PERCEPTUAL DEPRIVATION - EFFECTS ON HUMAN SLEEP

THE EFFECTS OF PERCEPTUAL DEPRIVATION ON SLEEP IN HUMAN SUBJECTS

By

WENDY K. POTTER, M.A.

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AUTHOR: Wendy K. Potter, B.A. (McGill University)
M.A. (McMaster University)

SUPERVISOR: Dr. W. Heron

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Eight human subjects underwent perceptual deprivation for either four or seven days. In addition, they slept in the laboratory for three nights before and three nights after the deprivation period. Continuous recordings of the electro-encephalogram, electro-oculogram, and electromyogram were made during these nights and during deprivation. The amounts of time spent in the awake state and in the various sleep stages were ascertained by the method of Dement and Kleitman (1957). Subjects slept much more than usual early in deprivation, but gradually returned to normal by about day four. The effects of deprivation on the various sleep stages are described and the implications of these for theories of sleep and sensory deprivation are discussed.

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CHAPTER 1

INTRODUCTION

What happens to a person who is put in a monotonous sensory environment? In 1951 at McGill University, an experimental program was begun which attempted to answer this question. Volunteer subjects were paid to spend twenty-four hours a day doing nothing; that is, they were asked to lie on a bed and to wear headphones over their ears, translucent goggles over their eyes, and cardboard tubes over their forearms and hands. This equipment ensured that the variety of the subjects' sensory experience was reduced to a minimum, a condition called by the McGill investigators perceptual deprivation.

Many functions have been studied under deprivation conditions, and generally, all have been shown to change, usually for the worse. These include the subject's emotional state, his motivation, his perception, and his cognitive abilities. One function that seems to have been overlooked by deprivation researchers, however, is sleep. This seems surprising since information about sleep during deprivation would appear relevant to the reticular theory of deprivation. Briefly, this states that deprivation symptoms can be attributed to dysfunction of the reticular activating system resulting from the monotonous nature of the incoming sensory information. In addition, an investigation of sleep under the special conditions of perceptual

deprivation would seem pertinent to several contemporary theories of the function of sleep, specifically to those proposing that sleep with rapid eye movements performs a cognitive function, or to those linking slow-wave sleep to recovery from physical fatigue.

The present study, then, is an investigation of sleep in human subjects exposed for long periods to perceptual deprivation.

CHAPTER 2

BACKGROUND

This chapter is intended to provide a review of the experimentation and theory relevant to the present research. The deprivation literature will be dealt with first, and will include a brief summary of some of the more striking of the deprivation symptoms, that is, cognitive, perceptual, and emotional changes, together with a consideration of some of the more prominent theoretical accounts. The next section of this chapter is concerned with sleep, beginning with a summary of current ideas about the neural mechanisms which control it, and continuing on to consider what its function is. Finally, several experiments bearing more directly on the present study of sleep under deprivation conditions are reviewed.

Deprivation

The first deprivation experiments, performed at McGill University in the 1950's, have had profound effects, not the least of which was to generate a huge amount of subsequent research. A recent bibliography of publications in this area (Weinstein et al., 1968) contains 1199 references. Perhaps the most surprising outcome of all this research is the amazing number and variety of activities that deprivation affects.

For a comprehensive review of these, a recently published book edited by Zubek (1969) is excellent. It is not possible to present a complete summary in this chapter, but several of the more prominent symptoms, including cognitive, perceptual, and affective changes, will be surveyed.

Cognition

There are two ways to assess any changes occurring in the subjects' cognitive abilities during deprivation; one is to listen to their own comments on the subject, and the other is to attempt to measure any changes directly. Subjects at McGill reported that they could not concentrate, and felt unable to think in a directed fashion, and that they occasionally felt confused. In fact, this has been an invariable report from all deprivation laboratories. When subjects are asked to describe their thought processes during deprivation, they state that they gradually cease to think in a directed and rational way, and instead spend most of their time day-dreaming. All of these indicate a rather dramatic degradation of cognitive capacity; objective tests support this conclusion to some extent. Isolated subjects are worse than non-isolated controls on the following tests: Kohs Blocks, Digit Symbol, Thurstone-Gottschaldt figures, Copying a Passage, Delta Blocks, and Picture Anomaly. In addition, they are impaired on word-making, number series, and anagrams, but show no deficits on digit span and analogies tests (Heron, Bexton, and Hebb, 1953; Heron, 1961).

Since these first studies at McGill, many more

experiments investigating cognitive phenomena have necessarily complicated the picture, supporting some of the above results and contradicting others. To illustrate, the deprivation laboratory under J.P. Zubek at the University of Manitoba has investigated the cognitive effects of variations in the conditions of isolation, as follows: sensory deprivation, or darkness and silence (Zubek, Sansom, and Prysiazniuk, 1960), perceptual deprivation, or unpatterned light and white noise (Zubek et al., 1962), and perceptual deprivation with required exercise (Zubek, 1963). Generally, the results indicate that perceptual deprivation leads to greater cognitive impairment than sensory deprivation (Zubek, 1964c), and also support McGill in showing deficits in deprived subjects on tests of numerical reasoning, verbal fluency, and abstract reasoning. Isolates had little or no trouble with digit span, rote learning, recall and recognition. One interesting result of the Manitoba experiments is that similar, though less severe, cognitive impairments can be produced by body immobilization alone (Zubek and Wilgosh, 1963; Zubek and MacNeill, 1966).

These few experiments suffice to give some idea of the cognitive effects produced by sensory monotony. Suedfeld (1969) has exhaustively reviewed the literature on this subject and attempted to provide a synthesis of the many results. If the tasks used by experimenters to objectively assess cognitive function are ranked according to their complexity - complexity as defined by Suedfeld being low for a memory test which demands

almost no active intellectual effort, and high for a test requiring unstructured creative thinking - then there is a positive correlation between the magnitude of the cognitive deficit observed and the degree of task complexity. In other words, it looks as though deprivation has a selective effect on cognition, not much harming simple well-learned behavior, but greatly impairing a subject's ability to deal creatively with novel unstructured material.

Perception

Early reports from McGill indicated that subjects experienced quite severe, though relatively short-lived, perceptual disturbances following isolation. Subjects reported difficulty in focusing, a tendency for objects to merge with their backgrounds and for the environment to appear two-dimensional, and super-saturation of colors (Bexton, Heron, and Scott, 1954; Doane et al., 1959). In addition to recording subjects' comments on the perceptual alterations they experienced, the McGill experimenters employed a large number of objective tests (Doane, 1955; Heron, Doane, and Scott, 1956). Those which showed a significant post-deprivation change included the Gottschaldt Embedded Figures, size constancy, color adaptation, figural after effects, autokinetic effect, Archimedes spiral after effect, and speed of copying a prose paragraph. No significant changes occurred in measures of critical flicker frequency, phi phenomenon, brightness contrast, brightness constancy, shape constancy, Necker cube reversals, tachistoscopic perception, and mirror drawing. There was a strong

suggestion of an increase in visual acuity.

As with the cognitive effects, some of these results have been confirmed and others have not. For example, the Manitoba laboratory reports no change in size constancy (Zubek et al., 1961; Zubek et al., 1962; Zubek, 1964b), thus disagreeing with the early reports from McGill. However, the two laboratories used different methods of measuring size constancy, so the seemingly contradictory reports might be accounted for by methodological differences. A deficit in color perception, on the other hand, seems to be an invariable result of deprivation; Vernon et al. (1961) and Zubek et al. (1962) agree with the McGill experimenters on this item.

All these results were obtained with multimodality deprivation. Single-modality deprivation experiments have been performed as well, using the visual (Zubek, Flye, and Aftanas, 1964; Zubek, Flye, and Willows, 1964; Schutte and Zubek, 1967), tactual (Heron and Morrison; Aftanas and Zubek, 1963a and b), and kinesthetic (Zubek et al., 1963; Zubek and Wilgosh, 1963; Zubek and MacNeill, 1966) sensory systems. In general, the findings of these studies indicate that single-modality deprivation is sufficient to produce many of the effects of multi-modality deprivation, including increases in tactual acuity, auditory discrimination, and pain sensitivity.

Perhaps the most striking perceptual alteration reported by the McGill investigators was the occurrence of hallucinations in their deprived subjects (Heron, 1961; Bexton, Heron, and Scott, 1954; Heron, Doane, and Scott, 1956). These occurred while the subjects

were unequivocally awake and consisted of definite "perceptions without object". They ranged in complexity from simple dots or lines to full-fledged scenes appearing in front of the subjects. Like the other perceptual and cognitive effects, these hallucinations have not gone unchallenged, and the general attitude toward them today is one of considerable skepticism. This skeptical attitude may be partly due to the fact that extremely short durations of deprivation have been reported to induce these hallucinatory phenomena (eg. Zuckerman et al., 1962). In addition, it has been found that subjects who receive pre-deprivation instructions such that they expect to experience vivid visual imagery are much more likely to report such experiences than those who are not so prepared (Pollard, Uhr, and Jackson, 1963).

Nevertheless, neither of these two factors can account for the McGill reports of hallucinations, since deprivation durations were long, and the experimenters, far from priming their subjects to report hallucinations, were in fact greatly surprised by their occurrence. It was only after several subjects had repeatedly asked the experimenters whether pictures were being projected on their goggles, and after one subject showed repeated head withdrawal when he thought he saw objects moving towards him that these reports began to be taken seriously (Heron, personal communication).

Zuckerman (1969) reviews the evidence pertaining to these phenomena and points out that though their incidence is much lower than that initially reported at McGill, they are nevertheless

genuine effects of the deprivation experience and cannot simply be dismissed as the imaginings of subjects previously "set" to report hallucinations.

Emotion

Subjects in the early deprivation experiments at McGill became bored and irritable, and said so without hesitation. In a complementary fashion, they seemed also to be very easily amused. In general, the deprivation experience seemed to produce an increase in emotional lability and a slight regression towards more childish emotional behavior, at which some subjects expressed considerable surprise (Heron, 1957). Another interesting result of these experiments, and one invariably replicated by subsequent investigators, was that not all subjects managed to stay in deprivation for as long as they had previously volunteered to stay. Zubek et al. (1961, 1962) find that only about two-thirds of all volunteers can endure sensory deprivation or perceptual deprivation for one week. This provides strong support for the proposal that deprivation is an unpleasant experience and perhaps makes subjects feel stressed and anxious. As with changes in cognition and perception, attempts have been made to assess the emotional changes experienced by deprived subjects objectively, with most of the emphasis being placed on measurements of stress and anxiety. When subjects rate themselves on these variables, a definite shift toward these negative emotional states is apparent (Zuckerman, Levine, and Biase, 1964; Zuckerman et al., 1966). Further investigations of which aspects of the deprivation

experience (loss of vision, loss of hearing, loss of social contact, confinement) are related to which emotional changes has revealed the following correlations: general complaints of discomfort are most common in a perceptual deprivation situation, but reports of fear and anxiety seem more related to the presence of white noise, whether or not vision is restricted as well (Leiderman, 1962). Confinement, even without sensory deprivation or social isolation, can produce significant subjective stress, but the greatest increase in anxiety results from the deprivation experience (Zuckerman et al., 1968). An interesting finding from the Manitoba laboratory (Zubek, 1963) is that physical exercise during perceptual deprivation can to some extent counteract the impairment found in intellectual and perceptual function, but that the proportion of quitters in this exercise group is about the same as in non-exercise perceptual deprivation groups. It seems, then, that exercise does not reduce the subjects' feelings of stress although it can produce rather dramatic improvement in other functions.

Clearly, then, when the measurement device is the subject's reports or self-ratings of emotional change, there emerges the consistent picture that deprivation produces stress and anxiety. However, sensory or perceptual deprivation apparently does not parallel other stressful situations in altering the activity of the adrenocortical and sympathetic- adrenomedullary systems. Murphy et al. (1955) report no change in the excretion of 11-oxycorticoids during deprivation; Zubek and Schutte (1966)

were unable to detect any difference between the amount of excreted catecholamines in experimental compared with recumbent control subjects. These results are puzzling. The only general conclusion that can be drawn is that the biochemical measures so far employed are not sensitive to the variety of affective disturbances reported by deprived subjects.

These, then, are a few of the effects produced in subjects undergoing sensory or perceptual deprivation. Much of the research produces conflicting results, much of it cannot even be compared because of methodological differences. Yet there is an enormous mass of data and, unexpectedly for a phenomenon with such wide-ranging and important symptoms, there is almost no theory available which attempts to synthesize it. With this discouraging thought in mind, it seems, Vernon named the last chapter of his 1963 book "Facts without a theory".

It is unfair and incorrect, however, to say that there are no theories at all, when, in fact, there have been three major ones advanced about the neural basis of the effects of deprivation. The McGill laboratory put forward a reticular system theory which states that the reticular activating system needs continuous varying input from the sensory receptors in order to properly perform its tonic activating function on higher brain centers. When it gets little input and when that little is unchanging, the cortex is deprived of its normal input and cannot continue in its usual fashion (Heron, 1961). This is also the gist of the theory put forward by Lindsley (1961), but with the

significant modification that the reticular formation serves as a kind of homeostat adjusting input-output relations. In order to perform these adjustments most efficiently, the reticular formation itself becomes "tuned" to ongoing activity within certain limits, in somewhat the same fashion as a band-pass filter deals with incoming frequencies only within its pre-set limits. In other words, the reticular formation can be said to be set at a certain adaptation level. When one element important to this regulatory function is drastically altered, for example, when sensory input ceases to vary in its usual manner, the adaptation level of the reticular formation is no longer appropriate, and the whole system malfunctions. Lindsley envisions the cortex as unaroused by the reticular activating system, and the subject, deprived of this activation, as inevitably becoming bored and inactive, and eventually falling asleep. Schultz also postulates a homeostatic sort of mechanism, based on a principle he calls sensoristasis. Sensoristasis is "... a drive of cortical arousal which impels the organism to strive to maintain an optimum level of sensory variation." (1965, p. 30). When sensory input is reduced in amount and variety, the reticular activating system adjusts to this state but the organism becomes very sensitive to incoming stimulation, and its arrival is considered to be reinforcing since it reduces the drive for sensoristasis.

These, then, are the three most comprehensive theories of deprivation phenomena, comprehensive in the sense that they attempt to encompass all of its effects and not just concentrate

on one or a few. Clearly, the three are variations on a single theme. All implicate the reticular activating system as the mechanism which is responsible for the production of deprivation symptoms. Briefly stated, the reticular theory attributes the effects of deprivation to an unaroused cortex.

The concept of arousal is central to any discussion of deprivation effects and the theories attempting to account for them. The McGill experiments were done just after the pioneer work of Moruzzi and Magoun (1949) and Lindsley et al. (1949, 1950) on the ascending reticular activating system, so it was quite natural for these deprivation workers to formulate their theories in these terms. Since then, however, much new information about the reticular system has emerged, with the result that concepts of both its structure and its function have been considerably modified. The arousal function of the reticular system is no exception, and the entire concept of arousal deserves re-examination. Dement and Kleitman (1957) demonstrated a clear dissociation between behavioral and EEG arousal with their recordings of low-voltage fast cortical activity from sleeping humans. Bradley (1958) showed that atropine produces a similar dissociation; animals given this drug are behaviorally awake and alert, yet show EEGs indicative of deep sleep. Further, Evarts (1967) has shown that it is not possible to predict the behavior of single neurons from the characteristics of the cortical EEG, since there are some cells which increase their spontaneous rate of discharge

from the waking to the sleeping state. These studies clearly demonstrate that the conventionally accepted equivalences between behavioral and EEG arousal and between EEG activity and the discharge of single neurons are no longer altogether valid. Other measures believed to indicate the state of arousal of an organism, such as activity, or various aspects of evoked potentials, or sensory thresholds, must also be similarly questioned.

Considerations of this kind urge a reappraisal of the usefulness of the concept of arousal, and in particular, of the simple form of it which prevailed at the time of the first deprivation experiments. In spite of this, however, present day investigators of deprivation phenomena still use the arousal concept as originally formulated, perhaps finding it difficult to discard so serviceable a theory. Their research has produced results considered to support the reticular theory of deprivation by indicating decreased levels of arousal, as well as those which do not seem to support it.

One piece of evidence which has been taken to support the reticular theory in that it seems to indicate a lowering of brain activity is a slowing of the alpha rhythm in deprived subjects. Heron describes the change as follows: "It is apparent that there was progressively slower activity during the isolation period. ... It should also be noted that the changes progressed regularly; that is, that there is more slow activity after 96 hours than after 48 hours in all cases." (1961, pp. 24-25).

In addition, this reduced frequency alpha rhythm persists for a considerable time after subjects have emerged from isolation. Heron (1961) measured the alpha frequency following four days of deprivation and found it still reduced at three and one-half hours afterwards. Others have found that this effect can last as long as ten days after a deprivation period of fourteen days (Zubek, 1964a). Though the slowing of the alpha rhythm itself has been interpreted as offerring support to the reticular theory in that it seems to indicate decreased arousal, the details of this process are more difficult to understand. In terms of reticular function, it is hard to understand why the changes occur so slowly and gradually, and also why they persist so long after deprivation.

Subsequent experiments on this phenomenon have been performed in North America (Zubek and Welch, 1963; Zubek, Welch, and Saunders, 1963; Marjerrison and Keogh, 1967), in England (Smith, 1962), in Japan (Nagatsuka and Kokubun, 1964; Sato and Kokubun, 1965; Ohyama, Kokubun, and Kobayashi, 1965), and in Russia (Lebedinsky, Levinsky, and Nefedov, 1964; Miasnikov, 1964; Gorbov, Miasnikov, and Yazdovsky, 1963; Agadzhanian et al., 1963). These experiments have added many more details to the finding that brain activity is slowed during deprivation, but none have failed to confirm this basic result.

There are several animal studies involving the surgical or chemical reduction of incoming sensory stimulation which are closely related to the deprivation experiments. The high voltage slow waves seen after massive deafferentation produced by brainstem

transection, as in the cerveau isolé preparation of Bremer (1935), cannot be attributed to the elimination of sensory input alone, but must be viewed primarily as the result of gross damage to the reticular formation (Lindsley et al., 1949, 1950). However, there are more recent reports which indicate that selective elimination of a single sensory modality can lead to an increase in EEG synchrony. Arduini and Hirao (1959) produced a reversible visual deafferentation by raising the intraocular pressure in cats with brainstem transections, and observed that this procedure was followed by cortical EEG sleep patterns. Both Hodes (1962) and Randt and Collins (1960) have reported EEG slowing in cats deprived of proprioceptive input by neuromuscular blocking agents.

These animal experiments, then, as well as those using human subjects, have been cited as support for the reticular theory of deprivation in suggesting that the arousal level is lowered during this experience. On the other hand, various other deprivation effects have been considered as strong support for the opposite contention, that arousal levels are heightened rather than depressed during deprivation.

Activity has been shown to increase during the period of deprivation. Smith, Myers, and Murphy (1962, 1967) found a significant and progressive increase in restlessness, or body movements, measured over four days of sensory deprivation. It is well known that patients hospitalized for eye surgery, a situation which could be considered as clinical sensory deprivation, often are very restless (Jackson, 1969). Observations of animals

reared in restricted environments may conceivably be related to the deprivation effect on activity. Both Thompson and Heron (1954) and Melzack and Scott (1957) have reported that dogs reared in this way tend to show an aimless hyperactivity. Goldfarb (1955) has observed similar hyperactivity and restlessness in institutionalized children.

Quite different from these activity measures, but thought to provide as strong support for the increased-arousal theory, are recordings of evoked potentials in deprived subjects. Gendreau (1969) reports that prison inmates subjected to a mild deprivation procedure for one week exhibited a decrease in the latency of visual evoked potentials when compared with non-deprived controls. Also, amplitudes of evoked responses tended to habituate less in the deprived group. However, it is possible that these results should be de-emphasized since the experiment included very few control subjects, and consequently the limits of normal variation in these measures of evoked potentials were not well defined.

Finally, there is considerable information on changes in acuity and sensitivity which also has been cited as support for an increased-arousal interpretation. Doane et al. (1959) reported an increase in tactual acuity following perceptual deprivation; Zubek (1964b) has replicated this finding. Increased pain sensitivity also seems to result from sensory deprivation (Vernon and McGill, 1961), but can occur after visual deprivation alone (Zubek, Flye, and Aftanas, 1964). Single modality deprivation can lead to increases in acuity in several modalities, not only

in the one restricted; these changes have been observed in the tactual (Zubek, Flye, and Willows, 1964), auditory (Duda and Zubek, 1965), and olfactory modalities (Schutte and Zubek, 1967) after visual deprivation, and in the tactile modality (Heron and Morrison; Aftanas and Zubek, 1963a and b) after tactual deprivation.

These data concerning activity, evoked potentials, and acuity changes, then, seem diametrically opposed to the reports of EEG slowing in that they are thought to reflect an increase, rather than a decrease, in arousal as a result of deprivation. Most of the cognitive and perceptual changes, except for the reports of increased acuity, can most easily be ascribed to a decrease in level of arousal. The subjective reports of affective change tend to support the idea of increased arousal, but the biochemical measures are not conclusive. All of these effects have been repeatedly demonstrated by experiment; none can be merely ascribed to accident. Both bodies of fact, interpreted as supporting either an increase or a decrease in arousal, must be accepted, leading to the paradoxical conception of deprivation as producing both an increase and a decrease in the level of excitation of the central nervous system. A possible resolution is provided by Beteleva and Novikova (1961). These authors produced olfactory deafferentation in the rabbit by thermocautery of the mucous membrane housing the olfactory receptors. EEG records taken from the visual and sensorimotor cortex showed a large decrease in amplitude and some decrease in frequency following this treatment. On the other hand, simultaneous recordings from the reticular

formation were increased in amplitude and showed a greater prominence of fast frequencies. The EEG slowing observed during deprivation might be the counterpart of the cortical depression observed by these authors, whereas the increase in reticular activity, if such an effect genuinely occurs in sensory and perceptual deprivation, might account for such effects as the increase in several sensory acuities and the progressive increase in activity. In fact, there is some evidence already available for reticular involvement in increased acuity. Fuster (1958) observed faster reaction times and generally improved performance on a tachistoscopically presented visual discrimination task in monkeys during stimulation of the reticular formation. Reticular stimulation also seems able to improve the resolving ability of the visual cortex for brief flashes of light (Lindsley, 1961).

