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BY

JOSEPH G. SALAMY

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AN INVESTIGATION INTO THE MOTIVATIONAL

PROPERTIES OF REM SLEEP

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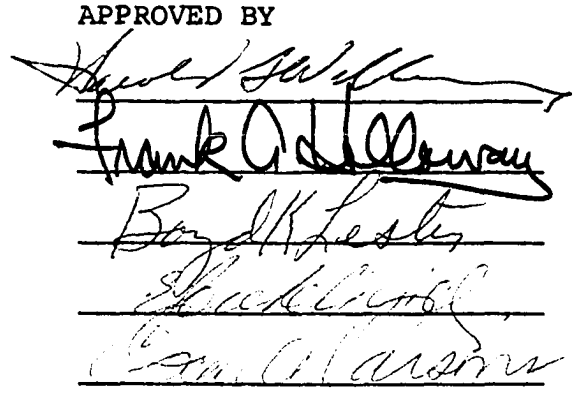

DISSERTATION COMMITTEE

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AN INVESTIGATION INTO THE MOTIVATIONAL PROPERTIES OF REM SLEEP

CHAPTER I

INTRODUCTION

Considerable progress in understanding sleep has been made in recent years. However, while sleep researchers have been diligently collecting empirical facts, they have remained essentially atheoretical (Webb, 1968). In this paper, an attempt will be made to fit the characteristics of REM sleep into the theoretical framework of motivation.

In this introduction, the electrophysiological stages of sleep will be briefly reviewed. Then a statement of the problem will be presented. This will be followed by a description of some important notions of motivation. Finally, there will be a review of the literature indicating supporting lines of evidence for considering REM sleep as a motivational variable.

The Stages of Sleep

The view that sleep is a state characterized by a drastic and passive diminution of activity has been eschewed. Sleep is now deemed an active process (see Koella, 1967) and an integrated activity involving the entire organism. It, thus, represents an orderly redistribution of activity. Within this context, REM sleep presents its own specific neural organization. Below is a brief description of the sleep cycles in man and in the cat.

As an individual descends into uninterrupted sleep, successive electroencephalographic (EEG) stages can be distinguished. The 8 - 12 cps alpha rhythm, characteristic of relaxed wakefulness, deminishes in amplitude and eventually disappears, giving way to the low voltage, fast irregular rhythm of stage 1 sleep. This is the borderland between waking and sleeping. If awakened at this point, subjects (Ss) assert that they were not really asleep.

After only a few minutes, the voltage increases (the EEG tracing becomes larger) and the EEG becomes slower in frequency. Occasionally, brief bursts or "spindles" of 12 - 15 cps appear. Such a record signifies the sleeper has reached stage 2. In this stage, the S is quite soundly asleep, yet he is easily awakened. This stage is described

as light to moderately deep sleep, and it occupies most of the night.

As stage 3 ensues, the voltage continues to increase and the frequency continues to decrease. Slow waves of 1 - 2 cps, which may reach 300 microvolts (compared to 60 microvolts of the waking alpha rhythm) begin to appear. The S's musculature is relaxed. Heart rate and blood pressure are falling, and respiration is even. This pattern continues until the spindling disappears completely. Stage 3 is sometimes considered to be a transitional stage between light and deep sleep. It usually only lasts for a few minutes.

Soon the record is dominated by almost continuous high voltage 0.5 - 1 cps delta waves. When this occurs, the sleeper has attained stage 4. Noises which caused arousal earlier in the night now leave the record unchanged.

Generally, it takes about 90 minutes to complete the descent through stage 4. Soon thereafter, stage 2 is typically resumed, followed closely by the irregular low voltage fast EEG of stage 1. However, in contrast to the rather slow rolling eye movements that occur in the initial stage 1, the eyes now move back and forth in rapid saccadic movements. These rapid eye movements are referred to as REMs.

They are typically associated with the unique state of consciousness in which vivid dreaming occurs. This stage is known as REM sleep. Aside from the brain waves and eye movements, this stage can be identified by the rather complete loss of muscle tonus. The muscle tension is usually recorded from electrodes placed under the chin (submental muscles). Sleeping Ss are unresponsive to external sounds in this stage. The autonomic activity which had been regular and steadily falling, is now marked by fluctuation and variability. A particularly noteworthy characteristic of REM sleep is that during this stage penile erections occur in males. This phenomenon has been observed in infancy through adulthood.

REM periods usually range from 3 - 50 minutes in duration. They account for about 20 percent of the nights sleep, and occur 3 - 6 times a night. They are shorter in duration in the beginning of the night and lengthen as morning approaches. By contrast, stage 4 periods are longer early in the night and become shorter as the night progresses (Oswald, 1962; Luce, 1965; Murray, 1965).

Thus, in man, sleep is represented by the five successively recurring EEG stages described above. In the sleeping cat, electrical brain activity can be divided into

two recurring patterns. In one, there is synchronized cortical activity consisting of spindles and/or high voltage slow waves. This is often referred to as slow wave sleep.

The other pattern is a low voltage fast cortical rhythm. This is called activated sleep or paradoxical sleep. This stage is assumed to correspond to REM sleep in humans. It occurs after a variable period of slow wave sleep and then appears periodically, during slow wave sleep. It occupies 20 - 25 percent of behavioral sleep and has a mean duration of 6 minutes.

The fast cortical activity is accompanied by a continuous 4 - 7 cps theta rhythm in the ventral and dorsal hippocampus. As in humans, this stage can be identified by the appearance of REMs and loss of muscle tonus (recorded from the neck muscles) (Jouvet, 1967c).

Statement of the Problem

Only recently have efforts begun to systematically conceptualize the EEG findings of sleep and related phenomena into behavioral theory. In his book "Sleep, Dreams, and Arousal", Murray (1965) proposed that the motive to sleep is comparable to other motives. He supported this view by drawing analogies between the neural mechanisms of

sleep and other behavior and relating them within the motivational context.

In this paper, an attempt will be made to assign the status of a motivational variable to REM sleep. Thus, while Murray dealt with a motive to sleep, this work is concerned with motivation to maintain a specific stage of sleep. As with Murray's sleep motive, the analysis of the "REM motive" begins with the assumption that REM sleep shares the basic characteristics of other primary physiological motives.

To justify this assumption, the following three criteria should be met: one should be able to show that REM sleep serves a biological function; that it operates through a physiological mechanism, and that its deprivation motivates learning and performance (see Murray, 1965).

There is ample evidence favoring the first two of these criteria. It will be discussed in a later section of this chapter. On the basis of this evidence, it was argued that the third criteria could be met too. Thus, the goal of this research is to demonstrate the motivational properties of REM sleep by showing that Ss will learn to perform a response in order to preserve that stage, and that such an effect will be enhanced by REM deprivation.

Motivation and Reinforcement

In this section, the lines of thought by which the above criteria were established will be discussed. A complete review of the literature is not intended.

The importance of a behavior for survival has been crucial in identifying motives. Thus, not infrequently motivation is approached with a discussion of organic or physiological needs. A need being defined as a deficiency or excess, which, if not corrected, is injurious to the organism or species (Hull, 1943; Hall, 1961).

Closely allied to motivation, and sometimes used synonomously with it (Brown, 1961) is the notion of drive. Drive is used to refer to the state of the organism that makes an activity occur (Bindra and Stewart, 1966; Valenstein, 1968). Primary drives are those motivational variables which produce their effects through physiological mechanisms (Brown, 1961).

Both drive and motivation are intervening variables. As such, they are not directly observed, but are identified with certain organismic antecedent conditions and inferred from behavior.

The demonstration of a need and drive for a given variable notwithstanding, not all theorists would classify

it as a motive on that basis alone. For example, Miller and Dollard (1941); Miller (1959); and Teitelbaum (1966, 1968) emphasize the potentiation of learning and performance as chief criteria for classifying a variable as a motive. Since oxygen deprivation apparently does not result in learning or performance of a response to obtain oxygen, Miller and Dollard would not classify the need for oxygen as a motive (Murray, 1965). Thus, the demonstration of a physiological need is in itself insufficient to lead to the postulation of a motive.

Teitelbaum (1968) points out that when an act is a completely automatic consequence of a stimulus, there is no need to speak of motivation; since a fixed built-in relation exists between stimulus and response, why infer the existence of a central motivational state underlying the response. One is justified in assigning motivational properties to behavior only when the existence of a central motivational state apart from the stimulus and the response is demonstrable. This can be achieved, according to Teitelbaum, by the learning process. The experimental situation is arranged so that the response results in a reinforcement and the organism learns the connection between them. Motivation is revealed once the relationship is

learned and the organism performs to obtain the reinforcement. By reserving the term motivation for situations in which an organism can learn an arbitrary response to obtain the appropriate goal object, it is possible to distinguish consummatory behavior related drive states from motivation.

Very simply then, an organism deprived of food will learn to perform a response to obtain food. This observation leads to the postulation of a hungar motive. Murray (1965) argues that if sleep is to be considered a motive, it must be shown to have the same relationship to learning and performance as do other motives. Williams, Lubin, and Goodnow (1959) have shown that sleep deprivation has a deleterious effect on learning and performance. Murray notes, however, that this is not analogous to the paradigmatic situation of a rat pressing a bar for food. The responses used in the sleep derivation studies are not relevant to the sleep motive; i.e., they do not lead to the reduction of the sleep motive. Thus, to test the validity of viewing REM sleep as a motive against the learning - performance criterion, the responses ideally should lead to an opportunity to obtain REM sleep. The term reinforcer, as used in an empirical sense, refers to events which have the effect of increasing the probability of the occurence of a response (Kimble, 1961).

