on the lack of relationship between scores on the psychological tests of introversion-extroversion and neuroticism of Eysenck and the degree of performance degradation in the five-choice test of serial reaction after total sleep loss of one night. Carewran (89) observed, however, that extroverts were affected more than introverts by loss of 60 hours of sleep, as measured by a pursuit tracking task in which excessive tracking errors were punished by loud noises.

Although there are some studies which linked paranoid psychotic reactions during total sleep deprivation to a pre-morbid personality that existed prior to total sleep loss, no definitive studies are available on interaction of personality and effects of total sleep deprivation (128 and 294).

(d) Repeated experience of sleep loss. Wilkinson (321) reported that repeated exposure to loss of total sleep increased the effects of such sleep loss in terms of task performance.

(e) Behavioral periodicity. A severity of effects of total sleep loss depends, in part, on the time of day that measures of performance are obtained. This is due to a behavioral periodicity. Sleep-deprived subjects

will look quite normal and perform very well in the early afternoon when most of them have peak behavioral efficiency, but the same subjects will reveal a marked deterioration in their performance in the very early morning when sleep loss effects combine with unusually low behavioral efficiency. Sleep loss itself may tend to enhance periodicity in behavioral efficiency, although evidence for enhanced periodicity is available only for EEG data (92). Ignorance of behavioral periodicity, which may have been enhanced by loss of total sleep, was undoubtedly a factor in those studies which misinterpreted an improvement of performance during a period of total sleep deprivation (111). If the measures of performance were taken at different times of the day, they would reflect the effects of increasing sleep debt as well as behavioral periodicity.

(2) Situational factors:

(a) Physical exercises. Physical exercises just prior to performing the tasks helped resist the decrement of performance caused by loss of total sleep (328). It should also be noted that the only sure way of staying awake for many sleep-deprived subjects was to take a walk. This indicates the importance of continuous activity to muscles.

(b) Noise. A noise, e.g., 90 to 100 decibels of white noise in an open field, lessened the effects of total sleep loss on performances of auditory vigilance and on the five-choice test of serial reaction (59 and 323).

(c) Temperature. A moderate ambient temperature of 30.5°C (87°F) did not increase the effects of total sleep loss (247). Occasional cold stresses, e.g., immersing the face in ice cold water and/or alcohol rubs with an electric fan blowing air onto the subject, helped combat frequent spells of extreme sleepiness (242). There are no conclusive data on the interactions of extreme heat or cold on effects of total sleep deprivation on performances (328).

(d) Drugs. Amphetamine appears to be the only effective drug which has been widely used in combating the effects of total sleep loss (295, 162, 176). In the study by Kornetsky *et al.*, oral intakes of 15 mg. dextro-amphetamine almost halved the behavioral impairment caused by 68 hours without sleep on the work-paced vigilance task. The dextro-amphetamine was effective in completely eliminating small effects of sleep loss which occurred in self-paced tasks. Depending upon the dosage, alcohol was found to counteract the effect of total sleep loss (330).

(e) Breathing apparatus. Hypoxia and inert gas narcosis (especially nitrogen narcosis) by themselves produced performance degradations which were quite similar to the effects of total sleep loss. Hypoxia and inert gas narcosis may potentiate the effects of total sleep loss on performances, although such contention has not yet been studied experimentally.

III. PARTIAL SLEEP LOSS

Partial sleep loss means that the subject has shorter hours of sleep in a 24-hour period. But there are infinite ways to achieve partial sleep loss and the methodological problems involved in the study of partial sleep deprivation are many and complex. Because the subject does get some sleep, some of the problems are: 1) the detection of minor effects of partial sleep loss on performance, 2) the infinite variations possible in hours the subject should be allowed to sleep at one time, and 3) the need to know sleep patterns and habits of the subject well enough so that partial sleep deprivation is equally effective from subject to subject. In the latter case, for example, a deprivation of 5 hours of sleep means quite a different degree of sleep disturbance for a subject who normally sleeps only 6 to 7 hours as compared to a subject who is a long sleeper that sleeps 9 or more hours.

The experimenter must decide how many hours of sleep will be allowed, at what time in the 24-hour period such sleep will take place, and whether or not the subject will be allowed to take all sleep in a single stretch or in segments, i.e., fractionated sleep or many "naps." Jones and Oswald (131) found two healthy insomniac subjects, each of whom required only 3 hours of sleep in a 24-hour period. Thus, the need for careful examination of subjects in terms of their sleep patterns should be emphasized.

Performance under Partial Sleep Loss

The earliest partial sleep deprivation study was by May Smith (385) who used the McDougall dotting task, the associated word task, speed of tapping, and learning and re-learning nonsense syllables. Smith served as the subject and practiced these tasks five days a week, except during vacations, for a period of over three years to establish her "normal variation." Her daily experiment was conducted at "approximately the same time of the day." The McDougall dotting task machine was a "mechanical device whereby a continuous band of paper tape about 1 inch wide is drawn behind an opening or window in the top of the desk by a weight-driven clock-work movement." On this band of paper were small red circles and the machine was adjusted to a speed so the circles passed the field of vision at 5.8 circles per second. The task was for the subject to dot these circles as they passed by. Smith reduced her hours of sleep over three consecutive nights to $1\frac{1}{2}$ hours, $3\frac{1}{2}$ hours, and $5\frac{1}{2}$ hours, respectively. The results of this partial sleep deprivation were either normal records, or records showing some improvement. However, her data showed significantly large and persisting decrements in performance during recovery days following the partial sleep loss.

Moore, Jenkins, and Barker (221) tested muscular efficiency by using a hand dynamometer on female subjects who slept between 6-9 hours each. They found that the longer the subjects slept, the better were the hand-dynamometer performances. In the same year, Robinson and Richardson-Robinson (267) reduced the hours of sleep over three consecutive nights in 28 subjects and found that Army Alpha Scores were not affected by such partial sleep loss. In 1936, Laird and Wheeler (177) conducted an experiment similar to that of May Smith. Three subjects shortened their sleep by 2 hours nightly for a week. The task used in this study was mental multiplication of three-digit numbers by three-digit numbers. The subjects practiced for several weeks before the partial sleep loss. After a week of shortened sleep, each subject could solve the problems in longer time than previously and

with undiminished accuracy - but with more expenditure of energy. Laslett (174) performed a partial sleep loss experiment on four subjects whose sleep was shortened by 40 per cent of normal for five consecutive nights. The results indicated a loss in efficiency of performance.

In the appendix of a popular book on sleep, Laird and Muller (171) described their study on partial sleep loss. The experiment called for the subjects on the first night to be awakened after one hour of sleep; the second night after two hours of sleep; after three, "then, four, five, six, then six again, then five, followed by four, three, two, and one." This schedule was chosen to avoid the practice effect, if there were such. To "return sleep function to par," on off-nights sufficient hours of sleep were allowed. Data were collected only two nights per week, and none of the shortened hours of sleep occurred on consecutive nights. Healthy, active college men were subjects. These men practiced the tasks for a month before the time of actual experiment to avoid any additional improvement on task performance which might occur during the partial sleep loss experiment. The first of the tasks was a simple reaction time task. The subjects were seated, and warned twice by the experimenter's "ready" and "now." These words were followed by a click. Each subject responded to the click by pressing a telegraph key. After 4 hours of sleep, the reaction time performance was such that additional sleep brought no further improvement. Thus, Laird and Muller concluded that four hours "in bed" each night would be adequate if the subjects desired to sustain their reaction speed. The second task was aimed at muscular coordination and balance by measuring the body sways when eyes were closed, arms outstretched, and the knees were held close together. After 4 hours of sleep, the subjects demonstrated as much muscular coordination as they had after a full night of sleep. The color discrimination task, matching the sample color with a mixed color by using a color mixer, showed that only one hour of sleep was needed to bring their ability to match the colors to the level achieved after a full night of sleep. The fourth task involved an ergographic task of lifting a 2.2 pound weight by the middle left finger until the subject was completely exhausted. The subjects fatigued as quickly after 6 hours of sleep as after one hour of sleep. In dot counting, the last task, Laird and Muller observed that "greatest improvement appeared between the first and second hours, and every added hour of sleep brought added improvement." In summary, the Laird and Muller study indicated the adequacy of 4 hours of sleep for simple task performances.

Owens (236) examined body sways and number of digit crossouts in five subjects who were awakened on successive nights after 2, 4, 6, or 8 hours of sleep. Body sways were worse after only 2 to 4 hours of sleep but 2 to 4 hours of sleep were too short to improve ability to cross out digits. Van Orner (300) tested two subjects who attempted to memorize lists of 12 nonsense syllables after 1, 2, 4, or 8 hours of sleep. He found that retention after 4 hours was as good as after 8 hours of sleep, but that retention after 2 hours of sleep was definitely poor and did not differ from the capability of retention after no sleep at all.

Most of the studies cited above covered relatively short periods of partial sleep deprivation. There are, however, a few studies on long-term partial deprivation of sleep. Frevonn and his wife (94) served as the subject and the watcher for 28 consecutive days. The amount of sleep was varied to include seven periods of 10, 8, 6, or 4 hours of sleep for every 24 hours. The examination of finger ocillation and the tasks of performing discrimination reaction, manual pursuit, and memory span were given twice daily, at 9 a.m. and 9 p.m. The results showed that performance remained at approximately the same level of achievement as that attained prior to the partial deprivation of sleep study. Hubbard (123), using himself as the subject, underwent month of fractionated partial sleep loss, i.e., sleeping from 11 p.m. to 2 a.m., and again from 5 a.m. to 8 a.m. He

examined head steadiness, discrimination, dynamometer testing, card sorting, body temperature, respiration rate, pulse rate, blood pressure, metabolism, body weight, and blood. None of these showed changes. He found that his work capacity and aptitude were unimpaired and he felt well. However, Husband's findings must be contrasted to the fractionated partial sleep loss study that Kleitman (152, p. 313) reported on. Kleitman's two subjects slept for two 2½-hour periods that were separated by a 4-hour period of wakefulness. Both subjects reported "a very low capacity for work during the 4-hour interval separating the two sleeps."

Webb and Agnew (310) used eight subjects who slept 3 hours per 24-hour period for 8 consecutive days. The tasks used were: 1) "a paced addition test" in which each subject added a pair of single digits presented at the rate of two digits every 2 seconds and then added 8 to the just obtained sum, 2) "an X test" in which each subject listened to tape recorded letters and pressed a signal button each time he heard X, and 3) a Pentagon Task in which the subject pressed a signal button each time a critical red light was flashed on one of the five positions arranged in a pentagon. The results indicated that performances deteriorated only after the seventh and eighth night but these degradations of performance were "neither uniform nor fully consistent."

While Wilkinson published preliminary results in 1966, he later reported an extended study involving 24 subjects. In these latter studies (329), an attempt was made to detect minor changes in performance resulting from a reduction of sleep from 7½ to 5, 3, 2, 1 or zero hours after two consecutive nights. From 7:45 a.m. to 10:35 p.m., auditory vigilance and addition tasks, alternately, were used with usual rest periods interspersed. The experiment called for each subject to sleep short hours for two nights (Tuesday and Wednesday) every week for 6 weeks, leaving the rest of the week to normal sleeping hours. The full details of partial sleep loss on the adding task were not included in Wilkinson's papers but the details of vigilance performance were given in a context of signal detection theory, i.e., statistic d (intrinsic capacity to discriminate the signals) and statistic β (willingness to report signal detection). By using tasks which were tailored for maximal sensitivity to small loss of sleep, Wilkinson succeeded in detecting a decrease in percentage of signal detection after 5½ hours of sleep loss. In other words, a partial sleep loss after one night in which each subject slept only 2 hours. Wilkinson stated that vigilance performance was not significantly impaired until sleep was reduced to 2 hours or less. After two nights of curtailed sleep, he found that performance was significantly impaired when sleep was shortened to less than 5 hours.

Usually, partial deprivation of sleep was accomplished by limiting the hours of sleep in each 24-hour period. However, partial sleep loss can be achieved by restricting the hours of sleep over a 48-hour period or a 72-hour period. Kleitman (152, pp. 175-177) described two subjects who slept 8 hours at night but only after they had stayed awake 39 to 40 hours, i.e., 50 per cent partial sleep loss over a 48-hour period. Two subjects were on this schedule of a "48-hour day" for 15 periods or 30 days. Marked sleepiness, low work capacity, and low efficiency were observed during the first night of each 48-hour day. Hence, Kleitman concluded that twice the usual 24-hour day would be unsuitable to sustain performance. On the other hand, Oswald (237, pp. 172-173) reported on two subjects who went through a similar routine of "saved" sleep for a period of one month. One of the subjects felt less well than usual while the other felt well and adapted to this extended sleep-wakefulness cycle without impaired work efficiency.

Pertinent to the study of partial sleep loss and performance were three published papers which agreed, in general, that reduction of sleep hours resulted in alterations in the kinds of sleep the subjects obtained during their limited hours of sleep. What were the kinds of sleep? In 1957, Dement and Kleitman (74) described the

stages of sleep in man, and their classification has been widely accepted (259). They found there were, in human subjects, five reliably identifiable stages of sleep, 1, 2, 3, 4, and REM (Figure 11.). Sleep Stages 1 through 4 were characterized by the appearance of more and more high amplitude slow brain waves, representing generally an increasing order of deepening sleep as measured by the arousal threshold. Stage REM, i.e., a period of rapid eye movement, was characterized by low voltage brain waves and was reported subjectively as the dreaming experience. Some of the studies on sleep physiology indicated a possible duality of sleep from Stage REM to NREM (non-rapid eye movement) sleep (135).

What was the usual sleep pattern? According to Williams, Agnew, and Webb (345), 16 male subjects, age 21 to 31 with a mean age of 24.2, usually had on the average of 24 per cent REM, 49 per cent Stage 2, and 21 per cent Stages 3 and 4 combined, of sleep during the night. When hours of sleep were reduced, did the subjects still obtain the comparable proportions of these sleep stages as they would have in their full night's sleep? Sampson (272) woke six subjects up for three consecutive nights after about 2½ hours of sleep. He found that more Stage REM tended to squeeze into their curtailed hours of sleep. Also noted in his study were the shortened REM latency and an evidence of competition between REM and NREM sleep. Under this shortened sleep, the subjects were not specifically deprived of NREM sleep, but the recovery night sleep showed more NREM sleep and that REM time compensation was delayed. Dement and Greenberg (71) curtailed the sleep of two subjects by 2½ to 3 hours every night for six consecutive nights. They found little or no increase in Stage 4 sleep during the partial sleep deprivation but a very curious increase in Stage 4 was observed during recovery nights. They repeated the same experiment with two different subjects. Partial loss of sleep was conducted for three consecutive nights only. They found again that there was no Stage 4 increase during the shortened hours of sleep but a rebound was noted on recovery nights. Webb and Agnew (310) permitted each of eight subjects to have only three hours of sleep for 8 consecutive days. They found that the pattern of shortened sleep was far different from what had been expected of the miniature edition of the usual sleep pattern. A comparison of the first 3 hours of the baseline sleep with the 3-hour sleep period indicated the shortened sleep had significantly more Stage 4 sleep, whereas there was less Stage 2, and the Stage REM began to show up more during this shortened sleep. Despite the abundance of slow wave sleep observed during the shortened sleep periods, a Stage 4 increase was noticed in sleep on recovery nights.

Therefore, the proportions of given sleep stages were altered by simply limiting the hours of allowed sleep. In other words, a change in the kind of sleep resulted from limiting the quantity (hours) of sleep. Webb and Agnew (311) have contributed a clarification of how partial sleep deprivation caused these changes, not only in the duration of sleep (which they attempted to alter by deprivation of sleep) but also in the kind (quality) of sleep.

One of the interesting observations of these investigators was the fact that the succession of sleep stages over a night's sleep was not entirely random. It was definitely cyclic and extremely stable within an individual's sleep, indicating a strong stability in intra-subject sleep pattern. Commonly this pattern was constructed so that 70 per cent of sleep Stage 4 occurred during the first third of the night, whereas sleep Stage REM occurred predominantly in the last third of sleep (Figure 12.). This cyclic "time of the night" effect, with respect to the kind of sleep, was found to resist attempts to modify it by changing sleep habits.

The presence of persistent and strong intra-sleep cycling of sleep stages has complicated research on partial sleep loss. The implications of stable sleep cycles were not fully explored until the works of Webb and Agnew.

In addition to the usual "time of the night" cycling of sleep stages, Webb and Agnew (311) suggested a presence of similar sleep cycling over a 24-hour period. In other words, the kind of sleep one got in the morning, at noon, or in the afternoon would differ due to this 24-hour cycling. A morning nap would be closed to the last third of nocturnal sleep in its sleep stage proportions than a late afternoon nap which would be closed to the sleep found during the first third of the night in its proportion of sleep stages.