The reticular theory of deprivation, then, seems to broadly fit and generally explain the effects of this procedure. In its present formulation, however, it is far from precise, making no attempt at all to define in more detail neural mechanisms which might underlie the various symptoms of deprivation. One additional limitation has been pointed out previously, that is, the persistence in theories of deprivation of an oversimplified concept of arousal. It seems to be a characteristic of all research in deprivation that it is not aimed at the development or elaboration of some theoretical point, but rather seems to accumulate in quite aimless fashion. Clearly, what are needed are research programs which set out to collect information about reticular system function under

both normal and deprived conditions. Equally clearly, one of the most obvious choices, and presently an area of intense research activity in its own right, is sleep.

Sleep

The reticular system has been implicated in regulating the various states of consciousness, including sleep, since its discovery by Moruzzi and Magoun in 1949. Their report that reticular stimulation leads to EEG arousal, coupled with Bremer's (1935) finding that cerveau isolé cats display only slow-wave EEG activity, led to the hypothesis that it was the reticular formation, rather than the classical sensory pathways as Bremer had initially supposed, that was responsible for regulating EEG arousal. Subsequent classic experiments by Lindsley et al. (1949, 1950) showed that reticular lesions alone were able to reproduce the effects of the cerveau isolé operation, but that lesions of the sensory pathways were not; the hypothesis was confirmed. Basically, this is the evidence which led to the proposal of what is now called the deafferentation theory of sleep. Briefly, it states that sleep is attributable to elimination of the waking influence of the reticular activating system, or, alternatively, that sleep occurs when the background activity of the cerebrum, which depends upon the tone of the ascending reticular formation, decreases below a critical level.

This deafferentation notion still forms the core of most theories of sleep, although much new information has forced

elaborations and modifications of it, and has often led to considerable confusion. For example, one of the more startling discoveries (Aserinsky and Kleitman, L955; Dement and Kleitman, 1957) was that sleep is not a homogeneous state, and that its EEG signs do not always consist of high voltage slow activity. All humans and many animals experience a phase of sleep characterized by low voltage fast EEG activity which greatly resembles the waking record, by great relaxation of certain body muscles, and by frequent rapid movements of the eyes under the closed lids. From these eye movements the stage has taken its name - REM (for Rapid Eye Movement) sleep.

Much additional information has become available which implicates many other brain structures besides the reticular formation in the production of sleep. Stimulation of many points in the diencephalon of cats revealed that natural sleep, complete with its preparatory treading and curling up, could be elicited regularly from points in and close to the midline thalamus (Hess, 1954). This behavioral resemblance between natural and stimulation-induced sleep is paralleled by the striking similarity in their EEG patterns (Akert, Koella, and Hess, 1952). In a similar fashion Clemente and Sterman (1967) were able to produce sleep in cats by stimulation of the preoptic area and the diagonal band of Broca, an area they refer to as the basal forebrain. These authors postulate a descending functional pathway from the orbital cortex to the brainstem and thalamus by way of the basal forebrain region and limbic system; this pathway is inhibitory and acts to

oppose the ascending reticular activating system. Moruzzi (1960, 1963) also believes in structures which act to oppose the activating system and promote EEG synchrony and sleep, but maintains that they are located in the lower brain stem. The fact that cats with midpontine pretrigeminal brainstem sections spend much more time in a state of EEG arousal than do normal intact cats (Batini et al., 1959) strongly supports the existence of these synchronizing structures. Magnes et al. (1961) speculate that they are located in the vicinity of the nucleus of the solitary tract, since it was from this region that they obtained widespread bilateral synchronization of the cortical EEG by low frequency stimulation. Moruzzi feels that this discovery is in good harmony with the deafferentation theory of sleep if it is accepted that the reticular barrage may decline because of active inhibition from the synchronizing mechanism as well as falling off passively. In fact, this hypothesis seems particularly able to explain those cases in which sleep is produced by stimulation of the brainstem (Favale et al., 1961) or by repetitive sensory stimulation. Finally, as a result of many years of experimentation, Jouvet (1963, 1967 a and b) has discarded the possibility of an electrical sleep mechanism in favour of what he calls "wet" neurophysiology, that is, the idea that sleep is dependent upon fluctuating levels of brain chemicals. More specifically, the monoamine serotonin contained in neurons of the raphe nuclei of the brainstem plays a part in the process of sleep, and is primarily concerned with slow-wave sleep. The neurochemical

system responsible for the control of paradoxical or REM sleep seems somewhat diffuse, and is only understood in a fragmentary fashion, but implicated are monoamine oxidase-containing and noradrenalin-containing neurons of the locus coeruleus.

It is obvious, then, that sleep is far from being a homogeneous function subserved by a single sleep "center". Far more likely is a system in which many brain structures interact and perform different roles in triggering or regulating various aspects of sleep. Koella (1967) is the major contemporary theorist who has attempted to survey all the evidence pertaining to sleep and to reconcile the various viewpoints about how it is produced. He believes that the hypnogenic area described by Hess in the thalamus is the "head ganglion" of sleep, that is, the only brain area which seems capable of controlling all aspects of sleep. Other areas which, on stimulation, have led to the production of sleep or, on lesion, to its disturbance he designates as subordinate structures, since all seem able to influence only certain aspects of sleep, and do not regulate the phenomenon as a whole. The nucleus of the solitary tract is conceived of as the brainstem synchronizing mechanism postulated by Moruzzi; its action is to oppose the activating or arousal system. The midbrain reticular formation also is considered to function in a subordinate manner during sleep, perhaps being involved in the control of phasic, short-lived episodes of sleep or deepening of sleep. The role of the basal forebrain is considered to be the organization of the adjustments and change in skeleto-motor tone

and autonomic output which are characteristic and to some extent necessary for sleep. The hippocampus too is a subordinate structure, which is very likely responsible for the presomnic phase of sleep; stimulation of this structure in cats elicits behavior which closely parallels normal pre-sleep behavior, including yawning, curling up, grooming, and relaxation of the nictitating membranes (Parmeggiani, 1962). That region of the pons which seems to be responsible for paradoxical or REM sleep also falls into Koella's subordinate category.

All this, however, constitutes an answer to the question "How?" of sleep; that is, it concerns the neural machinery responsible for sleep. Very little attention has been paid to the question "Why?". In fact, Jouvet believes that "very seldom in the history of physiology has so much effort been devoted to the description, quantification, classification, and delimitation of such a complex phenomenon of almost totally unknown function" (1969, p. 32). Nevertheless, some theorists have very recently been much preoccupied with possible functions of sleep. On the face of it, the question "Why do we sleep?" seems ridiculous. The obvious answer is because we are tired, and sleep is the recovery process from this tired state. Indeed, the investigations of Hess and Moruzzi and Magoun dwelt on this problem not at all, considering it already answered by the recovery from fatigue idea. Given that this is a sensible answer, the next question would have to be, "What exactly is it that gets tired and needs sleep to recover?". The common sense answer to this question is a little less obvious

than that to "Why do we sleep?" but nevertheless carries the same intuitive appeal. It cannot be true that we sleep solely in order to rest our muscles, since simply lying still is able to overcome muscle fatigue. The obvious alternative, then, is that it is the nervous system which needs sleep. This view draws an analogy from the observed states of rest and exercise in skeletal muscles in implying that sleep is a period of neuronal quiescence when compared with the intense activity of wakefulness. In other words, a sleeping brain is an inactive brain.

Recent evidence has shown, however, that cerebral neurons are not inactive during sleep. Evarts (1962) has shown that the average discharge frequency of neurons in the cat's visual cortex during stage I REM sleep is approximately the same as that observed when the cat is awake and attending to its environment, but that this frequency is twice that of waking in darkness. High levels of activity during REM sleep have also been reported for neurons in the brainstem (Huttenlocher, 1961), the somatosensory cortex (Evarts, 1963), and the motor cortex (Evarts, 1964). It appears, then, that sleep is not an inactive state, but an active one, which probably functions to allow recovery from some brain activity, and not simply to overcome muscular fatigue.

However, some experiments have shown an increase in synchronized or slow-wave sleep after physical exercise. Hobson (1968) exercised cats in a treadmill before permitting them to sleep, and found both an increase in the time spent in slow-wave sleep and an earlier onset of this stage than in non-exercised

cats. Matsumoto et al. (1968) have performed an identical experiment using rats, and report exactly the same results. In consequence, the theory that slow-wave sleep, at least, is related to physical fatigue has been seriously proposed. Hauri (1968 a and b, 1969) has failed to replicate these effects in humans. Six hours of physical exercise before sleep did not influence time to sleep onset, amount of slow-wave sleep, or time to the first slow-wave sleep period. Webb and Friedmann (1969) did not attend specifically to slow-wave sleep, but report that rats raised to 120 days of age in cages with or without activity wheels show no differences in either total sleep or diurnal distribution of sleep. There appears, then, to be evidence which both supports and contradicts the theory linking physical fatigue and slow-wave sleep. The current belief, however, is that it is unlikely that sleep serves only as a recuperative process from physical fatigue.

Far more in vogue is the alternative that it is some mental process which needs the sleep time to recover. It is impossible to neatly classify theories of sleep function according to whether they are primarily concerned with REM sleep or slow-wave sleep or both. Some theorists have concentrated specifically on the function of REM sleep, ignoring synchronized sleep, while others have spoken of sleep as though it were homogeneous, ignoring its two distinct phases.

Snyder (1966) has suggested that REM sleep functions to allow periodic arousal and activation, and so permit a quick orientation to the outside environment if the organism is in need

of it. Ephron and Carrington (1966, 1969) propose that the REM state, with its intense brain activity, serves to reverse the partial deafferentation occurring during slow-wave sleep, and preserves an optimum level of "cortical tonus". A quite different interpretation has been advanced by Berger (1968) who feels that REM sleep provides a mechanism for establishment of the neuromuscular pathways serving binocularly coordinated eye movements, and in adult life maintains this binocular coordination during sleep. A primarily developmental role has been assigned to the REM state by Roffwarg et al. (1966) and by Dement (1965); its intense neural discharge, probably from a pontine center, provides endogenous input necessary for the maturation of the neonatal central nervous system.

Probably the most attractive hypothesis, and one advanced by a number of authors, is that REM sleep, or sleep as a whole, is necessary at some stage of the learning-memory-consolidation process. Moruzzi (1966) seems to have been the first to advance this hypothesis and provide some indirect experimental support of its plausibility. He postulates that sleep is necessary, not for the whole brain, but only for those neurons whose synapses show plastic changes during wakefulness (learned synapses). It is possible that some metabolic substance slowly accumulates during the waking activity of these neurons or synapses and can only be dispersed by long slow processes of recovery. Other neurons or synapses, by contrast, do not need long recovery periods; like the neurons of the respiratory or vasomotor centers, their recovery

takes place in the intervals between cell discharges. Similar theories, but framed in the terminology of computer science, have been advanced by several other authors (Evans and Newman, 1964; Newman and Evans, 1965; Gaardner, 1966; Shapiro, 1967; Dewan, 1968, 1969). Essentially, these authors propose that sleep provides an opportunity for the processing of newly acquired information and its integration with older information, for discarding information that is irrelevant and unneeded, and for "reprogramming" for the arrival of new information.

Some experimental evidence in support of these cognitive theories of sleep has recently been published. Greenberg and Dewan (1968) report significantly greater amounts of REM sleep in aphasics improving in comprehension and production of speech as compared with those who are not improving. Feldman and Dement (1968) deprived subjects of REM sleep for one night and of non-REM sleep for another night and required them to perform a serial-anticipation learning task either before or after these nights. They report that REM deprivation is associated with lower savings scores for material learned following sleep and relearned several days later, but that non-REM deprivation is not. Other investigators have reported an increase in the amount of REM sleep following the wearing of distorting spectacles, an observation lending considerable support to the hypothesis that the REM stage is involved in learning, and specifically in this case in perceptual learning (Zimmerman, Stoyva, and Metcalf, 1970). A different kind of evidence is provided by Feinberg and Evarts (1969) who carefully

trace changes with age in certain sleep variables. For example, they report that the total amount of sleep, and the amounts of REM sleep and slow-wave sleep change relatively little during the mature years, compared to the changes which occur during childhood and old age. They hypothesize that these sleep variables "...reflect brain processes which underlie such cognitive activities as information acquisition and retrieval." (1969, p. 336). On the other hand, such variables as the high-voltage component of slow-wave sleep, and the frequency with which sleep is interrupted by waking change appreciably during maturity, and these authors consider that "...they might be related to those brain processes which underlie intellectual power as manifested by problem-solving and creativity, or general plasticity, as reflected by the ability to acquire languages or complex psychomotor skills." (pp. 336-337).

There is also some evidence from studies involving animals. Stern (1969a and b) reports that five days of REM deprivation impairs the acquisition of active and passive avoidance tasks in rats, and that this impairment can be attenuated by the administration of drugs which potentiate the action of norepinephrine in the central nervous system. This transmitter substance is thought to be crucial for the normal development and regulation of REM sleep (Jouvet, 1969). Similarly, Fishbein (1969a, b, and c) has postulated a very specific function for REM sleep, namely, that it is a mechanism which secures and maintains information for long-term memory stores. This author relates his view of REM sleep to current theoretical speculations on the nature of memory, which suggest that certain neural

transmitters play a vital role in both its formation and maintenance, and postulates that deprivation of REM sleep could deplete these transmitters. He reports that mice deprived of REM sleep between the learning and testing phases of a one-trial passive avoidance task showed amnesia for the initial learning, unless they are also permitted to recover from the loss of REM sleep. Inferring from this result that the memory trace had not dissipated completely, but was held in labile form during the period of REM deprivation, Fishbein postulated that electroconvulsive shock should disrupt the memory trace of one-trial passive avoidance training if the mice were REM deprived, but not if they were permitted to sleep normally. Experimental findings confirmed this possibility.

The various cognitive theories of sleep, supported by evidence like that cited above, seem to presently be the most popular, and to generate the most research. By no means, however, do they exclude acceptance of one or several other theories. For example, the view that REM sleep plays a role in the development of the fetal and infant nervous system is quite compatible with any or all of the cognitive theories. There need not be a single function of sleep, or even two, one for each of its two stages; it is quite possible that sleep serves many functions.

Deprivation and Sleep

The previous sections of this chapter have dealt with studies which investigate sensory deprivation and sleep independently. This final section will review the very few studies in which an

attempt is made to study the interaction between these two.

Kripke and O'Donoghue (1968) isolated five subjects in a dimly lit soundproof chamber for 36 hours. Subjects were permitted eight hours of sleep in the first twelve hours, but were asked to stay awake for the final 24 hours. They were unable to do so, and instead fell asleep repeatedly in a cyclic pattern resembling the periodicity of REM occurrences during nocturnal sleep. The authors interpret this finding as demonstrating the persistence during waking hours of a biological cycle previously identified only during sleep. In another experiment (Van der Kolk and Hartmann, 1968), subjects underwent either three or five hours of perceptual deprivation immediately before going to sleep. This treatment produced no significant changes in sleep variables, but there was a tendency toward increased D-time (Dream or REM time) and D-percent, and a decreased D-latency. Several investigators at Tohoku University in Japan have also examined EEG records from subjects isolated for 48 hours (Nagatsuka and Kokubun, 1964), for 24 hours (Sato and Kokubun, 1965), and for 18 hours (Ohyama, Kokubun, and Kobayashi, 1965). One minute of EEG was recorded at fifteen minute intervals throughout these deprivation periods, and similar results were reported for all three experiments. The EEG records typically showed a predominance of high amplitude middle slow waves throughout, with extremities of arousal waves and sleep waves appearing infrequently. These results are difficult to interpret since these experimenters did not record continuously from their deprived subjects, and used a method for classification

of EEG records which is obsolete as it was developed before the discovery of REM sleep.

Finally, after the present study was completed, a similar study was reported by Steinberg and Russo (1970). These authors confined ten subjects in groups of two or three for 21 days. No attempt was made to perceptually deprive these subjects, but their task activity and recreational material was minimal, and they had no outside contact, so their environment was certainly monotonous and boring. EEG records were obtained for the first three days (days 1-3), the middle three days (days 10-12), and three days toward the end of confinement (days 18-20). In addition, recordings were made of three baseline nights prior to, and three nights following the confinement period. All of these polygraph records were classified by a system very similar to that used in the present study.

The results obtained, with the figures in parentheses being percentages of 24 hours, are as follows: (i) Total daily sleep time for each subject had a mean of 10.34 hours during the first three days of confinement, and subsequently decreased to 9.21 hours during days 10-12 and to 8.63 hours for days 18-20. (ii) Stage IV values increased from a mean of 21.1 minutes (1.5%) for days 1-3 to 29.2 minutes (2.0%) for days 10-12, and then remained relatively constant at 28.1 minutes (2.0%) for days 18-20. Preconfinement measures were lower than any of these at 17.5 minutes (1.2%), and postconfinement measures higher at 32.6 minutes (2.3%). (iii) Stage III time, both during and after confinement, did not change appreciably from

preconfinement values. (iv) The mean amount of time spent in stage I REM was 102.1 minutes (7.1%) before and 83.9 minutes (5.8%) after confinement. During the first three days of isolation, this stage occupied 172.8 minutes (12.0%), dropping to 137.3 minutes (9.5%) for days 10-12 and then remaining relatively unchanged at 133.2 minutes (9.3%) for days 18-20.

It is unfortunate that these authors chose to average their data over three day periods rather than present the results obtained on each separate day. The calculation of an average value inevitably involves some loss of information, and this loss is especially regrettable for days 1-3 of confinement, since it precludes any knowledge of the changes which might be expected to take place during the early part of the isolation period. It is obvious that the experimental conditions devised by Steinberg and Russo differ considerably from the perceptual deprivation situation used in the present study. Nevertheless, the two have enough elements in common to warrant a comparison of the results obtained. A detailed comparison will be delayed until the discussion section, after a review of the results obtained in the present study.

At the moment, then, there is virtually no detailed information available about the sleep of subjects exposed specifically to a perceptual deprivation environment. Many questions occur: Might deprived subjects sleep more than normal, or do they maintain a normal cycle of sleep and wakefulness? Could deprivation cause them to sleep less? Regardless of any changes in total amount of sleep, is it possible that deprivation could have selective effects on one or

several of the various sleep stages?

The present study attempts to provide some answers to these questions, and to relate them to theories of sleep and of deprivation.

CHAPTER 3

PROCEDURE

Subjects

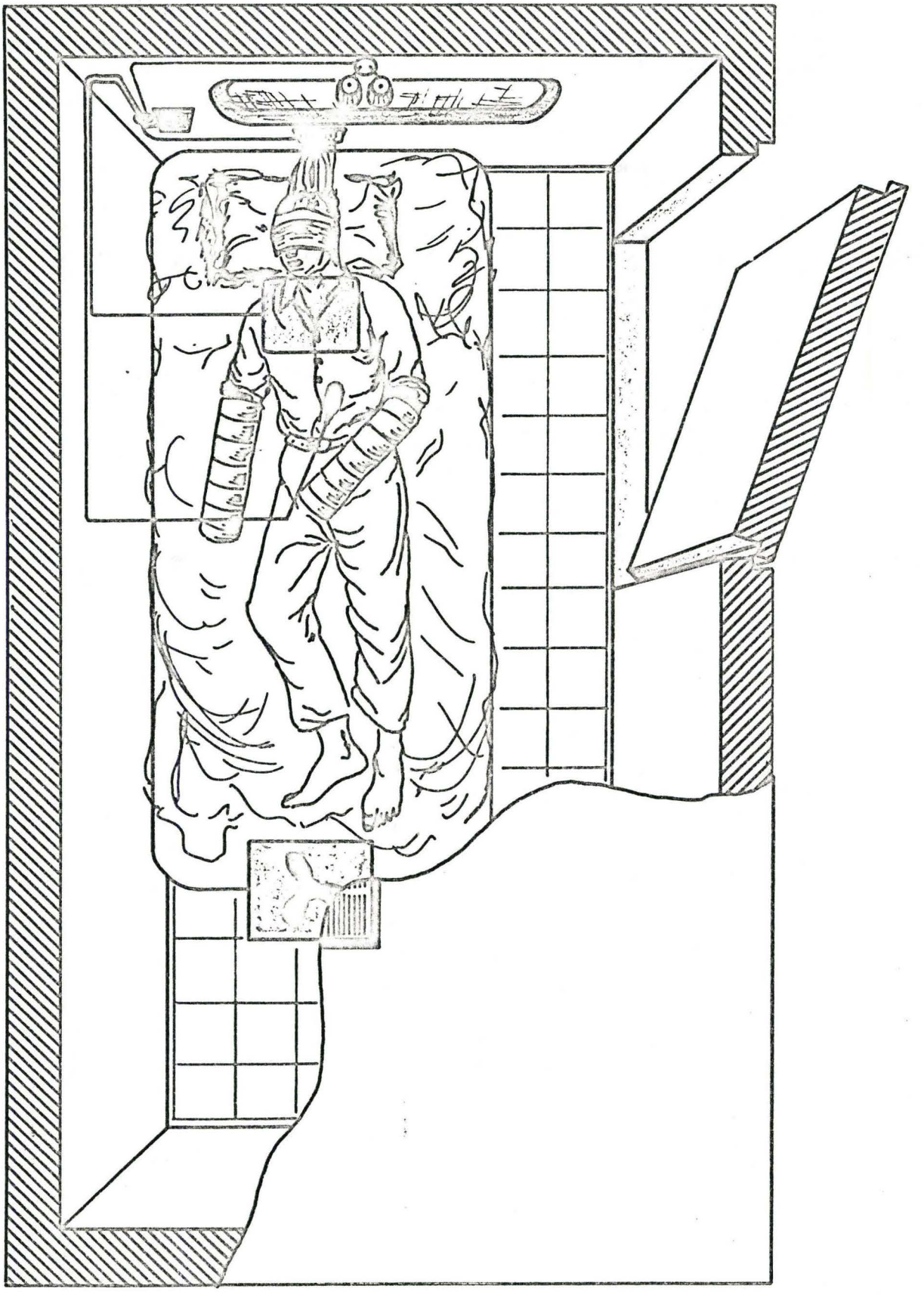
Eight adult humans, seven male and one female, between the ages of 21 and 27, served as subjects.