A persistent issue in behavioral theory has been whether behavior is a result of motivation or reinforcement. Bolles (1967) recognizes certain equivalences between the two, and suggests motivational phenomena are isomorphic with reinforcement phenomena. Thus, lists of reinforcers, drives, and needs could be compiled that would correspond to each other in a one - to - one fashion (Hull, 1943; Hall, 1961; Millenson, 1967). For example, a starved organism has a need for food and food will serve as a reinforcer.

There are many ways by which reinforcement is thought to come about, e.g., need reduction, drive reduction, drive-stimulus reduction, behavioral responding, stimulus intensity reduction, brain stimulation, etc. Miller (1959) suggested that a reinforcing effect results whenever events allow for the occurence of a response which has been activated but blocked. This is termed response-release reinforcement. The act of eating will be reinforcing to an animal primed to eat food, but prevented by deprivation (Livingston, 1967). This notion of reinforcement can easily be generalized to the present study. Thus, a response which allows consummation of the REM state once it has been aroused, but blocked by awakening should be reinforced. In this case, responses which lead to the continuance of the REM state

(positive stimulation) result in positive reinforcement. Awakening, which leads to the cessation of the REM state would serve as punishment.

If an experimental situation was arranged such that responding to a signal while in REM sleep would prevent the appearance of the noxious stimulation (awakening) an avoidance paradigm would be established.

This is the procedure by which the demonstration of the motivational properties of REM sleep will be attempted. It is hypothesized that if REM sleep possesses motivational qualities, then subjects should learn to perform a response in order to preserve it.

Deprivation is the cardinal operation that affects the value of a reinforcer or the strength of a drive (Millenson, 1967). One way in which the relationship between drive and response acquisition can be investigated is to compare the performance of groups learning a task under different drive levels. The present experimental design will permit such comparisons. It was argued that if there is a need for REM sleep, partial deprivation should exacerbate this need and lead to increased efforts to alleviate it. Thus, REM deprived subjects should show superior performance in a situation which will result in

the satisfaction of the drive.

In the next section, the literature suggesting that REM sleep may have motivational properties will be reviewed. While the neuroanatomy and biochemistry of the reward system do not correspond one - to - one to those of the REM system, there is evidence that there are some elements in common to the two systems. The review of the literature will be separated into three sections: neuroanatomy, biochemistry, and behavior. In each section, an attempt will be made to elucidate the elements common to the REM and reward systems. The sections on neuroanatomy and biochemistry will be elaborated in Appendices A and B respectively, and contradictory evidence will be raised there.

Review of the Literature

The effects of an activity are often examined by depriving an organism of that activity. For the most part, REM deprivation experiments have addressed themselves to the questions of the necessity and functions of dreams or REM sleep.

The classic experiment of this type was performed by Dement (1960), who used a method of forced awakenings. This consisted of arousing Ss upon the appearance of EEG

stage 1 and REMs. This procedure results in about a 70 percent reduction in REM time. The major effects of this experiment treatment included: increased attempts to enter the REM state, about a 50 percent average increase in REM sleep during recovery (uninterrupted sleep), and mild behavior disturbances, such as increase anxiety and appetite, and difficulty in concentration. None of these effects were found in control subjects aroused from NREM (non-REM sleep).

Kales, Hoedemaker, Jacobson, and Lichtenstein (1964) achieved approximately a 95 percent reduction in REM sleep by awakening subjects at the point of loss of muscle tonus (this usually precedes REMs). They did not report "psychological disturbances" in two Ss who were deprived for ten nights. However, the other effects were confirmed.

The findings of increased attempts to enter the REM state with repeated interruptions, and a rebound during recovery from deprivation implies a critical need for this stage. This suggests that REM sleep may possess motivational properties such as those of other physiological needs.

REM and Reward Systems: Neuroanatomical Relations

Some evidence by Dement (1965) makes the relationship between motivated behavior and REM sleep apparent. Six of twelve cats REM deprived from 30 - 70 days became hypersexual.

With REM deprivation alone, these animals ate only slightly more than controls, but upon starvation, motivation to eat increased markedly. In addition, the REM deprived cats revealed difficulty in learning a simple maze, a decrease in electroconvulsive threshold (increased CNS excitability, and increased restlessness and grooming. Dement suggested that with REM deprivation some metabolite accumulates in the nervous system which causes a general increase in excitability and potentiates drive-oriented behavior.

Fisher (1967) pointed out that the behavioral disturbances produced by REM deprivation resemble those resulting from bilateral ablation of the temporal lobes; i.e., the Kluver-Bucy syndrome. He suggested a possible relationship between the hypersexual behavior of the REM deprived cat and the normal hypersexuality (penile erections which typically accompany REM sleep) of the REM state. He postulated that during REM sleep the drive centers within the limbic system concerned with self and species preservation, i.e., sexual, oral, aggressive behavior, are released from inhibition.

The various structures involved in self and species preservation are to a considerable extent connected by the medial forebrain bundle (MfB), (see Appendix A). The area which parallels the course of the MfB provides one of the

major anatomical substrates of the rewarding effects of electrical stimulation (Olds and Milner, 1954; Olds and Olds, 1963; McCleary and Moore, 1965). Olds and Milner (1965) obtained self stimulation with electrodes implanted in the septum and other limbic structures. MacLean (1968a) reports that he once asked Olds if he had ever observed penile erections in these animals. Olds replied that in about one-third of the animals, he noticed erections, and almost invariably when erections were obtained, so was self-stimulation along with enchanced grooming.

These observations imply that activities related to self and species preservation are manifested in the REM state and exaggerated by REM deprivation. The anatomical location of these mechanisms corresponds to some degree to the structures of the limbic system which are connected by the MfB and which yield positive reinforcement upon self-stimulation.

While much of this evidence for the intersect of the REM and reward systems is indirect, the limbic-midbrain circuits role in REM sleep has to some extent been substantiated. Jouvet and Jouvet (1963) found that lesions in the septum, subthalamic region, interpenduncular region, and medial portion of the anterior pontine tegmentum suppressed

totally or partially the fast cortical activity and the hippocampal theta rhythm. On the other hand, mesencephalic lesions which interrupted the ascending reticular activating system suppressed cortical arousal, but not the tonic cortical desynchronization observed during REM sleep.

Ellman and Steiner (1969a) found direct evidence for an association between REM sleep and lateral hypothalamic reward centers. They argued that if such a link did in fact exist, a reciprocal relationship between REM sleep and electrical self-stimulation to the lateral hypothalamus should be demonstrable; i.e., REM deprivation should lower self-stimulation thresholds and increase response rates. This hypothesis was experimentally verified. After only one day of REM deprivation, the rate intensity function was shifted significantly to lower values. That is, rats responded more with lower intensity stimulation. In a second experiment, Ellman and Steiner (1969b) showed that lateral hypothalamic self-stimulation could affect REM sleep patterns. They found that REM deprived rats on an ad lib self-stimulation schedule displayed a 21% increase in REM time during recovery from deprivation, whereas control subjects (no electrical stimulation) showed a 53% REM rebound upon recovery.

Biochemical Relations

While the chemistry of the REM and reward systems is complex and requires further elucidation, there is some evidence for possible convergence. This evidence centers around the point that the catecholamines play a role in both the REM and reward systems.

Jouvet (1967b) induced a desynchronized EEG with injections of 3, 4 dihydroxphenylalanine (DOPA). DOPA was used because it is a precurser of norepinephrine and it will penetrate the blood brain barrier. REM sleep can be obtained after some time with DOPA after suppression with reserpine (reserpine depresses the catecholamine levels of the brain). That is, all the tonic phenomena associated with REM sleep reappear once the normal catecholamine level of the brain is re-established (Jouvet, 1967b).

Mandell, Brill, Mandell, Rodnick, Rubin, and Sheff (1966) have shown that the urinary output of catecholamine metabolites increases several times during the night usually following REM periods. Plasma levels of free fatty acids increase following a release of catecholamines and have been found to be higher in REM sleep (Hartmann, 1967). Finally, Stern (1969) using drugs which increase CNS levels of norepinephrine found a significant improvement in active

and passive avoidance performance following REM deprivation. This effect was not obtained with non REM-deprived controls.

Stein (1966, 1967, 1968) investigating some neurochemical substrates of reinforcement found that drugs which release norepinephrine from stores in the brain facilitate self-stimulation, whereas drugs like chlorpromazine which block adrenergic transmission and deplete brain norepinephrine inhibit self-stimulation. Incidentally, chlorpromazine depresses activity in the hypothalamus (Zukauskas and Machne, 1964) and decreases REM time (Hartmann, 1967). Hillarp, Fuxe, and Dahlstrom (1966) mapped catecholamine - containing neurous and located their sites or origin in the ventromedial mesencephalon (limbic - midbrain area). Fibers ascend from this area and terminate in the lateral hypothalamus, limbic lobe, and neocortex. By lesioning the MfB and assaying for norepinephrine, Heller, Seiden, and Moore (1966) concluded that the adrenergic fibers are arranged in an ascending system, since only structures rostral to the lesion showed a reduction of norepinephrine. Stein (1968, 1968b) suggested this ascending adrenergic system might be identified with at least part of the positive reinforcement system. While direct evidence for the ascending adrenergic systems role in REM sleep is lacking, it is clear that

catecholamines play an important role in both the REM and reward systems (see Appendix B).