Then, it would be reasonable to assume, until shown otherwise by experiments, that the shorter the hours of sleep permitted for non-chronic partial sleep deprivation subjects, the greater the sleep Stage REM deprivation would be. This apparently selective deprivation of the sleep Stage REM with partial deprivation procedure was utilized by Wilkinson (329) to explain the effects of partial deprivation of sleep on the statistical d and beta measures of auditory vigilance. Wilkinson concluded that the slow wave sleep had more to do with the capacity to discriminate and that REM sleep was closely tied up with willingness to report the signal detection.

Many sleep researchers are concerned with the changed quality of sleep due to partial sleep loss because they assume that each stage of sleep has unique biological and psychological functions. Such concern with the sleep stages would be well taken if certain stages of sleep are indeed critical in maintaining human task performance. However, at this moment the function of sleep stages is far from being clear-cut and much more research is necessary before a definite conclusion can be drawn on the biological and psychological necessities of each sleep stage.

IV. SELECTIVE SLEEP LOSS

Unlike the total and partial sleep deprivations, selective sleep loss does not occur in its purest form in our normal daily routine. Selective deprivation of sleep stages can be seen only as a result of laboratory interruption of sleep.

Selective sleep deprivation is the withholding of a particular stage of sleep. Hence, in the Stage REM deprivation, the subject will be prevented from getting REM sleep by his being awakened, although he is permitted to obtain all other types of sleep. Figure 13. is a polygraphic record of the transition period from sleep Stage 2 to REM. The top two channels are left and right eye movements (electrooculographic) channels. Electromyographic (EMG) activity on and beneath the chin is shown on channel 3. Three EEG leads are shown on the 4th, 5th, and 6th channels, respectively. All of these were referred to the opposite ear. Autonomic variables of skin potential (SP), heart rate (HR), with its calibration in beats per minute, and finger pulse (FP) are recorded on channels 7, 8, and 9. With the onset of Stage REM sleep, there was "concomitant appearance of relatively low voltage, mixed frequency EEG activity and episodic REM" (259, p. 7). Also there was a diminished tonic mental-submental EMG. Thus, the subject could be aroused from his sleep to prevent him from REM stage sleep at the points marked by either 4 dots or 6 dots.

Selective sleep stage deprivations were attempted in two ways; either deprivation of sleep Stage REM or deprivation of sleep Stages 3 and 4, i.e., slow wave sleep (64, 70, 139, 272, 65, 2, 3, and 347). Stage 1 and 2 deprivations were not attempted because such withholding would have interfered with the subject getting permissible sleep under the stage deprivation schedule. The combination of two-stage deprivation, e.g., deprivation of both REM and slow wave sleep, remains untried.

The underlying basis for any selective sleep deprivation study is a conviction that each stage of sleep may have a unique function whose lack will result in a heightened "press" for a selectively deprived sleep stage. It has been shown after selective sleep stage deprivation that sleep Stage REM or slow wave sleep increases or "rebounds" over pre-deprivation level during the nights of recovery sleep. Figure 14. illustrates a rebound in the first recovery night (R3) after two nights of total sleep loss and two nights of deprivation of slow wave sleep.

The rebound effect after selective sleep deprivation indicated that: 1) a lack of REM or slow wave sleep could not be compensated for by other sleep stages, and 2) REM and slow wave sleep could be interpreted as providing unique yet undefined functions for human organisms.

Performance under Selective Sleep Loss

A study of selective sleep deprivation of slow wave or Stage REM in six subjects for seven consecutive nights showed no performance decrement as measured by the paced addition, the strength of grip, or the pursuit rotor task (3 and 347). Although there had been obviously successful stage deprivation in these studies as judged by amounts of rebound during recovery night sleep, a lack of performance degradation was observed. In contrast to the overall lack of performance decrement because of selective sleep loss, either REM or slow wave sleep, a majority of studies on selective deprivation showed a variety of not too reliable changes in the

psychological functions of man. Although Kales and his associates (139), for example, failed to observe psychic changes as a result of REM deprivation. One common feature of selective sleep stage deprivation was that psychological changes, if there were any, occurred only after a relatively long period, e.g., roughly one week (68). Psychological testing must be extensive before psychological changes due to selective sleep loss can be detected (273). Thus, in the Williams, Agnew, and Webb study (347), the personality changes of the subjects were measured by the Minnesota Multiphasic Personality Inventory (MMPI), the Pensacola Z Scale, the Tyler Manifest Anxiety Scale, and Cattell's 16 PF Test. These investigators found that during Stage 4 deprivation "the subjects became physically uncomfortable, withdrawn and less aggressive, and manifested concern over vague physical complaints and changes in bodily feelings -- hypochondriacal reaction" (347). However, during REM deprivation the subjects became less well-integrated and less interpersonally effective. Clemens and Dement (54) used a variety of tests, including the color-word test, Walsh Figure Preference Test, Hotzman Ink Blot Test, TAT-styled pictures, Nowlis-Green Mood Check List, and others, on six subjects who underwent six nights of REM deprivation and six nights of Non-REM deprivation. They found that the REM deprivation caused "an increase in need and feeling intensity with a drop in certain ego-control functions." In other words, selective sleep stage deprivation of the relatively long duration of seven days caused "distinct unhealthy changes in the personality profiles" (347).

If there are performance tasks which are detrimentally effected by those mood and personality changes, as observed upon occasion after selective sleep stage deprivation, intensive study must be taken to detect such degradation. However, the literature on selective sleep loss has been very consistent in not finding any task performance deterioration. The results of studies at the Navy Medical Neuropsychiatric Research Unit with Laverne C. Johnson and A. Lubin (Figures 2. through Figure 10.) confirmed a similar lack of performance degradation due to selective sleep stage deprivation. In that series of experiments, the subjects were deprived of total sleep for two nights to create sufficient amounts of sleep debt resulting in definite performance degradation. Then, for two nights the subjects were divided into groups: Group A was permitted to sleep 8 uninterrupted hours (the Control group); Group S was deprived of slow wave sleep, while Group R was deprived of REM sleep. Thus, counting the total sleep loss of two nights, the subjects in Group S had actually 4 nights of sleep without slow wave sleep and the Group R subjects had 4 nights of REM deprived sleep. In terms of performances in immediate recall, addition, Plus 7, X crossouts, continuous counting, and auditory vigilance, the selective sleep stage deprivations did not cause performance degradation.

In 1965, Fisher summarized that the REM-deprived subjects showed disturbances in motor coordination (dropping small objects), in memory (forgetting appointments), difficulty in concentration, and a few others. These psychological changes are not fully substantiated by later studies, and these observed psychological degradations may have been due to subject-experimenter interaction in the course of REM deprivation procedures. Indeed, Snyder (287), Kales and his colleagues (139), and Vogel (302) observed only the minimal psychological changes, at least, in human subjects.

BIBLIOGRAPHY ON SLEEP DEPRIVATION¹

- 1* Adams, O. S., and Chiles, W. D. Human performance and the work-rest schedule. In E. Bennett, J. Degan, and J. Spiegel (Eds.), Human factors in technology. New York: McGraw-Hill, 1963, Pp. 38-64.
- 2 Agnew, H. W., Jr., Webb, W. B., and Williams, R. L. The effects of stage four sleep deprivation. Electroenceph. Clin. Neurophysiol., 1963, 17, 68-70.
- 3 Agnew, H. W., Jr., Webb, W. B., and Williams, R. L. Comparison of stage four and 1-REM sleep deprivation. Percept. Motor Skills, 1967, 24, 851-858.
- 4* Agnew, H. W., Jr., Webb, W. B., and Williams, R. L. Sleep patterns in late middle age males: An EEG study. Electroenceph. Clin. Neurophysiol., 1967, 23, 168-171.
- 5 Agostini, C. Sui disturbi psichici e sulle alterazioni del sistema nervoso centrale per insonnia assoluta. Riv. Sper. Freniat. 1898, 24, 113-125.
- 6 Alluisi, E. A., Chiles, W. D., and Hall, T. J. Combined effects of sleep loss and demanding work-rest schedules on crew performance. ANRL-TDR-64-63, Aerospace Medical Lab., Wright-Patterson AFB, Ohio, 1964. Pp. 63.
- 7 Alluisi, E. A., Chiles, W. D., and Smith, R. P. Human performance in military systems: some situational factors influencing individual performance. ITR-64-1, Performance Research Laboratory, Department of Psychology, University of Louisville, Kentucky, 1964. Pp. 24.
- 8* Alluisi, E. A., and Chiles, W. D. Sustained performance, work-rest scheduling, and diurnal rhythms in man. Acta Psychol., 1967, 27, 436-442.
- 9 Anderson, L. H., and Corfein, D. S. A case of prolonged sleep deprivation. J. Gen. Psychol., 1964, 71, 291-292.
- 10 Arrington, J. C., and Mitnick, L. L. Electroencephalogram and sleep deprivation. J. Appl. Physiol., 1959, 14, 247-250.
- 11 Ax, A., and Luby, E. D. Autonomic responses to sleep deprivation. Arch. Gen. Psychiat., 1961, 4, 55-59.
- 12 Ax, A. F., Fordyce, W., Loovas, I., Meredith, W., Pirajnikoff, L., Shavonian, B., and Wendahl, R. Quantitative effects of sleep deprivation. U.S. Army Quartermaster Res. Development Res. Rep., 1957.
- 13 Baraldi, M. Le modificazioni umorali indotte nell'organismo di epilettici sottoposti ad insonnia sperimentale. Riv. Sper. Freniat., 1940, 64, 165-172.
- 14 Barry, J., and Boussfield, W. A. A quantitative determination of euphoria and its relation to sleep. J. Abnorm. Soc. Psychol., 1935, 29, 385-389.
- 15 Bast, T. H., and Loewenhart, A. S. Studies in exhaustion due to lack of sleep. I. Introduction and methods. Amer. J. Physiol., 1927, 82, 121-126.
- 16 Bast, T. H., Schacht, F., and Vanderkamp, H. Studies in exhaustion due to lack of sleep. III. Effect on the nerve cells of the spinal cord. Amer. J. Physiol., 1927, 82, 131-139.
- 17 Bast, T. H., and Bloemendal, W. B. Studies in experimental exhaustion due to lack of sleep. IV. Effects on the nerve cells in the medulla. Amer. J. Physiol., 1927, 82, 140-146.
- 18 Bast, T. H., Supernaw, J. S., Lieberman, B., and Munro, J. Studies in exhaustion due to lack of sleep. V. Effect on the thyroid and adrenal glands with special reference to mitochondria. Amer. J. Physiol., 1928, 85, 135-140.

¹Basic reading materials pertinent to sleep in general are identified by the asterisk. For the most comprehensive and latest bibliography, refer to "Sleep and Dream Research A Bibliography" by Rechtschaffen and Eskin, published in 1969 by Brain Information Service, UCLA, Los Angeles, California 90024. A continuing bibliographic service on sleep and sleep deprivation is available, upon request, from the UCLA Brain Information Service as Sleep Bulletin.

- 19 Bennett, D. R. Sleep deprivation and major motor convulsions. *Neurology*, 1963, 13, 953-958.
- 20 Bennett, D. R., Mattson, R. H., Ziter, F. A., Calverley, J. R., Liske, E. A., and Pratt, K. L. Sleep deprivation: neurological and electroencephalographic effects. *Aerospace Med.*, 1964, 35, 888-890.
- 21 Berger, R. J., and Meier, G. W. The effects of selective deprivation of states of sleep in the developing monkey. *Psychophysiology*, 1966, 2, 354-371.
- 22 Berger, R. J., and Oswald, I. Effects of sleep deprivation on behavior, subsequent sleep and dreaming. *J. Ment. Sci.*, 1962, 108, 457-465.
- 23* Bills, A. G. Blocking: New principle of mental fatigue. *Amer. J. Psychol.*, 1931, 43, 230-245.
- 24 Ejermer, B. Alpha depression and lowered pulse rate during delayed actions in a serial reaction test: a study in sleep deprivation. *Acta Physiol. Scand.*, 1949, Suppl. No. 65, Vol. 19. Pp. 93.
- 25 Blake, H., and Gerard, R. W. Brain potentials during sleep. *Amer. J. Physiol.*, 1937, 119, 692-703.
- 26 Blake, H., Gerard, R. W., and Kleitman, N. Factors influencing brain potentials during sleep. *J. Neurophysiol.*, 1939, 2, 48-60.
- 27 Bliss, E. L. Sleep in schizophrenia and depression - Studies of sleep loss in man and animals. In S. S. Kety, E. V. Everts and H. L. Williams (Eds.), *Sleep and altered states of consciousness*. Proc. Ass. Res. Nerv. Ment. Dis., 1965. Baltimore: Williams and Wilkins, 1967. Pp. 195-210.
- 28 Bliss, E. L., Clark, L. D., and West, C. D. Studies of sleep deprivation - Relationship to schizophrenia. *Arch. Neurol. Psychiat.*, 1959, 81, 348-359.
- 29 Boren, J. J. Decrement in performance during prolonged avoidance sessions. *J. Exp. Anal. Behav.*, 1960, 3, 201-206.
- 30 Boufield, W. A. Further evidence of the relation of the euphoric attitude to sleep and exercise. *Psychol. Rec.*, 1938, 2, 336-344.
- 31 Boufield, W. A. Relation of euphoric attitude to quality of sleep. *J. Psychol.*, 1940, 9, 393-401.
- 32 Bowen, J. H., Ross, S., and Andrews, T. G. A note on the interaction of conditioned and reactive inhibition in pursuit tracking. *J. Gen. Psychol.*, 1956, 55, 158-162.
- 33 Bowers, M. B., Jr., Hartmann, E., and Freedman, D. X. Sleep deprivation and brain acetylcholine. *Science*, 1966, 153, 1416-1417.
- 34 Brauchi, J. T., and West, L. J. Sleep deprivation. *J. Amer. Med. Assoc.*, 1959, 171, 11-14.
- 35 Bredland, E. Effect of sleep deprivation on certain human activities. Unpublished doctoral dissertation, New York University, 1955.
- 36 Bridger, W. H. The neurophysiological accompaniments of sensory and sleep deprivation and their role in the production of psychological disturbances. In J. Wortis (Ed.), *Recent advances in biological psychiatry*, Vol. 6. New York: Plenum Press, 1968. Pp. 105-110.
- 37 Brodan, V., Kuhn, E., and Valak, J. [Effect of sleep deprivation on oral sugar tolerance curves.] *Can Lek Cong.*, 1968, 107, 412-414.
- 38 Bronck, J. Sleep deprivation, nutritional deficit, and some Soviet work on stress. In *Symposium on medical aspects of stress in the military climate*, 1964. Washington, D.C.: Walter Reed Army Institute of Research. Pp. 211-242.
- 39 Bruce, L. C. Some observations upon the general blood pressures in sleeplessness and sleep. *Scottish Med. Surg. J.*, 1900, 2, 109-117.
- 40 Bunch, M. E., Cole, A., and Frerichs, J. The influence of twenty-four hours of wakefulness upon the learning and retention of a maze problem in white rats. *J. Comp. Psychol.*, 1937, 22, 1-12.
- 41 Bunch, M. E., Frerichs, J. B., and Licklider, J. R. An experimental study of maze learning ability after varying periods of wakefulness. *J. Comp. Psychol.*, 1938, 22, 499-514.
- 42 Burch, M. R., and Greiner, T. H. Drugs and human fatigue: GSR parameters. *J. Psychol.*, 1938, 41, 3-10.
- 43 Byck, R., and Meerst, E. Adjustment of monkeys to five continuous days of work. *Science*, 1962, 132, 43-44.
- 44* Caldwell, D. F., and Domino, E. F. Electroencephalographic and eye movement patterns during sleep in chronic schizophrenic patients. *Electroenceph. Clin. Neurophysiol.*, 1967, 22, 414-420.