Apparatus

The deprivation environment used was similar to that described by investigators at McGill (Bexton, Heron, and Scott, 1954). The audiometric room which served as the deprivation chamber was manufactured by Eckel Industries of Morrisburg, Ontario. Its inside dimensions were 4' 0" by 7' 4" by 6' 8" high, large enough to snugly hold a single bed. This, as well as the other apparatus contained in the deprivation chamber, can be seen in Figure 1. Two hooded 40 watt bulbs supplied light; during deprivation, as measured through the subject's mask, the intensity of the light was 1.2 log foot lamberts. All inner walls of the room were painted gloss white to reflect well. A speaker which supplied 80 db. white noise from a Grason-Stadler model 901B noise generator was attached to the ceiling above the heads of the prone subjects. Communication between subjects and experimenters was via a two-way system; the subject's microphone inside the chamber was suspended about three feet

FIGURE 1

Diagram of a subject in the deprivation chamber, showing his mask, cuffs, and EEG electrodes. The positions of the chamber microphone, white noise speaker, communication speaker, chamber lights, and ventilation fan are also illustrated. (After Heron, 1957)



above his chest, while his speaker was mounted on the window wall of the chamber, slightly above and to the right of his head. The temperature of the chamber was kept at a level which was comfortable for the subject by an airconditioner connected to the intake fan of the chamber ventilation system. This temperature ranged from 68 to 74 degrees for the various subjects.

During deprivation, subjects wore masks which permitted only unpatterned visual stimulation. These were constructed of draughtsmans tracing paper cemented between two sheets of clear flexible plastic, and were padded around the edges with foam rubber for comfort and to minimize light leaks. They were held on with elastic around the back of the head. To restrict tactile sensation, subjects wore heavy cotton gloves and cardboard tubes extending from the elbow to beyond the finger tips. The tubes were secured with elastic ties around the wrists. Clothing was left to the discretion of the subjects, with the restriction that it cover arms and legs.

Lavatory and washing facilities were available in the same room which housed the deprivation chamber, so that subjects were never required to leave this one room.

The electrodes used were chlorided silver discs covered with felt pads. These were filled with a commercially available electrode cream, EKG Sol, and attached to the scalp with collodion. The International Federation 10-20 system of electrode placements was used (Jasper, 1957), and the following electrodes applied, all located over the right hemisphere: F4 (frontal), C4 (central),

T4 (temporal), P4 (parietal), and O2 (occipital). For two subjects, either F4 or T4 was dropped in order to record from the occipital area of the left hemisphere (O1). Recordings were monopolar with reference to the right ear (A2), or, in the case of left hemisphere placements, with reference to the left ear (A1). An electrode applied to the vertex (Cz) served as ground. In addition, an electrode was fixed at the outer canthus of each eye to provide a bipolar record of eye movements (electro-oculogram or EOG), and two additional electrodes approximately one cm. apart over the submental muscles gave the electromyogram or EMG.

Monopolar EEG derivations and bipolar EOG and EMG were recorded on a Grass model 330 P 8-channel polygraph, and from there were fed to an Ampex model SP 300 7-channel tape recorder. The EMG was not recorded on tape since the seventh channel was needed for voice. A Tektronix 502A dual beam oscilloscope was available for monitoring of the EEG as it was recorded on tape.

Procedure

A Screening Procedures

Volunteers for the deprivation experiment were asked to come into the laboratory for an initial interview with the experimenters. Almost all of them had some misconceptions about the experiment, so considerable care was taken to inform them of its exact nature before proceeding any further. Since another aspect of this experimental program concerned the alpha rhythm and its changes during deprivation, short recording sessions to

establish whether or not volunteers were alpha producers were usually also run at this initial meeting. Only those volunteers showing a strong occipital alpha rhythm were accepted as subjects. Once it had been established that the volunteers were alpha producers and were still willing to serve as subjects, they were asked whether they would undergo deprivation for four days or for seven days, and were told that the pay scale was $20 per day. In addition, they were informed that they would be required to sleep in the laboratory for three nights prior to, and three nights immediately after the deprivation period. $10 per night was the rate for these additional nights. Since the deprivation period for a 4-day subject actually lasted for four days and five nights, he would earn a total of $150. Similarly, a 7-day subject would earn $210.

Before their final acceptance as subjects, volunteers were asked to undergo further tests and interviews, including a physical check-up by a doctor, an interview with a psychiatrist, and an MMPI administered by a clinical psychologist. Depending, of course, on favourable reports from these three, the date was set for the first night's sleep in the laboratory.

B Pre- and Postdeprivation Sleeps

Procedures were identical for the three predeprivation and the three postdeprivation sleep nights. Subjects arrived in the laboratory approximately one and one-half hours prior to their normal bedtimes, having abstained from excess coffee, tea, or

alcohol. On the first night of the three predeprivation sleeps, the subjects' heads were measured and the placement of the electrodes was marked. Small patches of hair about one-quarter inch in diameter were cut at these spots to promote good electrical contact with the scalp. All electrodes were moistened with a commercial non-irritative EEG paste and fixed to the skin with collodion.

On these pre- and postdeprivation nights, subjects slept in the same audiometric chamber that was used for deprivation. The chamber was illuminated by a single 40 watt bulb aimed at the ceiling, so that the experimenter could observe the subject while he slept. This single bulb provided a light level of .6 log foot lamberts.

Fifteen minutes before retiring, subjects were given a sweet drink, which was usually lemonade or orange juice, but on occasion was coffee, tea, or milk if the subject requested one of these and was in the habit of drinking it before going to bed. At least two level teaspoons of sugar were added to the drink, enough to raise the blood sugar level to the high end of the normal range. This precaution was taken against the possibility that several hours of food deprivation, with the resulting drop in blood sugar, would affect the alpha frequency.

Before the subject settled down for sleep, a series of eyes open and eyes closed recordings were taken, and recorded on tape. Preceding these, the subject was asked to do several simple mental arithmetic problems (eg. 24 divided by 6 times 5 plus 7)

or, alternatively, some counting (eg. count backwards from 67 by 6's). This procedure was used to ensure a relatively comparable state of alertness during all such eyes open and eyes closed recording sessions.

During the night a continuous paper record of the subject's EEG, EMG, and EOG were taken on the polygraph, which ran at 15 mm. per sec. In addition, frequent samples were recorded on tape.

In the morning, at the time he had requested, the subject was awakened by the experimenter. For all subjects, the times requested were between 0730 hours and 0800 hours. After awakening, subjects were given another sweet drink, and, fifteen minutes later, after the usual mental arithmetic or counting, a second series of eyes open and eyes closed recordings were taken. The subject then got up, the electrodes were removed, and he was free to leave the laboratory. Subjects were asked to refrain from sleeping outside the laboratory, and to report any deviations from this request.

C Deprivation

The deprivation period started at the subject's usual bedtime, so that the preparatory routine was exactly the same as it was for the pre- or postdeprivation sleep nights, with the exception that the subject wore mask, cuffs, and gloves, and listened to white noise in the chamber. Before retiring, subjects were assured that an experimenter would be present in the room at

all times during the deprivation period, and would attend to their needs on request. The subjects were reminded that they were under no obligation to stay in isolation for the entire period if they wished not to; they had only to clearly state that they wanted to end the experiment for it to be ended.

Meals were regularly given the subject at approximately 8 am., 1 pm., and 7 pm. No attempt was made to extend sensory monotony to the diet by permitting only bland foods. As far as was possible, subjects were given foods that they had previously stated were their normal fare, or which they enjoyed. At meal times, subjects were permitted to smoke, and usually washed their hands and faces and brushed their teeth as well. Routine checks of electrode resistances were made at every meal; all electrodes were filled with paste or replaced as necessary. Subjects were encouraged to report any dreams they remembered; these were recorded by the experimenters.

Before and after each meal, an eyes open - eyes closed session was run and taped in order to track the changes in the alpha rhythm. Always before a meal, and fifteen minutes before the eyes open - eyes closed session, subjects were given a sweet drink.

Meals and trips to the lavatory constituted the major interruptions in the deprivation experience; others included the replacing of high resistance electrodes and the retrieving of fallen pillows. All together, these interruptions occupied an average of 3.5 hours out of every twenty-four.

Except for these 3.5 hours, the polygraph ran continuously. Subjects were encouraged to report the occurrence of any visual imagery they experienced, and the ongoing EEG was taped during these phenomena.

At the end of the deprivation period, subjects came out of the chamber at the usual breakfast time, but instead of eating, were settled in a chair prior to removal of their masks. They were asked to provide the experimenters with a running commentary on what they saw, how they felt, and so on, after the mask was removed. In addition, they were asked to write an essay describing their experiences during the deprivation period.

D EEG Analysis

The polygraph records were visually analysed in to the following categories: Awake, Stage I sleep, Stage II sleep, Stage III sleep, Stage IV sleep, and Stage I REM sleep. This is the classification system proposed by Dement and Kleitman (1957) and, for the most part, their definitions of the various stages were used as well. These are as follows: the essential characteristic of stage I is an absolute lack of spindle activity. In general, it is a low voltage, relatively fast pattern; any activity between full wakefulness and the appearance of spindles is included in stage I. Stage II is characterized by the presence of spindle activity with a low voltage background, including a small amount of slower activity in the 3-6 per second range. K-complexes occur in this stage. Stage III is an intermediate

stage characterized by the appearance of high voltage slow waves with some spindling superimposed. For borderline cases, records with an average of less than two waves over 100 μv. and 1-2 hz. or slower in ten seconds are assigned to stage II. Records with more than half over 100 μv. and 1-2 hz. or slower are assigned to Stage IV. Stage I REM, as the name implies, is characterized by a stage I EEG in conjunction with episodes of rapid eye movements. The submental EMG is also depressed during this stage.

Several of the modifications of this classification scheme proposed by Rechtschaffen and Kales (1968) were found to be helpful. For example, the present investigators felt that activity that clearly was stage I but for the occasional brief low amplitude spindle or K-complex was more properly classified as stage I than stage II. Accordingly, the criterion suggested by Rechtschaffen and Kales (1968) to deal with these borderline states was adopted; provided that the spindle burst or K-complex did not exceed 0.5 sec. duration, its inclusion in stage I was justified. Also the amplitude criterion for stages III and IV was lowered from 100 μv. to 75 μv. by these authors. Since waves of 100 μv. were not commonly seen even in the records of stage IV sleep during the present experiment, possibly because a reference electrode ipsilateral to the active electrode was used, the 75 μv. criterion was used.

Each page of the polygraph record, representing 20 sec., was assigned to one of these categories. In addition, a further breakdown of stage I REM sleep into periods with and without eye movements, named, respectively, REM sleep and NM sleep (for No

Muscle activity), was made. Following this page by page analysis, the amount of time spent in each EEG stage was converted to a percentage. For deprivation, these percentages were calculated over 24 hour periods which extended from 0800 hours to 0800 hours. In the case of the pre- and postdeprivation sleep nights and the first night of deprivation, percentages were also based on 24 hour periods.

E Reliability of EEG Analysis

Since all of the polygraph records were scored by one individual, it was necessary to have some measure of the reliability of this individual's analysis. Accordingly, for each of the eight subjects, 50 pages which had been assigned to each of the six EEG stages by the first individual were randomly selected for rescoring by a second rater. Thus, 2400 pages were reanalysed, and these pages compared for agreement or disagreement between the two raters.

CHAPTER 4

RESULTS

The comparison between the analyses of the two individuals who rated the polygraph records will be presented first, followed by the experimental results.

Table 1 shows, on the diagonal, the number of pages independently rated as belonging to the same EEG stage, and for each stage expresses this number as a percentage of the total pages rated. It can be seen that the agreement between the two raters is excellent; the total number of pages similarly rated was 1988 out of 2400, or approximately 83%. This consistency was not unexpected, since Monroe (1969) has shown that the scores of 28 different raters for a single sleep record also agree relatively well, though these individuals are from 14 different laboratories.

Figures 2 to 7 and Tables 2 to 8 represent the amounts of time, expressed as percentages of 24 hour periods, spent in the various EEG stages by all subjects. These will be dealt with in turn.

Figure 2 and Table 2 show the time spent in the awake state, the 7-day group being on the left of the figure and the 4-day group on the right. During the predeprivation period, subjects spent an average of 72.0% of each day awake, and,

TABLE 1

Comparison between ratings of two individuals for 2400 pages of EEG records. Numbers refer to the total pages similarly or dissimilarly rated.

RATER NUMBER 2	RATER NUMBER 1					
	Awake	St. I	St. I REM	St. II	St. III	St. IV
Awake	380 95%	76	2	1		
St. I	18	296 74%	6	25		
St. I REM	2	19	382 96%	6	1	
St. II		9	10	342 86%	63	2
St. III				25	260 65%	70
St. IV				1	78	328 82%

Total number of pages similarly rated: 1988 or 83%.

FIGURE 2

Time spent in the awake state by each subject, as a percentage of 24 hours.

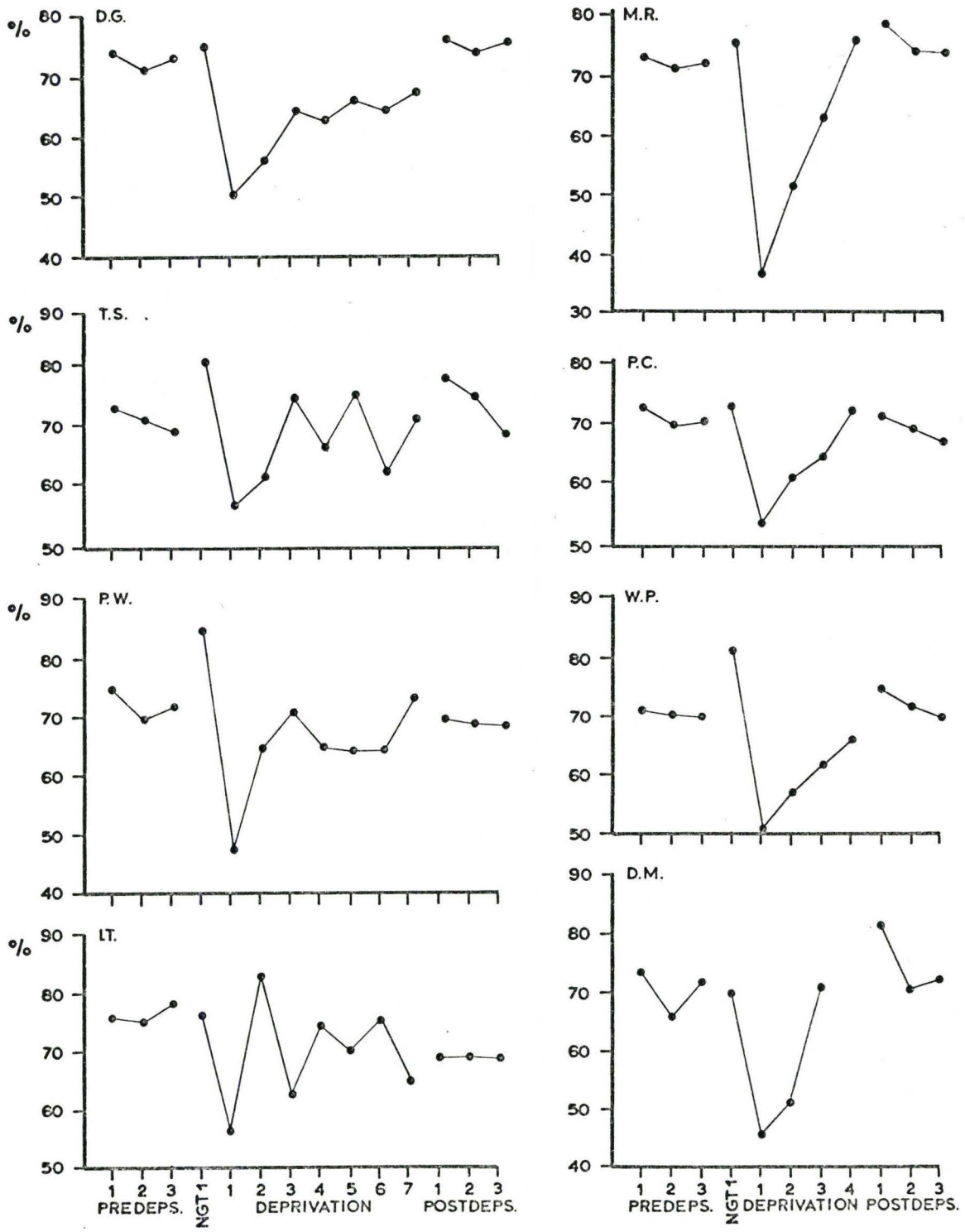

%
D.G.
M.R.
T.S.
P.C.
P.W.
W.P.
LT.
D.M.
PREDEPS.
NGT1
DEPRIVATION
POSTDEPS.
AWAKE

TABLE 2

Time spent by each subject in the awake state, as a percentage of 24 hours.

	Predeps.			Deprivation								Postdeps.		
Subject	1	2	3	Nt.1	1	2	3	4	5	6	7	1	2	3
D.G.	74.3	71.5	73.2	75.2	50.4	56.3	64.8	63.1	66.2	64.9	67.8	76.6	74.2	76.1
T.S.	74.0	71.9	69.7	81.9	57.4	62.4	75.5	67.1	76.4	63.7	72.0	79.1	76.3	69.8
P.W.	75.2	70.0	72.1	85.3	47.4	64.7	70.7	64.8	64.1	64.3	73.1	69.9	69.0	68.8
I.T.	76.1	75.0	77.9	76.4	56.1	83.0	63.4	74.4	70.2	75.6	65.2	69.6	69.6	69.2
M.R.	72.6	70.2	71.3	74.7	36.2	51.0	62.9	75.2				78.3	73.5	73.4
P.C.	71.8	69.2	69.4	72.0	52.4	60.2	63.8	71.7				71.0	68.8	66.4
W.P.	71.1	70.3	69.7	81.4	51.0	56.7	61.4	65.6				74.5	71.9	69.8
D.M.	73.8	66.3	72.1	69.8	45.7	51.4	70.7					81.5	70.6	72.6
Average	73.6	70.6	71.9	77.1	49.6	60.7	66.7	68.8	69.2	67.1	69.5	75.1	71.7	70.8

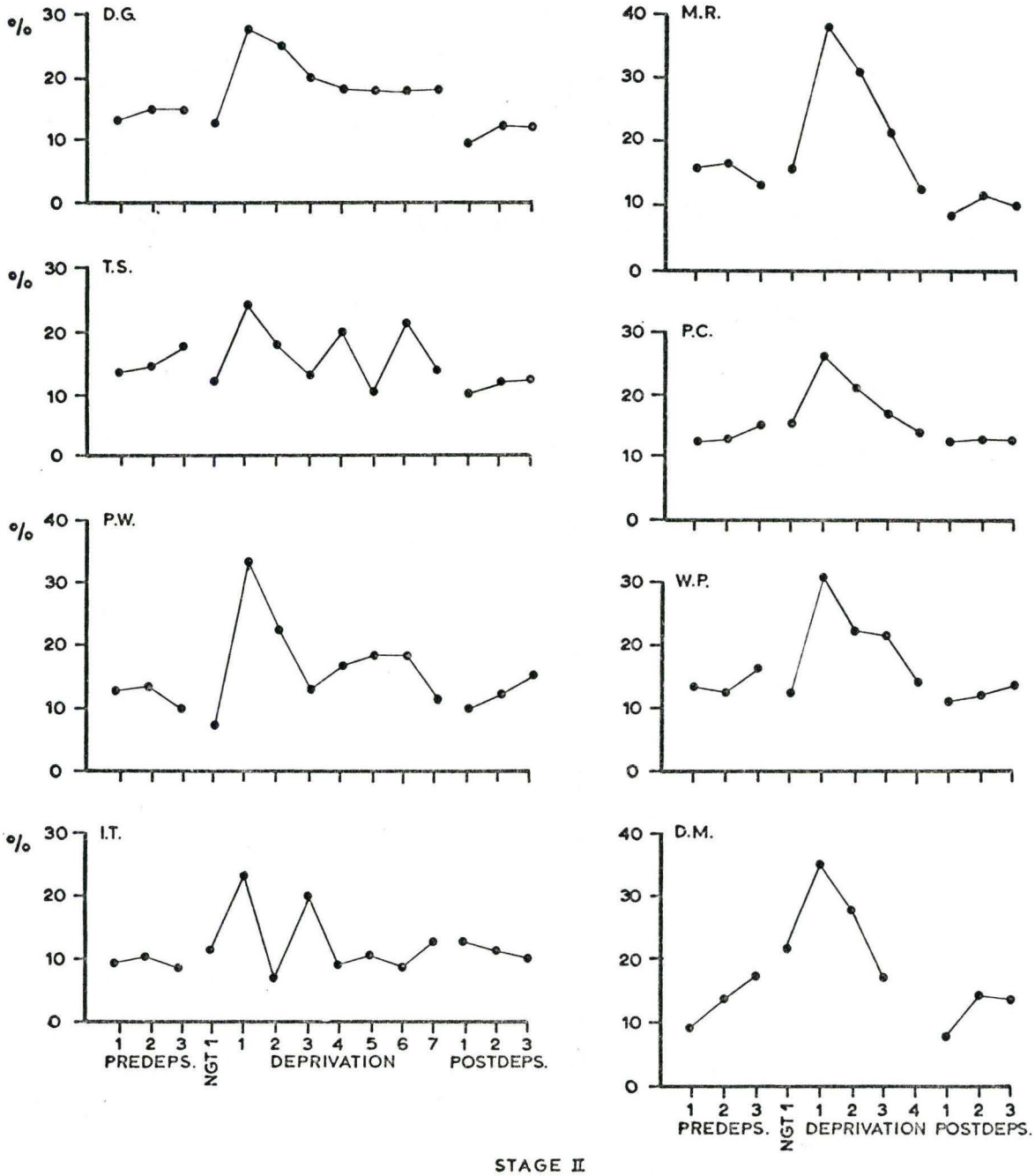
consequently, slept for 28.0%. For the postdeprivation period, these values were much the same, and represent a normal night's sleep of approximately seven hours.

Generally, subjects also slept a normal amount on the first night of deprivation. Three subjects (T.S., P.W., and W.P.) showed small elevations in the time awake on this first night; that is, they slept less than a normal amount. On day 1 of deprivation, the time awake dropped quite sharply, and on the average, subjects only remained awake for 50% of the time. As deprivation progressed, there was a gradual recovery from this excessive sleep and a return to the normal amount of awake time by day 3 or 4. For the additional deprivation days undergone by the 7-day group, the awake time remained relatively constant, showing no systematic tendencies toward further increases or decreases. Subject I.T. was the single exception to this pattern. He slept very poorly on the third night of deprivation, and this is reflected in the high value for awake time on day 2 and in the general irregularity of the entire graph.