Behavioral Relations

There is considerable evidence demonstrating that human Ss can make specified responses during sleep (Oswald, Taylor, and Treisman, 1960; Granda and Hammack, 1961; Williams, Morlock and Morlock, 1963, 1966; Antrobus and Fisher, 1965; Salamy and Williams, 1969). Williams et al. (1963, 1966) investigated the effect of reinforcement on behavioral responding during sleep. Prior to sleep Ss were instructed to close a switch taped to their hand whenever a tone was presented. After three nights on instruction alone, contingent punishment was introduced. Failure to respond resulted in the following sequence of events: a high intensity fire alarm was sounded, followed by flashing lights and shocks to the leg. While all Ss displayed the capacity to respond, the strongest reinforcement effect occured in stage REM, where the proportion of correct reponses went from 10% on instruction to 50% on avoidance nights. Many of these responses occured without EEG evidence of prior awakening. That auditory discrimination can occur in stage 2 without prior awakening was demonstrated by

Oswald, et al, (1960), who found that meaningful stimuli were more likely to evoke K complexes and behavioral responses than were non-meaningful stimuli.

There is some evidence that sleeping Ss can discriminate different stages of sleep upon awakening (Antrobus and Antrobus, 1967). Salamy and Janes (1968) found that stage 2 was as readily and reliably identified as stage REM. Antrobus et al. (1965) found that S could respond on a switch to both dream and non-dream conditions during sleep. In this study no external signal was used, Ss were merely instructed to respond when they were dreaming and not dreaming. This research was extended by Salamy and Williams (1969) who used awakening as punishment. They instructed Ss to close a switch four times when they started dreaming. The Ss were awakened for 4 minutes if they did not respond within 3 minutes of REM onset. Very few responses were obtained on an instruction night, and there was an insignificant increase on the first avoidance night. On the second avoidance night, there was a highly significant improvement in performance. However, on the third avoidance night, there was an almost as spectacular decrease in correct responding. Performance was slightly better on the extinction night compared with the third avoidance

night, and significantly superior to the instruction night.

Clearly, the Ss learned to discriminate and respond to their internal states in order to avoid awakening. This was achieved, in most instances, without EEG signs of arousal. Extrapolating, these data are amenable to the following possible interpretation: the greatest amount of responding occured on the second avoidance night; this effect was thought to be due to a substantial degree of REM deprivation incurred on the first avoidance night and continued on the second. Possibly this increased the drive for REM sleep and made REM stimuli more salient, allowing REM sleep after a correct response to serve as an adequate reinforcer. That is, Ss were motivated to perform the avoidance response in order to obtain REM sleep and curtail deprivation. The precipitous decline in performance on the third avoidance night was thought to result from a partial satiation of the drive.

Ellman (1969) provides further evidence suggesting that REM sleep has motivational properties. Ss were given 5 - 10 baseline nights, 6 - 11 deprivation nights and 7 - 13 recovery nights. After five hours of uninterrupted sleep (by which time 50% of the nights REM time had elapsed), human Ss were deprived of stage REM. During recovery,

75% - 80% of total REM time (TRT) occured in the first five hours of the night. This reversal of the usual cycle persisted for two to three recovery nights. In a second study, 2 - 4 nights of uninterrupted sleep were interspersed among the deprivation nights, to establish an intermittent reinforcement schedule. On recovery nights, again 75% - 85% TRT occured within the first five hours compared to 50% on baseline nights. However, this pattern endured for 7 - 13 recovery nights compared to the 2 - 3 nights on the continuous reinforcement schedule. Thus, as expected, the partial reinforcement schedule created greater resistance to extinction. Ellman concluded that REM sleep patterns can be experimentally modified, and suggested that learning may be able to account for the modification.

The research cited above suggests a functional relationship between the REM and reward systems, and provides the basis for the hypotheses to be tested in this experiment. Specifically, these hypotheses are: (1) If REM sleep possesses motivational qualities, then Ss should learn to perform a response in order to preserve that stage; (2) partial REM deprivation should enhance such an effect.

CHAPTER II

METHOD

Subjects

Thirty-two male paid volunteers between the ages of 21 and 28 years served as subjects (Ss). In addition to these 32 Ss, 4 Ss were rejected because they had great difficulty sleeping. Each S spent one night in the laboratory. Most of the Ss were medical students, all were college students (graduate and undergraduate).

Procedure and Apparatus

The experiment was programmed as a 2 x 2 x 2 factorial design, with two levels for each factor and four observations per cell, in the following manner:

	Factor	Level 1	Level 2
A.	stage of sleep	stage REM (REM)	stage 2
B.	reinforcement	instructions (inst)	avoidance (avoid)
C.	deprivation	REM deprived (RD)	non-REM deprived (NRD)

Thus, Ss were randomly assigned to one of the following eight groups:

1. RD - inst. - REM
2. RD - avoid. - REM
3. RD - inst. - stage 2
4. RD - avoid. - stage 2
5. NRD - inst. - REM
6. NRD - avoid. - REM
7. NRD - inst. - stage 2
8. NRD - avoid. - stage 2

These groups were selected such that the stage 2 and REM groups permitted comparisons between these two stages of sleep. Stage 2 was considered to be a control. The inst. and avoid. groups allowed the determination of an operant level (inst.) to be compared to the reinforced level (avoid.) of performance. Thus, the effectiveness of the punishment (awakening) could be assessed. The RD and NRD groups were regarded as high and low drive groups respectively. Thus, the effect of drive level on performance could be examined.

Thus, the inst. vs. avoid. groups provide information relevant to the hypothesis that Ss will learn to perform a response in order to maintain REM sleep. The RD vs. NRD comparisons are pertinent to the hypothesis that RD should improve performance in the REM groups. The stage 2 vs. REM comparisons permitted examination of the specificity of the

experimental effects with regard to REM sleep.

Factor A (stage of sleep) refers to the stage in which tones were presented. Factor B (reinforcement) indicates whether Ss received only instructions to respond in a given stage (inst.) or if, in addition, they were negatively reinforced by awakenings for response failure (avoid.). The inst. groups suffered no consequences for response failure. The punishment associated with the avoid. groups was awakening, which lasted for 1.5 minutes. It occured 5 seconds after the offset of the 3 second duration tone, and was effected by calling Ss names over an intercom. Tone presentations were confined to the stage of sleep to which Ss were assigned within the final 2.5 hours of the run. The response required was three presses on a microswitch taped to S's preferred hand.

Factor C (REM deprivation) was carried out to within 2.5 hours before the Ss had to get up in the morning (in RD groups only), and was achieved by the method of forced awakenings. These awakenings lasted from 30 - 60 seconds.

REM sleep was identified by the presence of a low voltage fast EEG, REMs, and the relative absence of chin muscle tonus. Occasionally, after several awakenings, muscle tonus remained at a very low level even when the

S was awake. In this situation the EEG and REMs were used to judge REMs sleep. If REMs and low muscle tension were present, but spindles were observable in the EEG, the S was considered not to be in REM sleep. In the event that the low voltage fast EEG and loss of muscle tonus evinced without the appearance of REMs, the S was not considered to be in REM sleep unless this pattern persisted for 3 minutes.

The tone level was adjusted to 35 db re: waking threshold, established before "lights out" for each S. Once trials had begun, an attempt was made to present a tone every 3 minutes. Three minutes was selected because it was felt that a longer period would give Ss too much REM sleep between trials, so that "motivation" would be reduced. Also, this period provided ample time for determination of the stage of sleep.

Before "lights out" all Ss received the following instructions:

> At certain times during the night you may be awakened, by calling your name over an intercom. These awakenings will only last from 30 - 60 seconds, but may occur close together. Also at certain times during the night, a tone will be presented over the speaker above your head. You are to press the switch taped to your hand three times when the tone is played.

The S was not told that response failures might be punished by interruption of sleep.

All recordings were obtained with a Grass Model 6 electroencephalograph at a chart speed of 15mm/sec. Grass silver disk electrodes were employed.

EEG tracings were taken from electrodes placed at $C_3 - A_2$, and $C_3 - O_1$, in accordance with the 10 - 20 international system. Muscle tension was recorded from leads placed on the submental muscles. Eye movements were obtained from placements on the outer canthi of each eye.

Tones were presented through a loud speaker approximately two feet above the S's head and were generated by a Hewlett-Packard Model 200AB Audio Oscilator at 500 cps. The duration of the tone was controlled by a Hunter Interval timer, the amplitude by a Hewlett-Packard 350c Attenuator set. Communications between S and experimenter were carried out over intercom.

The data were treated by analysis of variance. In cases where the data were in the form of proportions, the proportions were transformed to arcsins, and statistics were carried out on the transformed scores.

The alpha rhythm was analyzed to see if it was more readily evoked by a particular treatment, and if correct

responding was more often accompanied by it. The alpha rhythm was used to indicate transient awakening. It was defined as the appearance of either a mixed or pure 8 to 12 cps EEG of at least four cycles duration. If the alpha activity could be detected during a trial, that trial was termed an alpha event.

The number of trials and responses for each S was independently scored and confirmed.

CHAPTER III

RESULTS

Table 1 shows the amount of time Ss spent in stages REM and 4, up to and following trials (tone presentations) for each treatment combination. The trials began 2.5 hours before Ss had to get up in the morning. Thus, the first column shows the amount of time that Ss spent in REM sleep before trials began. It will be noted that the NRD Instruction stage 2 group, spent considerably less time in stage REM before trials began than the other NRD groups. This is thought to reflect the "laboratory effect". That is, Ss often do not exhibit their characteristic sleep patterns on the first night in the laboratory. While this is most conspicuous in the NRD Instruction stage 2 group, many Ss displayed slightly disturbed sleep.

