EFFECTS OF SENSORY DEPRIVATION ON SLEEP IN ADULT CAT

THE EFFECTS OF SENSORY DEPRIVATION ON SLEEP AND OTHER REGULATORY PROCESSES IN ADULT CAT

by

CHARLES BRECKENRIDGE, B.A.

A Thesis

Submitted to the School of Graduate Studies

in Partial Fulfilment of the Requirements

For the Degree

Doctor of Philosophy

McMaster University

January 1978

DOCTOR OF PHILOSOPHY (1978)
(Psychology)

McMASTER UNIVERSITY
Hamilton, Ontario

TITLE: The Effects of Sensory Deprivation on Sleep and Other Regulatory Processes in Adult Cat

AUTHOR: Charles Breckenridge, B.A. (University of Saskatchewan)

SUPERVISOR: Dr. W. Heron

NUMBER OF PAGES: xix; 400

ABSTRACT

It is known that human subjects deprived of meaningful visual, auditory and somatosensory stimulation (sensory deprivation), spend a larger portion of their time asleep (increased Stage I & II) than when in a normal environment. Furthermore, that phase of sleep which is characterized by the presence of rapid eye movements (REM sleep) is altered under these conditions, with REM density (number of rapid eye movements per minute) being greatly increased. However, little progress was being made toward understanding the conditions needed to obtain these effects, or the neural mechanisms underlying them. Therefore, the effects of sensory deprivation on sleep of adult cat were studied, since if the results from the human experiments could be duplicated, various experimental manipulations could be performed to elucidate the mechanisms.

It was found that adult cat responded to sensory deprivation in a manner analagous to human subjects since both REM density and the amount of time spent in the light phases of sleep increased during deprivation. The increase in REM density was not due to a change in the frequency with which the neural generator of REMs initiated eye movements since PGO wave density remained constant across conditions. Rather, it appeared to be due to the fact that the proportion of large amplitude, high velocity REMs increased during deprivation concomitantly with the change in REM density. In agreement with this, a positive correlation between REM density and average EOG amplitude was found under normal conditions and during sensory deprivation. The increase in sleep time during deprivation was found to be most similar to that described by others following sensory deafferentation. Thus, neither the proportion of deep SWS nor REM sleep was altered, while that of light SWS was greatly increased. The minimum condition needed to obtain these effects was found to be the lid suture procedure used to prevent vision during deprivation. Thus, lid suture alone and lid suture coupled with total light deprivation had equivalent effects while light deprivation alone caused only marginal changes. These results were contrasted to the human experiments where large sleep effects were obtained by requiring the subject to wear a translucent mask.

Finally, a preliminary account of the effects of sensory deprivation on other regulatory mechanisms was given. It was found that for some animals, food consumption and body weight increased and body core temperature decreased during deprivation.

PREFACE

In Chapter I, evidence indicating that sensory deprivation has an effect on structure and function of the nervous systems of adult human and cat was contrasted against data which suggests that the functional organization of the visual system becomes permanently established by the end of the critical period. Since the effects of sensory deprivation on human sleep were large and reliably obtained, these were selected for study. The experiments were performed on adult cat in the hope that some progress could be made toward understanding the conditions needed to obtain the sensory deprivation effects and the neural mechanisms involved. However, as the amount of data generated in these experiments was large and the analysis time consuming, this thesis deals mainly with a description of cat's sleep under normal laboratory conditions and during sensory deprivation. Accordingly, the literature reviewed in Chapter I dealt mainly with the neural mechanisms controlling sleep.

Chapter II was divided into four experimental sections. In Experiment I, the effects of sensory deprivation on sleep were described and compared with those obtained in the human sensory deprivation experiments. Experiment II focused on one parameter (REM density) that was found to change during sensory deprivation, in an attempt to elucidate the mechanisms underlying this effect. Experiment III was aimed at determining the minimum conditions needed to obtain the effects of sensory deprivation on sleep and Experiment IV provided a preliminary account of the effects of deprivation on other regulatory systems.

Chapter III provided a critical appraisal of the experimental work described in this thesis. The relevance of these results to theoretical issues and hypotheses found in the research literatures on sleep and sensory deprivation was discussed and new directions for future research were suggested.

TABLE OF CONTENTS

CHAPTER THREE

LIST OF FIGURES

LIST OF TABLES

CHAPTER 1

SECTION 1

GENERAL INTRODUCTION

It is well known that a certain amount of sensory stimulation is essential for normal development of brain function in human and subhuman species (Riesen, 1966). Similarly, it appears that there is an optimum amount of patterned sensory input required by the adult organism since sensory deprivation studies have demonstrated changes in cognitive (Scott, Bexton Heron & Doane, 1959; Suedfeld, 1969), perceptual (Bexton, Heron & Scott, 1954; Doane, Mahatoo, Heron & Scott, 1959; Zubek, 1969a) and physiological processes (Heron, 1957; 1961; Zubek, 1969b) following brief exposures of human subjects to a sensory environment where the amount of patterned input had been drastically reduced. The developmental studies, however, have been more successful in discovering the locus of the effects of sensory deprivation and the mechanisms leading up to changes in function than have studies on sensory deprivation in humans. The explanation for this discrepancy in progress between these two areas of research appears to be the successful use of animal models in the studies on development. The objective of the research program described in this thesis has been to obtain an animal model of the human sensory deprivation

experiment in the hope that some progress toward an understanding of the mechanisms and locus of effect of sensory deprivation in adult animals would be made.

I. Sensory Deprivation: Introduction

(A) Objective

The first human sensory deprivation experiment was conceived in terms of a comprehensive theory of perception and cognition which attempted to relate what was known about psychological processes to information about neural function (Hebb, 1949). Hebb postulated that conscious experience was dependent on neural activity in complex networks of functionally connected neurons called cell assemblies which themselves were organized temporally into phase sequences. The formation of cell assemblies and phase sequences depended on repeated exposure to the conditions which first gave rise to the particular pattern of neural activity. Once the organization was established, the phase sequence could be elicited by activation of individual cell assemblies, although the cell assemblies needed to be activated individually from time to time if they were to remain part of the phase sequence. Since cell assemblies and phase sequences were established under conditions of constant sensory flux to which motor activity contributed, it was postulated that the elimination of flux in this background sensory input would lead to a disruption in the organization of phase sequences. Thus, it was predicted that perceptual and thought processes would be altered by sensory deprivation.