- 45 Cappon, D., and Banks, R. Preliminary study of endurance and perceptual change in sleep deprivation. Percept. Motor Skills, 1960, 10, 99-104.
- 46 Cappon, D., and Banks, R. Studies in perceptual distortion: Opportunistic observations on sleep deprivation during a talkathon. Arch. Gen. Psychiat., 1960, 2, 346-349.
- 47 Carlson, V. R. Effects of sleep deprivation and chlorpromazine on size-constancy judgments. Amer. J. Psychol., 1961, 74, 552-560.
- 48 Carmichael, L., and Kennedy, J. L. Some recent approach to the experimental study of human fatigue. Science, 1949, 110, 445.
- 49 Carmichael, L., Kennedy, J. L., and Mead, L. Some recent approaches to the experimental study of human fatigue. Proc. Nat. Acad. Sci., 1949, 35, 691-696.
- 50 Cartwright, R., Monroe, L., and Palmer, C. Individual differences in response to REM deprivation. Arch. Gen. Psychiat., 1967, 16, 297-303.
- 51 Chile, W. D. The effects of sleep deprivation on performance of a complex mental task. WADC Tech. Rep. 55-423, Wright Air Development Center, Wright-Patterson AFB, Ohio, 1955. Pp. 13.
- 52 Clark, R. E., and Warren, N. The effect of loss of sleep on visual tests. Amer. J. Ophthalmol., 1939, 16, 20-25.
- 53 Clark, R. E., and Warren, N. A photographic study of reading during a sixty-five hour vigil. J. Educ. Psychol., 1940, 31, 383-390.
- 54 Clemens, S., and Dement, W. Effect of REM sleep deprivation on psychological functionings. J. Nerv. Ment. Dis., 1967, 144, 485-491.
- 55 Cohen, H., and Dement, W. Sleep: Changes in threshold to electroconvulsive shock on rats after deprivation of "paradoxical" phase. Science, 1965, 150, 1318-1319.
- 56 Cohen, H. S., Duncan, R. F., and Dement, W. C. Sleep: the effect of electroconvulsive shock in cats deprived of REM sleep. Science, 1967, 156, 1646-1648.
- 57 Cooperman, M. R., Mullin, F. J., and Kleitman, N. Studies on the physiology of sleep. XI. Further observations on the effects of prolonged sleeplessness. Amer. J. Physiol., 1934, 107, 589-593.
- 58 Corcoran, D. W. J. Individual differences in performance after loss of sleep. Unpublished doctoral dissertation, Cambridge, England, 1963.
- 59 Corcoran, D. W. J. Noise and loss of sleep. Quart. J. Exp. Psychol., 1962, 14, 178-182.
- 60 Corcoran, D. W. J. Changes in heart rate and performance as a result of loss of sleep. Brit. J. Psychol., 1964, 55, 307-315.
- 61 Crile, G. W. Studies in exhaustion. Arch. Surg., 1921, 3, 196-220.
- 62 Cuthbertson, D. P., and Knox, J. A. C. The effects of anaesthetics on the fatigued subject. J. Physiol., 1947, 106, 43-68.
- 63 Davis, M., Davis, P. A., Harvey, E. H., and Hobart, G. Human brain potentials during the onset of sleep. J. Neurophysiol., 1938, 1, 24-38.
- 64 Dement, W. C. The effect of dream deprivation. Science, 1960, 131, 1705-1707.
- 65 Dement, W. C. Experimental dream studies. In J. Masserman (Ed.), Science and psychoanalysis: Scientific proceedings of the Academy of Psychoanalysis, Vol. 7. New York: Grune and Stratton, 1964. Pp. 150-162.
- 66 Dement, W. C. Recent studies on the biological role of rapid eye movement sleep. Amer. J. Psychiat., 1968, 125, 404-408.
- 67 Dement, W. C. An essay on dreams: The role of physiology in understanding their nature. In T. Newcomb (Ed.), New directions in psychology, Vol. 2. New York: Holt, Rinehart and Winston, 1965. Pp. 157-257.
- 68 Dement, W. C. Studies on the function of rapid eye movement (paradoxical) sleep in human subjects. In M. Jouvet (Ed.), Aspects clinico-fonctionnels de la physiologie du sommeil. Paris: Centre National de la Recherche Scientifique, 1965. Pp. 572-611.
- 69 Dement, W. C. Psychophysiology of sleep and dreams. In S. Arieti (Ed.), American handbook of psychiatry, Vol. 2. New York: Basic Books, 1966. Pp. 200-222.
- 70 Dement, W. C., and Fisher, C. Experimental interference with sleep cycle. Canad. Psychiat. Assoc., 1963, 8, 400-408.

- 71 Dement, W. C., and Greenberg, S. Changes in total amount of stage four sleep as a function of partial sleep deprivation. Electroenceph. Clin. Neurophysiol., 1966, 20, 523-526.
- 72 Dement, W. C., Greenberg, S., and Klein, R. The effect of partial REM sleep deprivation and delayed recovery. J. Psychiat. Res., 1966, 4, 141-152.
- 73 Dement, W., Henry, P., Cohen, H., and Ferguson, J. Studies on the effect of REM deprivation on humans and on animals. In S.S. Kety, E. V. Everts, and H. L. Williams (Eds.), Sleep and altered states of consciousness, Proc. Assoc. Res. Nerv. Ment. Dis., 1965. Baltimore: Williams and Wilkins, 1967. Pp. 456-468.
- 74° Dement, W. C., and Kleitman, N. Cyclic variations of EEG during sleep and their relation to eye movements, body motility and dreaming. Electroenceph. Clin. Neurophysiol., 1967, 2, 673-690.
- 75 Dawson, J. H., III., Dement, W. C., Wagner, T. E., and Nobel, K. Rapid eye movement sleep deprivation: A central-neural change during wakefulness. Science, 1967, 156, 403-406.
- 76 De Renzi, E., and Faglioni, P. [The influence of sleep deprivation and work on performance in vigilance tests.] Arch. Psicol. Neurol. Psichiat., 1966, 27, 552-566.
- 77 Desoille, M., Pinchon, R. A., Faivre, G., and Bourguignon, A. [Effects of fatigue due to sleep deprivation on the human electroencephalogram.] Arch. Malad. Prof., 1964, 23, 303-314.
- 79 Dusan-Peyrethon, D., Peyrethon, J., and Jouvet, M. [A quantitative study of the phasic EEG phenomena of paradoxical sleep during and after its instrumental deprivation.] C. R. Soc. Biol., 1967, 161, 2530-2533.
- 80 Dusan-Peyrethon, D., Peyrethon, J., and Jouvet, M. [Selective suppression of paradoxical sleep in the cat by alpha methyl DOPA.] C. R. Soc. Biol., 1968, 162, 116-118.
- 81 Eagles, J. B., Halliday, A. M., and Redfearn, J. W. T. The effect of fatigue on tremor. In W. F. Floyd and A. T. Welford (Eds.), Fatigue. London: Lewis, 1953. Pp. 41-58.
- 82 Edwards, A. S. Effects of the loss of one hundred hours of sleep. Am. J. Psychol., 1941, 54, 80-91.
- 83 Faivre, G. Effects de la privation de sommeil sur la reactivite corticale. Paris: These, 1964.
- 84° Feinberg, I., Korecko, R. L., and Meller, M. EEG sleep patterns as a function of normal and pathological aging in man. J. Psychiat. Res., 1967, 5, 107-144.
- 85 Feldman, G. L. [The effect of deprivation of sleep on electrical activity and other indices of cerebral activity in animals.] Soviet Physiol. J. USSR, 1961, 47, 186-196.
- 86 Ferguson, J., and Dement, W. C. The effect of variations in total sleep time on the occurrence of rapid eye movement sleep in cats. Electroenceph. Clin. Neurophysiol., 1967, 22, 2-10.
- 87 Fisher, C. Psychological significance of the dream-sleep cycle. In H. A. Witkin and H. B. Lewis (Eds.), Experimental studies of dreaming. New York: Brunner House, 1967. Pp. 76-127.
- 88 Fishgold, H., Laverhne, J., and Blanc, C. [Sleep, insomnia, and sleep debt in aeronautic medicine.] Aviation Res., 1967, 7, 391-395.
- 89 Fiorion, V., Higgins, E. A., Impietro, P. F., Latogola, M. T., and Davis, A. W. Physiological responses of man during sleep deprivation. J. Appl. Physiol., 1968, 24, 167-176.
- 90 Foulkes, D., Pivik, T., Abrams, J. B., and Bannion, E. M. Effects of "dream deprivation" on dream content: an attempted cross-night replication. J. Abnorm. Psychol., 1968, 74, 403-418.
- 91 Fortune, R. F. Sleep and muscular work. The effect of sleep on the ability to perform muscular work. Austral. J. Psychol., 1956, 4, 36-40.
- 92 Frank, G., Milberg, F., Warner, R., Matthews, J., Johnson, E., Grevan, H., and Andrus, V. Circadian periodicity, adrenal corticosteroids, and the EEG of normal man. J. Psychiat. Res., 1966, 4, 73-86.
- 93 Frederick, W. F. Physiological aspects of human fatigue. Arch. Indust. Health, 1959, 20, 297-302.
- 94 Froman, G. L. Compensatory reinforcements of muscular tension subsequent to sleep loss. J. Exp. Psychol., 1952, 45, 267-268.
- 95 Froman, G. L., and Lohy, E. D. Some biochemical findings in sleep deprivation. In Progress in medical aspects of stress in the military climate. Washington, D.C.: Walter Reed Army Institute of Research, 1964. Pp. 208-210.

- 96 Fruchtgott, E., and Willingham, W. W. The effect of sleep-deprivation upon thresholds of taste. Amer. J. Psychol., 1956, 69, 111-112.
- 97 Gersham, S. C., Webb, W. B., and Williams, R. L. Alcohol and caffeine: effects on inferred visual dreaming. Science, 1963, 140, 1226-1227.
- 98 Gieseck, C. F., Williams, M. L., and Labin, A. The effect of sleep deprivation upon information learning. Amer. Psychologist, 1957, 12, 406.
- 99 Gieseck, C. F., Williams, M. L., and Labin, A. A generalization of Bill's concept of "blocks" to performance lapses in reaction time during sleep loss. Amer. Psychologist, 1958, 13, 422.
- 100 Gifford, S., and Murawski, B. J. Minimal sleep deprivation alone and in small groups: Effects on ego-function and 24-hour body temperature and adrenocortical patterns. In Symposium on medical aspects of stress in the military climate. Washington, D.C.: Walter Reed Army Institute of Research, 1964. Pp. 157-185.
- 101 Goldie, L., and Green, J. M. Paradoxical blocking and arousal in the drowsy state. Nature, 1960, 187, 952-953.
- 102 Goodhill, V., and Tyler, D. B. Experimental insomnia and auditory acuity. Arch. Otolaryngol., 1947, 46, 221-224.
- 103 Greenberg, R. Dream interruption insomnia. J. Ment. Nerv. Dis., 1967, 144, 18-21.
- 104* Greenberg, R., and Pearlman, C. Delirium tremens and dreaming. Amer. J. Psychiat., 1967, 124, 133-142.
- 105* Gross, M. M., Goodenough, D., Tobin, M., Malpert, E., Lepore, D., Perlstein, A., Sirote, M., Dibianco, J., Fuller, R., and Kirchner, I. Sleep disturbances and hallucinations in the acute alcoholic psychoses. J. Nerv. Ment. Dis., 1966, 142, 493-514.
- 106* Gross, M. M., and Goodenough, D. R. Sleep disturbances in the acute alcoholic psychoses. Psychiat. Res. Rep., 1968, 24, 132-147.
- 107 Gulevich, G., Dement, W., and Johnson, L. C. Psychiatric and EEE observations in a case of prolonged (264 hours) wakefulness. Arch. Gen. Psychiat., 1966, 15, 29-35.
- 108* Gulevich, G. D., Dement, W. C., and Zarcone, V. P. All-night sleep recordings of chronic schizophrenics in remission. Gen. Psychiat., 1967, 2, 141-149.
- 109* Gunderson, E. K. E. Adaptation to extreme environments: Prediction of performance. Unit Report No. 66-17, Navy Medical Neuropsychiatric Research Unit, San Diego, California, 1966. Pp. 41.
- 110* Hamilton, R. W., Macinnis, J. B., Noble, A. D., and Schriener, H. R. Saturation diving at 650 feet. Tech. Memo. B-411, Ocean Systems, Inc., Tonawanda Research Laboratory, Tonawanda, New York, March, 1966.
- 111 Handbook of Human Engineering Data. Medford, Mass.: Tufts College, 1949.
- 112 Harris, S. J. The effect of sleep loss on component movements of human motion. J. Appl. Psychol., 1960, 44, 50-55.
- 113* Hartman, B. O., and Controll, G. K. Sustained pilot performance requires more than skill. Aerospace Med., 1967, 38, 801-803.
- 114 Hartman, B. O., and Controll, G. K. NDL: Crew performance on demanding work/rest schedules compounded by sleep deprivation. SM-TR-67-99, USAF School of Aerospace Medicine, Brooks Air Force Base, Texas, 1967. Pp. 29.
- 115* Hartmann, E. Dauerwach. A polygraphic study. Arch. Gen. Psychiat., 1960, 11, 99-111.
- 116 Haselmann, M., Schaff, G., and Metz, B. [Respective influences of work, ambient temperature and sleep deprivation on urinary excretion of catecholamines of normal man.] Z. Exp. Biol., 1960, 114, 197-201.
- 117* Hasty, C. T. Psychophysiological problems of manned space vehicles. In Lectures in Aerospace Medicine, 11-14 January 1960. USAF School of Aerospace Medicine, Brooks Air Force Base, Texas, 1960. Pp. 51.
- 118 Hasty, C. T., and Payne, R. B. Behavioral and physiological consequences of 30 hours of sustained work. Amer. Psychologist, 1957, 12, 406.
- 119* Hernandez-Peon, R., and Sterman, M. B. Brain functions. Ann. N.Y. Acad. Sci., 1966, 17, 363-394.
- 120 Hwey, F. Cardiovascular effects of experimental insomnia. Amer. J. Physiol., 1942, 124, 65-70.

- 121 Hinkle, L. E., and Wolff, H. G. Communist interrogation and indoctrination of "enemies of the state." Arch. Neurol. Psychiat., 1956, 76, 115-174.
- 122 Nösl, L., Monnier, M., and Koller, Y. Humoral transmission of sleep and wakefulness. I. Method for dialysing psychotropic humors from the cerebral blood. Pflügers Arch., 1965, 282, 54-59.
- 123 Husband, R. W. The comparative value of continuous versus interrupted sleep. J. Exp. Psychol., 1935, 18, 792-796.
- 124 JAMA Editorials. Sleep deprivation and mental health. J. Amer. Med. Assoc., 1968, 204, 166.
- 125 Jerison, H. J. Activation and long-term performance. Acta Psychol., 1967, 27, 373-389.
- 126 Johnson, L. C. Galvanic skin response after sleep deprivation. Psychol. Rep., 1964, 15, 549.
- 127 Johnson, L. C. Sleep and sleep loss -- their effect on performance. Naval Res. Rev., 1967, August, 16-22.
- 128 Johnson, L. C. Psychological and physiological changes following total sleep deprivation. In Symposium proceedings. Philadelphia: Lippincott, in press.
- 129 Johnson, L. C., Slye, E. S., and Dement, W. Electroencephalographic and autonomic activity after prolonged sleep deprivation. Psychosom. Med., 1965, 27, 415-423.
- 130 Johnson, F., and Webb, W. B. Microsleep responses in the rat. Psychom. Sci., 1965, 3, 499-500.
- 131* Jones, H. S., and Oswald, I. Two cases of healthy insomnia. Electroenceph. Clin. Neurophysiol., 1968, 24, 378-380.
- 132* Jouviet, M. Paradoxical sleep -- A study of its nature and mechanism. In K Akert, C. Bally, and J. P. Slude (Eds.), Sleep mechanisms. Amsterdam: Elsevier, 1966. Pp. 20-62.
- 133* Jouviet, M. Etude de la dualite des etats de sommeil et des mecanismes de la phase paradoxale. In M. Jouviet (Ed.), Aspects anatomo-fonctionnels la physiologie du sommeil. Paris: Centre National de la Recherche Scientifique, 1968. Pp. 397-449.
- 134 Jouviet, M. [Recent findings on experimental insomnias in the cat.] Rev. Neurol., 1966, 118, 454-456.
- 135* Jouviet, M. Neurophysiology of the states of sleep. Physiol. Rev., 1967, 47, 117-177.
- 136* Jouviet, M. Mechanisms of the states of sleep: a neuropharmacological approach. In S. S. Kety, E. V. Everts and M. L. Williams (Eds.), Sleep and altered states of consciousness. Proc. Ass. Res. Nerv. Ment. Dis., 1965. Baltimore: Williams and Wilkins, 1967. Pp. 86-126.
- 137 Jouviet-Monnier, D., Vinont, P., and Delorme, F. [Study of the effects of sleep deprivation in the adult cat.] J. Physiol., 1965, 57, 624-637.
- 138* Kales, A., and Berger, R. J. Psychopathology of sleep. In C. G. Costello (Ed.), Aspects of psychopathology. New York: Wiley, in press.
- 139 Kales, A., Noszka, F. S., Jacobson, A., and Lichtenstein, E. L. Dream deprivation: an experimental reappraisal. Nature, 1964, 204, 1337-1338.
- 140* Kales, A., Jacobson, A., Kales, J. D., Kim, T., and Weisbach, R. All-night EEG sleep measurements in young adults. Psychom. Sci., 1967, 7, 67-68.
- 141 Kales, A., Kales, J. D., Malmstrom, E. J., Tan, T-L., and Allen, C. Drug induced REM deprivation: clinical implications and treatment considerations. Amer. J. Psychiat., in press.
- 142 Kales, A., Tan, T-L., Kollar, E. J., Mitoh, V., Malmstrom, E. J., and Schart, M. S. Sleep patterns following 206 hours of sleep deprivation. Psychosom. Med., in press.
- 143* Kales, A., Wilson, T., Kales, J. D., Jacobson, A., Paulsen, M. J., Kollar, E., and Walter, R. D. Measurements of all-night sleep in normal elderly persons: effects of aging. J. Amer. Geriatrics Soc., 1967, 15, 408-414.
- 144* Kaniya, J. Behavioral, subjective and physiological aspects of drowsiness and sleep. In D. W. Fisher and S. P. Maddi (Eds.), Functions of varied awakenings. Homewood, Ill: Dorsey, 1961. Pp. 149-174.
- 145 Karadic, V., and Dement, W. C. Heart rate changes following selective deprivation of rapid eye movement (REM) sleep. Brain Res., 1967, 9, 796-788.
- 146 Katz, S. E., and Lewis, C. Psychologic and physiologic phenomena during a prolonged vigil. Arch. Neurol. Psychiat., 1935, 34, 307-316.
- 147 Kavamu, J. L. An improved method for deprivation of sleep. J. Biol. Physiol., 1962, 17, 375-377.