Table 2 presents the number of trials, the number of successes, the average percent of success (see Guilford, 1965, p. 63), the number of awakenings (due to the deprivation procedure, and punishments in the avoidance procedure)

TABLE 1

REM AND STAGE 4 TIME UP TO AND DURING LAST 2.5 HOURS, TOTAL REM AND STAGE 4 TIME, AND MEAN TOTAL SLEEP TIME FOR ALL GROUPS

	REM Time up to 2.5 hrs. before end of run	REM Time in last 2.5 hrs.	Total REM Time	Stage 4 up to 2.5 hrs. before end of run	Stage 4 in last 2.5 hrs.	Total Stage 4 Time	Mean Total Sleep Time
Stage 2							
NRD Inst.	28	209*	238*	264	34	298	358.25
NRD Avoid.	186	218*	404*	244	0	244	392.00
RD Inst.	1	217*	217*	308	20	328	376.50
RD Avoid.	0	208	208*	205	25	230	347.75
Stage REM							
NRD Inst.	123	186	309*	229	49	278	402.00
NRD Avoid.	116	126	242*	229	25	254	402.75
RD Inst.	2	231	233*	174	33	207	379.00
RD Avoid.	6	200	206*	257	33	290	382.50

* Some Ss in that group were still in REM at end of run.

TABLE 2

NUMBER OF TRIALS, NUMBER OF SUCCESSES, MEAN PERCENT SUCCESS
NUMBER WAKE-UPS, AND TIME AWAKE
FOR EIGHT GROUPS AND TWO
SLEEP STAGES

Stage and Treatment	Trials	Successes	Mean % Successes	Wake-Ups		Time Awake
				Punishment	Deprivation	
Stage 2						
NRD Inst.	46	21	43.33	0	0	0
NRD Avoid.	41	16	38.93	25	0	57
RD Inst.	45	10	22.00	0	31	50
RD Avoid.	36	13	37.17	23	23	82
Stage REM						
NRD Inst.	58	8	13.88	0	0	0
NRD Avoid.	33	13	38.21	20	0	90
RD Inst.	71	1	1.35	0	72	132
RD Avoid.	61	49	84.95	12	28	113

and the time (in minutes) Ss were awake due to awakenings (includes wake-up time plus time to return to sleep) for each treatment and stage of sleep.

An arcsin transformation was applied to the proportion of correct responses for each S and a three-way analysis of variances was performed (Winer, 1962).

Table 3 provides the summary of the analysis of variance.

TABLE 3

SUMMARY OF ANALYSIS OF VARIANCE

Source of Variation	SS	df	MS	F
A (stage of sleep)	0.39	1	0.39	1.28
B (reinforcement)	3.09	1	3.09	9.99*
C (deprivation	0.04	1	0.04	0.13
AB	4.01	1	4.01	12.96*
AC	0.87	1	0.87	2.82
BC	2.67	1	2.67	8.62*
ABC	0.56	1	0.56	1.81
within cell (experimental error)	7.43	24	0.30	
TOTAL	19.10	31		

*p/.01

The main effect for reinforcement condition (B), the stage by reinforcement (AB) interaction, and the rein-

forcement by deprivation (BC) interaction were all significant at the .01 level of confidence (F .99 (1, 24) = 7.82). The significant main effect for reinforcement indicates a significant difference between the instruction and avoidance conditions. Table 4 gives the analysis of variance for the simple effects associated with the AB and BC interactions.

The stage for instruction, reinforcement for REM, reinforcement for REM deprivation, and deprivation for instruction simple effects were statistically significant.

TABLE 4

ANALYSIS OF VARIANCE FOR SIMPLE EFFECTS

Source of Variation	SS	df	MS	F
A for b1 (stage for instruction)	3.47	1	3.47	11.21a
A for b2 (stage for avoidance)	0.94	1	0.94	3.04
B for a1 (reinforcement for REM)	7.08	1	7.08	22.86a
B for a2 (reinforcement for 2)	0.02	1	0.02	0.09
B for c1 (reinforcement for RD)	5.76	1	5.76	18.60a
B for c2 (reinforcement for NRD)	0.00	1	0.00	0.02
C for b1 (deprivation for inst.)	1.68	1	1.68	5.44b
C for b2 (deprivation for avoid.)	1.02	1	1.02	3.31
within cell	7.43	24	0.30	

a p/.01
b p/.05

The analysis of the stage by reinforcement interaction revealed that there was no difference between stage 2 and REM under the avoidance treatment, but the stage 2 group performed significantly better than the REM group under instructions alone. The difference between instruction and avoidance treatments for stage 2 was negligible, but highly significant for stage REM. This finding supports the hypothesis that S's will learn to perform a response in order to obtain REM sleep, since performance improved under the avoidance treatment compared to instructions. That is, the punishment had a "motivating effect" in stage REM, but not in stage 2.

The analysis of the reinforcement by deprivation interaction revealed that under the NRD condition, there was no difference between the reinforcements; however, the difference is significant for the RD condition. This difference is larger for REM than it is for stage 2. There was no difference in deprivation conditions under the avoidance procedure, but a significant difference was found under instructions, with the NRD condition eliciting better performance. Thus, REM deprivation had a suppressing effect on performance under instructions alone. This was found for both stages of sleep.

Figure 1 depicts the average proportion of successes plotted for the two stages of sleep and for both deprivation conditions under each reinforcement. An a posteriori comparison between the NRD instruction groups for stage 2 and REM did not quite attain significance ($F = 4.16$ $F_{.95}(1, 24) = 4.26$). However, the stage 2 groups superior performance is evident. In this figure, it is also evident that REM deprivation had an inhibitory effect on performance under instructions, but facilatated it under the avoidance regime. These data support the expectation that REM deprivation would potentiate performance in the stage REM Ss.

In Figure 2 the average proportion of success is plotted for reinforcement and deprivation conditions in each stage of sleep. The avoidance treatment had little effect under the RD condition in stage 2, but a profound effect on performance was obtained in stage REM. Neither the REM - NRD instruction vs. avoidance, nor the stage 2 - RD instruction vs. avoidance a posteriori comparisons were significant ($F = 1.53$, and 0.33, respectively).

Williams et al.'s (1966) paper raised a question as to the effect the presence or absence of the alpha rhythm may have on performance for each reinforcement condition

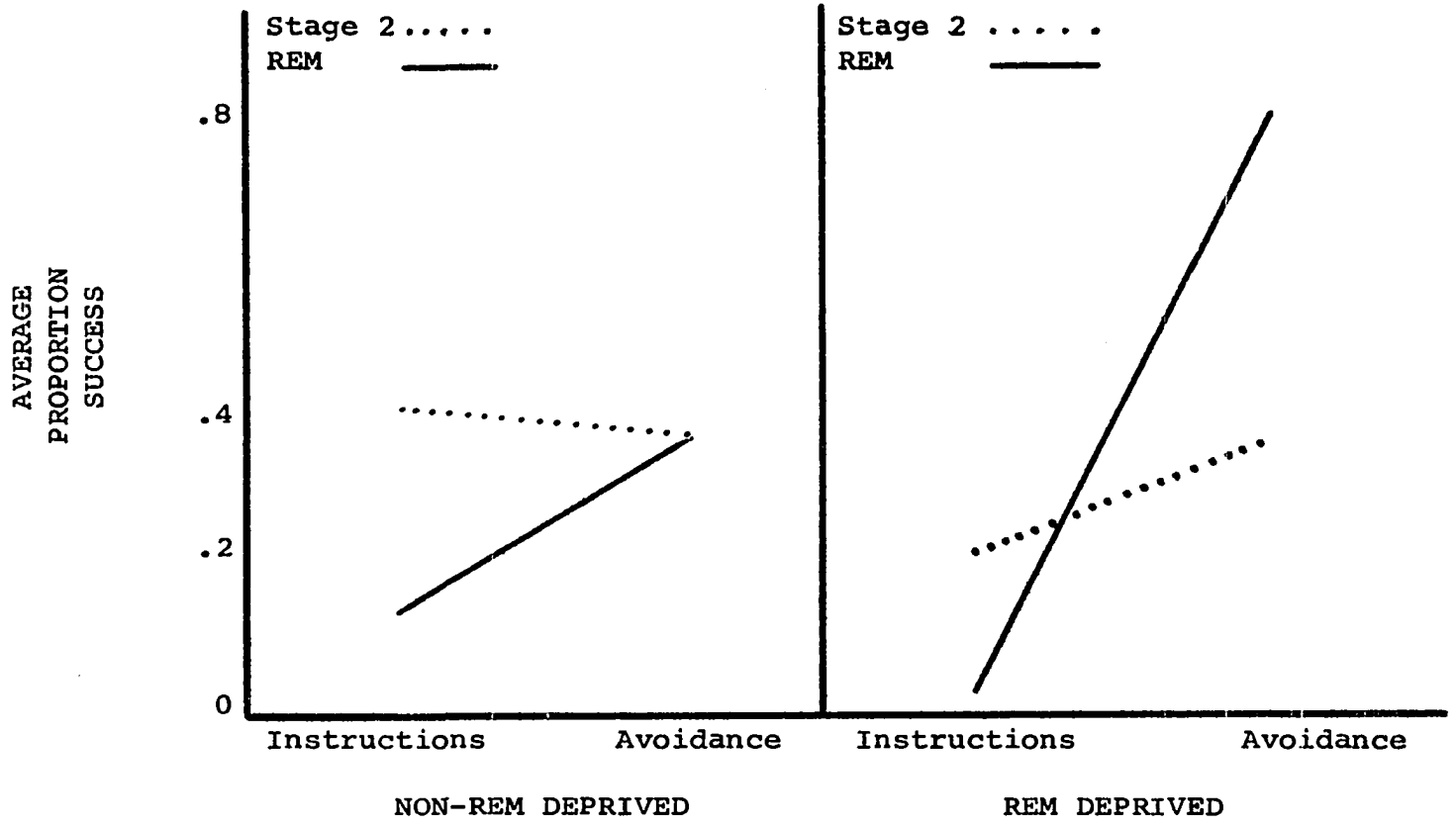

Figure 1 - Mean proportion of success plotted for two stages of sleep and both deprivation conditions under each type of reinforcement.