(B) Methodology

Bexton, Heron & Scott (1954) attempted to reduce "background sensory flux" by eliminating all external sources of patterned sensory input. Subjects were required to lie quietly on a bed in a partially soundproof chamber. A masking noise from an air conditioning unit prevented patterned auditory stimulation, translucent goggles eliminated patterned visual input, while cotton gloves and cardboard cuffs which extended from below the elbow beyond the fingertips reduced tactile input. This technique of reducing the amount of patterned input without substantially altering the net amount of sensory stimulation has been called perceptual deprivation while the procedure which aims at reducing the amount of sensory stimulation to zero has been called sensory deprivation (Kubzansky, 1961). Subsequently, the term sensory deprivation has been used more generally, to refer to any procedure which produces an impoverished sensory environment (Suedfeld, 1975).

II. Sensory Deprivation: Effects on Humans

(A) Cognitive Effects

Scott, Bexton, Heron & Doane (1959) reported that during sensory deprivation most subjects experience difficulty in sustaining coherent sequences of organized thought. Instead, their thoughts tended to jump from one topic to another in a random manner and often blank periods ensued when subjects reported having difficulty even finding things to think about. This inability to concentrate also manifested itself in terms of performance deficits on a variety of tests like those requiring the subject to do mental arithmetic, solve anagrams, construct geometric patterns identical to those illustrated in models (Kohs Block Design), identify geometric patterns in more complex figures (Thurstone-Gottschaldt Figures) or identify incongruencies in pictures (Picture Anomaly Test). These effects of sensory deprivation on cognitive function have, in general, been confirmed by subsequent research (Suedfeld, 1969).

(B) Perceptual Effects

Doane, Mahatoo, Heron & Scott (1959) reported that visual hallucinations were commonly experienced during sensory deprivation and that a variety of perceptual distortions occurred for a short time after subjects were returned to a normal environment. In addition, stimulus aftereffects were

exaggerated (figural aftereffect, Archimede's spiral after-effect, color adaptation), size constancy was decreased and the autokinetic effect was increased. No significant effects were noted on measures of critical flicker fusion, the phi phenomenon, brightness contrast, brightness constancy, shape constancy, Necker cube reversal or tachistoscopic perception. In general, subsequent research has indicated that hallucinatory behavior and perceptual distortion occur less frequently and less severely than was originally reported (Zubek, 1969a). Some of the effects of deprivation on the objective tests of perception have been substantiated, particularly those on stimulus aftereffects and color matching tests while contradictory data exist on most of the other measures.

(C) Physiological Effects

(i) EEG Studies

Heron, Doane & Scott (1956) and Heron (1957) reported that during sensory deprivation, the alpha rhythm recorded from the occipital region of the brain, gradually slowed down in frequency and became smaller in amplitude. This effect took about three days to reach a maximum reduction of 1.5 Hz to 2.0 Hz after which the frequency of the alpha rhythm stabilized. At the end of the deprivation, three days were again required before alpha frequency returned to its pre-deprivation level (Heron, Tait & Smith, 1972). A number of environmental factors appear to be important in determining

how large a reduction in alpha frequency is attained during sensory deprivation. Zubek & Welsh (1963) found that subjects who were exposed to unpatterned light and white noise during deprivation had a significantly greater reduction in alpha frequency ($\bar{x}$ = 1.21 Hz) than subjects exposed to total light deprivation and silence ($\bar{x}$ = 0.85 Hz). Control subjects who were required to lie in a prone position like experimental subjects, but who were in an otherwise normal environment did not show a change in alpha frequency. Subjects who underwent perceptual deprivation but who were required to perform a series of calisthetic exercises six times a day had a significantly smaller reduction in alpha frequency ($\bar{x}$ = 0.48 Hz) than a no-exercise group of subjects ($\bar{x}$ = 1.21 Hz, Zubek, 1963) while subjects who were immobilized in stocks in a recumbent position but who were allowed to see and hear the experimenter with whom they had a degree of social interaction, had a mean reduction in alpha frequency of 0.59 Hz (Zubek & MacNeill, 1966).

(ii) Endocrine and Metabolic Effects

It is frequently assumed that subjects in sensory deprivation experiments are under a condition of chronic stress (Zuckerman, 1964). Subjective reports of unpleasantness and the fact that a proportion of subjects always ask to leave the experiment before completion substantiates this view. Experiments where indices of adrenal cortical response

were measured during sensory deprivation have provided some support for this hypothesis. Murphy et. al. (1955) failed to find any change in the urinary excretion of 11-deoxycorticoid of subjects deprived of patterned sensory input for up to six days but Mendelson et. al. (1960) found that subjects who asked to be let out of modified tank type of respirators, significantly increased their urinary excretion of epinephrine. Zubek & Shutte (1966) found a similar increase in subjects who asked to quit a seven-day deprivation experiment after the second day of deprivation. Subjects who endured the entire seven days and subjects in a recumbent control group both decreased their urinary excretion of epinephrine and norepinephrine. These results indicate that sensory deprivation may indeed be stressful to a proportion of the population but these subjects tend to quit the experiment before completion. The fact that urinary excretion of epinephrine and norepinephrine decreased in recumbent subjects as well as sensory deprived subjects indicates the recumbency pre se may cause an alteration in the pattern of endocrine secretion. This interpretation is supported by the reported decrease in body temperature (Winget et. al., 1972) and increase in the concentration of serum triiodothyronine (T_3) during prolonged bed rest (Vernikos-Danellis et. al., 1972). These results are of great interest since some workers have reported dramatic decreases in body weight during brief

periods of sensory deprivation (Vernon et. al., 1961; Zubek, 1969b, p275). Although Heron (1961) reported that basal metabolic rate and oral temperature did not change during sensory deprivation a more complete analysis of the metabolic effects of recumbency and inactivity would be appropriate.

(iii) Effects on Sleep

Heron (1961) reported that from records of body activity, it appeared that subjects slept more during the initial period of sensory deprivation than later on. This observation was confirmed in a subsequent experiment by Potter & Heron (1972), where it was found that subjects slept approximately 25% more on the first day of deprivation than they did on either three pre-deprivation or post-deprivation days. This effect on sleep gradually became smaller until by the fourth deprivation day, subjects were sleeping about the same amount as they did on control days. The additional time spent asleep during the first few days of deprivation was spent mainly in stage II sleep. The amount of time spent in stage I sleep increased slightly while there was a reduction in the amount of time spent in REM sleep on deprivation day one and an increase of roughly the same magnitude on the second day of deprivation. (This REM sleep rebound phenomenon is a characteristic response obtained when subjects are asked to sleep in an unfamiliar

environment.) Stage III and stage IV were unchanged by deprivation. Accompanying these changes in the amount of time spent asleep were changes in REM density which refers to the number of rapid eye movements per minute of REM sleep. Potter & Heron found that REM density increased from 200% to 500% during sensory deprivation and returned to baseline by the end of the first post-deprivation day.