- 148 Khazan, N., and Sawyer, C. H. "Rebound" recovery from deprivation of paradoxical sleep in the rabbit. Proc. Soc. Exp. Biol. Med., 1963, 114, 536-539.
- 149 Kiyono, S., Kawamoto, T., Sakakura, H., and Iwama, K. Effects of sleep deprivation upon the paradoxical phase of sleep in cats. Electroenceph. Clin. Neurophysiol., 1965, 19, 34-40.
- 150 Kleitman, N. Studies on physiology of sleep. I. The effects of prolonged sleeplessness on man. Amer. J. Physiol., 1923, 66, 67-92.
- 151 Kleitman, N. Studies on physiology of sleep. V. Some experiments on puppies. Amer. J. Physiol., 1927, 84, 386-395.
- 152* Kleitman, N. Sleep and wakefulness. (2nd ed.) Chicago: University of Chicago Press, 1963.
- 153 Kleitman, N., and Jackson, D. P. Body temperature and performance under different routines. J. Appl. Physiol., 1950, 3, 309-328.
- 154 Kleitman, N., and Schreider, J. Sleepiness and diplopia. Amer. J. Physiol., 1940, 129, 398.
- 155* Koella, W. P. Sleep: Its nature and physiological organization. Springfield, Ill.: Thomas, 1966.
- 156 Koella, W. P., Feldstein, A., and Czicman, J. S. The effect of para-chlorophenylalanine on the sleep of cats. Electroenceph. Clin. Neurophysiol., 1968, 25, 481-490.
- 157 Kollar, E. J., Slater, G. R., Palmer, J. O., Docter, R. F., and Mandell, A. J. Stress in subjects undergoing sleep deprivation. Psychosom. Med., 1966, 28, 101-113.
- 158 Kollar, E. J., Namerow, N., Pasnau, R. O., and Naitoh, P. Neurological findings during prolonged sleep deprivation. Neurology, 1968, 18, 836-840.
- 159 Kollar, E. J., Pasnau, R. O., Rubin, R. T., Naitoh, P., Slater, G., and Kales, A. Psychological, psychophysiological and biochemical correlates of prolonged sleep deprivation. Amer. J. Psychiat., in press.
- 160 Koranyi, E. K., and Lehmann, M. E. Experimental sleep deprivation in schizophrenic patients. Arch. Gen. Psychiat., 1960, 2, 534-544.
- 161* Koreesco, R., Snyder, F., and Feinberg, I. "Dream time" in hallucinating and non-hallucinating schizophrenic patients. Nature, 1963, 192, 1118-1119.
- 162 Kornetsky, C., Mirsky, A. F., Kessler, E. K. and Dorff, J. E. The effects of dextro-amphetamine on behavioral deficits produced by sleep loss in humans. J. Pharmacol. Exp. Therap., 1959, 127, 46-50.
- 163 Kornfield, D. S., Zimberg, S., and Mals, J. R. Psychiatric complications of open-heart surgery. New England J. Med., 1965, 273, 287.
- 164 Kreider, M. B. Effects of sleep deprivation on body temperature. Fed. Proc., 1961, 20, 213.
- 165 Kubie, L. S. The concept of dream deprivation: a critical analysis. Psychosom. Med., 1963, 25, 62-65.
- 166 Kuhn, E., Brodan, V., Brodanova, M., and Kordac, V. [Influence of sleep deprivation on the daily rhythm of plasma iron.] Czech J. Physiol., 1967, 10, 1342-1343.
- 167 Kuhn, E., Brodan, V., Brodanova, M., and Friedman, S. Influence of sleep deprivation on iron metabolism. Science, 1967, 155, 1041-1042.
- 168 Kuhn, E., Hysanek, E., and Brodan, V. [Tryptophan metabolism during sleep deprivation.] Czech J. Physiol., 1968, 107, 424-424.
- 169 Kushanova, O. I., and Lagirov, V. V. [Changes in the ascorbic acid content of the adrenals of rats during experimental insomnia (a histochemical study).] Bull. Exp. Biol. Med., 1968, 68, 50-52.
- 170 Laird, D. A. Effects of loss of sleep on mental work. Indian. Psychol., 1926, 1, 427-428.
- 171 Laird, D. A., and Muller, C. G. Sleep: Why we need it and how to get it. New York: The John Day Co., 1938. Pp. 714.
- 172 Laird, D. A., and Mueller, W. What it costs to lose sleep. Indian. Psychol., 1926, 1, 424-426.
- 173 Laslett, H. R. An experiment on the effects of loss of sleep. J. Exp. Psychol., 1924, 2, 43-54.
- 174 Laslett, H. R. Experiments on the effects of the loss of sleep. J. Exp. Psychol., 1926, 11, 370-396.

- 175 Laties, V. G. The modification of affective response, social behavior and group performance by sleep deprivation and medication. Unpublished doctoral dissertation, University of Rochester, 1954.
- 176 Laties, V. G. Modification of affect, social behavior, and performance by sleep deprivation and drugs. *J. Psychiat. Res.*, 1961, 1, 12-25.
- 177 Loughlin, H. P. Research on sleep deprivation and exhaustion: an invitation to further observation and study. *Intern. Rec. Med. Gen. Pract. Clin.*, 1953, 166, 305-310.
- 178 Leake, C., Grab, J. A., and Senn, M. J. Studies in exhaustion due to lack of sleep. II. Symptomatology in rabbits. *Amer. J. Physiol.*, 1927, 92, 127-130.
- 179 Lee, M. A. M., and Kleitman, N. Studies on the physiology of sleep. II. Attempts to demonstrate functional changes in the nervous system during experimental insomnia. *Amer. J. Physiol.*, 1923, 67, 141-152.
- 180* Legendre, R. The physiology of sleep. *Rep. Smithsonian Inst.*, 1911, 12, 587-602.
- 181* Leonard, J. A. Advance information in sensori-motor skills. *Quart. J. Exp. Psychol.*, 1953, 5, 141-149.
- 182 Levitt, P. A. The sleep need: sleep deprivation in the rat. Unpublished doctoral dissertation, University of Florida, 1965.
- 183 Levitt, R. A. Sleep deprivation in the rat. *Science*, 1966, 152, 85-87.
- 184 Levitt, R. A. Paradoxical sleep: Activation by sleep deprivation. *J. Comp. Physiol. Psychol.*, 1967, 63, 505-509.
- 185 Levitt, R. A., and Webb, W. B. Effect of aspartic acid salts on exhaustion produced by sleep deprivation. *J. Pharmacol. Sci.*, 1964, 53, 1125-1126.
- 186 Lewis, H. E. Sleep patterns on polar expeditions. In G. E. W. Solstenholme and M. O'Connor (Eds.), *Ciba foundation symposium on the nature of sleep*. Boston: Little, Brown and Co., 1961. Pp. 322-328.
- 187 Liberson, W. T. Problems of sleep and mental disease. *Digest Neurol. Psychiat.*, 1945, 13, 93-108.
- 188 Licklider, J. C. R., and Bunch, M. E. Effects of enforced wakefulness upon the growth and maze-learning performance of white rats. *J. Comp. Psychol.*, 1946, 39, 329-350.
- 189 Lindsley, D. B. Psychophysiology and motivation. In M. R. Jones (Ed.), *Nebraska symposium on motivation*. Lincoln, Neb.: University of Nebraska, 1957.
- 190 Loveland, N. T., and Singer, M. T. Projective test assessment of the effects of sleep deprivation. *J. Proj. Tech. Per. Assessm.*, 1959, 23, 323-334.
- 191 Loveland, N. T., and Williams, H. L. Adding, sleep loss, and body temperature. *Percept. Motor Skills*, 1963, 16, 923-929.
- 192 Lubin, A. Performance under sleep loss and fatigue. In S. S. Kety, E. V. Evaris, and H. L. Williams (Eds.), *Sleep and altered states of consciousness*, Proc. Ass. Res. Ner. Ment. Dis., 1965. Baltimore: Williams and Wilkins, 1967. Pp. 506-513.
- 193 Lubin, A., and Williams, H. L. Sleep loss, tremor, and the conceptual reticular formation. *Percept. Motor Skills*, 1959, 9, 237-238.
- 194 Luby, E. D., and Caldwell, D. F. Sleep deprivation and EEG slow wave activity in chronic schizophrenia. *Arch. Gen. Psychiat.*, 1967, 17, 361-364.
- 195 Luby, E. D., Frohman, C. E., Grisell, J. L., Lemzo, J. E., and Gottlieb, J. S. Sleep deprivation: effects on behavior, thinking, motor performance, and biological energy transfer systems. *Psychosom. Med.*, 1960, 22, 182-192.
- 196 Luby, E. D., and Gottlieb, J. S. Sleep deprivation. In S. Arieti (Ed.), *American handbook of psychiatry*, Vol. 3. New York: Basic Books, 1966. Pp. 406-419.
- 197 Luby, E. D., Grisell, J. L., Frohman, C. E., Lees, H., Cohen, B. D., and Gottlieb, J. S. Biochemical, psychological, and behavioral responses to sleep deprivation. *Ann. N.Y. Acad. Sci.*, 1962, 96, 71-79.
- 198 Luby, E. D., Rosenbaum, G., Grisell, J. L., Cohen, B., and Gottlieb, J. S. Relationship between perceptual-motor deficits in sleep deprivation and chronic schizophrenia. In J. Wortis (Ed.), *Recent advances in biological psychiatry*, Vol. 6. New York: Plenum Press, 1963. Pp. 88-95.
- 199* Luce, G. *Current research on sleep and dreams*. U.S. Department of Health, Education and Welfare Publication No. 1389, 1965. Pp. 125.

- 200* Luce, G. G. and Segal, J. *Sleep*. New York: Coward-McCann, 1966.
- 201 Magnussen, G. The sleep function and sleep disturbances. *Ment. Hygiene*, 1953, 37, 89-118.
- 202 Malmö, R. B. Measurement of drive: An unsolved problem in psychology. In M. R. Jones (Ed.), *Nebraska symposium on motivation*. Lincoln, Neb.: University of Nebraska Press, 1958. Pp. 229-265.
- 203 Malmö, R. B., and Surwillo, W. W. Sleep deprivation: Changes in performance and physiological indicants of activation. *Psychol. Monogr.*, 1960, 74, No. 15 (Whole No. 502).
- 204 Malmö, R. B., and Belanger, D. Related physiological and behavioral changes: What are their determinants? In S. S. Kety, E. V. Everts, and H. L. Williams (Eds.), *Sleep and altered states of consciousness*, Proc. Ass. Res. Nerv. Ment. Dis., 1965. Baltimore: Williams and Wilkins, 1967. Pp. 288-313.
- 205* Manacine, A. de *Sleep: Its physiology, pathology, hygiene, and psychology*. London: Walter Scott, Ltd., 1899. Pp. 341.
- 206* Mandell, A. J., Brill, P. L., Mandell, M. P., Rodnick, J., Rubin, R. T., Sheff, R., and Chaffey, B. Urinary excretion of 3-methoxy-4-hydroxy-*o*-arylmandelic acid during dreaming sleep in man. *Life Sci.*, 1966, 5, 169-175.
- 207 Mandell, A. J., Kollar, E. J., and Sabbot, I. M. Starvation, sleep deprivation, and the stress responsive indole substance. In J. Wortis (Ed.), *Recent advances in biological psychiatry*, Vol. 6. New York: Plenum Press, 1963. Pp. 96-104.
- 208 Mandell, A. J., Sabbot, I. M., Mandell, M. P., and Kollar, E. J. The stress responsive substance in sleep deprivation. *Arch. Gen. Psychiat.*, 1964, 10, 299-305.
- 209* Mandell, M. P., Mandell, A. J., Rubin, R. T., Rodnick, J., Brill, P. L., Sheff, R., and Chaffey, B. Activation of the pituitary-adrenal axis during rapid eye movement sleep in man. *Life Sci.*, 1965, 5, 583-588.
- 210 Mangold, R., Sokoloff, L., Corner, E., Kleinsman, J., Therman, P. O. G., and Kety, S. S. The effects of sleep and lack of sleep on cerebral circulation and metabolism of normal young men. *J. Clin. Invest.*, 1955, 34, 1092-1100.
- 211 Matsunaga, H. Experimental studies of the function of concentration maintenance (IAF). 3. Influence of the deprivation of sleep. *Tokushima J. Exp. Med.*, 1965, 12, 71-78.
- 212 Mattson, R. H., Pratt, K. L., and Calverley, J. R. Electroencephalograms of epileptics following sleep deprivation. *Arch. Neurol.*, 1965, 13, 310-315.
- 213* Maulsby, R. L. Electroencephalogram during orbital flight. *Aerospace Med.*, 1966, 37, 1022-1026.
- 214 Meddis, R. Human circadian rhythms and the 48-hour day. *Nature*, 1968, 218, 964-965.
- 215* Medical problems in submarines. In *Submarine medicine practice*. Washington, D.C.: Bureau of Medicine and Surgery, Department of the Navy, 1956. Pp. 293-310.
- 216 Miles, W. R., and Laslett, H. R. Eye movement and visual fixation during profound sleepiness. *Psychol. Rev.*, 1931, 38, 1-13.
- 217 Mirsky, A. F., and Bloch, S. Effects of chlorpromazine, secobarbital and sleep deprivation on attention in monkeys. *Psychopharmacologia*, 1967, 10, 338-339.
- 218 Mirsky, A. F., and Cardon, P. A comparison of the behavioral and physiological changes accompanying sleep deprivation and chlorpromazine administration in man. *Electroenceph. Clin. Neurophysiol.*, 1962, 14, 1-10.
- 219* Monnier, M., and Hölsi, L. Humoral regulation of sleep and wakefulness by hypnogenic and activating dialysable factors. In K. Akert, C. Bally, and J. P. Shade (Eds.), *Sleep mechanisms*. Amsterdam: Elsevier, 1965. Pp. 118-123.
- 220* Monroe, L. J. Psychological and physiological differences between good and poor sleepers. *J. Abnorm. Psychol.*, 1967, 72, 255-264.
- 221 Moore, L. M., Jenkins, M., and Barker, L. Relation of number of hours of sleep to muscular efficiency. *Amer. J. Physiol.*, 1922, 59, 471.
- 222 Morden, B., Conner, R., Mitchell, G., Dement, W., and Levine, S. Effects of rapid eye movement (REM) sleep deprivation on shock-induced fighting. *Physiology and Behavior*, 1968, 3, 425-432.
- 223 Morden, B., Mitchell, G., and Dement, W. Selective REM deprivation and compensation phenomena in the rat. *Brain Res.*, 1967, 5, 339-349.