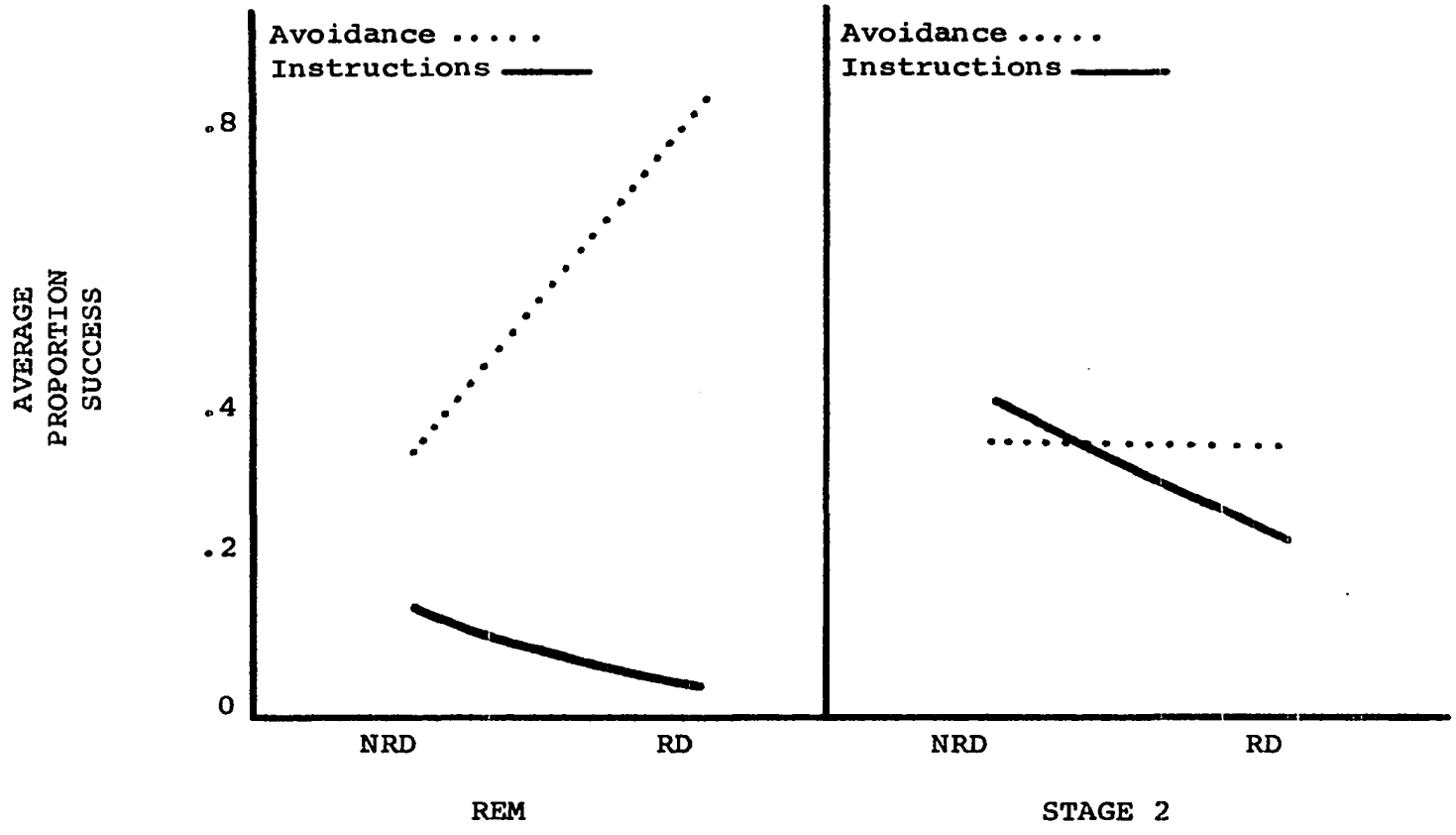

Figure 2 - Mean proportion of success plotted for reinforcement and deprivation conditions in each stage of sleep.

and each stage of sleep. To investigate this issue, a three-way analysis of variance was performed on the proportion of successes accompanied by alpha events and the proportion of correct responses to non-alpha events for each S. Arcsin transformations were applied to these proportions. The ANOVA was formulated into a reinforcement X alpha (success with and without alpha events) X sleep stage design. See Table 5.

TABLE 5

MEANS OF ARCSIN TRANSFORMED PROPORTIONS FOR REINFORCEMENTS X ALPHA X STAGE ANOVA

	REM		Stage 2	
Reinforcement	Alpha	No Alpha	Alpha	No Alpha
Instructions	0.56	0.19	1.75	0.89
Avoidance	2.09	0.98	1.33	1.17

F tests revealed significant main effects for reinforcements and alpha, with performance better under the avoidance treatment (as was found above) and with the alpha rhythm present (F = 5.86 and 7.66 respectively). The reinforcement X stage (AB) interaction was also significant (F = 8.29) (this was discussed above).

Table 6 presents the summary of this analyses.

TABLE 6

SUMMARY OF ANALYSIS OF VARIANCE FOR PERFORMANCE AND ALPHA EVENTS

Source of Variance	SS	df	MS	F
Between subjects				
A (reinforcements)	4.75	1	4.75	5.86a
B (stage)	1.75	1	1.75	2.16
AB	6.07	1	6.07	7.49a
Subj. w. groups (error between)	22.69	28	0.81	
Within subjects				
C (alpha)	6.21	1	6.21	8.48b
AC	0.00	1	0.00	0.00
BC	0.20	1	0.20	0.27
ABC	2.06	1	2.06	2.82
C X subj. w. groups (error within)	20.51	28	0.78	

a p/.05
b p/.01

In order to see if a particular treatment combination predesposed Ss to exhibit the alpha rhythm a reinforcement X deprivation X stage analysis of variance was performed. The proportion of trials manifesting alpha events of the total trials, for each S were transformed to arcsins.

Table 7 gives the means of the arcsin transformed proportions. This analysis included any alpha event occuring during a trial.

TABLE 7

MEANS OF ARCSIN TRANSFORMED PROPORTIONS OF ALPHA EVENTS DURING TRIALS

	REM		Stage 2	
	RD	NRD	RD	NRD
Instructions	1.25	1.92	1.16	1.97
Avoidance	2.32	1.68	1.21	1.23

Table 8 provides the summary of the analysis of variance and simple effects. Only the reinforcement by deprivation (AB) interaction attained statistical significance ($F = 4.93$). The simple effects revealed this to be due to the significant difference between the NRD and RD for instructions (B for a1) with the NRD groups displaying more alpha events.

In spite of the insignificant main effects several a posteriori comparisons were made. It was found that the RD avoidance REM group displayed the alpha rhythm more frequently than the RD instruction REM group ($F = 5.17$),

TABLE 8

SUMMARY OF ANALYSIS OF VARIANCE FOR
OCCURRENCE OF ALPHA EVENTS
IN EACH CONDITION, AND
SIMPLE EFFECTS

Source of Variation	SS	df	MS	F
A (reinforcements)	0.00	1	0.00	0.02
B (deprivation)	0.37	1	0.37	0.86
C (stage	1.28	1	1.28	2.93
AB	2.16	1	2.16	4.93*
AB	1.14	1	1.14	2.61
BC	0.31	1	0.31	0.72
ABC	0.13	1	0.13	0.31
Within cell (error)	10.51	24	0.43	
Simple effects				
A for b1 (RD)	1.23	1	1.23	2.81
A for b2 (NRD)	0.94	1	0.94	2.14
B for a1 (inst.)	2.17	1	2.17	4.96*
B for a2 (avoid.)	0.36	1	0.36	0.83

* p/.05

and both the RD avoidance and instruction stage 2 groups ($F = 5.62$ and 6.08 respectively). It thus appears as though the RD avoidance REM group was more readily aroused by a significant stimulus. Since the difference between the RD instructions and avoidance groups was insignificant in stage 2, this effect was probably not due to REM deprivation alone. It looks as though the punishment made the stimulus (tone) significant and the REM deprivation enhanced the significance in stage REM but not in stage 2.

The results with respect to the a posteriori comparisons should be treated cautiously particularly in the light of insignificant main effects. Moreover, the N's were small and one S could exert considerable influence on the outcome. This is important in view of the fact that Ss were not adapted to the laboratory. Appendix C contains the raw data of this experiment.

CHAPTER IV

DISCUSSION

The present investigation supports the hypothesis that Ss will perform an instrumental response in order to preserve REM sleep. It was found that responding was very infrequent in REM sleep under the instructions treatment. However, performance improved markedly when response failures were punished by interruptions of REM sleep. This is in agreement with the findings of Williams et al. (1966). Those investigators also obtained improvement in other stages of sleep under an avoidance paradigm. In the present experiment, punishment did not result in improved behavioral responding for the stage 2 groups. Perhaps the divergent results obtained for the avoidance condition alone can be explained by the more aversive negative reinforcement employed by Williams et al. Furthermore, Williams' Ss were informed that response failure would lead to aversive consequences, and that only one response was required.