Most of this extra sleep time early in deprivation appears to have been spent in stage II, which is plotted in Figure 3 and Table 3. Normal values for this stage, obtained on the three predeprivation nights and expressed as percentages of 24 hours, average 13.5%; that is, about three hours out of every 24 were normally spent in stage II. Values for the three postdeprivation nights were very similar. The mean amount of time spent in stage II was 12.0% of 24 hours. On the first night of deprivation

FIGURE 3

Time spent in stage II sleep by each subject, as a percentage of 24 hours.



STAGE II
TOTAL TIME

TABLE 3

Time spent by each subject in stage II sleep, as a percentage of 24 hours.

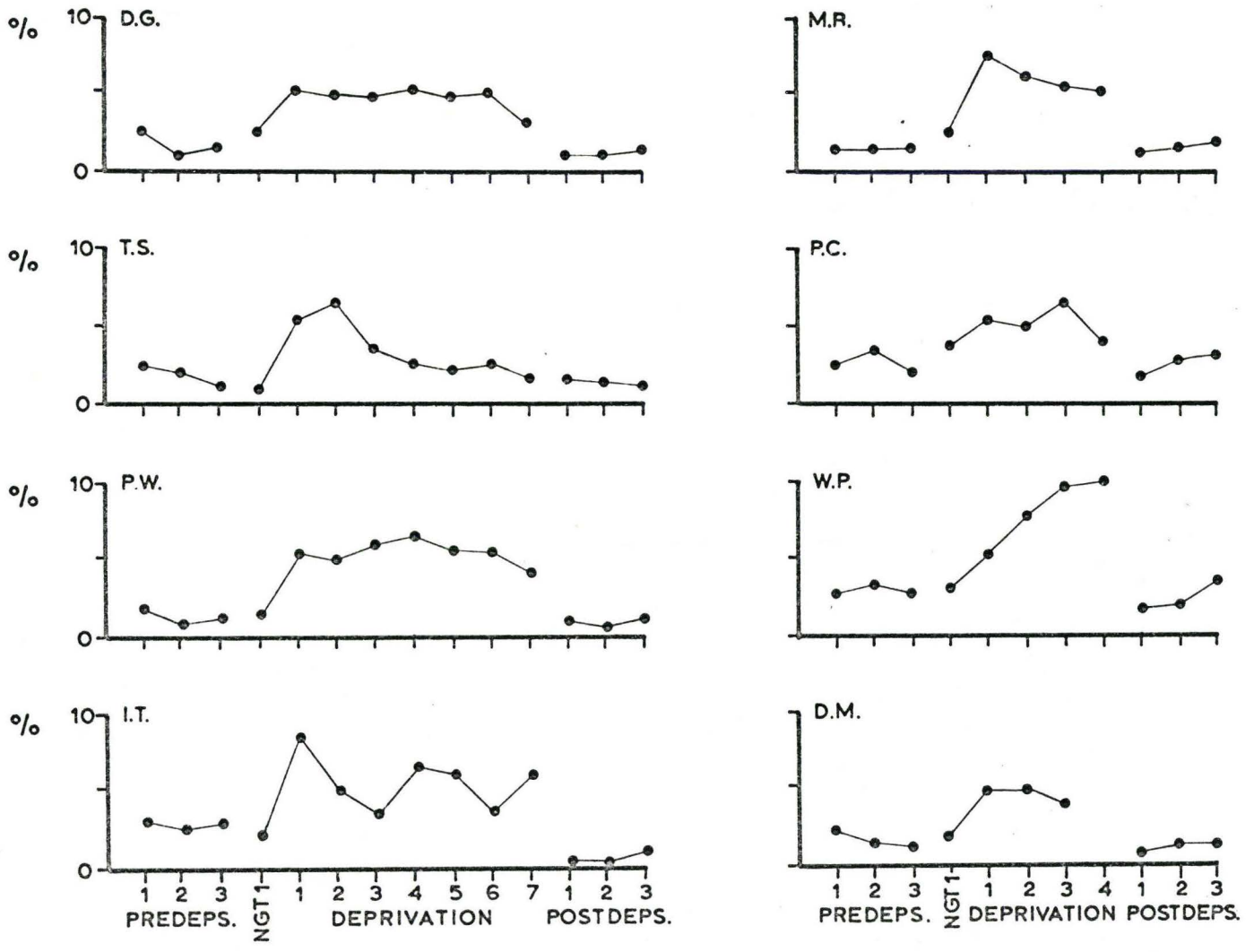
	Predeps.			Deprivation								Postdeps.		
Subject	1	2	3	Nt.1	1	2	3	4	5	6	7	1	2	3
D.G.	13.1	14.7	14.4	12.4	27.5	24.8	19.9	18.3	18.0	17.8	18.2	9.5	12.3	11.9
T.S.	13.5	14.4	17.5	11.8	23.9	17.6	12.8	19.5	10.3	21.2	13.9	10.1	12.0	12.2
P.W.	12.9	13.8	10.1	7.5	33.4	22.6	13.0	17.1	18.4	18.4	11.4	10.3	12.9	15.8
I.T.	9.5	10.6	8.4	11.9	23.2	7.2	20.3	9.4	11.2	9.1	13.2	13.3	12.0	10.7
M.R.	15.5	16.0	13.0	15.7	38.5	31.0	21.4	12.7				8.9	11.7	11.2
P.C.	12.4	13.1	15.3	15.2	25.7	20.8	17.1	13.7				12.7	13.1	13.0
W.P.	14.0	12.9	16.5	12.4	31.0	22.6	20.7	14.4				11.2	12.1	13.5
D.M.	9.6	14.0	17.8	21.8	35.3	27.8	17.4					8.5	14.5	14.0
Average	12.6	13.7	14.1	13.6	29.8	21.8	17.8	15.0	14.5	16.6	14.2	10.6	12.6	12.8

individual subjects showed small changes either up or down from their baseline values, but these were not consistently in the same direction. Generally, night 1 seemed much the same as the pre- and postdeprivation nights; a mean value for stage II of 13.6% was obtained. Day 1, however, showed a large increase to levels well above baseline for all subjects, with the mean value being 29.8%. Restated, subjects spent an average of 7.2 hours in stage II sleep on the first day of deprivation, roughly two and one-half times as much as they did normally. On the following days of the deprivation period, stage II values systematically decreased until they reached baseline levels by day 3 or 4. For the 7-day subjects, these baseline values were maintained relatively constant until deprivation was terminated. This stage II pattern of day 1 increase, gradual decrease, and subsequent levelling off parallels quite precisely the changes occurring in time spent awake. Again, the failure of subject I.T. to show this pattern is attributable to his extremely restless night on day 2. It is interesting to note that subject D.G., although showing the typical pattern, did not return to baseline levels late in deprivation, but instead maintained stage II at a slightly elevated value. Consistent with this is the finding that this subject did not return to baseline amounts of awake time either. Rather, he consistently spent slightly less than his baseline amount of time awake during the last four days of deprivation.

Changes in stage I sleep are shown in Figure 4 and Table 4.

FIGURE 4

Time spent in stage I sleep by each subject, as a percentage of 24 hours.



STAGE I
TOTAL TIME

TABLE 4

Time spent by each subject in stage I sleep, as a percentage of 24 hours.

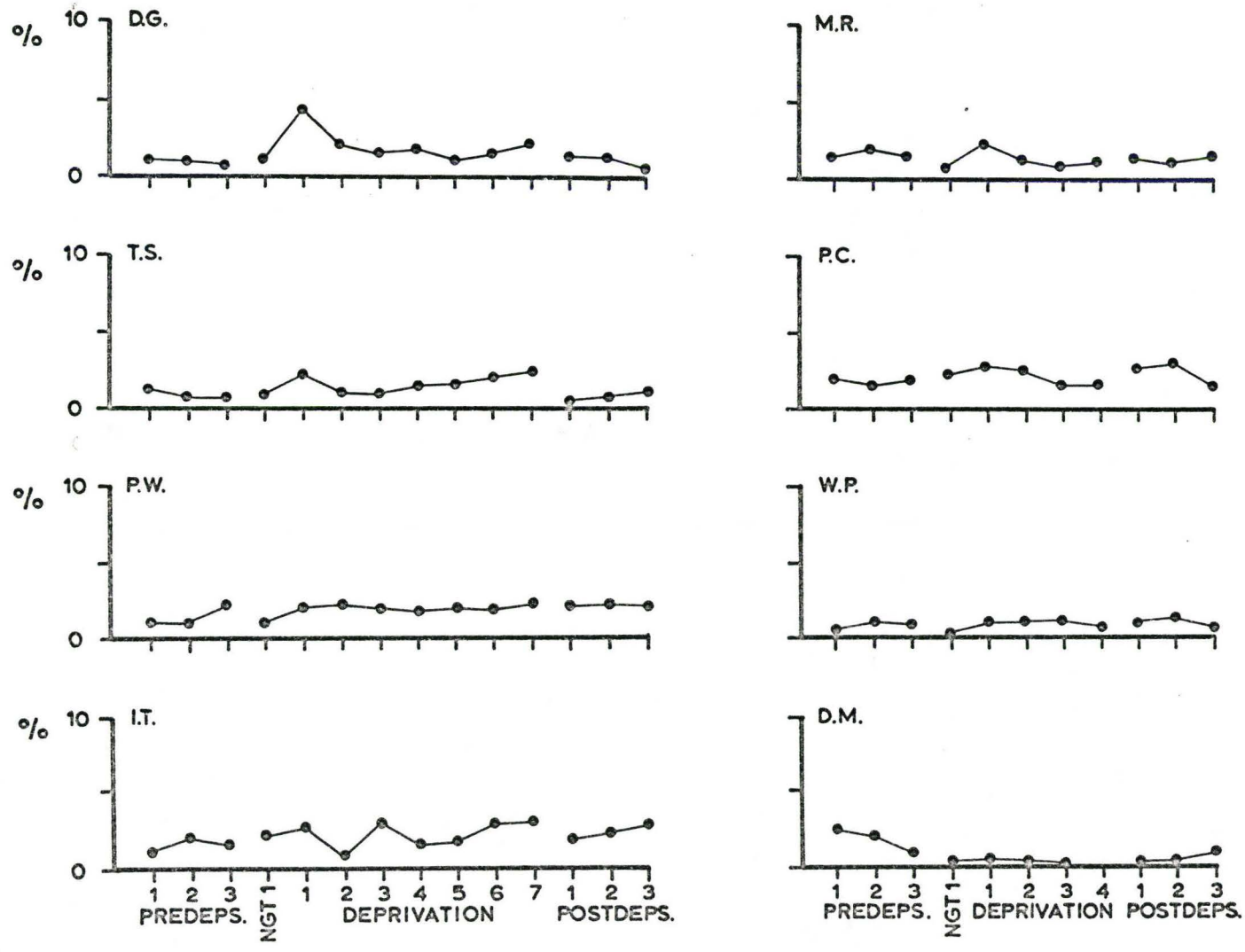
	Predeps.			Deprivation								Postdeps.		
Subject	1	2	3	Nt.1	1	2	3	4	5	6	7	1	2	3
D.G.	2.4	1.0	1.4	2.5	5.3	4.9	4.8	5.4	4.8	5.1	3.1	1.0	1.0	1.3
T.S.	2.6	2.1	1.3	1.3	5.9	6.5	3.1	2.3	2.0	2.3	1.7	1.8	1.2	.9
P.W.	2.1	.8	1.3	1.5	5.4	5.1	5.9	6.8	5.8	5.6	4.1	1.3	.7	1.3
I.T.	3.1	2.4	2.7	2.2	8.6	4.9	3.4	6.4	5.9	3.7	6.1	.3	.4	1.2
M.R.	1.5	1.6	1.3	2.5	7.7	6.3	5.8	5.2				1.2	1.6	1.9
P.C.	2.8	3.6	2.2	3.7	5.8	5.2	6.7	4.0				1.9	2.8	3.3
W.P.	2.9	3.2	2.8	3.3	5.3	7.9	9.5	10.0				1.8	2.1	4.0
D.M.	2.3	1.5	1.3	1.8	5.2	5.2	3.8					1.3	1.6	1.5
Average	2.5	2.0	1.8	2.4	6.2	5.8	5.4	5.7	4.6	4.2	3.8	1.3	1.4	1.9

The amounts of time spent in this stage were much the same on the pre- and postdeprivation nights; mean values were 2.1% and 1.6% respectively, indicating that approximately 30 minutes out of 24 hours was typically spent in stage I. Values obtained on the first night of deprivation were consistent with these baseline levels, the mean for all subjects being 2.4%. During the rest of the deprivation period, most subjects tended to show a small and relatively constant increase in stage I time. Exceptions are subject T.S., who showed an early increase and then a decrease to approximately baseline level, and subject W.P., who showed a linear increase in this stage as deprivation progressed. On the whole, however, stage I time appeared to be increased on the first day of deprivation to a level that was two and one-half to three times baseline and to be maintained at this level throughout. That is, the normal 30 minutes of each day that was spent in stage I was increased to 75 - 90 minutes during deprivation.

The effect of deprivation on slow-wave sleep, or stages III and IV, will be dealt with next. Figure 5 and Table 5 show stage III. The amounts of time spent in this stage during the pre- and postdeprivation periods were very similar. The mean value for both of these was 1.4%. There was virtually no change from the baseline level for any subject on the first night of deprivation, nor during the subsequent days. Stage III appears to be maintained at normal levels during the entire deprivation period, as can be seen in Table 5. Stage IV sleep is shown in

FIGURE 5

Time spent in stage III sleep by each subject, as a percentage of 24 hours.



STAGE III
TOTAL TIME

TABLE 5

Time spent by each subject in stage III sleep, as a percentage of 24 hours.

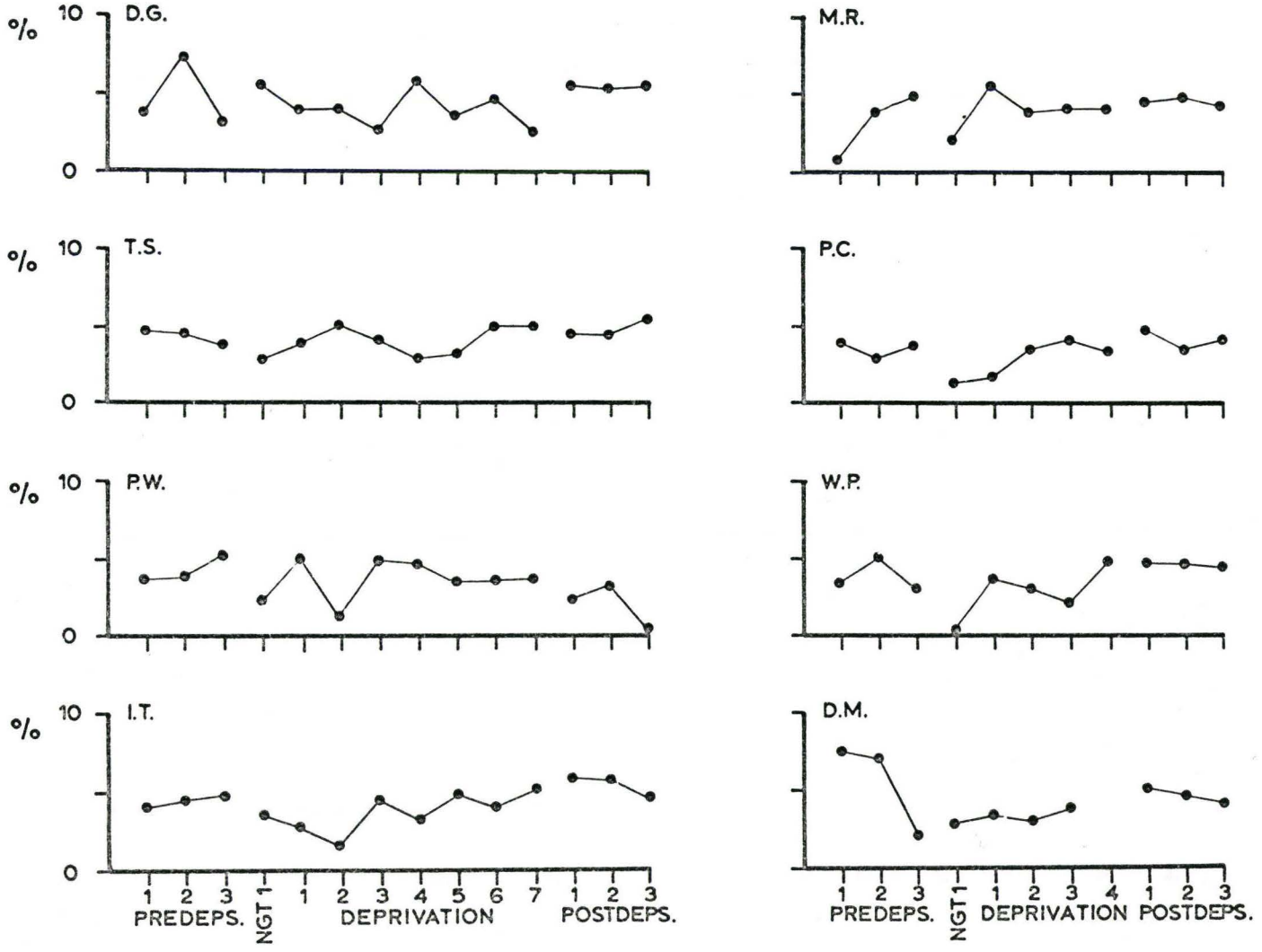
	Predeps.			Deprivation								Postdeps.		
Subject	1	2	3	Nt.1	1	2	3	4	5	6	7	1	2	3
D.G.	1.2	1.1	.7	1.1	4.3	2.2	1.5	1.8	1.0	1.6	2.2	1.3	1.3	.6
T.S.	1.3	.7	.6	1.1	2.3	1.0	1.0	1.6	1.5	2.0	2.6	.4	.7	1.1
P.W.	1.2	1.2	2.3	1.2	2.0	2.3	2.0	1.7	2.1	1.9	2.2	2.2	2.1	2.0
I.T.	1.2	2.2	1.6	2.3	2.7	.9	3.0	1.7	1.8	2.9	3.0	1.8	2.2	2.7
M.R.	1.5	1.9	1.4	.8	2.3	1.3	.8	1.1				1.3	1.1	1.4
P.C.	1.8	1.5	1.8	2.3	2.7	2.5	1.5	1.6				2.6	3.0	1.4
W.P.	.5	.9	.8	.3	1.1	1.1	1.2	.7				1.1	1.3	.5
D.M.	2.6	2.1	1.1	.4	.5	.4	.2					.1	.2	.6
Average	1.4	1.5	1.3	1.2	2.2	1.5	1.4	1.5	1.6	2.1	2.5	1.4	1.5	1.3

Figure 6 and Table 6. As with stage III, there was good agreement between the pre- and postdeprivation periods with regard to the amounts of time spent in stage IV. The mean percentage for all subjects on the predeprivation nights was 4.2%, and on the postdeprivation nights, 4.4%. On the first night of deprivation, there was a tendency for stage IV to go slightly below these baseline levels; the average amount of time spent in this stage was 2.6% of 24 hours. For the rest of the deprivation period, however, stage IV, like stage III, seemed to change very little. Individual subjects showed small fluctuations in the amounts of time they spent in this stage, but no systematic change could be detected.

The only sleep stage not yet reviewed is stage I REM, which is shown in Figure 7 and in Tables 7, 8, and 9. While all subjects responded to deprivation with the same general pattern of changes in the other sleep stages, they varied considerably with respect to stage I REM. The mean percentage of 24 hours spent in this stage on the predeprivation nights was 6.9%, or approximately 1.6 hours. All subjects showed a decline on night 1 of deprivation, and an increase on the first day, possibly because of some rebound effect. After this, however, there was considerable variability. Thus, all 4-day subjects showed a drop in the amount of time spent in stage I REM as deprivation progressed, and two of the 7-day subjects, T.S. and P.W., showed this same tendency early in deprivation. These two, however, displayed a later increase and then appeared to

FIGURE 6

Time spent in stage IV sleep by each subject, as a percentage of 24 hours.



STAGE IV
TOTAL TIME

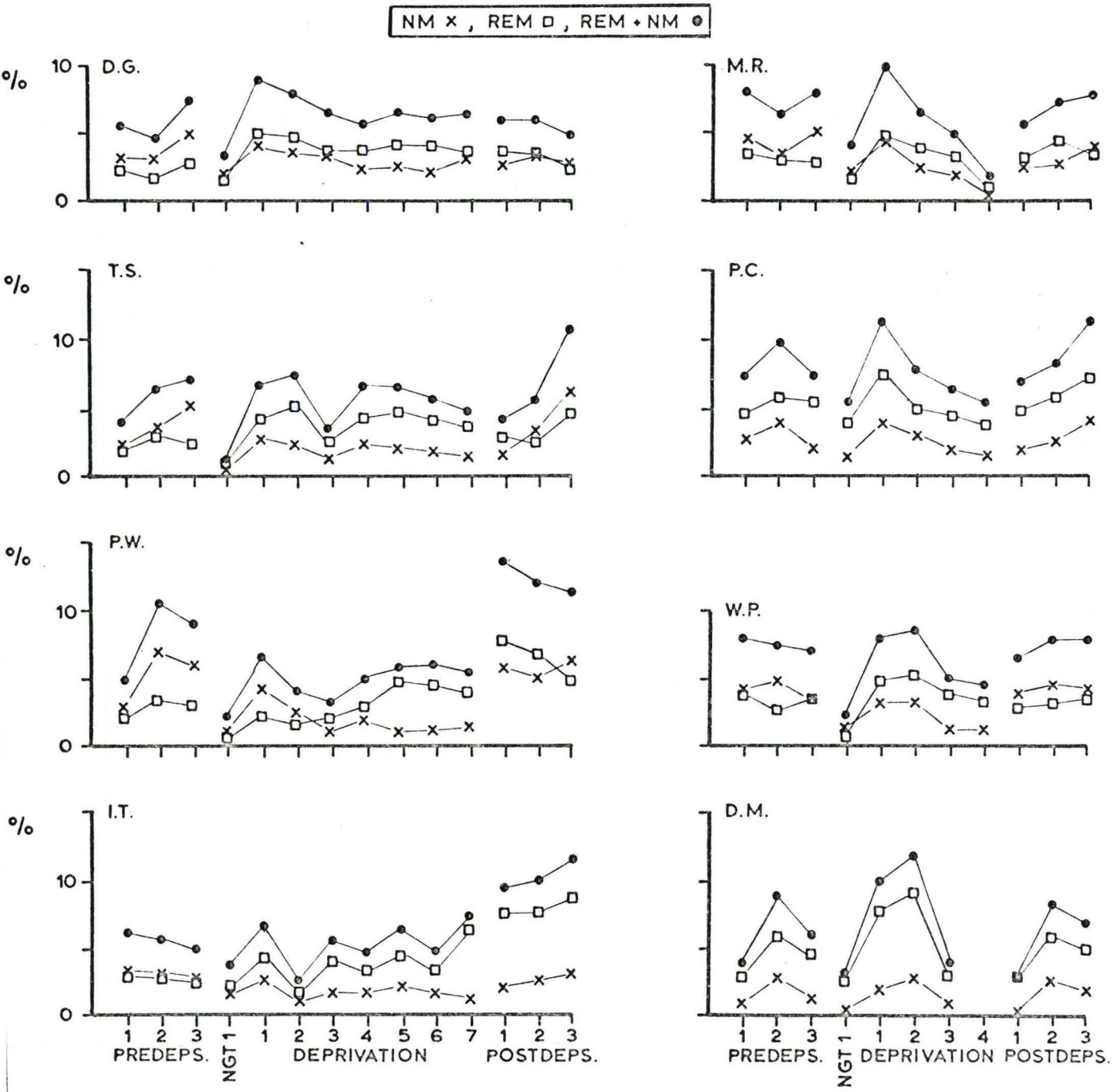
TABLE 6

Time spent by each subject in stage IV sleep, as a percentage of 24 hours.