Aserinsky (1969; 1971; 1973) obtained similar results when he instructed subjects to lie quietly in a dark room and try to sleep as much as they could. Under these conditions, Aserinsky (1973) found that the average subject slept 65.2% of the first day (a 35% increase) and 46.5% of the second day (a 20% increase) of enforced sleep. REM density increased by 200% during the first 12 hours and remained elevated throughout the remaining 42 hours. It is not surprising that subjects sleep more than usual when they are instructed to do so but the fact that REM density also changed suggests that Aserinsky's enforced sleep technique is similar to sensory deprivation. Thus, Aserinsky's subjects, isolated in a darkened, sound-attenuated chamber, instructed to move around as little as possible, would be expected to manifest all the symptoms of sensory deprivation even if the explicit instructions regarding sleep were omitted.

Aserinsky's results differed from those obtained during sensory deprivation in that he found a positive

correlation between REM density and the amount of time spent in non-REM sleep, whereas during sensory deprivation, REM density continued to increase after deprivation day one despite the fact that the proportion of non-REM sleep gradually returned to normal. Aserinsky may have failed to observe this latter effect though, since his experiments were always terminated at the end of the second day.

(D) Discussion

From the material presented in the previous sections, it is clear that sensory deprivation has a diverse and bewildering number of effects on psychological and physiological processes in human subjects. It is surprising how this relatively innocuous procedure can produce such large changes while at times it is difficult to detect behavioral or physiological effects of more robust procedures like lesioning the brain.

A likely explanation for the diversity of the effects of sensory deprivation is the complexity of the technique. Thus, sensory deprivation not only reduces the amount of sensory flux available to the subject, but also imposes on him social isolation, changes in activity patterns and postural changes. In addition, the subject brings to the situation a unique set of variables which are likely to interact with experimental variables in complex ways to produce even greater diversity in the results (Zuckerman, 1969). Heron (1961) has dealt with this diversity by grouping the effects of sensory deprivation into one of three categories; modality specific effects, effects which are not specific to a sensory modality and the effects on cognition and perception. Implicit in such a scheme is the assumption that effects of deprivation which are placed in the same category will be more likely to have a common origin than effects listed in

different categories. Similar assumptions are made by theories which deal with the mechanisms and locus of the effects of sensory deprivation since one mechanism is often postulated to account for all the data (Doane, 1956; Lindsley, 1961; Schultz, 1965). One of the objectives of this thesis will be to evaluate the extent to which the symptoms of sensory deprivation have a common etiology.

III. Sensory Deprivation: Animal Studies

(A) Introduction

Most of the research on the effects of sensory deprivation in animals has centered on determining what effect early experience, or lack of it, has on subsequent development. Thompson & Schaefer (1961) have identified two different theoretical positions which have guided much of the experimental work. One position was Hebb's view that the ability to correctly interpret sensory stimulation is slowly acquired through a process of learning during development (Hebb, 1949). Support for this hypothesis came from Senden's (1960) report of impairment in the perceptual ability of patients who had cataracts removed from their eyes when they were adults and Riesen's (1947) report of severe visual impairment of chimpanzees that were reared in the dark. The second theoretical position was held by Lorenz who suggested that the developing organism had an inherent ability to interpret and respond to particular stimulus arrays in a species-specific manner (Lorenz, 1937). According to this view, an animal's behavior would be appropriately elicited by the correct stimuli if it is raised in its normal environment. By raising the animal in a contrived environment, however, it would be possible to disassociate the response from its correct releaser such as when a baby duck is imprinted on an

inanimate object (Hess, 1959).

Both Hebb and Lorenz believed that the developing organism was more susceptible to changes in its environment than the adult. Hebb, apparently, believed this to be true because of the greater intellectual impairment obtained after brain damage in young individuals than in adults and because of the painstaking, slow improvements in perceptual ability of adults who acquired the faculty of vision late in life. Lorenz's belief rested on the fact that there exists a critical period in the life of the developing organism when certain types of behavior are permanently established. These observations are in accord with Roux's (1895-cited by Riesen, 1966) two stage theory of development, which postulates that development consists of "(a) an early period of intrinsic growth and self differentation, and (b) a later period of growth that is dependent upon functional activity" (Riesen, 1966, p118). Implicit in this hypothesis is the assumption that the animal is no longer susceptible to changes in "functional activity" when the second period of growth has ended (i.e. adulthood).

The problem of whether an organism is more susceptible to changes in its environment during development than in the adult state is directly relevant to the present work since one of the objectives of this thesis is to demonstrate that neural function in adult animals is not as immutable to

environmental changes as the foregoing discussion would suggest. Thus, this section will be devoted to evaluating the extent to which sensory deprivation has different effects in immature and adult animals.

(B) Effects of Sensory Deprivation on the Visual System: Evidence for a Critical Period

Visual deprivation during development, usually achieved either by placing the animal in total darkness or by sewing the eyelids of one or both eyes shut, has some well documented effects on morphology and function within the visual system. In the lateral geniculate, neurons in laminae receiving input from the deprived eye typically are smaller and stain less intensely for Nissl substance than neurons in undeprived lamina (Wiesel & Hubel, 1963a) or in the geniculate of normally reared cats (Hickey et. al., 1977; Sherman & Wilson, 1975). Neurons in the visual cortex, while showing no evidence of gross morphological change after sensory deprivation, undergo clear functional modification. Thus, a proportion of neurons found, either cannot be excited by visual stimulation, or else give weak responses which fatigue easily (Ganz, Fitch & Satterburg, 1968; Kratz & Spear, 1976; Wiesel & Hubel, 1965a). Receptive field size tends to be larger than normal (Ganz et. al., 1968; Singer & Tretter, 1976) and the neurons respond less selectively to changes in stimulus orientation (Buisserst & Imbert, 1976; Sherk &

Stryker, 1976) and retinal disparity (Pettigrew, 1974). In addition, following monocular deprivation or any procedure that cause the two eyes to receive different inputs, the proportion of neurons that can be driven by providing visual stimulation to either eye is greatly reduced.