The finding that REM deprivation depressed performance in both stages of sleep under instructions, but facilitated responding under the avoidance regime supported the hypothesis that REM deprivation would enhance the effect produced by avoidance alone. This facilitation was significant only for the REM group. Thus, REM deprivation appears to have a specific and selective effect on REM sleep.

Since REM deprivation depressed performance under instructions alone, the hypothesis that improved performance results from general activation induced by REM deprivation rather than by motivational properties can be ruled out. The analysis of alpha events supports the view that REM deprivation did not produce a propensity towards arousal. It was found that trials accompanied by alpha events occured more frequently under the NRD than the RD instruction conditions. However, under the RD avoidance REM treatment, there was a trend toward alpha activation. Unlike Williams et al. (1966) who found the effect of reinforcement on responding stronger for non-alpha than alpha events, the present study obtained significantly more correct responses when the alpha rhythm was present compared to when it was absent.

Inasmuch as neither the avoidance nor deprivation treatments significantly improved performance in stage 2, the arguments that increased responding results from the loss of sleep in general and not only REM sleep or to the aversive effect of awakening are not very compelling.

The present investigation provides a behavioral basis for demonstrating the motivational nature of REM sleep. The findings suggest that interruption of REM sleep is both biologically and psychologically annoying. The former because it interferes with a primed and ongoing state and the latter because consummation of the state is an internal satisfaction. This view is consonant with the response-release mechanism of reinforcement. That is, a reinforcing effect results whenever events allow for the occurrence of a response which has been activated but blocked. Routtenberg (1966) makes the pertinent observation that while the common element in REM sleep, self-stimulation, and exploratory behavior remains to be elucidated, a possible working hypothesis is that they are all related to some reinforcing consequence.

Routtenberg (1966, 1968) has distinguished between two electroencephalographic desynchronizing systems. One is identified with the reticular activating system, and the

other with the limbic system. The former is referred to as arousal system I, the latter as arousal system II. The MFB is the major component of arousal system II. These two systems as postulated by Routtenberg (1968) provide an organizational framework for understanding reinforcement which is seen as part of a reciprocal relation between arousal system I (drive) and arousal system II (incentive). These two systems will now be briefly summarized and used as a basis for interpreting the findings of this experiment.

1) Arousal system I is regarded as predominately concerned with drive or the activation and organization for response(s). This system, according to Routtenberg, must be active for the production and selection of the appropriate response. Therefore, response occurrence, whether approach or withdrawal, is more probable when arousal system I is active and less probable when arousal system I is inactive.

2) Arousal system II is believed to be a reward system, and important in reinforcement, thatis, increasing the probability that a particular response will be repeated.

3) Hypothalamic stimulation is thought to be rewarding because it activates both arousal systems, while septal stimulation is rewarding because it activates arousal

system II and suppresses arousal system I activity.

4) Dorsal midbrain stimulation is considered aversive due to its dampening effect upon the activity of arousal system II.

5) Arousal system I and II are mutually inhibitory. Both systems are constantly active, reciprocal suppression allows a dynamic equilibrium, first one predominating, then the other.

6) Finally, Routtenberg takes reward or incentive to refer to the stimulus population which activates arousal system II, while reinforcement refers to the consequences of arousal system II activity, viz., the suppression of arousal system I.

It has been suggested in the introduction that at least some of the neural activity in REM sleep may correspond to activity in arousal system II. If this is true, it is possible that in REM sleep, arousal system I is relatively inhibited (5 above). REM sleep is thus assumed to be pleasurable (2 and 3 above) since both the hypothalamus and septal regions are activated in REM sleep (see introduction).

Waking up subjects from REM sleep would be tantamount to dorsal midbrain stimulation and is aversive

because it attenuates activity in arousal system II (see 4 above). In other words, REM sleep is considered reinforcing because it corresponds with arousal system II activity and arousal system I suppression.

If interruption of a primed and ongoing state represnets a negative reinforcement, then interference with NREM sleep can be considered aversive. Most will agree that being awakened at any time is annoying. It was noted that the awakening procedure used in this experiment was not very effective in stage 2 sleep. After all, Ss usually had completed approximately five hours sleep including most of their stage 4 before trials began. Based on the model presented above, it could be argued that the disruption of NREM sleep was tantamount to dorsal midbrain stimulation, i.e., activation of arousal system I. This would be aversive because it upsets the existing equilibrium between arousal systems I and II by facilitating the "negative incentive" system. Activity in both arousal systems I and II are presumed to be at a low level in NREM sleep. Thus, NREM sleep may possess motivational properties of its own. A conclusion in this regard cannot be drawn from the present study, since a procedure of stage 2 deprivation equivalent to REM deprivation was not carried out.

There is a need for both REM and NREM sleep. The case for a drive for sleep has been established by Murray (1965). The present paper has looked at the special attributes of REM sleep. It was argued that the specific organization of neural activity in REM sleep is analogous to that of other drives. Such activity is admittedly diffuse; i.e., not relegated to a single structure or center. However, Grossman (1968) presents the proposition that motivational processes such as hunger and thirst are not necessarily regulated by autonomous hypothalamic centers. Instead complex pathways possibly including the entire limbic system and associated subcortical nuclei are involved. As evidence for this view, he reports numerous studies in which damage or stimulation (chemical) produced hyperphagia or aphagia. Valenstein (1968) and associates have demonstrated the lack of specificity between a behavior and the stimulation of a discrete hypothalamic locus. They suggest that the electrical stimulation activates a physiological state which allows associations to be created with well-established behavior patterns. This hypothesized state is considered to have much in common with a general drive construct.

The physiological activation of the REM state also

bears some resemblance to a general drive in that deprivation leads to hyperreactivity, increased restlessness, motivation to eat, CNS excitability, and hypersexuality (Dement, 1965). However, in the absence of deprivation, the REM state presents a complicated picture of increased upward ergotropic discharges interrelated with increased downward trophotropic discharges including decreased EMG, decreased heart rate and blood pressure, etc. The trophotropic discharge of REM sleep is part of the central discharge which determines this phase of sleep (Gellhorn, 1967). Thus, the readiness to perform motor acts characteristic of a general drive or activation is absent in the REM state.

While controversy on the issue of the existence of drive stimuli persists, there is at least an intimation of their presence in REM sleep. Antrobus et al. (1965) found subjects could produce behavioral responses during sleep to the instruction to close a switch when you start dreaming. On a task requiring subjects to response to internal signals associated with the REM state, Salamy and Williams (1969) found REM deprivation markedly improved performance. As REM time was regained, performance dropped off. The possibility that the increased drive made REM stimuli more salient was therefore suggested. That performance on the

extinction night was better than on the instruction night, implies the stimuli may be conditionable.

CHAPTER V

SUMMARY AND CONCLUSIONS

While the importance of a behavior for survival has been important in identifying motives, many theorists emphasize the potentiation of learning and performance as the chief criterion for classifying a variable as a motive. This research tested the validity of viewing REM sleep as a motive against this learning-performance criterion.

The hypotheses that Ss will learn to perform an instrumental reponse in order to obtain REM sleep, and that REM deprivation will enhance this effect received experimental support in this study.

The procedure involved the comparison of response rates of Ss under two reinforcement treatments (instructions and avoidance) and two deprivation conditions (REM deprived and non-REM deprived) for stage REM and stage 2 sleep. The task required the S to close a microswitch, which was taped to his hand, three times within 5 seconds of the offset of a 3 second tone. The tones were presented every 3 minutes,

in the designated stage of sleep, during the last 2.5 hours of bed time. REM deprivation was achieved by the method of forced awakenings and continued up to 2.5 hours before the end of the run. Under the instruction condition, there was no consequence for response failure; while under the avoidance treatment, Ss were awakened for 1.5 minutes if they did not respond. They were, however, not informed as to the reason for the awakenings.

It was found that with ininterrupted sleep, the stage 2 Ss performed better than the REM group. However, the avoidance procedure significantly improved performance of a REM group, but failed to promote better responding in the stage 2 group.

The combination of REM deprivation and the avoidance procedure resulted in pronounced improvement in stage REM, but only slightly increased responding in stage 2. REM deprivation suppressed responding in both stages under instructions alone.

The data militated against the alternative hypotheses that improvement in performance in the REM groups was due to the aversive effects of awakening; the loss of sleep in general and not only REM sleep loss; and the activating effects of REM deprivation.

The results were discussed in relation to other studies of behavioral responding during sleep. An attempt was made to formulate the findings within the framework of Routtenberg's organizational model of reinforcement. The amenability of the data to interpretation within the structure of the "two arousal hypothesis" served to emphasize the functional relationship between the REM sleep and reward mechanisms.

It was concluded that REM sleep possesses motivational properties which are analogous to other physiological drives.

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APPENDIX A

ANATOMICAL CONSIDERATIONS: REWARD SYSTEMS, REM SYSTEMS

This appendix contains a brief review of structural considerations important to the understanding of the anatomical interrelationship of the REM and reward systems, and provides the bases for many of the implicit assumptions made in this study.

The reward system refers to those parts of the brain which yield reward and/or aversive effects upon self-stimulation. The classical experiments were performed by Olds and Milner (1954) and Delgado, Roberts and Miller (1954) respectively. Reward effects can be obtained from stimulation in a zone which parallels the course of the medial forebrain bundle (MFB). The negative reinforcement areas reside within the mid-brain reticular formation and central gray substance (McCleary and Moore, 1965).