	Predeps.			Deprivation								Postdeps.		
Subject	1	2	3	Nt.1	1	2	3	4	5	6	7	1	2	3
D.G.	3.7	7.2	2.9	5.4	3.8	3.9	2.6	5.7	3.5	4.5	2.3	5.4	5.3	5.4
T.S.	4.6	4.4	3.8	2.8	3.8	5.1	4.2	3.0	3.3	5.0	5.1	4.5	4.4	5.4
P.W.	3.6	3.8	5.2	2.4	5.0	1.3	5.0	4.7	3.7	3.7	3.8	2.6	3.3	.7
I.T.	4.1	4.5	4.8	3.6	2.8	1.6	4.5	3.3	4.8	4.0	5.2	5.8	5.7	4.6
M.R.	.7	3.8	4.8	2.1	5.6	3.8	4.0	4.0				4.5	4.7	4.2
P.C.	3.8	2.8	3.7	1.3	1.7	3.4	4.1	3.3				4.7	3.5	4.2
W.P.	3.6	5.2	3.1	.3	3.7	3.1	2.2	4.8				4.7	4.6	4.4
D.M.	7.7	7.0	1.4	2.8	3.4	3.1	3.9					5.2	4.4	4.3
Average	4.0	4.8	3.7	2.6	3.7	3.2	3.8	4.1	3.8	4.3	4.1	4.7	4.5	4.2

FIGURE 7

Time spent in stage I REM sleep, and in its two subcategories, REM sleep and NM sleep, by each subject, as a percentage of 24 hours.


NM × , REM □ , REM + NM ⊙
%
10
0
D.G.
M.R.
T.S.
P.C.
P.W.
W.P.
I.T.
D.M.
1 2 3
PREDEPS.
NGT 1
1 2 3 4 5 6 7
DEPRIVATION
1 2 3
POSTDEPS.

TOTAL TIME

TABLE 7

Time spent by each subject in stage I REM sleep, as a percentage of 24 hours.

	Predeps.			Derrivation							Postdeps.		
Subject	1	2	3	Nt.1	1	2	3	4	5	6	7	1	2	3
D.G.	5.4	4.5	7.3	3.3	8.8	7.8	6.4	5.7	6.5	6.1	6.4	6.0	5.9	4.8
T.S.	4.1	6.4	7.1	1.2	6.7	7.4	3.4	6.5	6.5	5.7	4.7	4.1	5.5	10.6
P.W.	5.0	10.5	9.1	2.2	6.7	4.1	3.3	5.0	5.9	6.0	5.4	13.7	12.0	11.4
I.T.	6.0	5.4	4.7	3.6	6.5	2.4	5.5	4.7	6.2	4.7	7.3	9.3	10.0	11.6
M.R.	8.2	6.5	8.2	4.2	9.8	6.7	5.1	1.8				5.8	7.4	7.9
P.C.	7.4	9.8	7.6	5.6	11.6	8.1	6.7	5.7				7.1	8.7	11.7
W.P.	7.8	7.4	7.1	2.3	8.0	8.6	5.0	4.5				6.6	7.9	7.8
D.M.	4.1	9.1	6.4	3.3	10.0	12.1	4.0					3.3	8.8	7.1
Average	6.0	7.5	7.2	3.2	8.5	7.2	4.9	4.8	6.3	5.6	6.0	7.0	8.3	9.1

TABLE 8

Time spent by each subject in the NM subcategory of stage I REM sleep, as a percentage of 24 hours.

	Predeps.			Deprivation								Postdeps.		
Subject	1	2	3	Nt.1	1	2	3	4	5	6	7	1	2	3
D.G.	3.2	3.0	4.7	1.9	4.1	3.4	3.3	2.1	2.5	2.0	3.0	2.4	3.0	2.5
T.S.	2.2	3.5	5.0	.4	2.7	2.3	1.1	2.3	1.9	1.6	1.2	1.4	3.2	6.2
P.W.	2.8	7.0	6.0	1.2	4.4	2.4	1.1	1.9	1.1	1.3	1.3	5.9	5.1	6.4
I.T.	2.8	2.8	2.4	1.7	2.5	.9	1.7	1.5	2.0	1.5	1.0	1.9	2.5	3.1
M.R.	4.6	3.4	5.3	2.2	4.8	2.6	2.2	.6				2.5	2.8	4.3
P.C.	2.7	3.9	2.2	1.6	4.0	3.1	2.1	1.6				2.1	2.7	4.2
W.P.	4.0	4.8	3.6	1.3	3.2	3.3	1.2	1.2				3.8	4.7	4.3
D.M.	1.0	2.9	1.4	.6	2.0	2.8	1.0					.3	2.8	2.1
Average	2.9	3.9	3.8	1.4	3.5	2.6	1.7	1.6	1.9	1.6	1.6	2.5	3.4	4.1

TABLE 9

Time spent by each subject in the REM subcategory of stage I REM sleep, as a percentage of 24 hours.

	Predeps.			Deprivation								Postdeps.		
Subject	1	2	3	Nt.1	1	2	3	4	5	6	7	1	2	3
D.G.	2.2	1.5	2.6	1.4	4.7	4.4	3.1	3.6	4.0	4.1	3.4	3.6	2.9	2.3
T.S.	1.9	2.9	2.1	.8	4.0	5.1	2.3	4.2	4.6	4.1	3.5	2.7	2.3	4.4
P.W.	2.2	3.5	3.1	1.0	2.3	1.7	2.2	3.1	4.8	4.7	4.1	7.8	6.9	5.0
I.T.	3.2	2.6	2.3	1.9	4.0	1.5	3.8	3.2	4.2	3.2	6.3	7.4	7.5	8.5
M.R.	3.6	3.1	2.9	2.0	5.0	4.1	2.9	1.2				3.3	4.6	3.6
P.C.	4.7	5.9	5.4	4.0	7.6	5.0	4.6	4.1				5.0	6.0	7.5
W.P.	3.8	2.6	3.5	1.0	4.8	5.3	3.8	3.3				2.8	3.2	3.5
D.M.	3.1	6.2	5.0	2.7	8.0	9.3	3.0					3.0	6.0	5.0
Average	3.1	3.5	3.4	1.9	5.1	4.6	3.2	3.2	4.4	4.0	4.3	4.5	4.9	5.0

maintain stage I REM at a relatively constant level until deprivation was terminated. The other two 7-day subjects, D.G. and I.T., showed virtually no change in this stage during deprivation.

Postdeprivation values for this stage also differed widely from subject to subject. Two individuals, P.W. and I.T., showed excessive amounts of stage I REM on these nights. The other two 7-day subjects, D.G. and T.S., showed similar values for the pre- and postdeprivation periods, as did the entire 4-day group.

As described in Chapter 3, stage I REM was further analysed into two subcategories, the first called REM sleep for periods with active eye movements (Figure 7, open squares; Table 9), and the second, NM sleep for periods without eye movements (Figure 7, crosses; Table 8). When the relationship between these two subcategories is examined, it is apparent that five of the eight subjects (D.G., T.S., P.W., M.R., and W.P.) spent more time in NM sleep than they did in REM sleep during the predeprivation period. A sixth subject, I.T., showed approximately equal amounts of these two subcategories. During deprivation, these six subjects showed a reversal of this relationship, with REM sleep occupying more of the stage I REM time than did NM sleep. In four subjects (D.G., P.W., M.R., and W.P.), this crossover effect occurred during the deprivation period, while in the remaining two (T.S. and I.T.), it was present even on the first night of deprivation. Moreover, this reversal is clearly an effect of deprivation, for

the relationship between REM sleep and NM sleep returned to the predeprivation state during the postdeprivation period, with subjects (I.T. is the single exception) again spending more time in NM sleep than in REM sleep. This pattern was not present in subjects P.C. and D.M. of the 4-day group. Both displayed a greater amount of REM sleep than NM sleep throughout the three phases of the experiment.

By way of summary, Figure 8 shows changes in each EEG stage for all 7-day subjects, and Figure 9, the same for all 4-day subjects. As with the previous figures, amounts of time spent in the various stages are plotted as percentages of 24 hours. Both groups spent much less than the normal amount of time awake on day 1 of deprivation, but gradually returned to a normal sleep-wakefulness cycle as deprivation progressed. Paralleling this decrease, then increase in awake time was the reverse pattern for stage II sleep. Most of the extra sleep early in deprivation appears to consist of this stage, but it returned to baseline levels at the same rate as the awake time. Both groups showed a small increase in stage I, which was maintained quite constant throughout deprivation. The curves for slow-wave sleep, or stages III and IV, are quite flat throughout deprivation and compare well with pre- and postdeprivation curves in all cases. These stages appear unaffected by deprivation. Both groups showed a decrease in stage I REM on night 1 of deprivation, and an increase on day 1. The 4-day subjects clearly displayed a depression in this stage as deprivation progressed,

FIGURE 8

Time spent in each EEG stage for all 7-day subjects, expressed as a percentage of 24 hours.

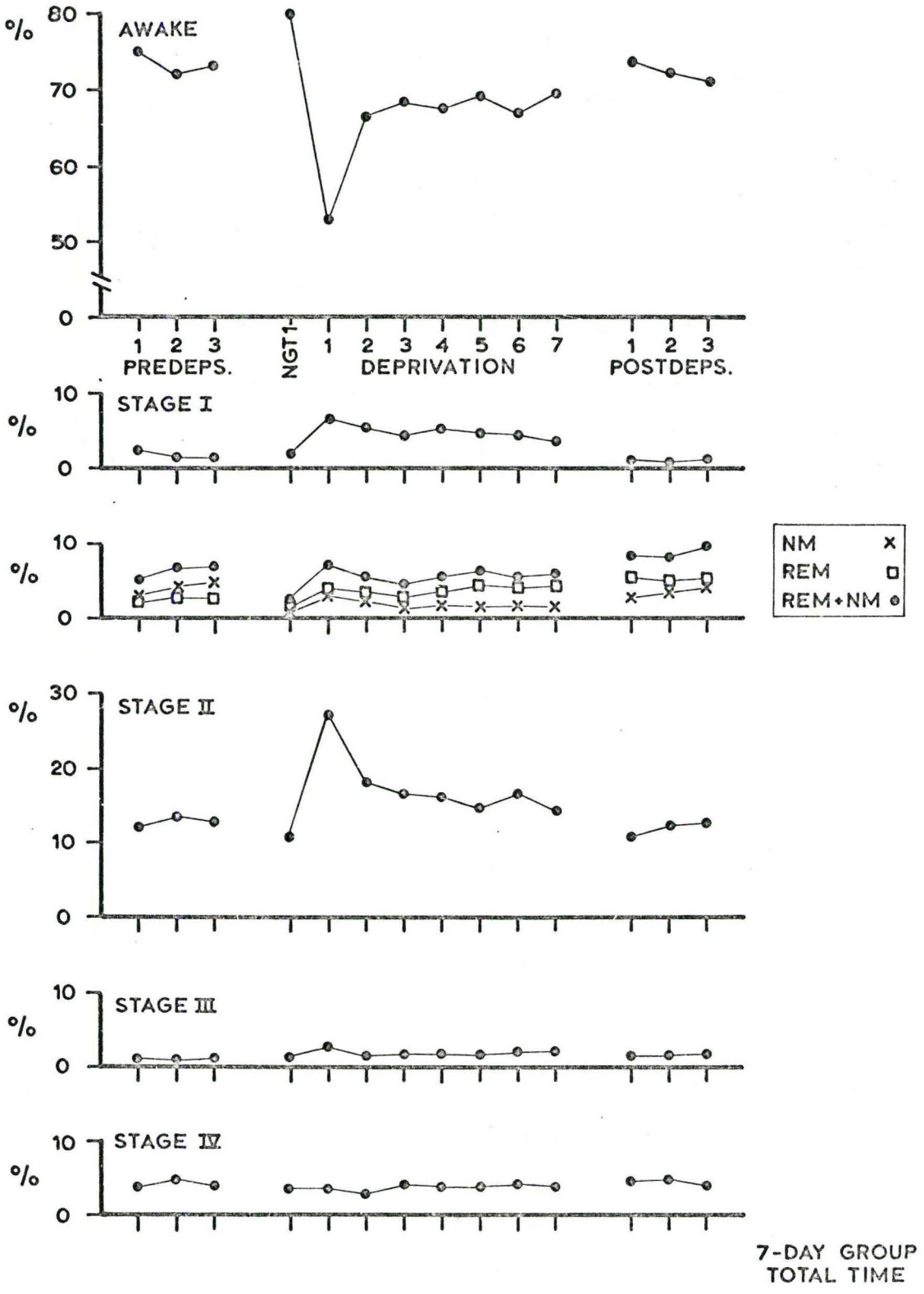
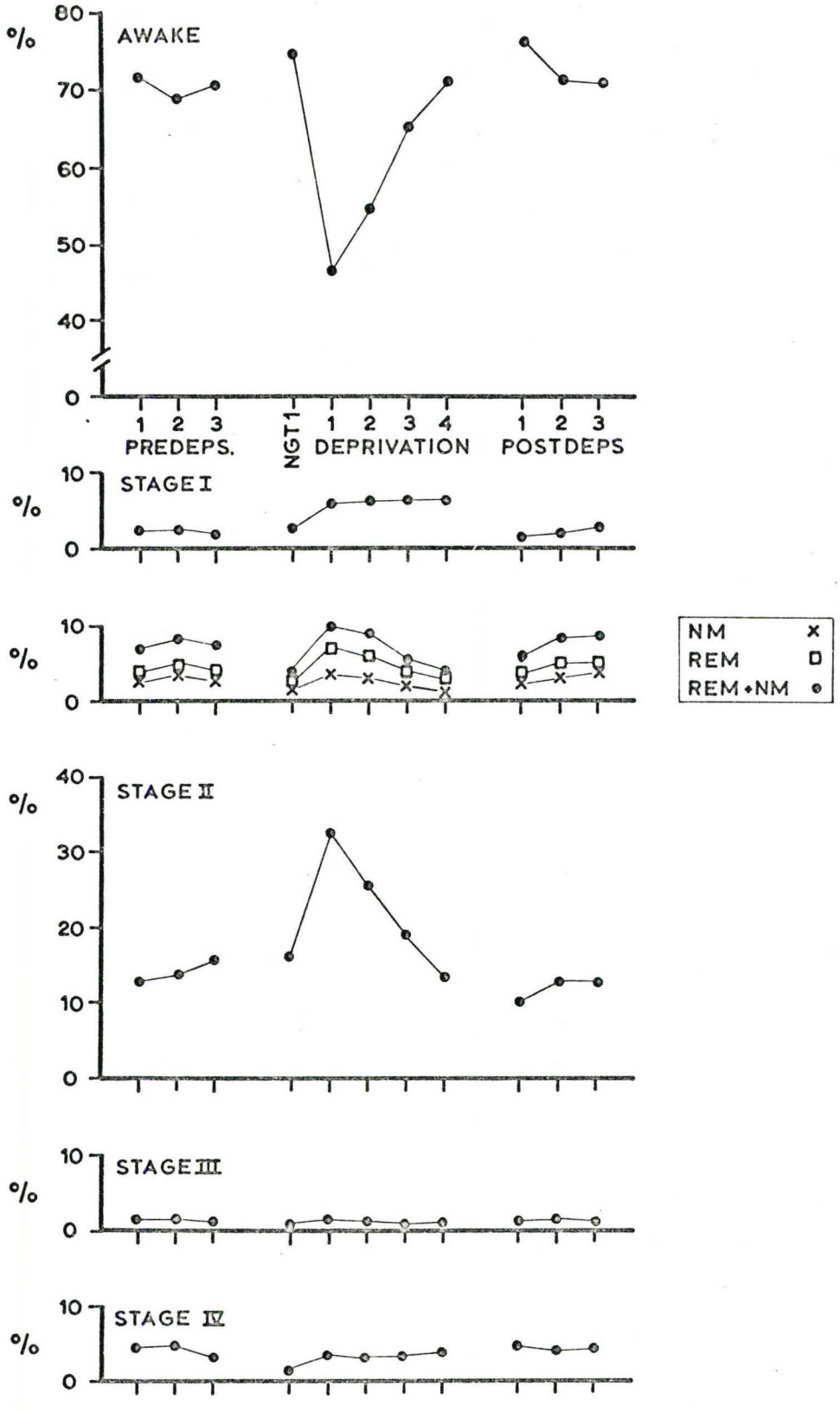

%
80
70
60
50
0
AWAKE
1 2 3
PREDEPS.
NGT1
1 2 3 4 5 6 7
DEPRIVATION
1 2 3
POSTDEPS.
10
0
STAGE I
NM
REM
REM+NM
30
20
STAGE II
STAGE III
STAGE IV
7-DAY GROUP
TOTAL TIME

FIGURE 9

Time spent in each EEG stage for all 4-day subjects, expressed as a percentage of 24 hours.



4-DAY GROUP
TOT. TIME

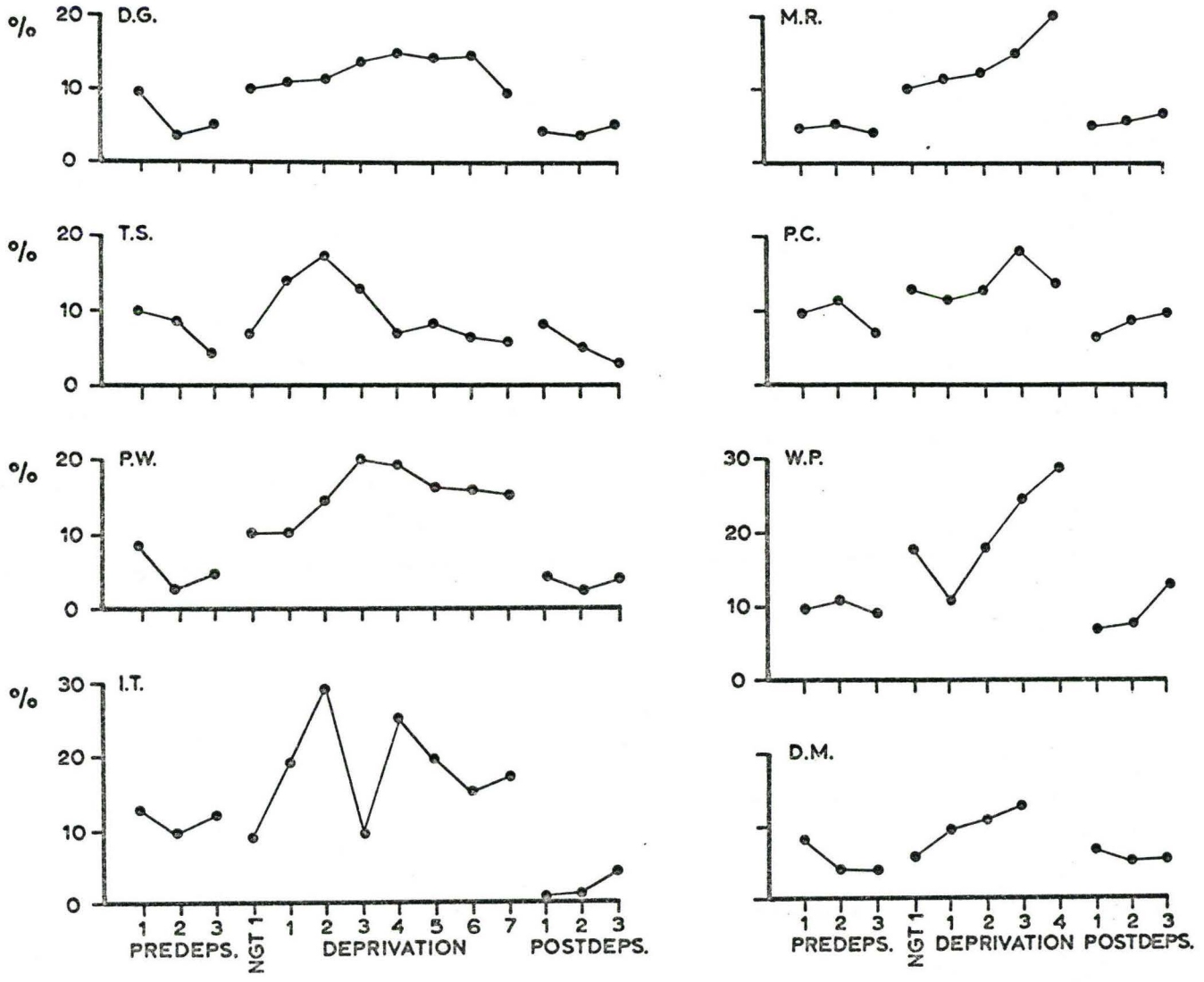
with this change being only hinted at in the 7-day group. On the other hand, the previously discussed crossover effect, with REM sleep occupying more time than NM sleep during deprivation but not during the pre- or postdeprivation periods, was shown only by the 7-day subjects.