Accompanying these changes observed in the structure and function of the visual system are clear signs indicating that the animal is severely impaired in visual perception. Thus, when the animals are first allowed to see through their deprived eye(s), they behave as if they are totally blind. In an open field situation, the animals move about cautiously with their bodies close to the floor. Despite this they frequently bump into objects in their path and when placed on a table, they walk off the edge apparently without perceiving the change in depth at the border. The animals fail to blink or flinch at a rapidly approaching object until it actually touches them and then they give an exaggerated startle response (Dews & Wiesel, 1970; Ganz & Fitch, 1968; Riesen, 1947; Rizzolatti & Tradardi, 1971; Van Hof-Van Duin, 1976a; Wiesel & Hubel, 1965b). Visual pursuit is absent unless the moving object is highly contrasted against the background such as a lighted bulb moving in an otherwise darkened room (Ganz & Fitch, 1968; See also Sherman, 1973; 1974; Vital-Durand, Putkonen & Jeannerod, 1974). Visual placing is absent although tactile placing

is normal (Van Hof-Van Duin, 1976b).

Until recently, there has been general agreement that these behavioral and physiological effects of sensory deprivation can be obtained only if the animal is subjected to sensory deprivation during a certain restricted portion of its development. Thus, Dews & Wiesel (1970) found that kittens were most susceptible to the effects of monocular visual deprivation between four weeks to four months of age. Although the degree of susceptibility was not uniform during this period, deprivation beginning at the end of the fourth month was ineffective in producing impairment in visual ability even when deprivation was extended to sixteen months duration (Hubel & Wiesel, 1970 - Also see Jones & Pasnak, 1970; Riesen, 1965; Slomin & Pasik, 1972; Wiesel & Hubel, 1963b). A similar period of susceptibility for the effects of deprivation on the morphology and function of lateral geniculate (Burke & Hayhow, 1968; Headon & Powell, 1973; Hubel & Wiesel, 1970, Wiesel & Hubel, 1963a) and cortical neurons (Hubel & Wiesel, 1970; Wiesel & Hubel, 1963b) has also been found.

Additional evidence for the existence of a critical period comes from experiments which show that the effects of deprivation can only be fully reversed if the animal's vision is restored early in the critical period. Termination of visual deprivation late in the critical period is less

effective, and prolonged visual experience provided at the end of the critical period does not promote any recovery (Blakemore & Van Sluyters, 1974; Movshon & Blakemore, 1974; Movshon, 1976a; b; Wiesel & Hubel, 1965b). There is some controversy over the permanence of visual impairment following sensory deprivation (Chow & Stewart, 1972; Dews & Wiesel, 1970; Van Hof-Van Duin, 1976b). However, in all the studies where improvement in visually guided behavior has been demonstrated, it has not been possible to rule out the hypothesis that improvement occurs simply as a result of animals learning response strategies which optimize the use of remaining visual capacity (Rizzolatti & Tradardi, 1971; Sherman, 1973; 1974).

(C) Effects of Sensory Deprivation on the Visual System: Evidence for Plasticity in the Adult

The strongest challenge to the claim that the functional organization of the visual system is permanently established once the critical period has ended comes from the work of Buchtel, Berlucchi & Mascetti (1972), Fiorentini & Maffei (1974) and Maffei & Fiorentini (1976). They found that the immobilization of one eye by cutting the lateral rectus muscle and by sectioning the 3rd and 4th cranial nerves of adult cat caused a decrease in the proportion of simple type cortical neurons which could be driven binocularly. Since this effect did not depend on whether the animal

was permitted vision during the period of eye immobility, Maffei & Fiorentini (1976) concluded that it was not caused by an asymmetry in visual input between the two eyes. Rather, it seemed to be due to an asymmetry in eye mobility since the immobilization of both eyes had no effect on ocular dominance.

Brown & Salinger (1975) and Salinger et. al., (1977a; b) have extended the experiments of Maffei & Fiorentini (1976) to the lateral geniculate where they have found some evidence for a decrease in the proportion of X cells relative to Y cells two weeks after the immobilization of one eye. The fact that the magnitude of this effect appeared to be larger in animals which had both eyes sewn shut prior to eye immobilization than in animals that were subjected to eye immobilization alone, has led Salinger et. al. (1977b) to propose that the eye immobilization procedure allowed them to "unmask" an effect of binocular deprivation which had gone undetected in other studies. However, at the present time, the most one can conclude from these studies on the effects of eye immobility is that some characteristics of visual system function, which previously have been thought to remain invariant after the end of the critical period, are susceptible to modification in the adult (Also see Creutzfeldt & Heggelund, 1975). This conclusion is supported by a few other studies where it has been found that the effect

of sensory deprivation on neurons in the visual cortex can be partially reversed by providing visual experience outside of the critical period (Cynader, Berman & Hein, 1976; Spinelli et. al., 1972) or by removing the undeprived eye following monocular deprivation (Kratz, Spear & Smith, 1976).

(D) Discussion

Most of the developmental studies reviewed in this section indicate that a critical period exists during development when the visual system is susceptible to the effects of sensory deprivation. By the end of the critical period, the organization of the system appears to be complete enough that sensory deprivation is no longer capable of causing changes in structure or function. The visual cortex, however, appears to retain a degree of plasticity since partial reversal of the effects of deprivation occurs as a result of visual experience acquired after the critical period has ended.

The research on the effects of eye immobility have clearly demonstrated that neurons in the visual cortex and possibly in the lateral geniculate retain a degree of plasticity which is comparable to that seen during development. However, the fact that an asymmetry in eye motility rather than an asymmetry in visual input appears to be critical for obtaining an effect, suggests that this phenomenon may have to be considered separately from the effects of sensory depriv-

ation, unless an asymmetry of eye motility is responsible for the sensory deprivation effects as well. Similarly, the human sensory deprivation experiments have provided clear evidence of functional plasticity in adult but again these data are difficult to relate to the animal studies since no comparable data exist in the two literatures. Thus, without any cross-links between the animal and human literatures, these literatures have grown up independent of each other, despite clear similarities in objectives, methodology and history of origin.