- 224 Morris, G. O., and Singer, M. T. Sleep deprivation: Transactional and subjective observations. Arch. Gen. Psychiat., 1961, 5, 453-461.
- 225 Morris, G. O., Williams, H. L., and Lubin, A. Misperception and disorientation during sleep deprivation. Arch. Gen. Psychiat., 1960, 2, 247-254.
- 226 Mrsulja, B. B., Bakic, L. M., and Radulovacki, M. The influence of deprivation of paradoxical sleep on glycogen content in various brain structures of the cat. Experientia, 1967, 3, 200-201.
- 227 Murawski, B. J., and Grabbe, J. Effect of sleep deprivation on plasma 17-hydroxycorticosteroids. J. Appl. Physiol., 1960, 15, 280-282.
- 228 Murray, E. J. Conflict and repression during sleep deprivation. J. Abnorm. Soc. Psychol., 1959, 59, 95-101.
- 229 Murray, E. J. Sleep, dreams, and arousal. New York: Appleton-Century-Crofts, 1965. Pp. 407.
- 230 Murray, E. J., Schein, E. H., Erikson, K. J., Hill, W. F., and Cohen, M. The effects of sleep deprivation on social behavior. J. Soc. Psychol., 1959, 49, 229-236.
- 231 Nichols, R. S. The relationship between chronic anxiety level and response to sleep deprivation and medication with secobarbital-amphetamine. Unpublished doctoral dissertation, University of Rochester, 1956.
- 232 Niimi, Y., Watanabe, T., and Mori, T. [Skin potential activities as a function of stages of sleep.] J. Physiol. Soc. Jap., 1968, 30, 231-244.
- 233 Nikaido, T. Chemical changes in brain tissue produced by sleep-deprivation in the rat. Psychiat. Neurol. Jap., 1961, 63, 246-255.
- 234 Okazaki, S. [An experimental study of the lack of sleep.] Shinkei Gaku Zasshi, 1925, 25, 55-100. (Psychol. Abst., 1928, 2, 628.)
- 235 Oller-Daurella, L. [Sleep deprivation as a method of E. E. G. activation in the epileptic.] Rev. Neurol., 1966, 115, 530-535.
- 236 Omsake, K. T. Effect of varying periods of sleep on nervous stability. J. Appl. Psychol., 1932, 16, 623-632.
- 237* Oswald, I. Sleeping and waking. Amsterdam: Elsevier, 1962. Pp. 232.
- 238* Oswald, I., and Priest, R. G. Five weeks to escape the sleeping pill habit. Brit. Med. J., 1965, 2, 1093-1099.
- 239* Oswald, I., and Thacore, V. R. Amphetamine and phemetraxine addiction: physiological abnormalities in the abstinence syndrome. Brit. Med. J., 1963, 2, 427-431.
- 240 Palmer, J. O. Alterations in Rorschach's experience balance under conditions of food and sleep deprivation: a construct validation study. J. Prof. Tech. Per. Assessm., 1963, 27, 208-213.
- 241 Papenheimer, J. R., Miller, T. B., and Goodrich, C. A. Sleep-promoting effects of cerebrospinal fluid from sleep-deprived goats. Proc. Nat. Acad. Sci. USA, 1967, 58, 513-517.
- 242 Pasnau, R. O., Naitoh, P., Stier, S., and Kollar, E. J. The psychological effects of 205 hours of sleep deprivation. Arch. Gen. Psychiat., 1968, 18, 496-505.
- 243 Patrick, G. T. W., and Gilbert, J. A. On the effects of loss of sleep. Psychol. Rev., 1896, 3, 469-483.
- 244 Paul, A. Effects of sleep deprivation on visual function. Aerospace Med., 1965, 36, 617-620.
- 245 Pegram, G. V. Changes in EEG, temperature, and behavior as a function of prolonged sleep deprivation. Unpublished doctoral dissertation, University of New Mexico, 1968.
- 246 Pepler, R. D. Environmental warmth and performance. Unpublished doctoral dissertation, University of Cambridge, England, 1956.
- 247 Pepler, R. D. Warmth and lack of sleep: accuracy or activity reduced. J. Comp. Physiol. Psychol., 1959, 52, 446-450.
- 248* Pieron, H. Le probleme physiologique du sommeil. Paris: Masson & Cie, 1913. Pp. 520.
- 249 Pivik, T., and Foulkes, D. Dream deprivation: Effects on dream content. Science, 1966, 153, 1282-1284.
- 250 Pokrovskii, A. A., Malakhov, I. E., and Kushmanova, O. D. [A method of producing sustained insomnia in small animals.] Bull. Exp. Biol. Med., 1967, 63, 123-125.

- 251 Pokrovskii, A. A., Malakhov, I. E., Kon'IIA, and Gapparov, NM-G. [Gamma-amino butyric acid and monoamine oxidate in the rat brain during experimental sleep deprivation.] Ukr. Biochem. J., 1967, 39, 604-607.
- 252 Pokrovskii, A. A. and Shaternikova, I. S. [Changes in certain aspects of lipid metabolism in rats with experimental insomnia.] Bull. Exp. Biol. Med., 1967, 64, 24-27.
- 253 Pratt, K. L., Mattson, R. H., Weikers, N. J., and Williams, R. EEG activation of epileptics following sleep deprivation - a prospective study of 114 cases. Electroenceph. Clin. Neurophysiol., 1967, 24, 11-15.
- 254 Pressey, A. W., and Kelm, H. Effects of sleep deprivation on a visual figural after-effect. Percept. Motor Skills, 1966, 23, 795-800.
- 255 Pujol, J-F., Mouret, J., Jouvret, M., and Glowinski, J. Increased turnover of cerebral norepinephrine during rebound of paradoxical sleep in the rat. Science, 1968, 159, 112-113.
- 256 Pujol, J-F., Mery, F., Durand, M., and Glowinski, J. [Increase in serotonin synthesis in the brainstem of the rat after selective augmentation of paradoxical sleep.] C. R. Acad. Sci., 1968, 267, 371-372.
- 257 Rakestraw, N. W., and Whittier, F. O. The effect of loss of sleep on the composition of the blood and urine. Proc. Soc. Exp. Biol. Med., 1923, 21, 5-6.
- 258* Ray, J. T., Martin, O. E., Jr., and Alluisi, E. A. Human performance as a function of selected studies. National Academy of Sciences - National Research Council Publication No. 882, 1961. Pp. 32.
- 259* Rechtschaffen, A., and Kales, A. (Eds.) A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects. Public Health Service, U.S. Government Printing Office, Washington, D.C., 1968. (NIH Publication No. 204)
- 260* Rechtschaffen, A., and Maron, L. The effect of amphetamine on the sleep cycle. Electroenceph. Clin. Neurophysiol., 1964, 16, 438-445.
- 261 Robinson, E. S., and Hermann, S. O. Effects of loss of sleep. I. J. Exp. Psychol., 1922, 5, 19-32.
- 262 Robinson, E. S., and Richardson-Robinson, F. Effects of loss of sleep. II. J. Exp. Psychol., 1922, 5, 93-100.
- 263 Rodin, E. A., Luby, E. D., and Gottlieb, J. S. The electroencephalogram during prolonged experimental sleep deprivation. Electroenceph. Clin. Neurophysiol., 1962, 14, 544-551.
- 264* Roffwarg, H. P., Dement, W. C., and Fisher, C. Preliminary observations of the sleep-dream pattern in neonates, infants, children and adults. In E. Harms (Ed.), Monographs on child psychiatry, No. 2. New York: Pergamon Press, 1964. Pp. 60-72.
- 265* Roffwarg, H. P., Muzio, J. N., and Dement, W. C. Ontogenetic development of the human sleep-dream cycle. Science, 1966, 152, 604-619.
- 266 Ross, J. J. Neurological findings after prolonged sleep deprivation. Arch. Neurol., 1965, 12, 399-403.
- 267* Rosvold, H. E., Mirsky, A. F., Sarason, I., Bransome, E. D., Jr., and Beck, L. H. A continuous performance test of brain damage. J. Counsel. Psychol., 1956, 20, 343-350.
- 268 Rothman, T., Goodman, J., and Tyler, D. B. Studies on experimental insomnia: Electroencephalographic changes during 112 hours of wakefulness. Trans. Amer. Neurol. Assoc., 1946, 71, 173-174.
- 269* Routtenberg, A. Neural mechanisms of sleep: Changing view of reticular formation function. Psychol. Rev., 1966, 73, 481-499.
- 270 Rubin, R. T., Kollar, E. J., Slater, G. G., and Clark, B. R. 17-hydroxycorticosteroid and vanillylmandelic acid excretion during 205 hours of sleep deprivation in man. Psychosom. Med., in press.
- 271 Rust, L. D. Changes in bar pressing performance and heart rate in sleep-deprived rats. J. Comp. Physiol. Psychol., 1962, 55, 621-625.
- 272 Sampson, H. Deprivation of dreaming sleep by two methods. I. Compensatory REM time. Arch. Gen. Psychiat., 1965, 13, 79-86.
- 273 Sampson, H. Psychological effects of deprivation of dreaming sleep. J. Nerv. Ment. Dis., 1966, 143, 305-317.
- 274* Sargant, W. Battle of the mind. New York: Doubleday, 1957. Pp. 263.

- 275 Schein, E. H. The effects of sleep deprivation on performance in a simulated communication task. J. Appl. Psychol., 1957, 41, 247-252.
- 276 Scholander, I. The effects of moderate sleep deprivation on the habituation of autonomic response elements. Acta Physiol. Scand., 1961, 51, 325-342.
- 277 Schulz, H., Meyer, A-E., and Cohen, R. [Experimental dream deprivation and psychic changes.] Nervenarzt, 1968, 39, 193-198.
- 278 Scrimshaw, N. S., Habicht, J. P., Pellet, P., Piche, M. L., and Sholakov, B. Effects of sleep deprivation and reversal of diurnal activity on protein metabolism of young men. Amer. J. Clin. Nut., 1966, 19, 313-319.
- 279 Sears, R. R., Hovland, C. I., and Miller, N. E. Minor studies of aggression. I. Measurement of aggressive behavior. J. Psychol., 1940, 2, 277-281.
- 280 Seymour, J. H. Some changes in psychometric, perceptual and motor performance as a function of sleep deprivation. Unpublished doctoral dissertation, New York University, 1956.
- 281 Shurley, J. T. Hallucinations in sensory deprivation and sleep deprivation. In L. J. West (Ed.), Hallucinations. New York: Grune & Stratton, 1962. Pp. 87-91.
- 282 Siegel, J., and Gordon, T. Paradoxical sleep: deprivation in the cat. Science, 1965, 148, 978-979.
- 283 Simon, C. W. Some immediate effects of drowsiness and sleep on normal human performance. Human Factors, 1961, 3, 1-17.
- 284* Sleep, wakefulness, dreams and memory. Neurosciences Research Program Bulletin, 1966, Vol. 4, No. 1. Pp. 103.
- 285 Smith, May. A contribution to the study of fatigue. Brit. J. Psychol., 1916, 8, 327-350.
- 286* Snyder, F. Progress in the new biology of dreaming. Amer. J. Psychiat., 1965, 122, 377-391.
- 287* Snyder, F., and Scott, J. Psychophysiology of sleep. In N. Greenfield and R. Sternbach (Eds.), Handbook of psychophysiology. New York: Hold, Rinehart and Winston, in press.
- 288 Svorad, D. Influence of sleep deprivation on hippocampal theta activity. In Proceedings of International Congress of Electroencephalography and Clinical Neurophysiology. Vienna: Wiener Med. Akad., 1965. Pp. 159-161.
- 289 Svorad, D., and Kohout, L. Eine methode der Ausloesung experimenteller Schlafloesigkeit auf Grund bedingter Schutzreaktion. Z. Ges. Exp. Med., 1959, 132, 342-345.
- 290 Svorad, D., and Novikova, V. [Effect of sleep deprivation on sleep and wakefulness rhythm.] Cesk Physiol., 1960, 9, 53-54.
- 291 Svorad, D., and Novikova, V. [The effect of experimentally induced insomnia on the neurotic state of rats.] Physiol. J. USSR, 1960, 46, 57-63.
- 292 Todd, C. E. A description and evaluation of electroencephalographic analysis techniques. Tech. Rep. MDC-TR-67-106, Air Force Missile Development Center, Holloman AFB, New Mexico, 1967.
- 293* Trumbull, R. Diurnal cycles and work-rest scheduling in unusual environments. Human Factors, 1966, 8, 385-398.
- 294 Tucker, R. P. A review of the effects of sleep deprivation. Univ. Michigan Med. Cent. J., 1968, 34, 161-164.
- 295 Tyler, D. B. The effect of amphetamine sulfate and some barbiturates on the fatigue produced by prolonged wakefulness. Amer. J. Physiol., 1947, 150, 253-262.
- 296 Tyler, D. B. Psychological changes during experimental sleep deprivation. Dis. Nerv. Syst., 1955, 16, 293-299.
- 297 Tyler, D. B., Goodman, J., and Rothman, T. The effect of experimental insomnia on the rate of potential changes in the brain. Amer. J. Physiol., 1947, 149, 185-193.
- 298 Tyler, D. B., Marx, W., and Goodman, J. Effect of prolonged wakefulness on the urinary excretion of 17-ketosteroids. Proc. Soc. Exp. Biol. Med., 1946, 62, 38-40.
- 299 Ukolova, M. A. [Experimental neurosis evoked by sleep deprivation.] Bull. Exp. Biol. Med., 1959, 47, 43-46.
- 300 Van Ormer, E. B. Sleep and retention. Psychol. Bull., 1933, 50, 415-439.

- 301 Vimont-Vicary, P., Jouvret-Maurier, D., and Delmore, F. Effects EEG et comportementaux des privations de sommeil paradoxal chez le chat. Electroenceph. Clin. Neurophysiol., 1966, 20, 439-449.
- 302 Vogel, G. W. REM deprivation. III. Dreaming and psychosis. Arch Gen. Psychiat., 1968, 18, 312-329.
- 303 Vogel, G. W., and Traub, A. C. REM deprivation. I. The effect on schizophrenia patients. Arch. Gen. Psychiat., 1968, 18, 287-300.
- 304 Vogel, G. W., Traub, A. C., Ben-Horin, P., and Meyers, G. M. REM deprivation. II. The effects on depressed patients. Arch. Gen. Psychiat., 1968, 18, 301-311.
- 305 Vojtechovsky, M., and Skala, J. Experimental psychoses induced by sleep deprivation and hallucinogenic drugs in abstaining alcoholic patients. In Proceedings of the 28th International Congress on Alcohol and Alcoholism, Vol. 1, Abstracts. Washington, D.C., P. 12.
- 306 Warren, N., and Clark, B. Blocking in mental and motor tasks during a 65-hour vigil. J. Exp. Psychol., 1937, 21, 97-105.
- 307 Webb, W. B. Antecedents of sleep. J. Exp. Psychol., 1957, 53, 162-166.
- 308 Webb, W. B. Some effects of prolonged sleep deprivation on the hooded rat. J. Comp. Physiol. Psychol., 1962, 55, 791-793.
- 309 Webb, W. B., and Agnew, H. W., Jr. Sleep deprivation, age, and exhaustion time in the rat. Science, 1962, 136, 1122.
- 310 Webb, W. B., and Agnew, H. W., Jr. Sleep: Effects of a restricted regime. Science, 1965, 150, 1745-1747.
- 311* Webb, W. B., and Agnew, H. W., Jr. Sleep cycling within twenty-four hour periods. J. Exp. Psychol., 1967, 74, 158-160.
- 312 Weiskotten, T. F. On the effects of loss of sleep. J. Exp. Psychol., 1925, 8, 363-380.
- 313 Weiskotten, T. F., and Ferguson, J. E. A further study of the effects of loss of sleep. J. Exp. Psychol., 1930, 13, 247-266.
- 314 Wellnerova, J., and Svorad, D. [Sleep deficiency (debt); its measurement and influence.] Cesk Physiol., 1959, 8, 136-137.
- 315 West, L. J. Psychopathology produced by sleep deprivation. In S. S. Kety, E. V. Evarts, and M. L. Williams (Eds.), Sleep and altered states of consciousness. Proc. Ass. Res. Nerv. Ment. Dis., 1965. Baltimore: Williams and Wilkins, 1967. Pp. 506-513.
- 316 West, L. J., Janszen, H. H., Lester, B. L., and Cornelisoon, F. S. The psychosis of sleep deprivation. Ann. N.Y. Acad. Sci., 1962, 96, 66-70.
- 317 Wilkinson, R. T. The effects of lack of sleep on perception and skill. Unpublished doctoral dissertation, University of Cambridge, England, 1957.
- 318 Wilkinson, R. F. The effects of sleep loss on performance. Report No. 323, Applied Psychology Research Unit, Medical Research Council, Cambridge, England, 1958. Pp. 36.
- 319 Wilkinson, R. T. Rest pauses in a task affected by lack of sleep. Ergonomics, 1959, 2, 373-380.
- 320 Wilkinson, R. T. The effect of lack of sleep on visual watch-keeping. Quart. J. Exp. Psychol., 1960, 12, 36-40.
- 321 Wilkinson, R. T. Interaction of lack of sleep with knowledge of results, repeated testing, and individual differences. J. Exp. Psychol., 1961, 62, 263-271.
- 322 Wilkinson, R. T. Muscle tension during mental task under sleep deprivation. J. Exp. Psychol., 1962, 64, 565-571.
- 323 Wilkinson, R. T. Interaction of noise with knowledge of results and sleep deprivation. J. Exp. Psychol., 1963a, 66, 332-337.
- 324 Wilkinson, R. T. After-effects of sleep deprivation. J. Exp. Psychol., 1963b, 66, 439-442.
- 325 Wilkinson, R. T. Effects of sleep deprivation on performance and muscle tension. In G. E. W. Wolstenholme and M. O'Conner (Eds.), CIBA Foundation symposium on the nature of sleep. Boston: Little, Brown and Co., 1963c. Pp. 329-342.
- 326 Wilkinson, R. T. Effect of up to 60 hours of sleep deprivation on different types of work. Ergonomics, 1964a, 7, 175-186.