Rather than calculating the amount of time spent in each EEG stage as a percentage of a 24 hour period, most sleep investigators prefer to use the total amount of time spent in sleep. For example, a subject who spent 1.6 hours in stage I REM out of 7 hours of sleep would have spent 22.8% of this sleep time in this stage. The comparable percentage of a 24 hour period would be 6.7%. The data collected in the present study have been calculated as percentages of sleep time as well as of 24 hours. Figures 10 to 14 represent the amounts of time spent by each subject in each of the stages of sleep, calculated as percentages of total sleep time.

Figure 10 and Table 10 show stage I. Pre- and post-deprivation amounts of this stage agree relatively well, averaging 7.5% and 5.7% respectively. Most of this small disparity is very likely due to the five subjects who spent slightly more time in stage I on the first predeprivation night than on any other baseline night. During deprivation all of the 4-day group, and subjects D.G. and P.W. of the 7-day group, showed a progressive increase in the percentage of sleep time spent in this stage. Subject T.S. showed this same tendency

FIGURE 10

Time spent in stage I sleep by each subject, as a percentage of total sleep time.



STAGE I
SLEEP TIME

TABLE 10

Time spent by each subject in stage I sleep, as a percentage of total sleep time.

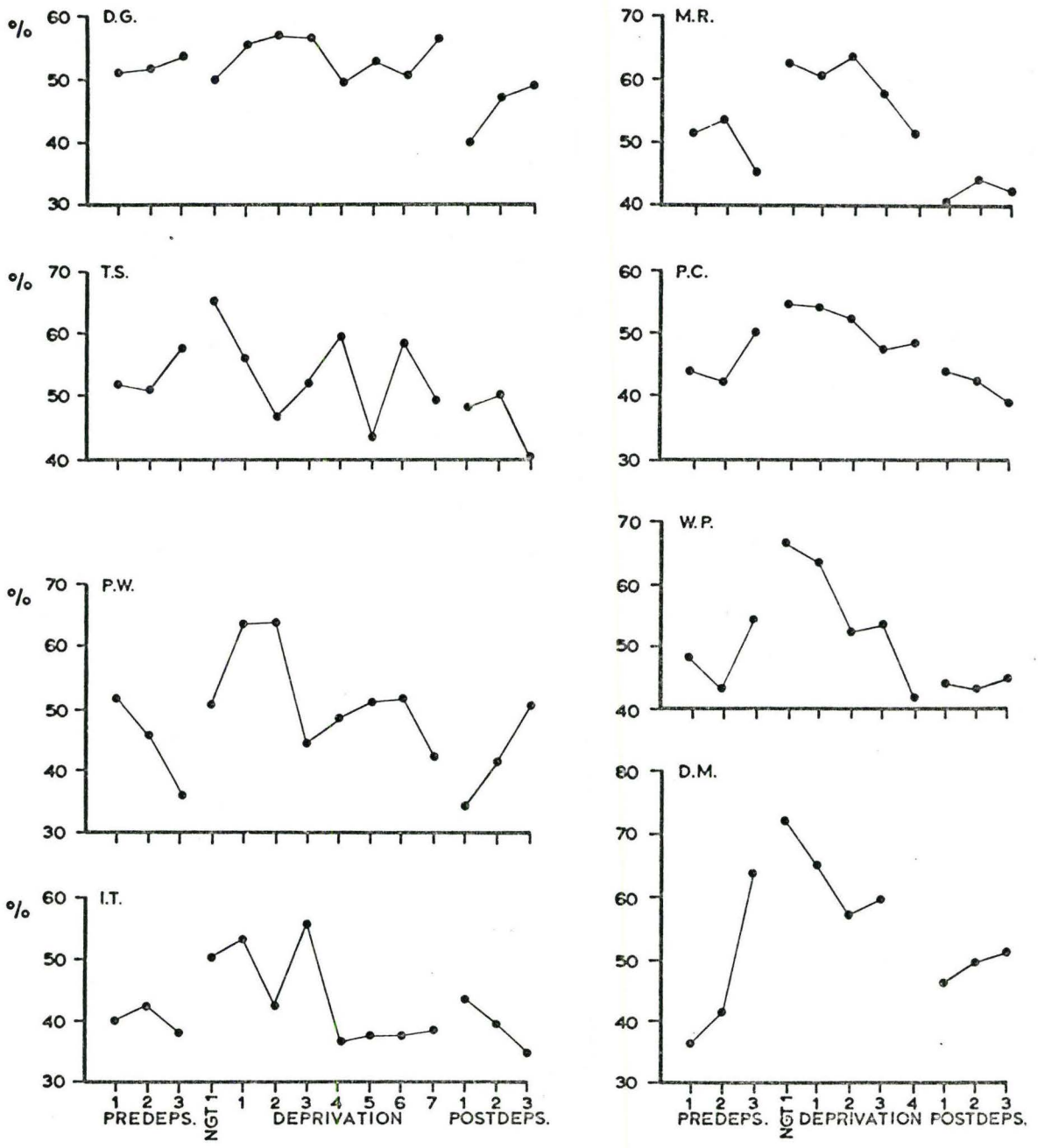
	Predeps.			Deprivation								Postdeps.		
Subject	1	2	3	Nt.1	1	2	3	4	5	6	7	1	2	3
D.G.	9.4	3.6	5.2	9.9	10.7	11.2	13.5	14.6	14.2	14.6	9.6	4.4	4.0	5.4
T.S.	9.9	7.3	4.3	6.9	13.9	17.3	12.7	7.1	8.3	6.4	5.9	8.4	5.2	3.0
P.W.	8.4	2.8	4.7	9.9	10.2	14.5	20.0	19.2	16.2	15.8	15.2	4.2	2.3	4.1
I.T.	12.8	9.5	12.2	9.2	19.6	28.9	9.4	25.2	19.7	15.0	17.4	1.1	1.4	3.9
M.R.	4.8	5.3	4.4	10.1	12.0	12.8	15.7	20.9				5.4	6.0	7.2
P.C.	9.9	11.6	7.0	13.1	12.1	12.9	18.6	14.1				6.7	9.1	9.9
W.P.	10.2	10.7	9.2	17.6	10.8	18.2	24.6	28.9				7.1	7.4	13.1
D.M.	8.6	4.6	4.5	6.0	9.5	10.7	12.9					6.9	5.4	5.6
Average	9.3	6.9	6.4	10.3	12.4	15.8	15.9	18.6	14.6	13.0	12.0	5.5	5.1	6.5

until day 2, then a decrease on days 3 and 4 which returned his stage I percentage to baseline levels. Subject I.T., as has been previously mentioned, slept very poorly on the third night of deprivation, and this is reflected in the irregularity of this curve for stage I as well as most of the others.

Stage II is shown in Figure 11 and Table 11. Generally, subjects spent more sleep time in stage II during the predeprivation than during the postdeprivation period. Mean values for these are 47.8% and 43.8% respectively. On night 1 of deprivation, all subjects except D.G. showed an increase in stage II to values well above those obtained on the predeprivation nights, the mean percentage for all subjects being 59.0%. Following this initial increase, all the 4-day subjects displayed a gradual decline in this stage as deprivation progressed, with values on day 3 or 4 back within the normal range. The 7-day subjects showed much more variability. Subject D.G. maintained approximately predeprivation amounts of stage II throughout the deprivation period. After the initial increase noted above, subject T.S. showed considerable fluctuations in this stage but no consistent trend toward either increase or decrease. Subject P.W. displayed further increases from the night 1 value on days 1 and 2 of deprivation, then abruptly returned to baseline on day 3 and maintained this level for the remaining days of deprivation. The curve for subject I.T. is distorted because of his abnormally restless night on day 2, but stage II was clearly maintained within the normal range for the last four days of the

FIGURE 11

Time spent in stage II sleep by each subject, as a percentage of total sleep time.



STAGE II
SLEEP TIME

TABLE 11

Time spent by each subject in stage II sleep, as a percentage of total sleep time.

	Predeps.			Deprivation								Postdeps.		
Subject	1	2	3	Ht.1	1	2	3	4	5	6	7	1	2	3
L.G.	51.0	51.6	53.7	50.1	55.4	56.8	56.5	49.8	53.3	50.7	56.5	40.7	47.6	49.7
T.S.	52.0	51.2	57.5	65.0	56.1	46.8	52.2	59.4	43.6	58.4	49.6	48.4	50.5	40.3
P.W.	52.0	45.9	36.3	50.8	63.6	63.9	44.5	48.6	51.2	51.6	42.3	34.4	41.7	50.7
I.T.	39.9	42.4	37.9	50.5	52.9	42.4	55.3	36.7	37.5	37.4	38.0	43.7	39.5	34.8
M.R.	51.6	53.7	45.3	62.2	60.3	63.3	57.7	51.4				40.9	44.1	42.2
P.C.	44.1	42.5	50.1	54.2	54.1	52.1	47.3	48.6				43.8	42.2	38.6
W.P.	48.4	43.5	54.3	66.6	63.2	52.2	53.5	41.8				44.0	43.2	44.7
D.M.	36.6	41.6	63.8	72.2	65.0	57.1	59.5					46.2	49.3	50.9
Average	47.0	46.6	49.9	59.0	58.8	54.3	53.3	48.0	46.4	49.5	46.6	42.8	44.8	44.0

deprivation period.

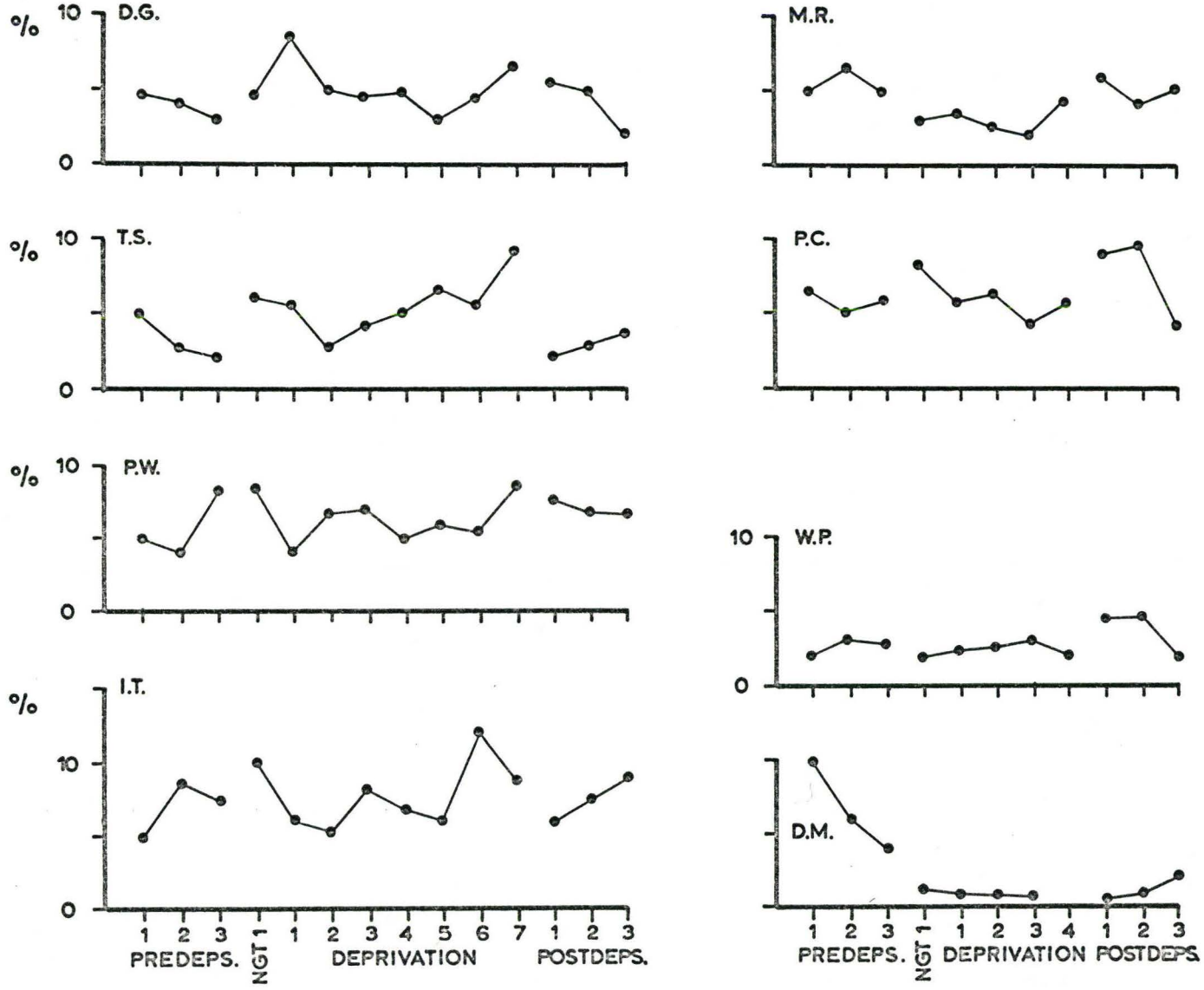
Figure 12 and Table 12 show stage III as a percentage of total sleep time. For seven of the eight subjects, pre- and postdeprivation values for this stage agree well, the means for all subjects being 5.0% and 4.9% respectively. D.M. is the single exception; his predeprivation values were the highest, and his postdeprivation values, the lowest, that were obtained from any subject. During deprivation, stage III changed very little and in general appeared to remain at approximately baseline levels throughout.

Figure 13 and Table 13 show stage IV. The mean percentage for this stage on the predeprivation nights was 15.4%, and on the postdeprivation nights, 16.7%. As with stage III, these values agree well, both on the average and for the individual subjects. On night 1 of deprivation, four subjects (M.R., P.C., W.P., and I.T.) showed a decrease in stage IV to subnormal levels, while the remaining four displayed approximately baseline amounts of this stage. The same four individuals who showed this initial drop appeared to progressively increase the percentage of sleep time they spent in stage IV during the remaining days of deprivation, until baseline levels were again reached toward the end of this period. For the other four subjects, stage IV values were maintained at roughly normal levels throughout deprivation.

Stage I REM and its two subcategories, REM sleep and NM sleep, are shown in Figure 14 and in Tables 14, 15, and 16. For all subjects, stage I REM averaged 24.3% of the total sleep time

FIGURE 12

Time spent in stage III sleep by each subject, as a percentage of total sleep time.



STAGE III
SLEEP TIME

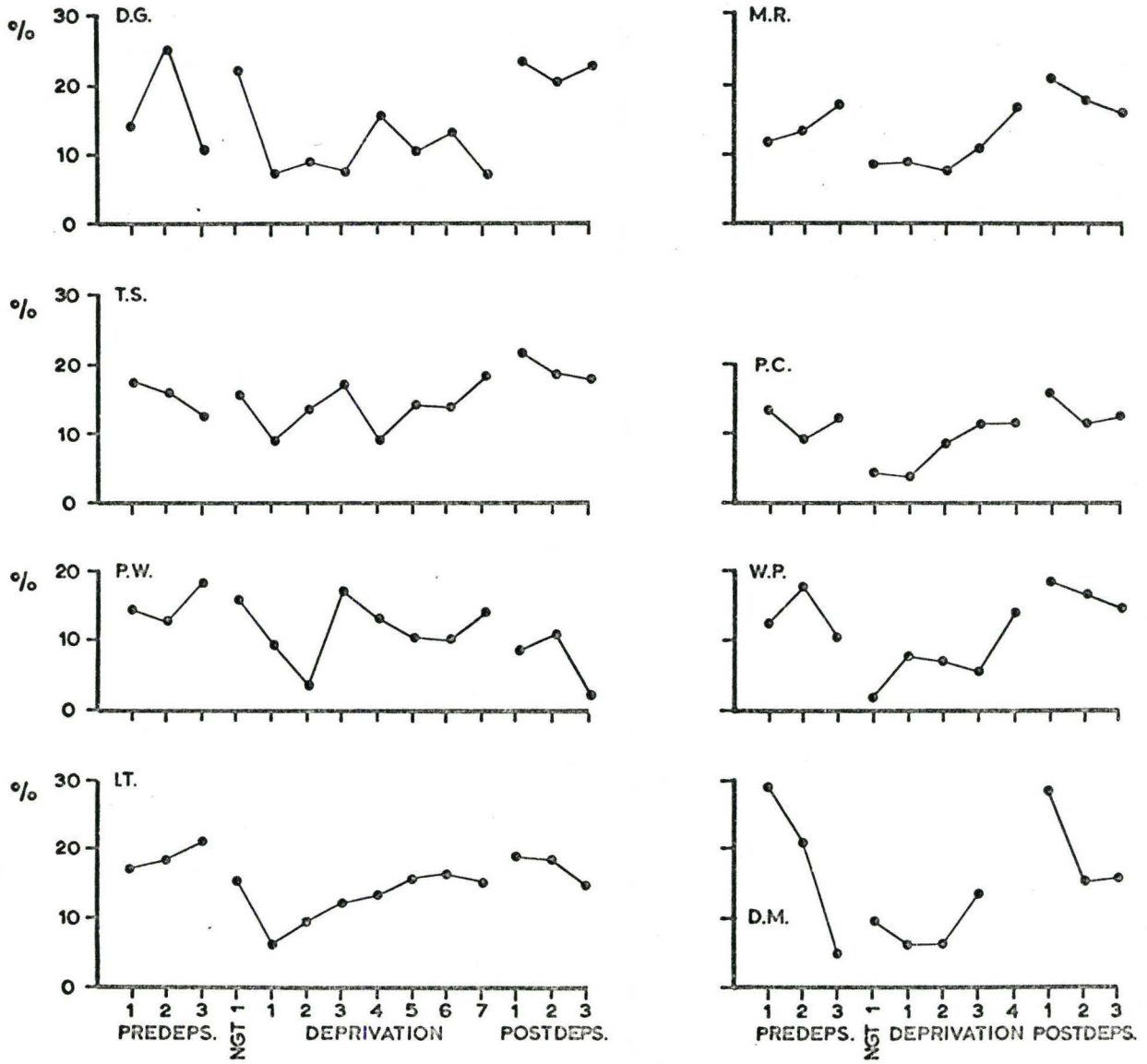
TABLE 12

Time spent by each subject in stage III sleep, as a percentage of total sleep time.

	Predeps.			Deprivation								Postdeps.		
Subject	1	2	3	Nt.1	1	2	3	4	5	6	7	1	2	3
D.G.	4.5	3.9	2.8	4.6	8.6	5.0	4.4	4.8	3.0	4.4	6.8	5.7	5.0	2.3
T.S.	4.8	2.6	2.1	6.0	5.4	2.7	4.0	4.9	6.5	5.5	9.2	2.1	2.8	3.7
P.W.	4.8	3.9	8.2	8.3	3.9	6.6	6.8	4.8	5.8	5.4	8.4	7.4	6.7	6.5
I.T.	4.9	8.6	7.3	9.8	6.1	5.3	8.2	6.7	6.1	11.9	8.7	5.9	7.4	8.8
M.R.	5.0	6.4	4.9	3.1	3.6	2.6	2.1	4.4				6.1	4.2	5.2
P.C.	6.4	5.0	5.8	8.2	5.7	6.3	4.2	5.6				8.9	9.5	4.1
W.P.	1.9	3.1	2.7	1.9	2.2	2.5	3.1	2.1				4.4	4.6	1.8
D.M.	9.9	6.1	4.1	1.2	.9	.8	.6					.5	.7	2.1
Average	5.3	5.0	4.7	5.4	4.6	4.0	4.2	4.8	5.4	6.8	8.3	5.1	5.1	4.3

FIGURE 13

Time spent in stage IV sleep by each subject, as a percentage of total sleep time.



STAGE IV
SLEEP TIME

TABLE 13

Time spent by each subject in stage IV sleep, as a percentage of total sleep time.

	Predeps.			Deprivation								Postdeps.		
Subject	1	2	3	Nt.1	1	2	3	4	5	6	7	1	2	3
D.G.	14.2	25.1	11.0	21.8	7.6	9.0	7.5	15.5	10.3	13.0	7.1	23.3	20.4	22.6
T.S.	17.5	15.8	12.6	15.6	8.8	13.6	17.1	9.1	14.2	13.9	18.3	21.7	18.5	17.9
P.W.	14.6	12.7	18.5	16.0	9.6	3.7	17.2	13.3	10.4	10.5	14.2	8.5	10.8	2.2
I.T.	17.3	18.0	21.6	15.3	6.5	9.5	12.2	12.9	15.9	16.3	15.0	19.0	18.6	14.9
M.R.	11.5	12.9	16.7	8.2	8.8	7.7	10.7	16.1				20.8	17.8	15.7
P.C.	13.4	9.0	12.0	4.5	3.7	8.4	11.3	11.5				16.0	11.3	12.6
W.P.	12.5	17.6	10.4	1.5	7.5	7.1	5.6	14.0				18.6	16.4	14.7
D.M.	29.3	20.9	5.0	9.4	6.3	6.3	13.5					28.3	14.9	15.5
Average	16.3	16.5	13.5	11.5	7.4	8.2	11.9	13.2	12.7	13.4	13.7	19.5	16.1	14.5

FIGURE 14

Time spent in stage I REM sleep, and in its two subcategories, REM sleep and NM sleep, by each subject, as a percentage of total sleep time.