Taking the physiological effects of sensory deprivation on humans as a starting point, we endeavoured in this work to bridge the gap between the human and animal sensory deprivation literatures. If a suitable animal model of the human sensory deprivation experiments could be obtained, it would then be possible to discover the conditions needed to produce the effects and the underlying mechanisms. With this information in hand, one could then decide whether any relationship exists between the effects of deprivation seen during development and those found in the adult animal.

Since the effects of sensory deprivation on human sleep were particularly robust phenomena, these were chosen for study. Adult cat was selected as subject since the sleep of cat and human are electrographically quite similar, and the sleep of both species was known to be affected by other

experimental treatments in an equivalent way (Dement, 1960; Ferguson & Dement, 1967). In addition, most of the basic neurophysiological data on sleep and on the effects of visual deprivation were obtained from cat. Thus, it was expected that a description of the effects of sensory deprivation on the sleep of adult cat, information which has never previously been reported, would be of interest to researchers in three major areas of study; the human sensory deprivation literature, the animal sensory deprivation literature and the sleep literature. Before proceeding with this description though, a review of the physiological basis of sleep in cat is necessary.

SECTION II

The developmental literature reviewed in Section I, in general, indicates that visual deprivation of adult cat does not have the same effect on structure or function in the visual system as does comparable periods of deprivation during development. However, as will be seen later, brief periods of sensory deprivation have large, reversible effects on the sleep of adult cat, similar to those described in the human experiments. Thus, while it was important to document the nature of the effect of deprivation on the visual system of adult cat because the sensory deprivation procedures used in the present work are very similar to those used in the developmental studies, attention will now have to be directed toward sleep and other regulatory processes which do change during sensory deprivation. Therefore, a description of sleep processes in cat will be presented here to prepare for the work which follows. Particular attention will be given to research dealing with the role of sensory input in modulating sleep behavior.

I. Description of Sleep Phenomena in Cat

The first stage of sleep a cat enters into when it goes to sleep has been called by Ursin (1968) light slow wave sleep (LSWS). Electrographically, this sleep is characterized by the periodic occurrence of 12 to 15 Hz "spindles" recorded best transcortically from the frontal pole of the brain and by the presence of synchronized cortical EEG. Behaviorally, the animal typically has its head resting on its paws or else directly on the floor. Alternatively, the cat may be curled up with its head tucked into its abdomen with the degree of curling being inversely related to the environmental temperature (Parmeggiani & Rabini, 1970; Parmeggiani & Sabattini, 1972). The animal is easy to arouse from this stage of sleep and frequentky wakes spontaneously to make slight postural adjustments.

As the sleep episode continues, the degree of cortical synchronization increases and the EEG record becomes dominated by large amplitude, 1 to 4 Hz slow waves. Frontal cortical spindles continue to occur but the frequency of occurrence increases. Muscle tonus in antigravity muscles is usually reduced as indicated by a flattening out of the electromyographic (EMG) record and the animal is more difficult to arouse. Ursin (1968) has labelled this phase of sleep deep slow wave sleep (DSWS).

The next stage of sleep to emerge is REM sleep. In the transitional phase between DSWS and REM sleep, the electrographic signs indicative of SWS begin to disappear, being replaced by indices of REM sleep. Thus, frontal-cortical spindles, cortical synchronization and muscle tonus abate and cortical desynchronization and muscle atonia take their place in the electrographic record. Additionally, the EEG of the dorsal hippocampus changes over from a desynchronized pattern to a 4 to 7 Hz synchronous rhythm (theta). Accompanying these changes in tonic electrographic activity are the appearance of phasically occurring phenomena. These include ponto-geniculo-occipital (PGO) waves, which can be recorded with macroelectrodes from the pontine reticular formation, lateral geniculate nucleus and occipital cortex, rapid eye movements (REMs), periodic twitching of the musculature and irregularity in respiration, heart rate and blood pressure. (For a more complete description of these phenomena see the reviews by Jouvet (1967)). At the end of the REM sleep episode, the animal may either awake or else lapse back into SWS.

II. Brain Mechanisms Controlling Slow Wave Sleep

(A) Role of Sensory Afferents and the Brain Stem Reticular Formation in Producing Cortical Desynchronization

One of the oldest theories about sleep is that it is a passive phenomenon that occurs whenever the active processes responsible for maintaining wakefulness can no longer sustain the conditions needed for wakefulness (Kleitman, 1939). One condition frequently defined as essential for wakefulness is the continual afferent bombardment of the cerebral cortex by sensory input (Moruzzi, 1964). Thus

> "(i) any drop of the afferent barrage, following muscular relaxation and closure of the eyes, any (ii) increase of fatigability of the cerebral cortex and of the subcortical structures involved in wakefulness would lead to a state of functional deafferentation of the cerebral cortex. This state would cause sleep merely because it is incompatible with wakefulness." (Moruzzi, 1964, p24)

Although deafferentation obtained by destroying primary sensory pathways does cause an increase in the proportion of sleep time (Hagamen, 1959; Hodes, 1962; Vital-Durand & Michel, 1971), the physiological mechanism for producing "functional deafferentation" during the course of falling asleep is probably more complex than this. Indeed, the important discovery by Moruzzi & Magoun (1949) that electrical stimulation of the brain stem reticular formation blocked both spontaneous and elicited cortical synchronization led to the hypothesis that

there exists an ascending reticular activating system (ARAS) which is responsible for the maintenance of wakefulness (See Moruzzi, 1963; 1971 for a review).

> "According to the reticular hypothesis, sleep is attributed to the elimination of the waking influence of the ascending reticular system." Sleep is still regarded, therefore, as absence of wakefulness. The main difference between the old and the new formulation of the hypothesis is that the maintenance of a waking state is attributed to a tonic barrage of ascending reticular impulses, while the steady flow of sensory messages coursing along the classical pathways appears to be without importance for the physiology of sleep." (Moruzzi, 1963, p234)

While in this new formulation sleep is still regarded as an absence of wakefulness, cessation of wakefulness is accomplished by damping down tonic activity in the ARAS by means of an active process. If the ARAS is disconnected from the cerebral cortex by a midbrain transection made just behind the oculomotor nerve (Bremer's (1935) cerveau isole preparation), the animal goes into a comatose state which resembles SWS with respect to EEG and ocular indices. This result contrasts markedly with that obtained from the encéphale isolé preparation (brainstem transected at the first cervical ganglion) which retains the ability to have a sleep-wake cycle (Bremer, 1937; 1938).