- 327 Wilkinson, R. T. Sleep deprivation. Acta Psychol., 1964b, 23, 276-277.
- 328 Wilkinson, R. T. Sleep deprivation. In O. G. Edholm and A. L. Bacharach (Eds.), The physiology of human survival. New York: Academic Press, 1965. Pp. 399-430.
- 329 Wilkinson, R. T. Sleep deprivation: performance tests for partial and selective sleep deprivation. In L. A. Abt and B. F. Riess (Eds.), Progress in clinical psychology. Vol. 8. New York: Grune and Stratton, 1968. Pp. 28-43.
- 330 Wilkinson, R. T., and Colquhoun, W. P. Interaction of alcohol with incentive and with sleep deprivation. J. Exp. Psychol., 1968, 76, 623-629.
- 331 Wilkinson, R. T., Edwards, R. S., and Haines, E. Performance following a night of reduced sleep. Psychon. Sci., 1966, 5, 471-472.
- 332 Williams, H. L. Decrement in performance due to sleep deprivation. In Symposium on medical aspects of stress in military climate. Washington, D.C.: Walter Reed Army Institute of Research, 1964a. Pp. 187-202.
- 333 Williams, H. L. Sleep starvation and you. Army Information Digest, 1964, June, 11-18.
- 334 Williams, H. L. States produced by manipulating environmental factors. In Sleep, wakefulness, dreams and memory. Neurosciences Research Program Bulletin. Vol. 4. 1968. Pp. 63-65.
- 335 Williams, H. L., Gieseck, C. F., and Lubin, A. Some effects of sleep loss on memory. Percept. Motor Skills, 1966, 23, 1287-1293.
- 336 Williams, H. L., Granda, A. M., Jones, R. C., Lubin, A., and Armington, J. C. EEG frequency and finger pulse volume as predictors of reaction time during sleep loss. Electroenceph. Clin. Neurophysiol., 1962, 14, 64-70.
- 337 Williams, H. L., Hammack, J., Daly, R., Dement, W., and Lubin, A. Responses to auditory stimulation, sleep loss and the EEG stages of sleep. Electroenceph. Clin. Neurophysiol., 1964, 16, 269-279.
- 338 Williams, H. L., Kearney, O. F., and Lubin, A. Signal uncertainty and sleep loss. J. Exp. Psychol., 1965, 69, 401-407.
- 339 Williams, H. L., and Lubin, A. Effects of acute sleep loss on performance. A talk given to Neuropsychiatry Division, Walter Reed Army Institute of Research, 8 January 1958. (Mimeographed paper)
- 340 Williams, H. L., and Lubin, A. Speeded addition and sleep loss. J. Exp. Psychol., 1967, 73, 313-317.
- 341 Williams, H. L., Lubin, A., and Goodnow, J. J. Impaired performance with acute sleep loss. Psychol. Monogr., 1959, 73, No. 14 (Whole No. 484). Pp. 26.
- 342 Williams, H. L., Morris, G. O., and Lubin, A. Illusions, hallucinations and sleep loss. In L. J. West (Ed.), Hallucinations. New York: Grune and Stratton, 1962. Pp. 158-165.
- 343 Williams, H. L., and Williams, C. L. Nocturnal EEG profiles and performance. Psychophysiology, 1966, 2, 164-175.
- 344 Williams, M., and Burge, W. E. The effect of sleeplessness on brain potential. Fed. Proc., 1945, 4, 77.
- 345* Williams, R. L., Agnew, H. W., Jr., and Webb, W. B. Sleep patterns in young adults: An EEG study. Electroenceph. Clin. Neurophysiol., 1964, 17, 376-381.
- 346* Williams, R. L., Agnew, H. W., Jr., and Webb, W. B. Sleep patterns in the young adult female: An EEG study. Electroenceph. Clin. Neurophysiol., 1966, 20, 264-266.
- 347 Williams, R. L., Agnew, H. W., Jr., and Webb, W. B. Effects of prolonged stage four and 1-REM deprivation. EEG, task performance, and psychological responses. Tech. Rep. SAM-TR-67-59, USAF School of Aerospace Medicine, Brooks Air Force Base, Texas, 1967. Pp. 10.
- 348* Yules, R., Freedman, D., and Chandler, K. The effect of ethyl alcohol on men's electroencephalographic sleep cycle. Electroenceph. Clin. Neurophysiol., 1966, 20, 109-111.
- 349* Yules, R. B., Ogden, J. A., Gault, F. P., and Freedman, D. X. The effect of ethyl alcohol on electroencephalographic sleep cycle in cats. Psychon. Sci., 1966, 5, 97-98.
- 350 Yules, R. B., Lippman, M. E., and Freedman, D. X. Alcohol administration prior to sleep - the effect on EEG sleep stages. Arch. Gen. Psychiat., 1967, 16, 94-97.
- 351 Zarcone, V., Gulevich, G., Pivik, T., and Dement, W. Partial REM phase deprivation and schizophrenia. Arch. Gen. Psychiat., 1968, 19, 194-202.

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LEGENDS FOR FIGURES

- Figure 1. Electroencephalographic (EEG) and electrocardiographic (EKG) records of a subject who developed visual hallucination during sleep loss. Before an episode of visual hallucination, EEG showed low voltage waves of mixed frequencies. A period of hallucination was accompanied by sustained high voltage alpha wave in EEG and increased heart rate.
- Figure 2. Unidimensional manual tracking of visual stimulus in 4 subjects, R. S., H. H., D. C., and J. L. Three channels on the top of the figure show sinusoidal tracking signal, tracking response, and tracking error of a subject, H. H., at 123rd hour of sleep loss. The lower graphs show the tracking errors only on 4 subjects. The more wavy the lines are, the more inaccuracies are present in tracking. R1 and R2 are the tracking responses after one and two night(s) of recovery sleep.
- Figure 3. Results of an immediate recall test before, during, and after 64 hours of total sleep deprivation followed by selective sleep stage deprivation. C denotes Control Group for selective sleep deprivation (N = 4). S denotes Slow Wave Sleep Deprived Group (N = 3). R denotes REM Sleep Deprived Group (N = 3). Perfect score for the task was 30 words recalled. B denotes Baseline Days, 1 through 4; D denotes Deprivation Days, 1 and 2; PR denotes Partial Recovery Days, 1 and 2; R denotes Recovery Days, 3 and 4.
- Figure 4. Results of task of adding five two-digit columns of numbers. C is Control (N = 4); S is Slow Wave Deprived (N = 3); R is REM Sleep Deprived (N = 4), and A is the No Sleep Deprived Group (N = 4).
- Figure 5. This is a polygraphic record showing EEG and autonomic concomitants of performing a task, Plus 7, before the deprivation of sleep. This record was taken from a subject, J. Y., 5 minutes after the start of the Plus 7 Task. TASK channel indicated smooth and rapid mental addition with the subject completing six correct additions in 30 seconds. EEG showed high amplitude alpha activity. Finger pulse volume was relatively small and well regulated. Respiration was deep. HR is Heart Rate; SP is Skin Potential.
- Figure 6. This is a polygraphic record of a subject, J. Y., 5 minutes after the start of the Plus 7 task but with an intervening sleep loss of two nights. TASK polygraph channel shows accurate mental additions but with a considerable slowing in addition. The subject completed only three additions in 30 seconds. EEG alpha was attenuated and intermittent. Heart rate was slowed and finger pulse volume was larger. Respiration was shallower.

- Figure 7. This shows the results of the Plus 7 Task performance. C denotes Control Group (N = 3); S denotes Slow Wave Sleep Deprived (N = 3); W denotes REM Sleep Deprived (N = 4); A denotes the No Sleep Deprived Group (N = 2). The task was 50 minutes long but this figure shows only the average performance during three periods (Minutes 1-10, 21-30, and 41-50).
- Figure 8. This figure shows the results of performance on the X crossout test. C denotes the Control Group (N = 4); S denotes Slow Wave Deprived Group (N = 3); R denotes the REM Sleep Deprived Group (N = 4); and A denotes No Sleep Deprived Group (N = 4).
- Figure 9. This figure shows the results of performance of Continuous Counting. C is Control Group (N = 4); S is Slow Wave Deprived Group (N = 3); R is REM Deprived Group (N = 4); and A is No Sleep Deprived Group (N = 4).
- Figure 10. These are the results of performance of auditory vigilance. C is Control Group (N = 4); S is Slow Wave Deprived Group (N = 3), and R is REM Deprived Group (N = 4).
- Figure 11. This figure shows the Stages of Sleep, 1, 2, 3, 4, and REM, plus W, the Wake Period. Calibrations for heart rate and for EEG appear at the lower bottom of the figure. LEOG is Left Electrooculogram; REOG is Right Electrooculogram. A is the reference created by connecting left and right mastoid processes.
- Figure 12. This shows the sleep cycles of two subjects, J. C. V. and H. L. C. The stages of sleep are shown on the vertical axis. W is Wake Period, R is REM Period. The horizontal axis shows the hours of sleep and also local clock time.
- Figure 13. This figure shows the physiological indicators used in the Stage REM sleep deprivation. LEOG and REOG are Left and Right Electrooculograms. EMG is Electromyogram taken on and beneath the muscular area of the chin. F3, C2, and J1 are EEG derivations after the 10-20 system. SP is Skin Potential; HR is Heart Rate; FP is Finger Pulse volume. A is the reference formed by interconnecting left and right mastoid processes. In REM Deprived Sleep, the subject was awakened at the time when chin EMG tones disappeared (4 dots), or at the beginning of the train of rapid eye movements (6 dots).
- Figure 14. These are the sleep profiles of a subject in 3rd baseline sleep, the second night of slow wave sleep deprivation and the first night of recovery sleep following two nights of total sleep loss and two nights of slow wave sleep deprivation.

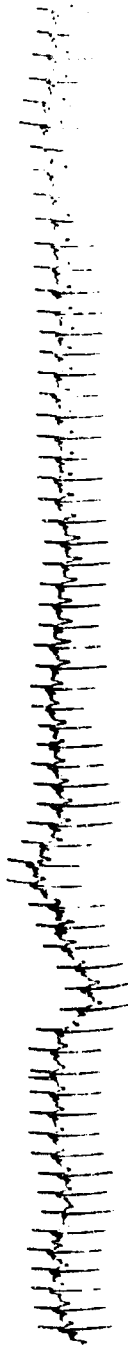
PARIETAL - OCCIPITAL EEG AND EKG OF SUBJECT R.S. BEFORE
AND DURING HALLUCINATORY EPISODE AT 170 HOURS
SLEEP DEPRIVATION

BEFORE

EEG



EKG



DURING

EEG



EKG



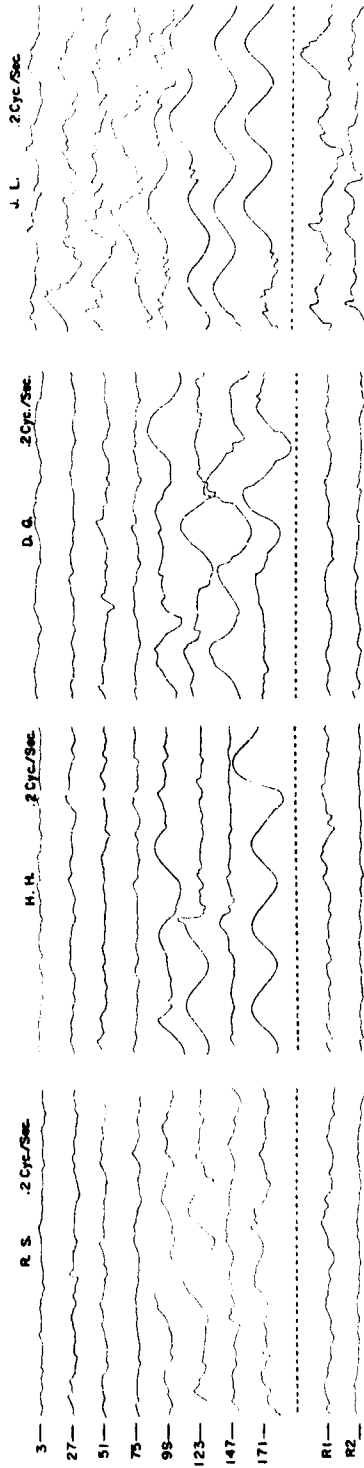
TRACKING SIGNAL (.2 Cye./Sec.)

TRACKING RESPONSE (H.H./23 Hz)

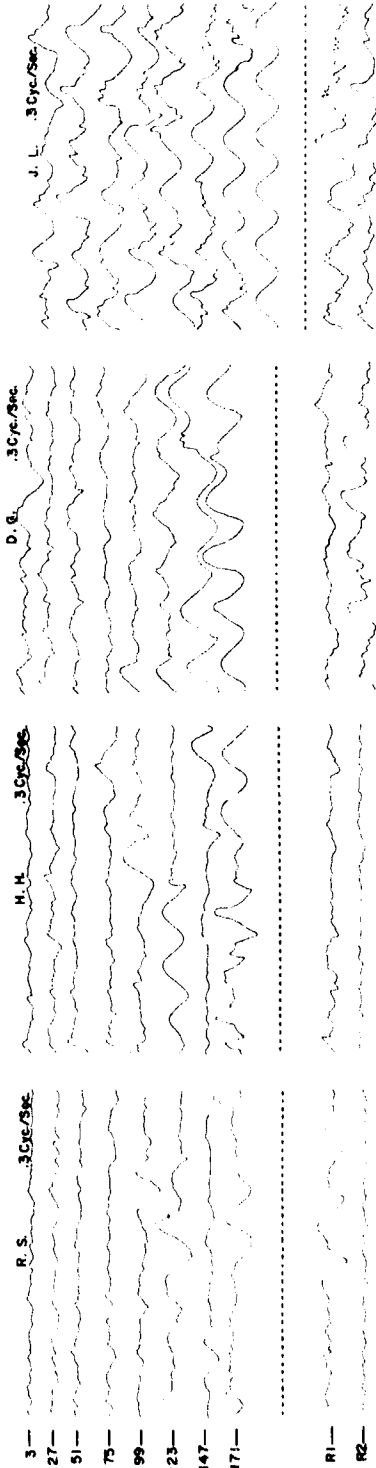
TRACKING ERROR

5 SECONDS

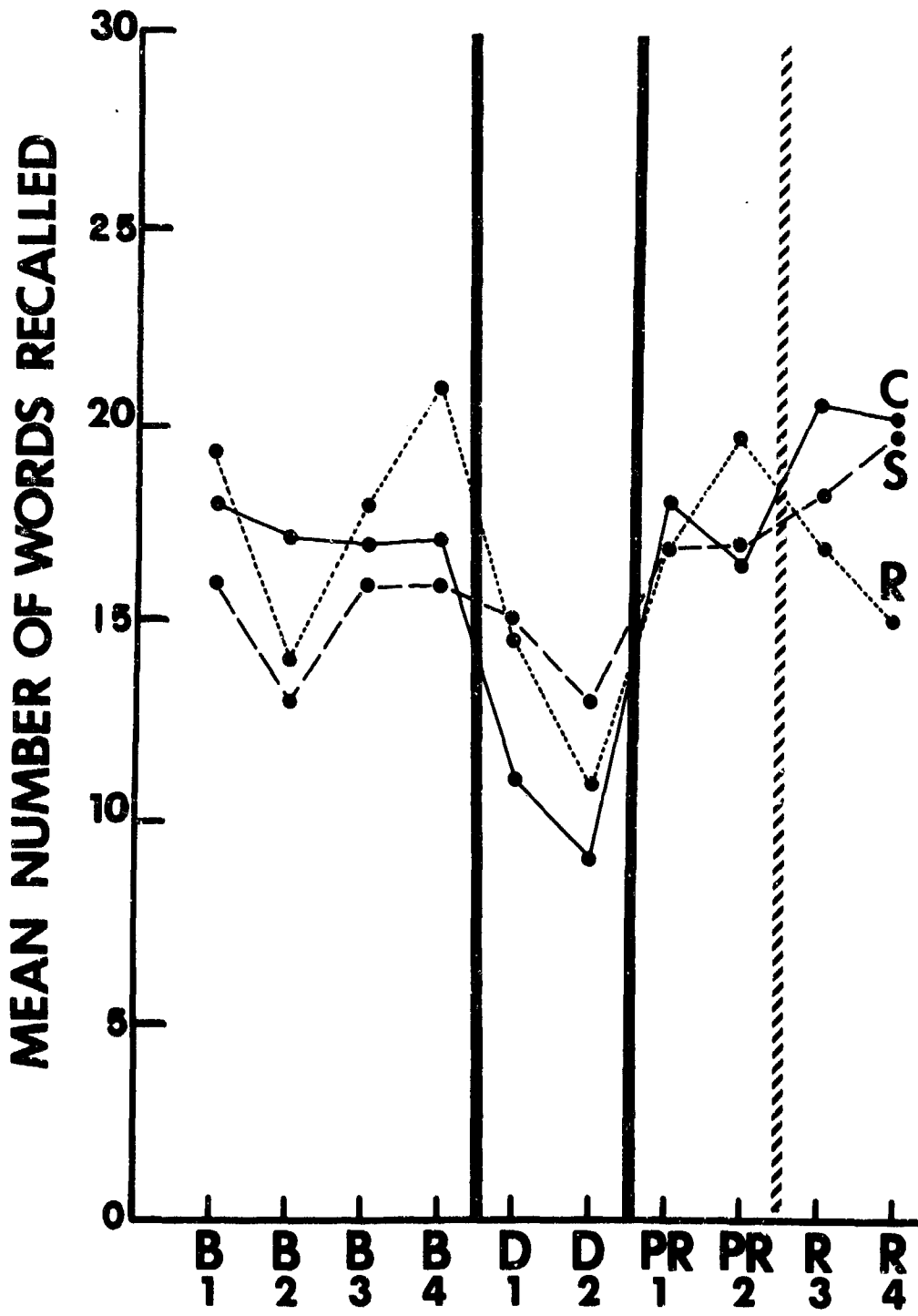
HOURS OF SLEEP DEPRIVATION
(9 A.M. SESSION)