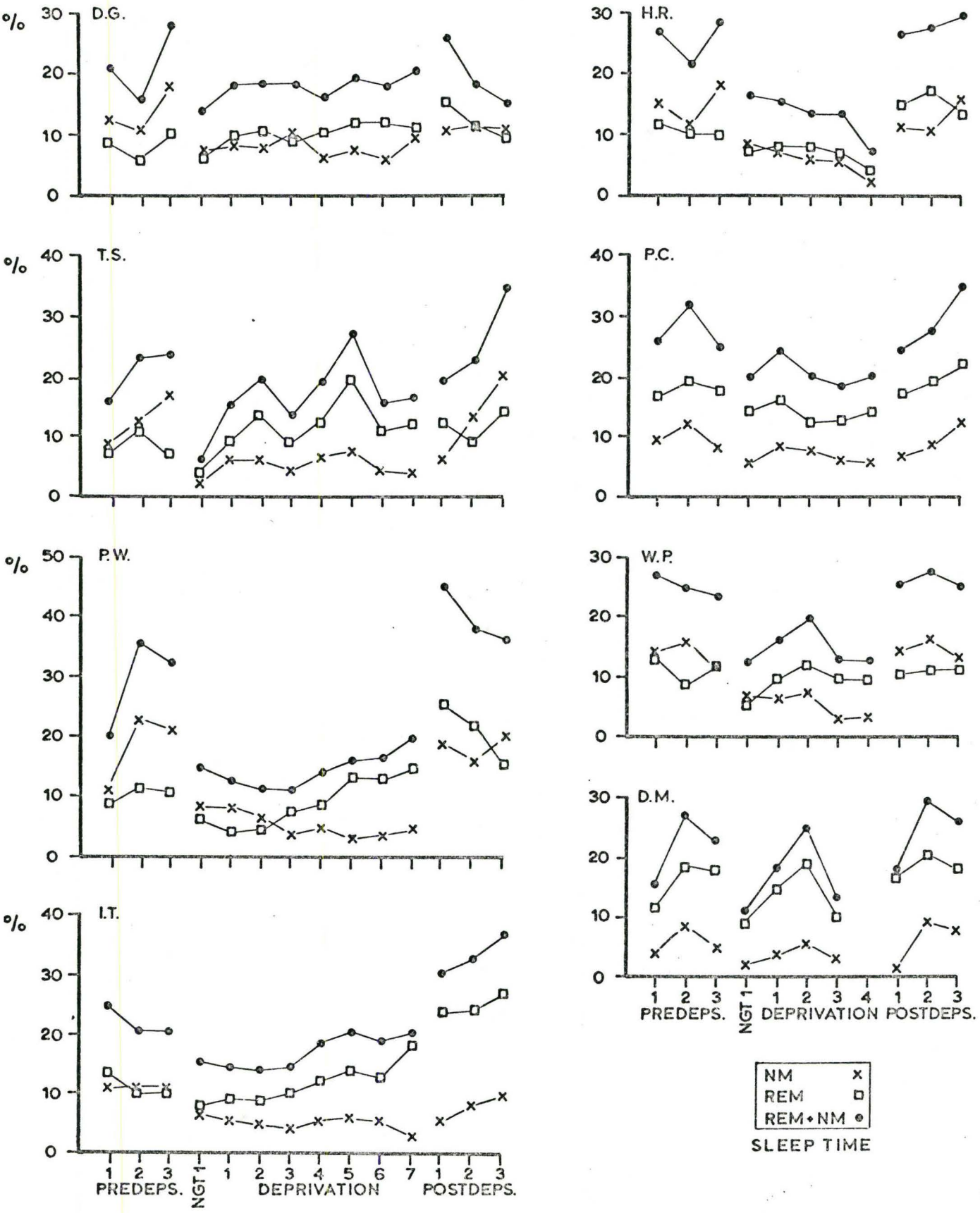

%
D.G.
H.R.
T.S.
P.C.
P.W.
W.P.
I.T.
D.M.
PREDEPS.
NGT 1
DEPRIVATION
POSTDEPS.
NM
REM
REM+NM
SLEEP TIME

TABLE 14

Time spent by each subject in stage I REM sleep, as a percentage of total sleep time.

	Predeps.			Deprivation								Postdeps.		
Subject	1	2	3	Nt.1	1	2	3	4	5	6	7	1	2	3
D.G.	20.9	15.9	27.4	13.5	17.7	18.0	18.2	15.5	19.1	17.4	20.0	25.9	23.0	20.0
T.S.	15.7	23.1	23.5	6.5	15.8	19.7	13.9	19.5	27.4	15.8	16.9	19.4	23.0	35.1
P.W.	20.3	35.8	32.3	14.9	12.7	11.4	11.5	14.2	16.3	16.7	20.0	45.5	38.5	36.5
I.T.	25.1	21.5	21.1	15.3	14.9	14.0	14.9	18.4	20.9	19.3	20.9	30.4	33.2	37.6
M.R.	27.1	21.7	28.6	16.4	15.4	13.6	13.8	7.3				26.8	27.9	29.6
P.C.	26.1	31.9	25.0	20.0	24.4	20.2	18.6	20.2				24.5	27.9	34.9
W.P.	27.1	25.1	23.4	12.5	16.3	19.9	13.1	13.1				25.9	28.3	25.7
D.M.	15.7	27.0	22.8	11.1	18.3	25.0	13.4					18.0	29.8	25.9
Average	22.3	25.3	25.5	13.8	16.9	17.7	14.7	15.5	20.9	17.3	19.5	27.1	29.0	30.7

TABLE 15

Time spent by each subject in the NM subcategory of stage I REM sleep, as a percentage of total sleep time.

	Predeps.			Deprivation							Postdeps.		
Subject	1	2	3	Nt.1	1	2	3	4	5	6	7	1	2	3
D.G.	12.5	10.5	17.7	7.8	8.2	7.8	9.4	5.7	7.3	5.6	9.4	10.4	11.6	10.5
T.S.	8.6	12.6	16.6	2.3	6.3	6.2	4.6	6.8	8.0	4.5	4.3	6.7	13.4	20.5
P.W.	11.3	23.3	21.3	8.3	8.4	6.7	3.9	5.3	2.9	3.5	4.9	19.5	16.4	20.6
I.T.	11.6	11.1	10.7	7.1	5.8	5.3	4.6	6.0	6.7	6.3	2.9	6.2	8.4	10.1
M.R.	15.1	11.4	18.4	8.5	7.6	5.3	6.1	2.6				11.5	10.4	16.2
P.C.	9.5	12.6	7.3	5.8	8.4	7.7	5.9	5.7				7.2	8.7	12.6
W.P.	14.0	16.3	11.8	7.0	6.5	7.6	3.2	3.4				14.7	16.9	14.1
D.M.	4.0	8.6	5.0	2.1	3.6	5.8	3.3					1.6	9.4	7.8
Average	10.8	13.3	13.6	6.1	6.9	6.6	5.1	5.1	6.2	5.0	5.4	9.7	11.9	14.1

TABLE 16

Time spent by each subject in the REM subcategory of stage I REM sleep, as a percentage of total sleep time.

	Predeps.			Deprivation								Postdeps.		
Subject	1	2	3	Mt.1	1	2	3	4	5	6	7	1	2	3
D.G.	8.4	5.4	9.7	5.7	9.5	10.2	8.8	9.8	11.8	11.8	10.6	15.5	11.4	9.5
T.S.	7.1	10.5	6.9	4.2	9.5	13.5	9.3	12.7	19.4	11.3	12.6	12.7	9.6	14.6
P.W.	9.0	11.5	11.0	6.6	4.3	4.7	7.6	8.9	13.4	13.2	15.1	26.0	22.1	15.9
I.T.	13.5	10.4	10.4	8.2	9.1	8.7	10.3	12.4	14.2	13.0	18.0	24.2	24.8	27.5
M.R.	12.0	10.3	10.2	7.9	7.8	8.3	7.7	4.7				15.3	17.5	13.4
P.C.	16.6	19.3	17.7	14.2	16.0	12.5	12.7	14.5				17.3	19.2	22.3
W.P.	13.1	8.8	11.6	5.5	9.8	12.3	9.9	9.7				11.2	11.4	11.6
D.M.	11.7	18.4	17.8	9.0	14.7	19.2	10.1					16.4	20.4	18.1
Average	11.4	11.8	11.9	7.7	10.1	11.2	9.6	10.4	14.7	12.3	14.1	17.3	17.1	16.6

on the predeprivation nights. For night 1 of deprivation, however, this value dropped to 13.8%, and for every subject, the percentage obtained on this first night was well below any recorded during the predeprivation period. In fact, stage I REM was considerably depressed throughout the entire deprivation period, as Table 14 shows. On the postdeprivation nights, subjects P.W. and I.T. showed substantial increases in their stage I REM percentages, to levels that not only exceeded those recorded during deprivation but also during the predeprivation period. The remaining six subjects also showed increases, but of a smaller magnitude; generally, these represented a return to baseline amounts of stage I REM. The average value for this stage on these postdeprivation nights was 28.9% of the total sleep time.

Turning now to examine the two subcategories of stage I REM in relation to one another, it can be seen that the previously described crossover of REM sleep and NM sleep is displayed in these graphs as well as those based on 24 hour periods.

To summarize as before, Figures 15 and 16 show changes in each EEG stage for all 7-day subjects and for all 4-day subjects. Like Figures 10 to 14, these two plot amounts of time spent in the various stages as percentages of the total sleep time per 24 hour period. Both groups showed roughly comparable amounts of stage I on the pre- and postdeprivation nights. During deprivation, both also showed an elevation in this stage. For the 4-day group, this progressively increased throughout deprivation, whereas the 7-day group appeared to level off after day 2 and then to maintain

FIGURE 15

Time spent in each sleep stage for all 7-day subjects, expressed as a percentage of total sleep time.

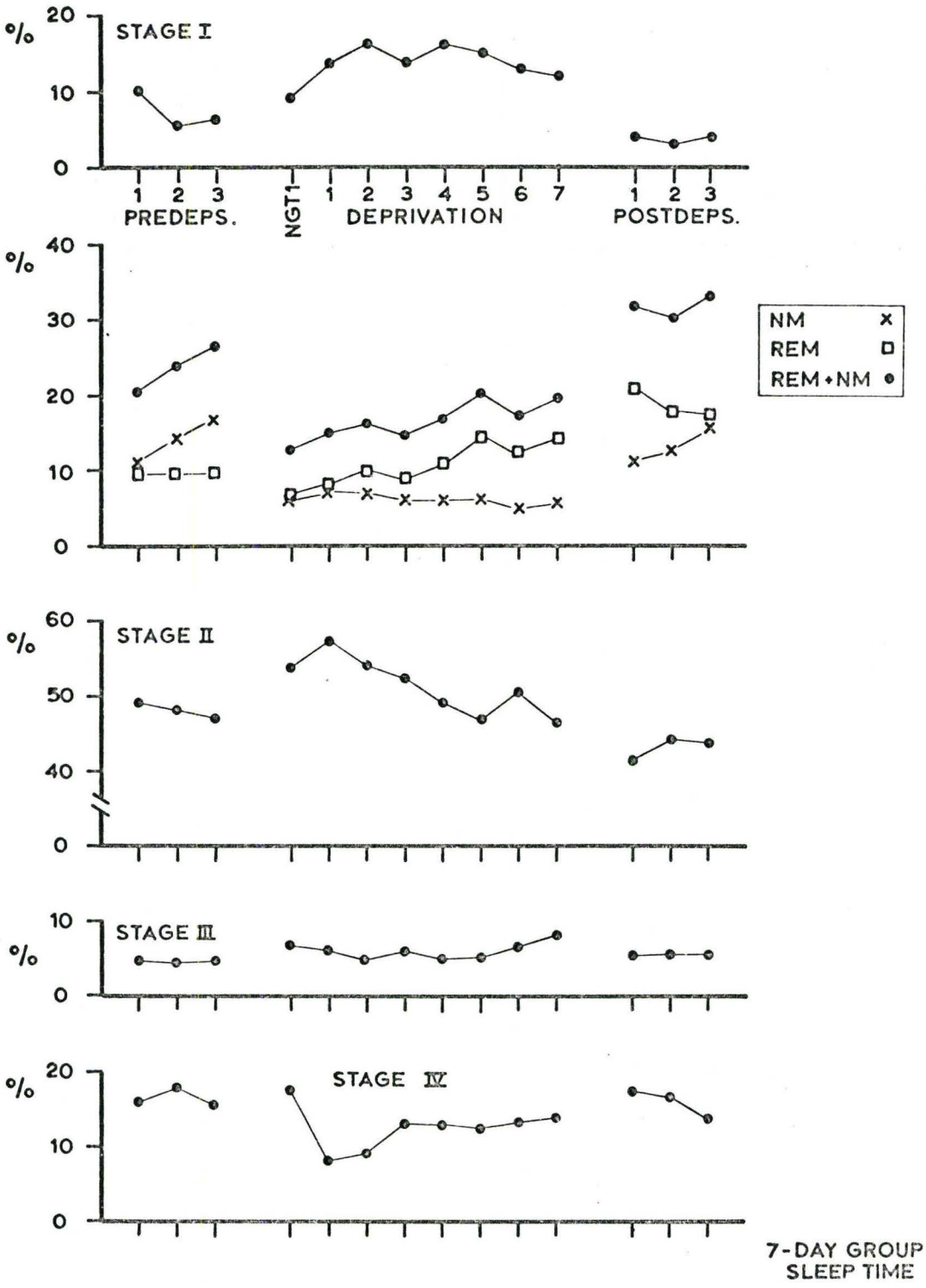
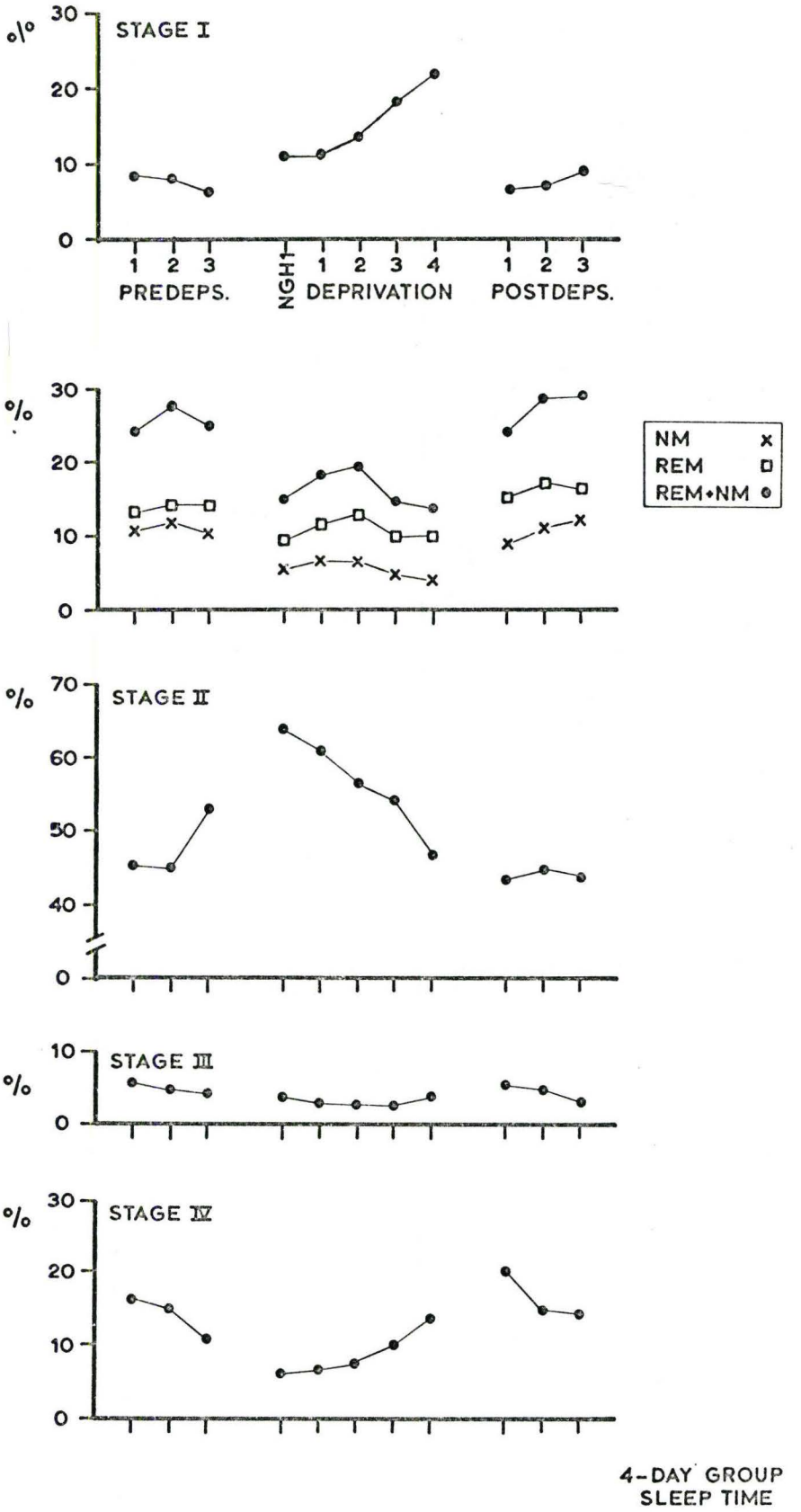

%
20
10
0
STAGE I
1 2 3
PREDEPS.
NGT1
1 2 3 4 5 6 7
DEPRIVATION
1 2 3
POSTDEPS.
%
40
30
20
10
0
NM
REM
REM+NM
%
60
50
40
0
STAGE II
%
10
0
STAGE III
%
20
10
0
STAGE IV
7-DAY GROUP
SLEEP TIME

FIGURE 16

Time spent in each sleep stage for all 4-day subjects, expressed as a percentage of total sleep time.


%
30
20
10
0
STAGE I
1 2 3
PREDEPS.
NGHT
1 2 3 4
DEPRIVATION
1 2 3
POSTDEPS.
%
30
20
10
0
NM
REM
REM+NM
%
70
60
50
40
0
STAGE II
%
10
0
STAGE III
%
30
20
10
0
STAGE IV
4-DAY GROUP
SLEEP TIME

a fairly constant amount of stage I. Percentages of sleep time spent in stage II were also similar for both groups on the pre- and postdeprivation nights, with, perhaps, a slight tendency toward higher values during the predeprivation period. There was a large increase in this stage early in deprivation for both, followed by a gradual return to baseline levels by day 3 or 4. For the remaining deprivation days undergone by the 7-day subjects, stage II was maintained approximately level. Stage III for both groups showed very little change during deprivation, and was generally maintained at the same level as was recorded during pre- and postdeprivation periods. Percentages of stage IV obtained on the pre- and postdeprivation nights showed good agreement for both groups, and the value recorded for the 7-day group on night 1 of deprivation was also much the same. Both showed a decline in stage IV early in deprivation, on night 1 for the 4-day group and on day 1 for the 7-day group. Following this, both appeared to increase their stage IV percentages until baseline levels were again reached on day 3 or 4. The 7-day group seemed not to return quite to baseline, but some stable level was clearly reached and maintained until deprivation was terminated. With regard to stage I REM, the graphs for both groups clearly show the depression of this stage on the first night of deprivation. For both also, the decrease in the percentage of sleep time occupied by this stage continued throughout the deprivation period. The 4-day group appeared to spend approximately equal amounts of time in stage I REM during both pre- and postdeprivation periods, while for the 7-day

group, the postdeprivation values were consistently higher. The reversal or crossover in the relationship of REM sleep and NM sleep during deprivation was shown only by the 7-day and not by the 4-day group.

To conclude, further analysis of the polygraph records collected in this experiment would be desirable. In particular, it would be of interest to examine the incidence during deprivation of the spindles and K-complexes characteristic of stage II sleep, and to compare this with their occurrence on the pre- and post-deprivation nights. Further analysis of the crossover between REM sleep and NM sleep should include some measurement of the density of the eye movements. This experiment, however, is part of an ongoing program of research in this laboratory. Power spectral analysis of the alpha rhythm of deprived subjects is nearly complete, and similar analysis of the EEG rhythms characterizing the various sleep stages is planned for the near future. It was felt that the detailed inspection of spindles, K-complexes, and eye movement density recommended above would be more appropriate in conjunction with the results of the power spectral analysis, and for this reason, these have not been included with the present results.

CHAPTER 5

DISCUSSION

As indicated in Chapter 1, the data collected in this study of sleep during deprivation are relevant both to previous research and theory in the area of deprivation and in the area of sleep. The present chapter is organized in this way, and will deal with the experimental findings first in relation to deprivation and then in relation to sleep.

Before that, however, a comparison of the present results with those obtained in similar studies would be appropriate. The experiments carried out at Tohoku University in Japan (Nagatsuka and Kokubun, 1964; Sato and Kokubun, 1965; Chyama, Kokubun, and Kobayashi, 1965) were mentioned in Chapter 2. These authors report that EEG records from subjects deprived for 48 hours show a predominance of "...chronic slow waves of middle levels as suppressed α-waves, spindle or hump waves reflecting light or drowsy sleep...." (Nagatsuka and Kokubun, 1964, p. 62). Though the records were not scored according to sleep stages, this finding might be interpreted as indicating that their subjects spent considerable time in stage II sleep, a result which is consistent with the present data.

The study of Steinberg and Russo (1970), although it does not mention stage II sleep and so precludes any comparison, is nevertheless in good agreement with respect to several other findings.

The same pattern of early increase and later decrease in total sleep time was observed in both studies, although the subjects of Steinberg and Russo seemed to persist in sleeping slightly more than usual even toward the end of their confinement. Stage III results from both studies also agree well, with neither observing any change from baseline levels during the experimental period. The values reported for stage IV sleep are low when compared with those obtained in the present study, but it is possible that this discrepancy could be accounted for by differences in the placement of the EEG electrodes, or in recording parameters. Aside from this, both studies agree in reporting low values for stage IV early in the experimental period. Steinberg and Russo consider that these are due to the arousing effects of the novel confinement situation, and propose that this factor also accounts for their low stage IV values during the preconfinement period. After the similar early depressions in stage IV, both studies also agree that little change was observed throughout the rest of the experimental period. With regard to stage I REM, the values reported by Steinberg and Russo are well within the normal range observed in the present study. They also observe an increase in this stage on days 1-3 of confinement that is similar to the day 1 increase seen in the present study. Following this, both studies report a decrease in stage I REM to a level which more closely approximates the normal baseline.