The lack of cortical desynchronization in the cerveau isolé preparation cannot be explained by the fact that animals with midbrain transections are more completely deafferented than encéphale isolé animals since Lindsley et. al. (1949;

1950) showed that lesioning the sensory pathways ascending through the brainstem did not reproduce the effects on cortical EEG that were obtained by lesions of the brainstem reticular core. This conclusion is also supported by the experiments of Batini et. al. (1959a; b; c) which showed that cat exhibits continually desynchronized cortical EEG if brainstem transection is made just in front of the trigeminal nerve (midpontine pretrigeminal preparation) instead of behind the oculomotor nerve (rostropontine pretrigeminal) as was the case in Bremer's cerveau isole preparation. The difference between these two preparations, therefore, is not in the degree of afferentation since in both preparations the first three cranial nerves were intact, but rather in terms of the integrity a small band of reticular tissue between the two sections which is occupied largely by the nucleus reticularis pontis oralis. Camacho-Evangelista & Reinoso-Suárez (1964) confirmed that lesioning this nucleus and the nucleus reticularis ventralis increases the amount of EEG synchronization found in the record relative to control periods. Lesions placed dorsal, lateral and caudal to these nuclear groups increased the proportion of desynchronized EEG obtained post-operatively relative to control observations made pre-operatively.

These results, however, do not rule out the importance of sensory afferents in producing a state of cortical activa-

tion since Claes (1939 - cited by Arduini & Hirao, 1959) reported that bilateral eye enucleation or optic nerve section in encephale isole cat led to an appearance of EEG sleep patterns in the visual cortex, an effect that was not duplicated by dark adaptation. Roger et. al. (1956) observed a similar effect after bilateral destruction of the Gasserian ganglion (viz. trigeminal nerve) in the encephale isole preparation but not after cutting the olfactory, optic, statoacoustic or vagus nerve, and Batini et al. (1959c) reported that optic and olfactory nerve ablation in the midpontine pretrigeminal preparation caused temporary cortical synchronization. Arduini & Hirao (1959) showed that reversible synchronization of the cortex could be obtained in the midpontine pretrigeminal preparation by raising intraocular pressure to a level that produced ischemic anoxia sufficient to block all optic nerve activity and then returning intraocular pressure back to normal. (Similar observations were made by Bizzi & Spencer (1962) in the cerveau isole preparation.) Since this effect could not be produced by placing the animal in total darkness, Arduini & Hirao concluded that the continual retinal discharge known to occur in total darkness (Arduini & Pinneo, 1962; Kuffler, Fitzhugh & Barlow, 1957; Rodieck, 1967)

> "is critical for the maintenance of an adequate level of activity in the ascending reticular system. The sudden withdrawal of this afferent retinal barrage would thus lead to a fall in the "tonus cortical", which may be eventually compensated in chronic preparations (Batini et. al. 1959a; b; c) but would certainly go without compensation during the short time available in our experiments of acute ischemic inactivation of the retina."
> (Arduini & Hirao, 1959, p151)

In a subsequent experiment though, Arduini & Hirao (1960) found that continual illumination of the retina also was capable of causing cortical synchronization in the mid-pontine pretrigeminal preparation. The interpretation the authors made of these results was that under continual illumination the tonic retinal discharge may actually be less than that obtained in total darkness; a hypothesis that receives some support from studies where direct measurements were made (Arduini & Pinneo, 1962; Kuffler et. al., 1957).

Thus, it appears that sensory input does play an important role in determining the degree to which cortical synchronization dominates the EEG. This conclusion, however, must necessarily be weakened by the observation that both the cerveau isolé preparation (Bastel, 1960; 1964; Villablanca, 1965; 1966a; b) and the midpontine pretrigeminal preparation (Slósarska & Zernicki, 1969; Zernicki, 1968) recover the capacity for producing desynchronized/synchronized EEG as post-surgical survival time is lengthened. Thus, the acute preparation, a preparation which Moruzzi (1972)

suggested is experiencing a type of surgical shock akin to spinal shock that follows spinal cord transection, may not be ideal for detecting additional effects of deafferentation. In addition, the procedure of looking for qualitative changes in the electrographic record following sensory deafferentation may not be as sensitive an index as a measure of the proportion of time over a twenty-four hour period that the EEG is synchronized. For these reasons, more weight must be given to the chronic deafferentation studies of Hagamen (1959) and Vital-Durand & Michel (1971) where deafferented cats were found to spend significantly more time asleep than normal animals.

(B) Cortical Synchronization Mechanisms

(i) Brainstem Synchronization Structures

The observation that the EEG is continually desynchronized following midpontine pretrigeminal brainstem transection, at least in the acute stages, led Batini et. al. (1958) to suggest that an EEG synchronizing influence is located in the brainstem caudal to the level of the trigeminal nerve. This hypothesis was supported by the experiment of Magni et. al. (1959) where the blood supply to the rostral pons, midbrain and upper cerebrum was separated from the supply to the caudal pons and medulla by clamping the basilar artery at midpontine level. Under these conditions, they found that small amounts of thiopental injected into the common carotid

leading to the rostral pons and forebrain caused previously desynchronized EEG to become synchronized for a brief period of time whereas, injecting thiopental into the vertebral artery which supplied the caudal brainstem caused previously synchronized EEG to become briefly desynchronized. The interpretation of this latter finding was that the short-acting barbiturate caused a temporary inactivation of the synchronization structures in the lower brainstem.

Further support for the existence of a brainstem synchronizing system has come from experiments where discrete surgical (Jouvet & Renault, 1966; Jouvet, 1969) and biochemical (Cohen et. al., 1973; Delorme et. al., 1966; Jouvet, 1969; Pujol et. al., 1971) lesions of the raphe nuclei caused almost total insomnia for the first few days after the lesion with some recovery being noted thereafter. In addition to the raphe nuclei, the area postrema (Bronzino et. al., 1972; Koella et. al., 1968) and the nucleus of the solitary tract (Bonvallet & Allen, 1963; Bonvallet & Bloch, 1961; Magnes et. al., 1961) have also been implicated since electrical stimulation in the region of the solitary tract nucleus provokes cortical synchronization (Favale et. al., 1961; Magnes et. al., 1961) as does chemical stimulation of the area postrema with topically applied serotonin (Bronzino et. al., 1972; Koella et. al., 1968) or serotonin injected into the local arterial circulation (Roth, Walton & Yamamoto, 1970). Electrolytic