HOURS OF SLEEP DEPRIVATION
(9 A.M. SESSION)

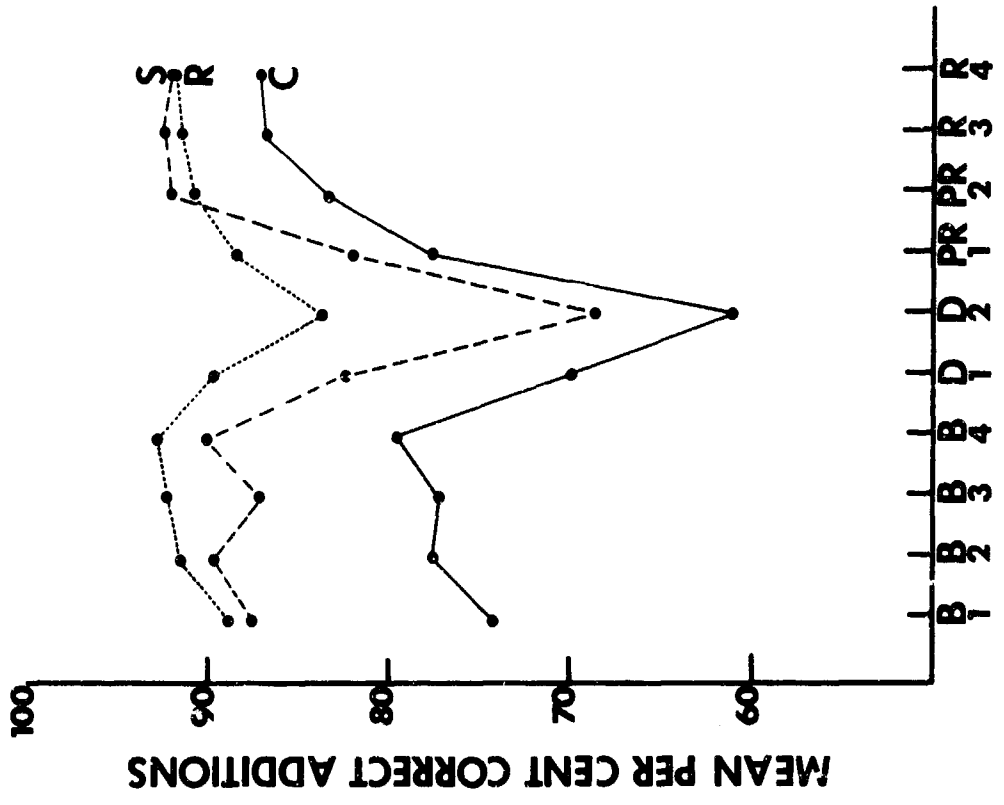
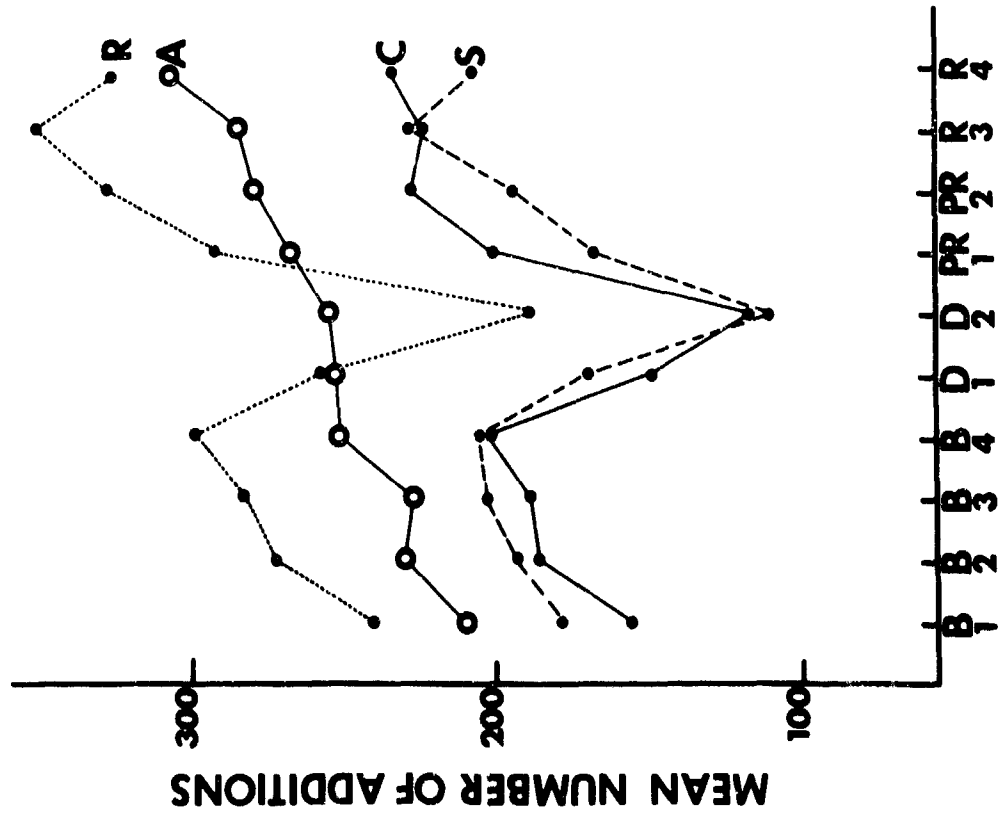