Returning now to a consideration of the present experimental findings alone, perhaps the most consistent and the most striking change seen in the present study is the great increase in sleep

early in deprivation. This finding was not unexpected and, indeed, is exactly what would be predicted by the reticular theory of deprivation. Lindsley, in fact, postulating that continuously varying stimulation is essential for normal functioning, states, "Without such stimulation, boredom, inactivity, and, ultimately, sleep, prevail." (1961, p. 176). This prediction is verified by the present experimental findings, but only for the early part of deprivation. Later on, and in spite of the persistence of lowered and monotonous sensory input conditions, the deprived subjects nevertheless return to a relatively normal sleep-wakefulness cycle. The reason for this is unknown, but speculation quickly leads to the notion that the effects of deprivation, paramount early in this experience, are later overridden by the normal biological rhythms. The Tohoku University investigators of deprivation phenomena have quite specifically drawn this conclusion. To quote, "In brief, the qualitative aspect of behaviors is influenced by the effect of S.D., but their quantitative aspect or intensity depends rather on the rhythm of the organism." (Nagatsuka and Kokubun, 1964, p. 62). That is, sensory deprivation appears to change the quality of the subjects' verbalizations, in that the early ones, such as reporting a dream or singing, were relatively neutral, while agressive and emotional overtones predominated in the later ones. On the other hand, measures such as the GSR, EKG, and respiration, which these authors believe indicate levels of arousal, oscillated up and down in a manner which corresponded closely to the subjects' day-night cycle. This conclusion was reached also by experimenters in Myers'

laboratory who were engaged in measuring restlessness during deprivation (Smith, Myers, and Murphy, 1962, 1967). Subjects were isolated for four days without any time cues, and yet showed a significant difference between day and night measures of restlessness, indicating that their diurnal cycles were maintained throughout the deprivation experience.

The specific time at which these biological cycles return to a completely normal state is also of interest. The sleep-wakefulness cycle of all subjects engaged in the present experiment had apparently returned to normal by day 3 or day 4. Some of the other changes which occur during deprivation also seem to stabilize at this same time. For example, data collected in this laboratory on the alpha rhythm of deprived subjects indicates that it becomes progressively slower during deprivation until day 3 or 4, and then appears to stabilize at this new slow frequency, and to show minimal change after this (Tait, unpublished data). Similarly, the activity measured by the Myers group (Smith, Myers, and Murphy, 1967; Myers, 1969) increased over a four day period and then seemed to be maintained at a constant level.

It can be argued that the effects of deprivation described in Chapter 4, namely, the great increase in sleep early in deprivation and the changes in the various sleep stages, result merely from providing subjects with the opportunity for large amounts of sleep. Admittedly, deprivation does provide this opportunity. Two possibilities exist then, namely, that deprivation is uniquely able to produce the effects described, or that they are due to the

opportunity for sleep inherent in the deprivation conditions. In order to decide between these two alternatives, one would have to use a control group provided with the same opportunity for sleep but not deprived. This, however, would seem to be extremely difficult. The widely used control conditions, such as employing recumbent subjects in groups, automatically involve a certain degree of deprivation, though such conditions may permit talking, radios, books, and so on. In fact, Myers (personal communication to W. Heron) has shown that subjects so confined not only show the alpha slowing typical of deprived subjects, but that the magnitude of this slowing is proportional to the size of the confinement chamber. Subjects confined in a small chamber show significantly greater alpha slowing than those in a larger one. Unfortunately, then, for lack of an entirely adequate control condition, it is difficult to decide what causes the excessive sleep observed early in the present experiment.

One can, however, exclude the possibility that deprivation symptoms are produced by gross disturbances in the amount of sleep or in the proportion of time spent in the various sleep stages. Since the sleep of deprived subjects had never been precisely monitored, it was unknown whether these were abnormal, slightly changed, or normal during deprivation. It was well known that sleep loss and even loss of REM sleep produce cognitive, perceptual and emotional changes that are very similar to those seen during deprivation (Bliss, Clark, and West, 1959; Morris and Singer, 1961; Dement, 1960). Thus the possibility, remote but

real, existed that deprivation produced its effects indirectly by disturbing sleep, which then was directly responsible for the observed symptoms. The results described in Chapter 4, however, obviate this possibility since they clearly indicate that sleep is really more normal than abnormal during deprivation, both in total amount and in the proportions of its stages.

The specific changes observed in the various stages of sleep will be dealt with next, including an examination of the first night of deprivation, of the increases seen in stage I REM on day 1 and in stage II early in deprivation, of the rise observed in stage I throughout, and of the crossover between REM sleep and NM sleep.

Chapter 4 has described several changes which were observed on the first night of deprivation. First, the mean amount of time spent in stage IV on this night was 2.6% of 24 hours, and clearly had decreased from the average of 4.2% observed during the predeprivation period. Second, all subjects showed a decline in stage I REM on night 1, the average value being 3.2% compared with 6.9% for the predeprivation nights. Third, three subjects slept less than a normal amount on this first night. These three measures probably indicate that the first night of deprivation was a somewhat uncomfortable one for most subjects. In fact, this effect of discomfort and novelty is well known among sleep researchers, who routinely observe it on a subject's first night in their laboratories, and call it the night 1 effect. We have not observed unequivocal indications of it on the first predeprivation night, but the first night of deprivation clearly shows it. Night 1 was

the subjects' first experience with mask, gloves, cuffs, and white noise. It is quite likely that these deprivation conditions, which were new to the subjects at this time, were responsible for the effects observed.

On the first day of deprivation, stage I REM showed an increase. For all subjects except T.S. and P.W., values obtained for this stage on the first day slightly exceeded those obtained on any of the predeprivation nights. As has been discussed previously, however, stage I REM showed a decrease on night 1. This may indicate that subjects experienced a slight degree of REM deprivation on night 1, and the day 1 increase could then be interpreted as the later compensation for this loss. However, a comparison of subjects who characteristically slept less than six and one-half hours per night with those who slept more than eight and one-half hours (short sleepers vs. long sleepers) revealed that the long sleepers spent more time in stage I REM than did the short sleepers (155 minutes as opposed to 96 minutes per night, or 11% as opposed to 7% of 24 hours) (Webb and Agnew, 1970). This raises the possibility that the increase seen in stage I REM on day 1 of the present experiment results from the great increase in total sleep time on this same day. The two factors, then, REM deprivation on night 1 and increased sleep on day 1, could be responsible for the day 1 elevation in stage I REM.

The single greatest change seen in any sleep stage during deprivation was the enormous increase in stage II shown by all subjects on day 1. In fact, almost all of the extra sleep time

early in the deprivation period was occupied by this stage. It is already known that both stage I REM and slow-wave sleep are fairly well regulated in amount since subjects deprived of these stages make up their losses during subsequent sleep periods (Dement, 1960; Berger and Oswald, 1962; Agnew, Webb, and Williams, 1964). In contrast, the lability found in stage II during the present study suggests that it is not regulated as stringently as these other two stages. This is also suggested by the recent study of Webb and Agnew (1970) which compared long sleepers and short sleepers. A significant difference between these two groups was found in the amount of time they spent in stage II. The short sleepers spent a mean of 168 minutes in stage II per night (12% of 24 hours) while the long sleepers spent a mean of 277 minutes in this stage (19% of 24 hours). Thus it would seem that prolongation of sleep time per se is able to produce a significant elevation in stage II, and that this result in the present experiment is a consequence of the increased sleep seen early in deprivation.

Though stage I REM and stage II show these increases on day 1 of deprivation, slow-wave sleep, or stages III and IV, seem not to change at all. Thus it is clearly not true that when sleep is increased, its stages also increase in a proportionate fashion. That is, a subject who sleeps twice as much as he does normally does not spend twice as much time in each of the sleep stages. The only stage for which this statement even approximates the truth is stage II, since the increase in stage I REM does not even approach what would be expected if it were increasing in proportion

to the total amount of sleep. It is already known that stage I REM and slow-wave sleep are prevented by regulatory mechanisms from falling below certain baseline levels (Dement, 1960; Berger and Oswald, 1962; Agnew, Webb, and Williams, 1964). Their failure to increase when sleep is greatly increased raises the interesting possibility that they also may be prevented from rising above these baseline amounts. Though this speculation seems plausible, by analogy with such homeostatic mechanisms as govern body temperature or blood pressure, it has yet to be empirically confirmed. A direct experimental demonstration would involve elevating stage IV or stage I REM above normal levels prior to measuring these during the post-treatment period. If the reverse of the REM deprivation or stage IV deprivation effects were seen, that is, if these stages were depressed to subnormal levels, the existence of an upper as well as a lower limit for them would be established.

The implication of the constant increase in stage I seen during deprivation is that subjects spend more time than normal in this intermediate zone between wakefulness and sleep. This is of interest in view of the subjects' frequent reports of non-directed, day-dreaming types of cognitive activity during deprivation. Since it is known that cognitive activity during stage I or generally during the presomnic phase of sleep also possesses these characteristics (Foulkes and Vogel, 1965; Roffwarg and Muzio, 1965), it is possible that at least some of these day-dream reports can be attributed to the increased stage

I time observed during deprivation.

As previously mentioned, there is an actual change in the waking EEG of deprived subjects. Their alpha frequency declines, they appear to produce less alpha than normal, and also, those segments of record identifiable as alpha seem less regular and sinusoidal in wave form than usual. It might be argued that all these changes would tend to make the waking record of a deprived subject look more like a stage I record than it did normally, and may perhaps have led to some errors in the categorization of these records. These errors, however, were unlikely to have occurred with any meaningful frequency, since two other independent indices of the EEG stage besides the EEG itself were available to aid in scoring the polygraph records. These were the electro-oculogram and the electromyogram. Eye movements during wakefulness occur much more frequently than during stage I, and also display a different wave form. During wakefulness they are rapid, and show sharp peaks of relatively high amplitude, while during stage I they are slow, low in amplitude, and sinusoidal, almost rolling, in shape. Body movements also occur more frequently when a subject is awake, and the baseline EMG is slightly higher than it is in stage I. It is probable, then, that scoring errors were relatively few, and hence that the increase seen in stage I during deprivation is genuine. This conclusion is supported by the relatively good agreement between independent ratings of this stage, as reported in Chapter 4.

Finally, the curious reversal during deprivation of the

relative amounts of the two subcategories of stage I REM, REM sleep and NM sleep, deserves comment. This reversal persists after the termination of deprivation; it can be seen from Figures 7 and 14 that one subject reverted immediately to the predeprivation state, two return on the second postdeprivation night, and two on the third. One subject persisted in showing more REM sleep than NM sleep for all three postdeprivation nights. In addition, this crossover does not consist merely of a reversal of the relative contributions of REM sleep and NM sleep to stage I REM as a whole. Rather, as a reexamination of Figures 7 and 14 will show, the proportion of REM sleep seems enhanced and that of NM sleep depressed, over and above the observed reversal. Though these two components of stage I REM have always been recognized, the distinction between them is usually glossed over in the human sleep literature on the assumption that they are simply two facets of the unitary REM state, though the work of Pompeiano and his associates on cats (eg. Pompeiano, 1967) has emphasized the difference. The crossover effect itself, however, and the enhancement-depression phenomenon pointed out above, would suggest that these two are at least partially independent of each other. The experiment of Zimmerman, Stoyva, and Metcalf (1970) on changes in stage I REM in subjects who wore distorting spectacles tends to support this view, since Stoyva (personal communication) has stated that the density of rapid eye movements is markedly increased following this treatment. These authors believe that the changes they observed in stage I REM, namely, both an increase in time spent in this stage and the increase in REM density, to be

caused by the perceptual learning their subjects undergo in adapting to their distorted visual experience.

Several of the modern theories of sleep function were reviewed in Chapter 2, including those which link REM sleep to cognitive processes. Zimmerman, Stoyva, and Metcalf (1970) have produced evidence broadly supporting this type of theory. More specifically, they propose that stage I REM sleep is strongly involved in perceptual learning. Thus they would seem to imply that stage I REM should be depressed during perceptual deprivation.

Examination of several other variants of the REM-cognition theory shows that they, too, would predict that stage I REM should decline in subjects exposed to perceptual deprivation. Shapiro states, "In brief, then, the proposed theory assumes that the total input of signals received by the brain in the waking state requires some further processing to prevent overloading of the available facilities for storing and processing information. ... It is not clear whether all the data processing is experienced as dreaming, but it is suggested that much of it is thus experienced, and that particularly critical and significant processing is necessarily experienced as dreaming." (1967, pp. 78-79). In the terms used by Shapiro, it is obvious that deprivation involves a large reduction in the total input of signals received by the brain. Consequently, the overload which the dream processing serves to prevent would probably be minimal. If this is the case, and the need for dream processing is reduced, it might be argued that dreaming itself would be reduced.

The "P"-hypothesis of stage I REM sleep advanced by Dewan (1968, 1969; Greenberg and Dewan, 1968) is similar. "P" stands for programming, a process which involves the integration of new information into past information stores. Programming is believed by Dewan to occur at least partly during REM sleep. If the deprivation conditions are considered within this theoretical framework, it would seem that very little new information is available. In consequence, the need for integration of new and old information, as programming, would be minimal, and stage I REM might be expected to decrease.

Feinberg and Evarts state, "The possibility that S-REM is especially important for the restructuring of memories is based, in part, on its high level of neuronal activity. ... Whatever the origin of the neuronal activity of S-REM, its intensity and altered spatio-temporal pattern are consistent with a role in either engram consolidation or decay." (1969, p. 341). Again, this particular theory shares much in common with those of Shapiro and Dewan. Though no mention is made specifically of external conditions which influence memory restructuring or engram consolidation and decay, it is implied that the initial impetus for these processes is the presentation of information to the organism. If conditions are such that very little information is presented, as in deprivation, very little restructuring would be called for, and it might be predicted that stage I REM would decline.

Although Fishbein (1969a, b, and c) has been more specific in detailing the function he proposes for stage I REM sleep, his

theory still is concerned with memory. "Paradoxical sleep may be a periodic mechanism for securing and maintaining information for long-term memory." (1969a, p. 225). The information presented to subjects undergoing perceptual deprivation is made as monotonous as possible, and it could be argued that this would be unlikely to be selected for long-term memory. If this is the case, stage I REM sleep would have very little data to secure and maintain for long-term storage, and might be expected to be reduced.

All these theories, then, when extended to encompass perceptual deprivation, could be construed as predicting that stage I REM would decline as deprivation progressed. Very early in the deprivation period, however, the invariant nature of the sensory input might not yet be perceived by subjects as monotonous, but would rather represent a quite novel stimulus situation. Newman and Evans, in presenting their theory of the function of REM sleep, quite specifically invoke this concept of novelty. "Perhaps the most important factor which needs to be considered is our proposition that dream clearance is, in fact, an examination of novel material collected by the system in the course of the day." (1965, p. 534). In fact, the various other theories which have been reviewed also recognize, either implicitly or explicitly, the importance of novel information. To illustrate, Zimmerman, Stoyva, and Metcalf (1970) provided a novel perceptual environment for their subjects by requiring them to wear distorting spectacles, and consider that "...REM sleep is strongly involved in adaptation

to novel sensory input, in perceptual learning." (p. 14). The "P"-hypothesis of Dewan (1968, 1969) states that stage I REM serves to integrate new information into past information stores. Broadly stated, then, all these theories consider that novelty has a positive relation to stage I REM sleep. It has been pointed out above that the early part of the deprivation period could be viewed as a novel experience, in that invariant sensory input is an extreme departure from the subjects' normal experience. Consequently, it might be expected that stage I REM would be elevated early in the deprivation period, although it would later tend to decrease as the novelty of the situation dissipated and monotony truly set in. Further, it might be argued that the normal perceptual environment, after several days of the monotony of deprivation, would itself then appear novel to deprived subjects. If this is the case, stage I REM would be expected to show an increase when measured after deprivation. More specifically, this effect might be seen most prominently on the first post-deprivation night, when the contrast between the normal and the deprivation environments, and hence the novelty of the normal environment, would be greatest.

The present study does not unequivocally support these predictions. With regard to the early part of deprivation, it is true that all eight subjects showed an increase in stage I REM on day 1. However, as has been discussed previously, there are two other quite plausible reasons, aside from the novelty theory, which could account for this. One is the fact that all subjects

showed a depression in stage I REM on night 1 of deprivation, which, if this depression indicates a slight degree of REM deprivation, would then make the day 1 increase appear as a rebound effect, as described by Dement (1960). The other reason concerns the greatly increased sleep shown by all subjects early in deprivation, since Webb and Agnew (1970) have shown that an increase in stage I REM is normally associated with an increase in sleep. The principle of parsimony would suggest, then, that it is not necessary to view the day 1 increase in stage I REM as the effect of a novel situation.

Nor is the prediction of an increase in stage I REM during the postdeprivation period entirely borne out by the present experimental evidence. Only two subjects showed excessive amounts of this stage following deprivation. Of these two, one showed a progressive decrease over the three postdeprivation nights, but the other showed a progressive increase. All other subjects showed postdeprivation values for this stage that were quite consistent with the baseline values obtained during the predeprivation period.

Finally, with regard to the predicted decline in stage I REM during the later part of the deprivation period, the present results are inconclusive. The individual subjects varied considerably in the amounts of time they spent in this stage. Thus, all 4-day subjects showed a decrease in this stage as deprivation progressed, and two of the 7-day subjects showed a similar tendency over days 1 to 3. These two, however, then showed an increase and appeared to maintain stage I REM at a relatively constant level until

deprivation was terminated, an observation that appears at variance with the prediction. The final two 7-day subjects showed almost no change in this stage throughout the deprivation period.

Another theorist who proposes that sleep is necessary for cognitive function is Moruzzi. He states, "Hence, sleep should not be regarded as a period of recovery for the entire cerebrum, but only (or mainly) as a period of recuperation for the synapses where plastic (macromolecular) changes occurred during wakefulness, as a consequence of higher nervous activities such as those involved in learning or conditioning." (1966, p. 376). This proposal differs from the others discussed in this chapter in that Moruzzi does not specify which stage of sleep is involved in the learning process, but instead speaks of sleep as a whole. Nevertheless, since deprivation provides little information and hence little opportunity for learning, it might be argued that sleep itself, or one of its stages, should be decreased in the later part of the deprivation period. Chapter 4 has shown that this is not the case. Thus, it would seem that this hypothesis is unsupported by the present experimental evidence.

Another class of theories concerned with the function of sleep was also reviewed in Chapter 2. These are fewer in number and less variable in details than the REM-cognition theories, and quite specifically propose that slow-wave sleep is related to recovery from physical fatigue. To quote Hobson, "... a functional role of synchronized sleep in recovery from fatigue is strongly suggested." (1968, p. 1505). Among the published experiments

concerned with this proposal can be found those which support it (Hobson, 1968; Matsumoto et al., 1968) and those which do not (Webb and Friedmann, 1969; Kauri, 1968 a and b, 1969). When applied to subjects undergoing perceptual deprivation, this theory would seem to predict a decrease in slow-wave sleep, on the basis of the following considerations. Deprived subjects are generally restricted to lying on a bed, and so are certainly much less active than normal. Even though they show a progressive increase in restlessness during deprivation (Smith, Myers, and Murphy, 1962, 1967), this is unlikely ever to exceed or even approach the activity level of a normal day. In consequence, deprived subjects are extremely unlikely to be physically fatigued, and might be expected to show a decline in slow-wave sleep. Results obtained in the present experiment do not confirm this prediction, since stages III and IV, which constitute slow-wave sleep, do not change at all during the deprivation period.

Very different from any of these theories of sleep and dream function, which are based on experimental evidence, is the psychoanalytic view of dreaming. As the originator of these theories, Freud (1953) regarded sleep, and especially dreams, as an opportunity to suspend the reality testing function and revert to a more primitive mode of thought, which he called primary process thought. There are several theories of sensory deprivation based in this psychoanalytic tradition (Rapaport, 1958; Goldberger and Holt, 1961) which consider that this primary process thinking is enhanced under these conditions, while secondary process

thinking is depressed. Hallucinations are one of the observable manifestations of primary process thought, appearing in the waking state only in psychotics, but transformed into the vivid imagery of dreams in normals. It is known that deprivation conditions can produce hallucinations in normal subjects, though this is by no means an invariant feature of the deprivation experience. One would expect a reciprocal relationship between the primary processes involved in hallucinations and dreams during deprivation, such that a difference between deprived subjects who do and do not hallucinate might be observed in some aspect of dreaming sleep, or stage I REM. This difference should appear as a decrease in stage I REM for hallucinators as opposed to non-hallucinators, since primary process material appears in the hallucinations and would then not need to be expressed as dreams. The present experiment included only one subject (P.C.) who experienced full blown hallucinations. It is of interest that this subject did not differ from the others, all non-hallucinators, in the amount of time he spent in stage I REM.

P.C. first reported complex hallucinations on day 3 of deprivation, but admitted he had experienced some vivid visual imagery prior to this, although he had not mentioned it. Frequently, the hallucinated scene was a landscape, and was observed by the subject from a bizarre position. For example, he viewed a high cliff as though he were suspended in the air, or an array of underground stalagtites and stalagmites as though his head were pressing against the cavern's roof. P.C. frequently

used the words grotesque and wierd to describe his experiences, as, for example, in relating a scene of a cobbled street, whose stones were not stones at all, but the heads of many primitively formed brown creatures. This evidence, then, seems not to support the psychoanalytic approach to hallucinations and dreams.

To summarize, the data collected in this experiment do not seem to support any of the current theories of sleep function, nor any simple form of the arousal interpretation of deprivation. More specifically, those theories which link stage I REM sleep to some aspect of cognitive function, either learning itself, or information processing, or memory storage, seem to generate inaccurate predictions when applied to the deprivation situation. Even when they make provision for the particular importance of novelty, they are unsupported by the present experimental data. Neither does this experiment confirm those theories which propose that slow-wave sleep plays a role in recovery from physical fatigue. Further, the proposal of psychoanalytic theory that stage I REM should decrease in hallucinating subjects seems to be untrue. Similarly, the arousal interpretation of deprivation would seem to have some difficulty in accounting for the finding that deprived subjects return to a normal sleep-wakefulness cycle as deprivation progresses.

In conclusion, then, it would seem that the recent emphasis of sleep theorists on both cognitive and physical fatigue functions has been misplaced. The present results suggest that sleep is still a biological phenomenon of unknown function,

regulated in a largely unknown way. As for the arousal interpretation of deprivation, it is clear that, in the oversimplified form in which it is sometimes used, it fails to account for the present findings. Although it is well known that the reticular formation is itself influenced by the cortex whose activity it regulates, few theories of deprivation phenomena take account of this downstream activity, preferring to concentrate on the ascending reticulo-cortical influences. An arousal theory which fails to recognize descending activity is unable to account for the present results. However, this is possible for the more sophisticated form of the theory (Lindsley, 1961), since it can be postulated that the reduced output of the reticular formation to the cortex evokes a compensatory cortico-reticular flow, thus tending to restabilize the entire system. However, the observation that the alpha rhythm declines very slowly during deprivation, and requires long periods afterwards to recover its normal frequency, still poses some difficulties for this interpretation. It would seem wise, then, to avoid a general application of the arousal theory to all deprivation phenomena.

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