lesions or localized cooling in the vicinity of the solitary tract nucleus, on the other hand, prolong the duration of spontaneous or evoked reticular activation of cortical EEG, whereas control lesions in the caudal medulla do not have this effect (Berlucchi et. al., 1964; 1965; Bonvallet & Allen, 1963; Bonvallet & Dell, 1965). Since the IXth and Xth cranial nerves constitute the main afferent input to the nucleus of the solitary tract (Bonvallet & Allen, 1963; Bonvallet & Sigg, 1958), it seems likely that the EEG synchronization produced by electrical stimulation of the vagus nerve (Bonvallet & Sigg, 1958; Chase et. al., 1967; Dell & Padel, 1965; Foutz, Ternaux & Puizillout, 1974) or by mechanical stimulation of the baroreceptors in the carotid sinus (Mazzella et. al., 1956) may be obtained as a result of activating this brainstem synchronization mechanism. The fact that cortical desynchronization can be obtained only under certain stimulus conditions (Chase et. al., 1966) has led Chase et. al. (1967) to suggest that there are at least two functional groups of afferents being carried in the vagus nerve; a hypothesis supported by the demonstration that desynchronization of the EEG was obtained only when a slow conducting group of fibers were activated by electrical stimulation whereas activation of a low threshold, rapid conducting system resulted in synchronization. The brainstem system is obviously more complex than this though, since Foutz, Ternaux

& Puizillout (1974) and Puizillout & Foutz (1976) have observed that REM sleep can be triggered directly by vago-aortic nerve stimulation in a semi-chronic encephale isole preparation; an effect which was facilitated by REM sleep deprivation prior to stimulation.

(ii) Basal Forebrain & Thalamic Synchronization Structures

The fact that both the cerveau isolé preparation of Bremer (1935) and the midpontine pretrigeminal preparation of Batini et. al. (1959a) recover the capacity to have recurrent episodes of synchronized and desynchronized EEG if maintained chronically for a long enough time (Batsel, 1960; 1964; Villablanca, 1965; 1966a; b) forces one to consider forebrain participation in the control of the sleep-wake cycle. It is clear that cerebral cortex, undercut amd isolated from other cortical tissue does not spontaneously shift from electrographic synchronization to desynchronization in a manner characteristic of an intact cortex (Sharpless & Halpern, 1962; Villablanca, 1967; But see Khananashvili & Bogoslovskii, 1976). Similarly, diencephalic and caudal brainstem structures do not exhibit normal cycling when separated from the cortex as they are in decorticate (Jouvet & Michel, 1959; Kleitman & Camille, 1932), decerebrate (Villablanca, 1966a) and diencephalic preparations (Villablanca & Marcus, 1972). On the basis of these results, one must conclude that the alternation between the two electrographic patterns is a property of

intact cerebrum probably depending on an interaction between neocortex, subcortical and diencephalic structures.

Although there have been a few reports on the role of telencephalic structures in sleep processes (Kim et. al., 1975; Villablanca et. al., 1976), most attention has been focused on thalamic and subthalamic areas. An intact thalamus has long been known to be essential for the occurrence of barbiturate spindles and spindles evident during sleep (Morison & Bassett, 1945; Villablanca & Salinas-Zeballos, 1972; Villablanca & Schlag, 1968). The medial and intra-laminar nonspecific thalamic nuclei have been implicated in the genesis of these phenomena since high frequency stimulation of the nonspecific thalamic nuclei results in cortical desynchronization similar to that observed following mesencephalic reticular formation stimulation (Jasper, 1949), whereas low frequency stimulation produces a cortical recruiting response which is electrographically similar to a sleep spindle (Dempsey & Morison, 1942; Morison & Dempsey, 1942). In addition, low frequency stimulation of the midline nonspecific thalamic nuclei in freely behaving cat has been reported to cause the animal to go to sleep (Akert, Koella & Hess, 1951; Hess, 1954; Parmeggiani, 1962). The validity of these latter results, however, has continued to be questioned (Bremer, 1954; Harrison, 1940; LoPiccolo, 1978), mainly on the ground that none of these experiments have controlled for the

tendency of the animal to go to sleep spontaneously. When such control experiments are done, no hypnogenic effects of thalamic stimulation can be demonstrated (LoPiccolo, 1978). This result is consistent with the report of Angeleri et. al. (1969), who found that selective ablation of the nonspecific thalamic nuclei had no effect on the amount of time the animal slept. Total thalamectomy (Villablanca & Salinas-Zeballos, 1972) and complete destruction of the neocortex, rhinencephalon and striatum (Villablanca & Marcus, 1972), however, cause a large permanent reduction in both SWS and REM sleep. On the basis of these results, Villablanca (1974) suggested that forebrain structures normally have a sleep enhancing effect on hypothalamic/reticular formation activating systems. Thus, removal of these structures was postulated to create an imbalance between hypnogenic and activating systems resulting in insomnia.

The hypothalamus has also been postulated as a likely candidate for the locus of forebrain sleep and waking centers since bilateral lesions in the posterior portions of the lateral hypothalamus (Ingram et. al., 1936; Ranson, 1939) and in adjacent parts of the midbrain tegmentum (Nauta, 1946) produce a condition of somnolence whereas bilateral lesions in the preoptic area cause insomnia (McGinty & Sterman, 1968; Nauta, 1946). In the case of posterior hypothalamic lesions, McGinty (1969a) has reported that a short-lasting

hyperactivity (4-8 hours duration) occurs immediately post-operatively, a result that was missed in the earlier studies because long-acting anaesthetics were used. This phase is followed by somnolence, a sleep-like state from which the animal can be briefly aroused by intense stimulation. Hypothermia, anorexia, emotional lability and in the case of cat, catalepsy, accompany the excessive sleepiness. Recovery from these symptoms occurs, to a greater or lesser extent, depending on factors like the species of animal used and the extent of the lesion (McGinty, 1969a; Ranson, 1939). In the case of preoptic lesions, the amount the animal sleeps (SWS & REM sleep) decreases progressively during the first two weeks post-operatively and gradually recovers, though not to pre-operative levels, during the next four weeks (McGinty & Sterman, 1968; Nauta, 1946). In some cases, the animal may not sleep at all post-operatively. Such animals do not survive longer than ten days, apparently dying in a state of total exhaustion. The insomnia produced by these lesions is usually accompanied by disturbances in thermoregulation and the regulation of food and water intake.