Olds and Olds (1963) have corroborated these earlier findings. They examined the effects of 96 electrode

placements. They conclude that the entire hypothalamus was involved in the reward system; the MFB elicited the greatest approach reactions; avoidance to hypothalamic stimulation resulted only from very intense or prolonged stimulation; and that pure negative reinforcement was elicited at diffusely scattered points through the thalamus, dorsal tegmentum and periventricular area of the midbrain.

Stimulation of the regions which have been implicated in positive and negative reinforcement phenomena has also been shown to elicit or inhibit hunger, thirst, sexual activity, and other drives. In addition, self-stimulation rate varies as a function of deprivation of most primary drives. Finally, the available evidence suggests that specific drives are closely related to the reward effect since self-stimulation rate at a particular electrode site correlates with only one specific drive (Grossman, 1967).

The "limbic-midbrain area" (Nauta, 1958); i.e., the central gray and paramedian reticulum of the midbrain, and nucleus of Gudden, provides the vital link between the limbic cortex and the lower brain stem and spinal cord. From these areas, the MFB ascends to the hypothalamus and further forward it biforcates. One stream turns laterally to the area of the amygdala. From there, fibers distribute

to the limbic cortex and frontotemporal region. The other stream runs medially to the septum, from which fibers are distributed by way of the fornix and cingulum to the hippocampus and cingulate gyrus respectively. Descending pathways run parallel to the ascending ones just mentioned (MacLean, 1968a) (there are also other ascending and descending connections not discussed). There is abundant evidence that the amygdalar circuit is related to the manner in which an animal feeds and protects itself (Kluver and Bucy, 1939; Pribram and Bagshaw, 1953; Schreiner and Kling, 1953; Fuller, Rosvold, and Pribram, 1957). For example, monkeys who normally prefer fruit will eat raw meat after ablation of this region. Wild animals become tame. Monkeys may repeatedly mouth a burning match; that is, they persist in doing things harmful to themselves. On the other hand, the septal circuit (including the septum, hippocampus and cingulate gyrus) seems to pertain to activities which are involved in preservation of species and reproductive behavior. Lesions in this area result in hypersexuality (Kluver and Bucy, 1939; Schreiner and Kling, 1953). Often this hypersexuality is of a bizarre nature, such as a cat copulating with a chicken.

The amygdala-oral and septal-genital pathways converge

in the area of the anterior hypothalamus and remain in close association back to the midbrain level. MacLean (1968b) points out the relation of these pathways and aggressive behavior. Stimulation above a point which elicited agnoistic behavior (where the pallido-hypothalamic tract dips over the medial aspect of the fornix in the squirrel monkey) results in complete erection. As the electrodie is lowered, there is, in addition, a showing of fangs and fear, pain, and anger vocalizations. Further advancement of the electrode elicits only the agonistic signs. Still further lowering of the electrode elicits only biting and chewing.

By plotting out these points, it can be seen that loci of face and mouth activity congregate in the area of the amygdala. Genital responses are grouped in the septum region. Both converge at the anterior hypothalamus. MacLean observes that fighting is frequently a preliminary to mating as well as feeding.

It will be recalled that REM deprived cats manifested hypersexuality, increased appetite upon starvation, increased CNS excitability and increased grooming. This led Fisher (1967) to suggest that the limbic system structures concerned with self and species preservation are released

from inhibition during REM sleep; and if REM sleep is prevented, this behavior presumably spills over to the waking state. Thus, REM deprivation was tantamount to a functional ablation of structures which inhibit drive centers of the hypothalamus. However, since many of these behaviors; e.g., penile erections, are a normal component of non-REM deprived REM sleep, the effects under discussion seem to be due to a stimulation action; thus, they appear in normal REM sleep and in REM deprived waking.

In the following paragraphs an attempt will be made to briefly review some recent advances in the neuroanatomy and electrophysiology of REM sleep.

Pompeiano (1967) presents experiments indicating that the rapid eye movements, the phasic discharges from the motor and somatosensory cortex, the myoclonic twitches, the phasic depression of the spinal reflexes, the phasic mydrasis, the phasic heart rate changes, and other tonic and phasic manifestations of desynchornized sleep (REM sleep) are critically related to the activity of the medial and descending vestibular nuclei. All these somatic and vegetative functions are abolished after destruction of these vestibular nuclei.

Jouvet (1967) cites evidence that the neocortex, and

all neural structures (including the hypothalamus and hypophysis) rostral to the pons fails to prevent the appearance of REM sleep. Animals with prebulbar transections, however, did not evince the decrease in EMG activity associated with REM sleep. Ocular and cortical activity was the same as the midpontine pretrigeminal preparations.

Coagulation of the mediolateral part of the caudal section of the nucleus reticularis pontis oralis (RPO) and the rostral part of the nucleus reticularis pontis caudalis (RPC) suppresses REM sleep in chronic cats (see Jouvet, 1967). Jouvet also reports that bilateral destruction of an area in the dorsal portion of the medio-lateral pontino tegmentum including the nucleus locus coeruleus suppresses the occurrence of muscular atonia during REM sleep. Jouvet concludes that the structures responsible for the triggering of REM sleep are thus located behind a prepontine transection.

The ascending structures responsible for cortical desynchronization in REM sleep remain to be delimited. Carli, Armengol, and Zanchetti (1965) placed lesions which interrupted the ascending limb of the libmic-midbrain circuit (including the ascending component of Schutz's dorsal longitudinal fasciculus, the mannillary peduncle, and the ascending component of the medial forebrain bundle),

and the descending limbic-midbrain pathways (including direct hippocampal projections, MFB fibers originating from lateral and preoptic hypothalamic regions, the mannillo-tegmental tract, the fasciculus retroflexus, and the descending component of Schutz's dorsal longitudinal bundle) in the cat. None of these lesions interferred with the electroencephalographic desynchronization characteristic of paradoxical sleep. The authors concluded that neither the ascending nor the descending components of the limbic-midbrain circuit exerted a significant influence on paradoxical behavior. Also, Jouvet (1967) cites evidence in which coagulations in the area of the limbic-midbrain circuit failed to prevent the cortical activation and thereby rejected this as a possibility. He also reports that neither destruction of the rostral reticular formation of the pons nor coagulation of the mesencephalic tegmentum suppressed this activation. The hippocampal theta rhythm is suppressed by septal destruction. Total section of the brain stem at the mesodiencephalic border destroying the posterior diencephalon does, however, suppress cortical activation during REM sleep. The peripheral signs of REM sleep remain. Gellhorn (1967) presents data indicating that the EEG changes characteristic of REM sleep still occur

after mesencephalic reticular formation lesions provided the interpenduncular region, hypothalamus, and septum remained intact. Thus, the limbic-midbrain circuits role in cortical activation during REM sleep is not completely precluded, the failure of coagulation of the limbic-midbrain area to suppress it notwithstanding. Obviously, the pathways by which the cortex becomes desynchronized from the initial triggering of REM sleep in the pons are quite diffuse.

This discussion can be summarized by some data from Jouvet and Jouvet (1963). Destruction of the nucleus RPC suppressed REM sleep. Lesions in the septum, subthalamic region, interpenduncular region, and medial portion of the anterior pontine tegmentum suppressed totally or partially the fast cortical activity and the hippocampal theta rhythm. Mesencephalic lesions interrupting the ascending reticular activiating system suppressed cortical arousal, but the possibility of cortical desynchronization during REM sleep was not eliminated.

There is a substantial body of literature favoring increased cortical excitability during REM sleep and the dissociation of such activating mechanisms from waking. For example, the unitary cortical arousal patterns are different, evoked potentials are different, and the

frequency and topography of hippocampal theta are different (see Jouvet, 1967).

These differences probably are, at least to some degree, a function of the activation patterns originating from the hypothalamus and reticular formation. Posterior hypothalamic coagulation causes somnolence without preventing cortical desynchronization in response to mesencephalic reticular and peripheral stimulation, which does not result in behavioral arousal (Ranson, 1939). While animals respond to visual and auditory stimuli behaviorally and also show desynchronization with midbrain reticular lesions (Feldman and Waller, 1962). Finally, Tokizane, Kawamura, and Imamura (1960) found near-threshold stimulation to the reticular formation produced greater neocortical than hippocampal arousal, whereas posterior hypothalamic stimulation created greater arousal of the hippocampus than the cortex. Gellhorn (1967) finds these data warrant the differentiation between hypothalamic-hippocampal and reticulo-neocortical arousal.

APPENDIX B

CHEMICAL CONSIDERATIONS

In this appendix an attempt will be made to point out similarities in the neurochemical substrates of the REM and reward systems. The role of the catecholamines in reinforcement is readily demonstrable. While their importance in REM sleep is indicated, their function remains to be made clear. In this section, points of convergence and divergence will be discussed.

Jouvet (1967b) using a neuropharmcological approach to the study of sleep mechanisms has shown that 5-hydroxytryptomine (5-HT, serotonin) induced a state resembling NREM sleep with REM depressed. On the other hand, injections of 3, 4 dihydroxphenylalanine (DOPA) produced a desynchronized EEG. Five-hydroxytryptophan (5-HTP) adminstered before or after DOPA counteracts its activating effects. Nialamide, a potent monoamine exidase (MAO) inhibitor has a long lasting suppressing effect on REM sleep.