WILLIAMS WORD MEMORY



WILKINSON ADDITION

60 MIN. TASK



PLUS 7

B4:JY
5min

Cz-A2



O1-A2

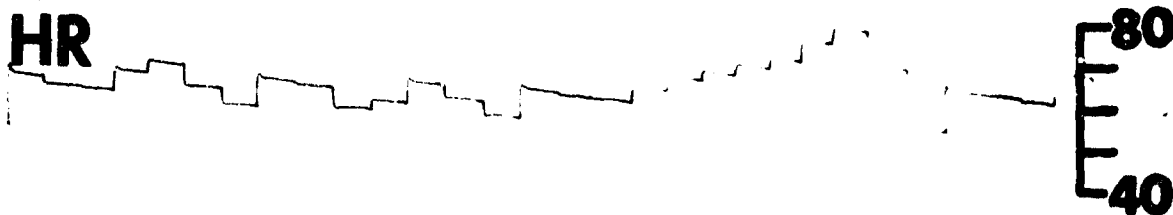


50µV
1sec

SP



HR



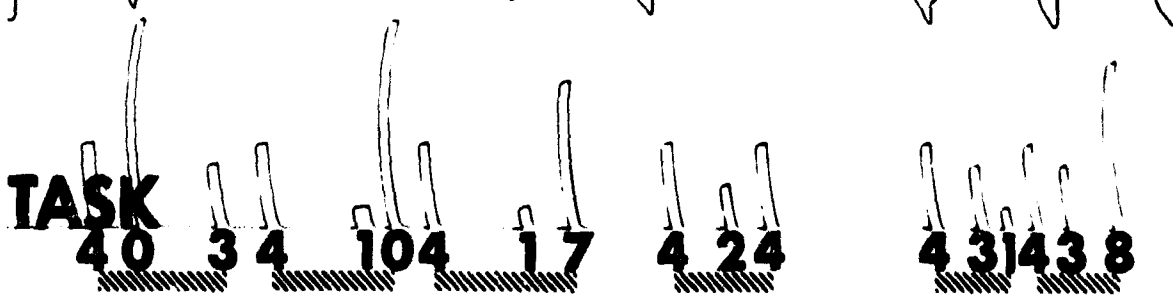
FP



Resp



TASK



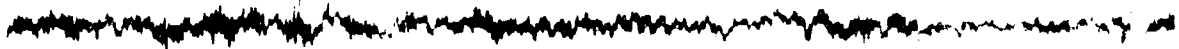
PLUS 7

D2:JY
5min

Cz-A2



O1-A2



50µV
1sec

SP



HR



80
40

FP



Resp

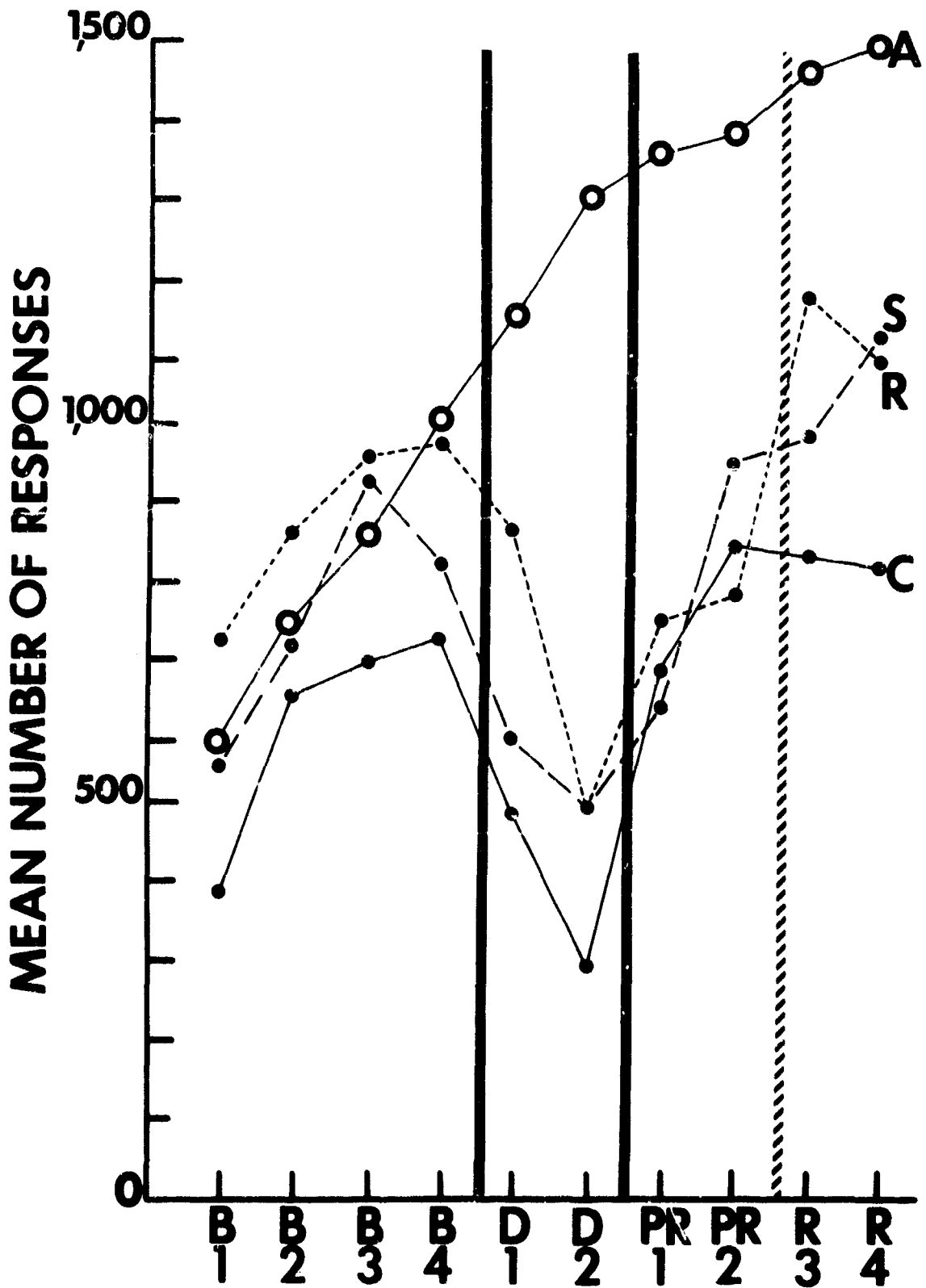


TASK

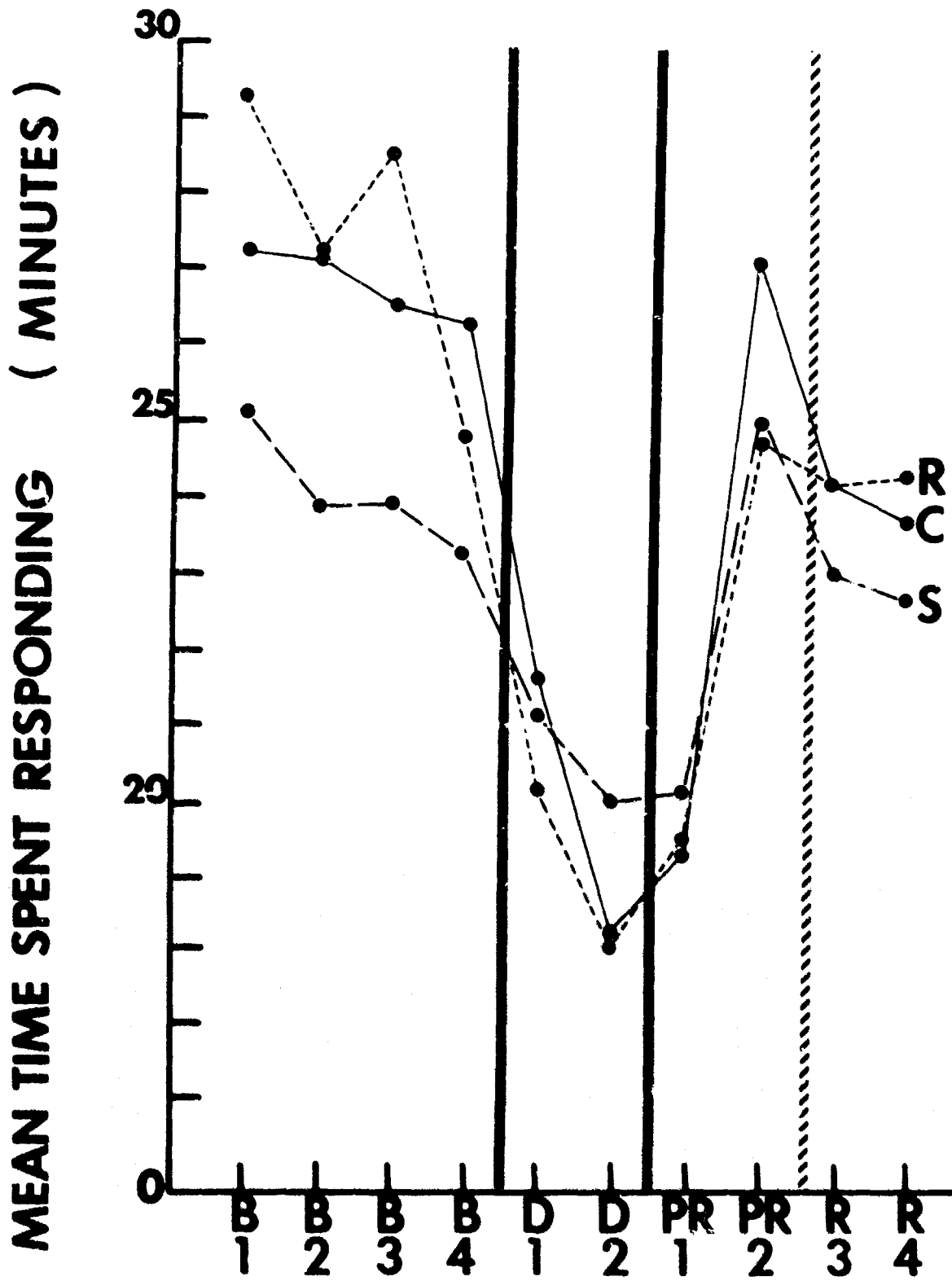


4 0 7 4 1 4 4 21

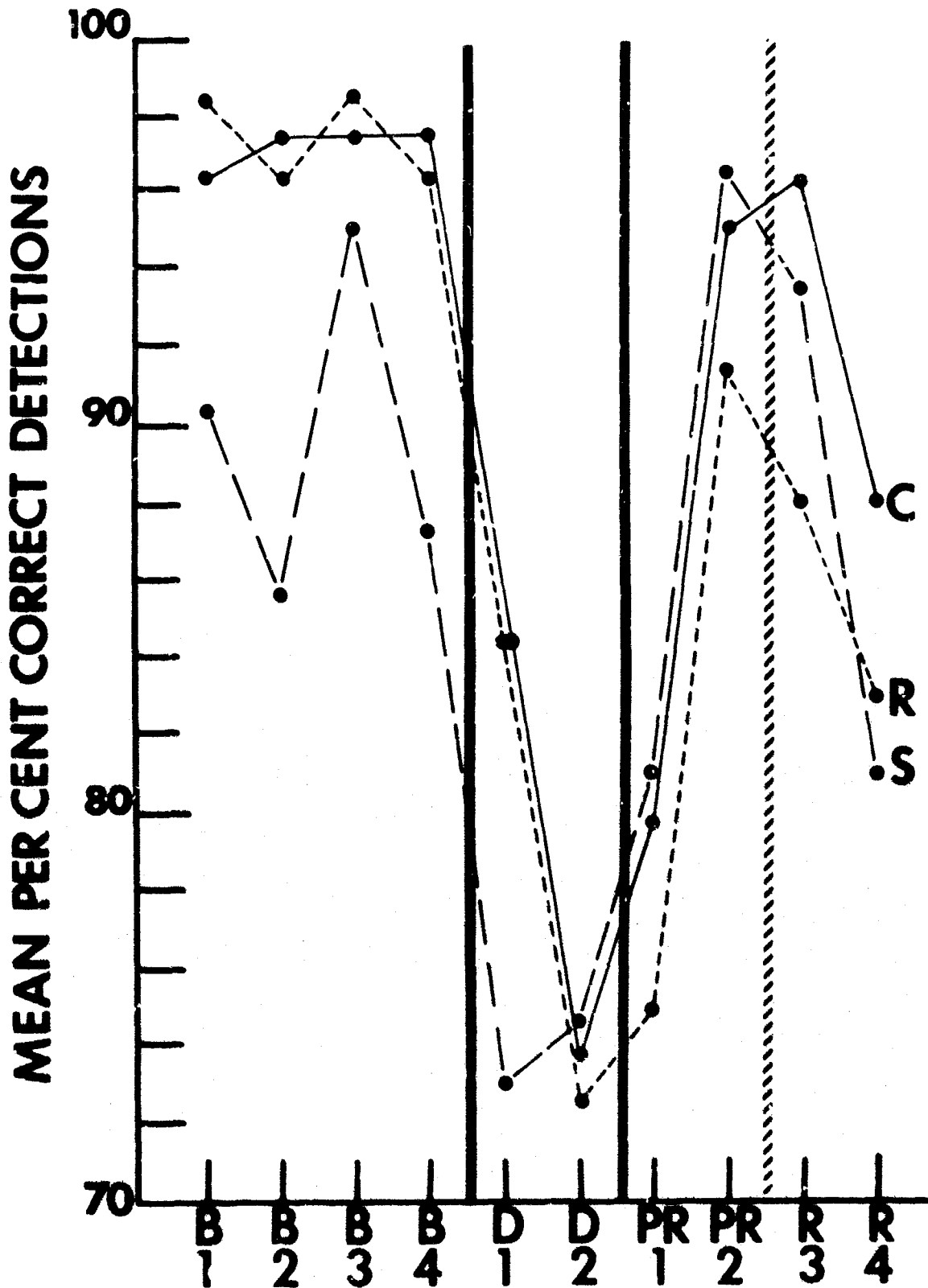
PLUS SEVEN



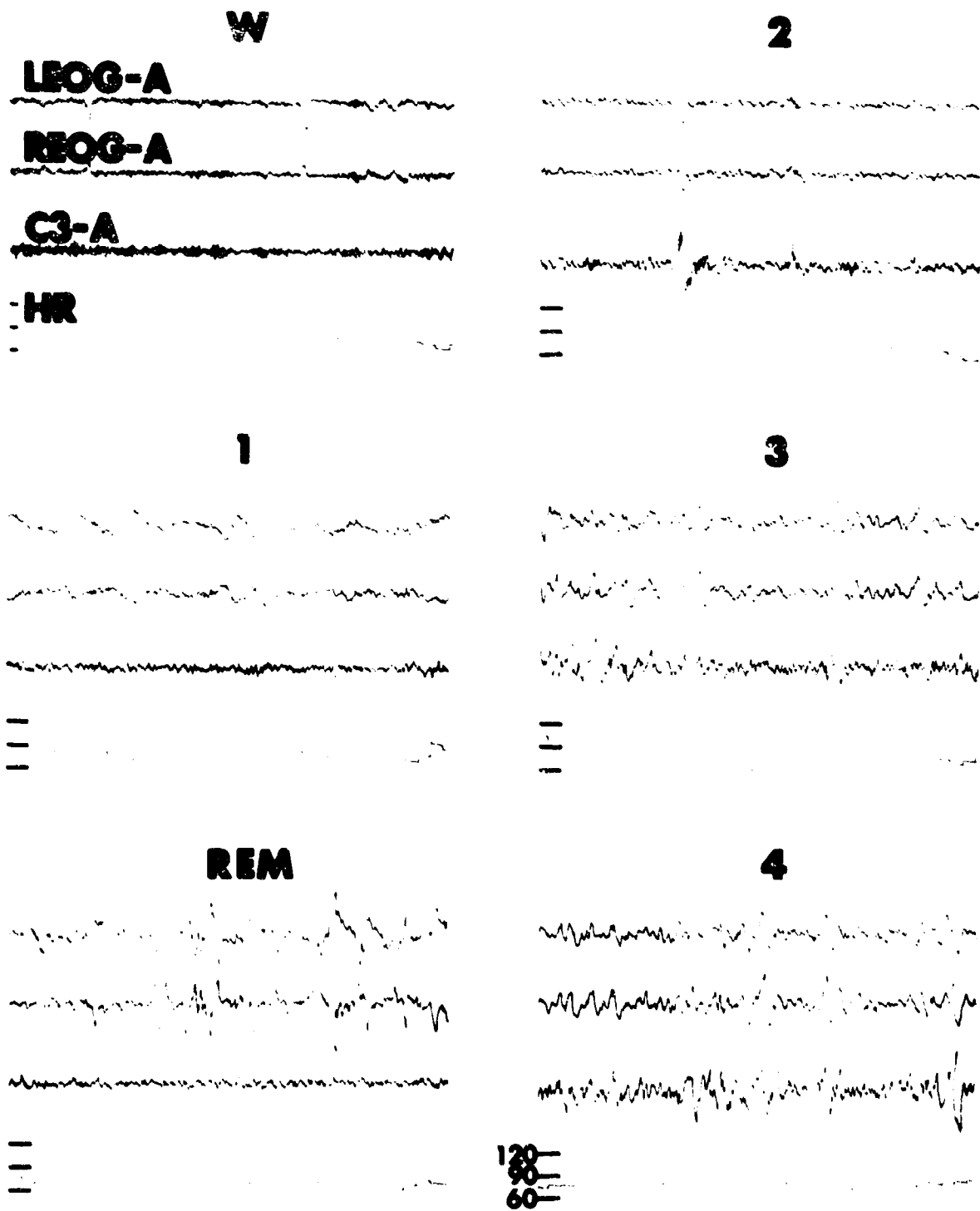
CONTINUOUS COUNTING



AUDITORY VIGILANCE



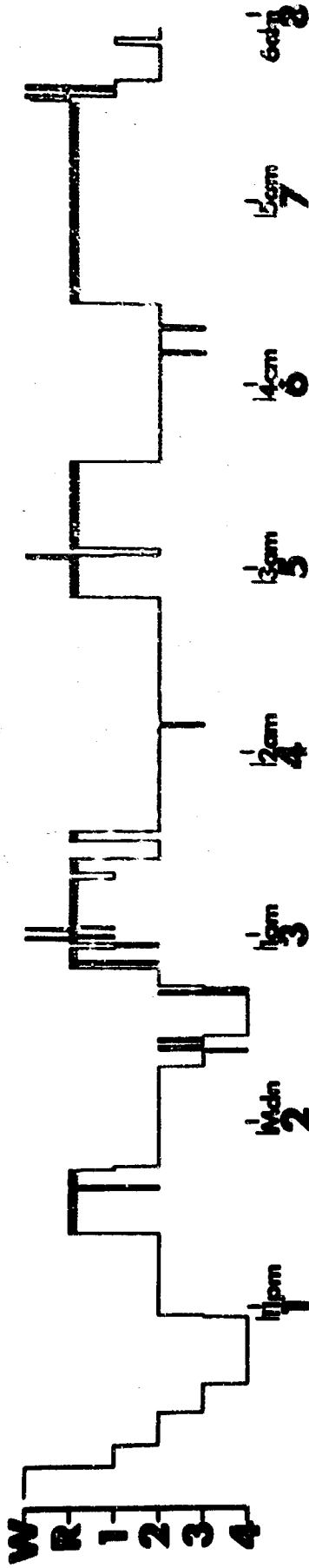
STAGES OF SLEEP [MAN]



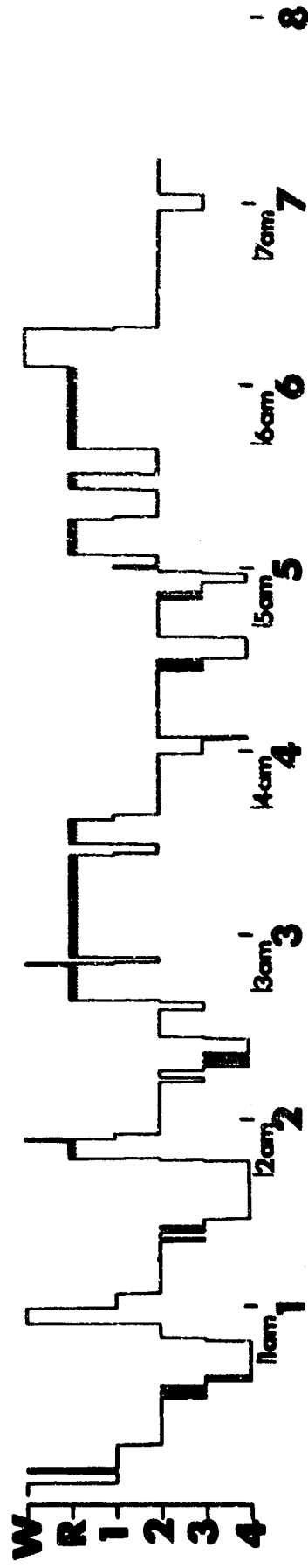
100µv | 250c

SLEEP CYCLES

JGV



HEC



HOURS OF SLEEP

LEOG-A

REOG-A

EMG

F3-A

C3-A

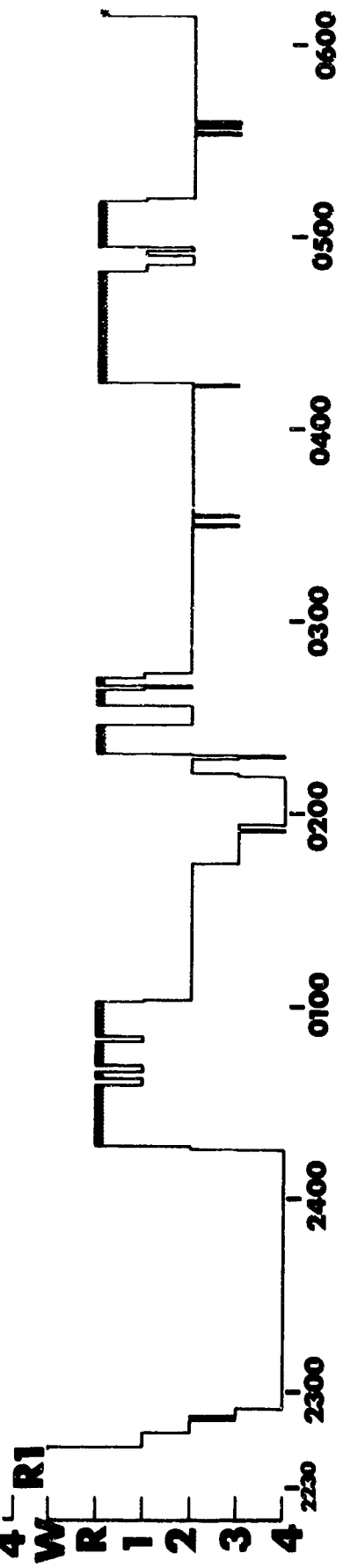
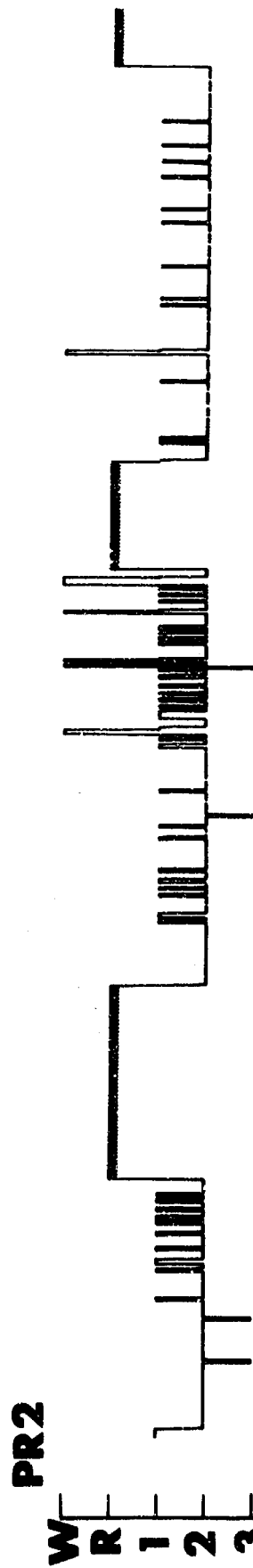
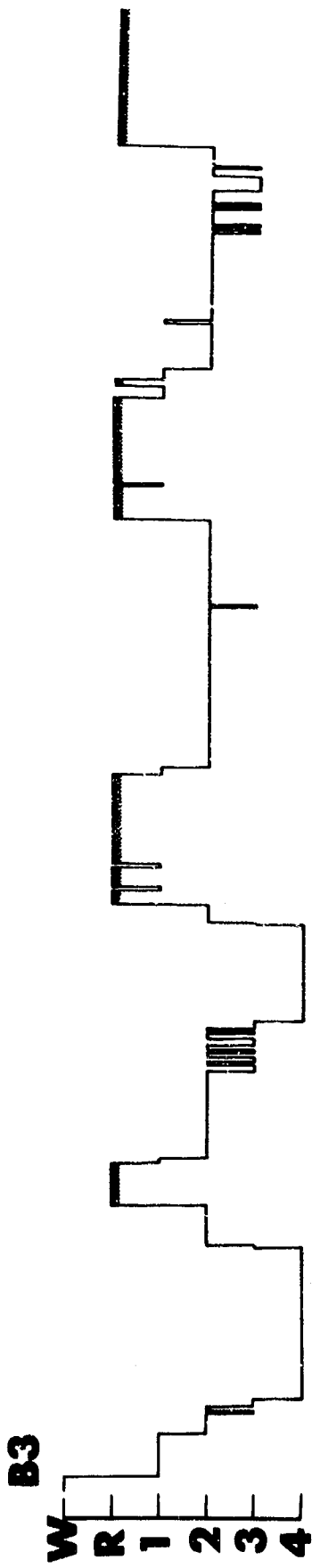
O1A

SP

HR

FP





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13. ABSTRACT The effects of sleep loss on human task performance were discussed under total, partial, and selective deprivations of sleep. Some of the frequently used psychological tasks in studies of total sleep loss were described in sufficient detail so that experimenters could choose, on the basis of materials presented in this monograph, adequate tasks to fit their experimental objectives. Factors which played critical roles in determining the degree of task sensitivity to total sleep loss were listed. Effects of shortened hours of sleep on human task performance were discussed. Effects of selected sleep deprivation on performance were also briefly commented upon. The commentary of this monograph covered almost all studies conducted on sleep loss under laboratory conditions, including a series of on-going experiments on total and selective sleep deprivations at the Navy Medical Neuropsychiatric Research Unit, San Diego. The commentary was followed by a bibliography on sleep deprivation with author and subject indices.		

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14. KEY WORDS	LINK A		LINK B		LINK C	
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Partial Sleep Deprivation						
Sleep Loss Sensitive Tasks						
Shortened Sleep						
Slow Wave Sleep Deprivation						
REM Deprivation						