Reserpine depresses the level of both 5-HT and

catecholamines in the brain. It also selectively induces PGO (an electrical harbinger of REM sleep) activity without affecting other tonic components of REM sleep. DOPA injected after reserpine will increase PGO activity and after 4 - 6 hours will induce NREM sleep followed by REM sleep. In other words, all the tonic phenomena of REM sleep reappear once the normal catecholamine level of the brain is re-established.

Pujol, Mouret, Jouvet, and Glowinski (1968) found a marked increase of turnover of norepinephrine to be associated with the enhancement of REM sleep characteristic of the rebound period. Their method involved labeling and did not alter the endogenous pools. The increased turnover of norepinephrine is taken to indicate the augmentation of synthesis and utilization of the amine.

Stern (1969) has found significant improvement in active and passive avoidance performance following REM deprivation using drugs which increase CNS levels of norepinephrine (imipramine, pargyline, amphetamine, and DOPA). This effect was only obtained with drugs which increased norepinephrine and/or prolonged its endogenous action.

Following a release of catecholamines, there is an increase in free fatty acids in the plasma. In REM sleep,

plasma levels of free fatty acids have been found to be elevated (see Hartmann, 1967). In addition, the urinary output of catecholamine metabolites increases several times during the night, usually following REM periods (Mandell, Brill, Mandell, Rodnick, Rubin, and Sheff, 1966).

Olds, Killam, and Bach-y-Rita (1956) found reserpine depressed self-stimulation rate more with the electrodes posterior and lateral to the anterior commissure than with electrodes in front of the commissure. Poschel and Nintemen (1963, 1964) found MAO inhibitors facilitated bar-pressing for posterolateral hypothalamic stimulation.

Stein (1966, 1967, 1968) investigated neurochemical substrates of reinforcement and found drugs that release norepinephrine from stores in the brain (amphetamine, MAO inhibitors) facilitate self-stimulation. Whereas drugs that block adrenergic transmission (chlorpromazine) and deplete brain norepinephrine inhibit self-stimulation. Incidentally, chlorpromazine has been shown to depress activity in the hypothalamus (Zukauskas and Machne, 1964); moreover, chlorpromazine decreases REM time (see Hartmann, 1967).

Thus, while it is not clear why amphetamine and MAO inhibitors increase self-stimulation and decrease REM

time (see Hartmann, 1967; Jouvet, 1967b), it is clear that catecholamines play an important role in both the REM and reward systems. Hillarp, Fuxe, and Dahlstrom (1966) mapped catecholamine-containing neurons, and located their sites of origin in the ventromedial mesencephalon (limbic-midbrain area). Fibers ascend from this area and terminate in the lateral hypothalamus, limbic lobe, and neocortex. By lesioning the MFB and assaying for norepinephrine, Heller, Seiden, and Moore (1966) concluded that the adrenergic fibers are arranged in an ascending system, since only structures rostral to the lesion showed a reduction of norepinephrine. Stein (1968a, 1968b) suggested that this ascending adrenergic system might be identified with at least part of the positive reinforcement system.

The ascending adrenergic system refers to the norepinephrine containing neurons originating in the limbic midbrain area, ascending in the medial forebrain bundle and terminating in the hypothalamus, preoptic area, amygdala, septum, hippocampus and neocortex. This system may be descending as well as ascending, and inhibitory as well as facilatory.

Hernandez-Peon (1965) and associates obtained synchronized sleep which eventuated into desynchornized sleep

by stimulation of points along Nautas' limbic circuit with cholinergic substances (acetylcholine, acetylcholine plus eserine, and carbamylcholine). Lesions or deposition of atropine in the caudal parts of the limbic midbrain circuit inhibited the effect obtained from more rostral acetylcholine stimulation. Thus, Harnandez-Peon concluded that the flow of excitation in this cholinergic-hypnogenic system is in a rostro-caudal direction. However, parts of this pathway also resulted in activation to cholinergic stimulation. Morgane (1969) extended this work and mapped both a cholinergic-hypnogenic system and a cholinergic arousal system. The two systems were topographically dissociated in the midbrain posterior to the interpeduncular nucleus. These systems are topographically intermixed in the lateral hypothalamic area such that stimulation of zones one millimeter apart could result in sleep or arousal. Both arousal and hypnogenic systems were thought to be contained in the median forebrain bundle.

These studies indicate the complexity of the chemical pathways involved in sleep and in REM sleep. Thus, while an ascending adrenergic system is directly involved in reinforcement, the picture is less clear in REM sleep.

APPENDIX C

RAW DATA

Amount of time spent in stage REM and stage 4, up to and following trials for each subject in each treatment combination:

S	REM Time up to 2.5 hrs before end of run	REM Time in last 2.5 hrs	Total REM Time	Stage 4 up to 2.5 hrs before end of run	Stage 4 in last 2.5 hrs	Total Stage 4 Time	Total Sleep Time
NRD - INSTRUCTIONS - STAGE 2							
a**	3	49	52	89	0	89	366
b	15	53	68	82	0	82	340
c	7	53	60	30	20	50	330
d	3	54	57*	63	14	77	397
$\bar{x}$							358.25
NRD - AVOIDANCE - STAGE 2							
e	49	44	93	64	0	64	402
f	37	51	88	31	0	31	414
g**	58	73	131	72	0	72	377
h	42	50	92*	77	0	77	375
$\bar{x}$							392.00
RD - INSTRUCTIONS - STAGE 2							

S	REM Time up to 2.5 hrs before end of run	REM Time in last 2.5 hrs	Total REM Time	Stage 4 up to 2.5 hrs before end of run	Stage 4 in last 2.5 hrs	Total Stage 4 Time	Total Sleep Time
i	0	72	72*	75	0	75	362
j	0	42	42	73	0	73	368
k	1	42	43*	83	0	83	395
l	0	61	61	77	20	97	381
$\bar{x}$							376.50
RD - AVOIDANCE - STAGE 2							
m	0	44	44	78	0	78	337
n	0	32	32*	63	17	80	348
o	0	94	94	12	8	20	334
p	0	38	38	52	0	52	372
$\bar{x}$							347.75

S	REM Time up to 2.5 hrs before end of run	REM Time in last 2.5 hrs	Total REM Time	Stage 4 up to 2.5 hrs before end of run	Stage 4 in last 2.5 hrs	Total Stage 4 Time	Total Sleep Time
NRD - INSTRUCTION - REM							
q	46	63	109	56	25	81	420
r	33	16	49*	62	0	62	374
s	28	61	89	76	0	76	442
t**	16	46	62	35	24	59	372
$\bar{x}$							402.00
NRD - AVOIDANCE - REM							
u	51	17	68	72	0	72	388
v**	8	21	29*	64	0	64	440
w**	22	59	81*	45	20	65	395
x**	35	29	64*	48	5	53	388
$\bar{x}$							402.75
RD - INSTRUCTIONS - REM							
y	0	30	30	12	0	12	368
z	0	50	50	18	5	23	358
aa	0	97	97	78	3	81	389
bb	2	54	56*	66	25	91	403
$\bar{x}$							379.00
RD - AVOIDANCE - REM							
cc	0	59	59	52	0	52	419
dd	3	66	69	69	26	95	393
ee	0	48	48*	99	7	106	414
ff	3	27	30*	37	0	37	304
$\bar{x}$							382.50

* S still in REM at end of run
** S received trials before last 2.5 hrs.

Responses in the presence and absence of the alpha rhythm for the stage 2 groups:

	Correct Response		No Response	
S	Alpha Present	Alpha Not Present	Alpha Present	Alpha Not Present
NRD - Inst. - stage 2				
a	7	0	0	0
b	4	2	3	7
c	2	2	2	8
d	6	1	2	1
NRD - Avoid. - stage 2				
e	3	3	2	2
f	1	1	0	4
g	0	1	1	10
h	4	3	5	1
RD - Inst. - stage 2				
i	2	2	4	1
j	1	2	0	7
k	0	1	2	8
l	2	0	3	10
RD - Avoid. - stage 2				
m	0	1	3	5
n	1	7	0	4
o	0	2	2	4
p	2	0	3	2

Responses in the presence and absence of the alpha rhythm for the stage REM groups:

	Correct Response		No Response	
S	Alpha Present	Alpha Not Present	Alpha Present	Alpha Not Present
NRD - Inst. - REM				
q	0	0	13	6
r	1	0	1	4
s	3	0	13	4
t	3	1	8	1
NRD - Avoid. - REM				
u	1	0	0	3
v	0	1	4	1
w	6	2	1	3
x	2	1	6	2
RD - Inst. - REM				
y	0	0	3	8
z	0	0	8	8
aa	0	0	1	27
bb	1	0	11	4
RD - Avoid. - REM				
cc	9	5	1	3
dd	15	0	4	0
ee	7	6	0	3
ff	8	0	1	0

Type and number of awakenings for each S in the stage groups

Wake - Ups

	Deprivation	Punishment
NRD Inst. Stage 2		
a	0	0
b	0	0
c	0	0
d	0	0
NRD Avoid. Stage 2		
e	0	4
f	0	4
g	0	11
h	0	6
RD Inst. Stage 2		
i	6	0
j	2	0
k	13	0
l	10	0
RD Avoid Stage 2		
m	3	8
n	0	4
o	7	6
p	13	2
NRD Inst. REM		
q	0	0
r	0	0
s	0	0
t	0	0

Wake - Ups

	Deprivation	Punishment
NRD Avoid REM		
u	0	3
v	0	5
w	0	4
x	0	7
RD Inst. REM		
y	14	0
z	16	0
aa	29	0
bb	13	0
RD Avoid REM		
cc	16	4
dd	5	3
ee	3	3
ff	4	1