Another piece of evidence implicating the anterior hypothalamic and preoptic areas in sleep processes is provided by reports which claim that electrical (Sterman & Clemente, 1962a; b) or chemical stimulation (Hernandez Peon & Chavez Ibarra, 1963) of these regions of the brain is capable

of inducing sleep. Although the validity of these experiments has been questioned (LoPiccolo, 1978), Roberts & Robinson (1969) have obtained convincing data from cat which strongly suggests that sleep can be elicited with short latency diathermic warming of the preoptic area. Since, in these studies, the animal typically awoke within 15 to 150 seconds of stimulus offset, it was possible to show, by using repeated stimulation and blank trials, that the phenomenon could not be attributed to a spontaneous tendency of the cat to go to sleep. Furthermore, since the threshold current needed to obtain this effect was about twenty-five percent lower than that required for lesion production and since lesioning the preoptic area produces insomnia, not sleep, Roberts & Robinson concluded that a localized change in temperature of the preoptic area was responsible for the effects they observed. This interpretation is consistent with the fact that there are neurons located in this region of the brain which undergo large changes in firing rate in response to thermal stimulation (Nakayama et. al., 1963; Wit & Wang, 1968). Thus, on the basis of these data, it appears that there is a thermosensitive sleep regulating system located in the basal forebrain. That such a system might exist is suggested by experiments which demonstrate that the amount an animal sleeps is positively correlated with environmental temperature, at least over a small range of temperature (Parmeggiani et. al.,

1975; Parmeggiani & Rabini, 1970; Schmidek et. al., 1972). The fact that total sleep time and particularly, REM sleep time decreases markedly at both high and low environmental temperatures appears to facilitate survival at extreme temperatures since it is known that thermoregulatory ability is severely impaired, if not totally absent, during REM sleep (Parmeggiani et. al., 1973; Parmeggiani & Sabattini, 1972; Shapiro et. al., 1974). The neural system regulating this temperature dependent effect, however, has not been described.

(C) Sleep Induced by Repetitive Sensory Stimulation

It is a commonly known fact that repetitive sensory stimulation is very effective in inducing sleep but this phenomenon has never attracted much scientific interest. Pavlov (1927) found that the repetitive presentation of a condition stimulus in the absence of reinforcement rapidly led to the development of "internal inhibition". This inhibition was postulated to occur first in the foci of cortical elements initially excited by the conditioned stimulus. When this inhibition spreads to other parts of the brain, sleep ensued.

> "Internal inhibition during the alert state is nothing but a scattered sleep of separate groups of cellular structures; and sleep itself is nothing but internal inhibition which is widely irradiated, extending over the whole mass of the hemispheres and involving the lower centers of the brain as well. Thus, internal inhibition in the alert state of the animal represents a regional distribution of sleep which is kept within bounds by the antagonistic nervous process of excitation."
> (Pavlov, 1927, p253)

The decrease in response that occurs to a specific stimulus upon repeated presentation has subsequently been called habituation but habituation cannot be regarded as the process underlying sleep induced by sensory stimulation. The critical difference between procedures that produce sleep and those that produce habituation is that in the former, the stimulus is hypnogenic only if repetition occurs at regular intervals (Oswald, 1959; 1960; See Kleitman (1939) for a review of the older literature) whereas, the only requirement for habituation to occur is that the same stimulus be presented several times (Sharpless & Jasper, 1956). Thus, habituation will occur to stimulation capable of inducing sleep (Gastaut & Bert, 1961), but sleep is not a necessary consequence of habituation.

Konorski (1948), in criticism of Pavlov's view of sleep, suggested that the conditions in Pavlov's experiments were such that the animal would have fallen asleep naturally. Thus, the repetition of the CS did not interfere with this tendency, but it certainly didn't facilitate the occurrence of sleep. This opinion has been echoed in the work of others (Kleitman, 1939, p198; Tizard, 1966), but even these experiments have been recently contested (Bohlin, 1971). In spite of this controversy over the sleep observed in Pavlov's conditioning experiment and in other experiments where the stimuli used were likely too insignificant to have much effect (Bohlin,

1971; Tizard, 1966), there have been a few clear demonstrations of the hypnogenic properties of repetitive stimulation. Oswald (1960) found that subjects which were exposed to intense, repetitive, synchronous auditory (800 cps tone), visual (four flashing 60 W light bulbs viewed from a distance of 60 cm with eyelids taped open) and somatosensory (electric shock applied to the leg intense enough to be painful and to elicit a leg flexion) stimulation, rapidly went to sleep. Since the conditions to which these subjects were exposed could not be considered amenable to sleep by any normal criteria, one must conclude that repetition of intense afferent barrages has the capacity to induce sleep.

There are several potential mechanisms which could underlie the phenomenon of sleep induced by repetitive stimulation. It is known that the cortical field potentials evoked by discrete auditory and visual stimuli consists of short latency rapid primary components followed by more variable multiple slow components occurring at a frequency of 5-8 Hz (Doty, 1958; Fleming & Evarts, 1959; Kawamura & Yamamoto, 1961; Kimura, 1962). Since these multiple slow waves (1) are blocked by mesencephalic reticular stimulation; (2) do not occur in animals which are highly aroused; and (3) have the same frequency spectrum and locus of origin as recruiting responses and spontaneously occurring spindles, many authors have drawn a parallel between this phenomenon

and sleep spindles. Thus, the hypothesis has been advanced that the multiple slow wave is a thalamo-cortical spindle burst triggered by sensory stimulation (Kimura, 1962). The observation by Moruzzi (1972) that in most experiments where sleep has been induced by repetitive sensory stimulation (Marczynski & Sherry, 1972) or electrical stimulation of the brain, low frequencies stimulation were used (See Roelofs et. al., 1963; Sterman & Clemente, 1974 for exceptions) may be related to the fact that low rates of sensory stimulation (6-8 Hz) are the most effective in eliciting the spindle-like multiple slow wave (Kimura, 1962).

There is no evidence on the question of how repetitive sensory volleys might activate thalamic synchronizing systems but the work of Pompeiano & Swett (1962a; b; 1963) suggests a way such stimulation could lead to sleep. They found that low frequency repetitive electrical stimulation of cutaneous nerve (Pompeiano & Swett, 1962a) or skin (Roitbak, 1960) was capable of inducing spindles in cat. In an analysis similar to that carried out by Chase et. al. (1967) on synchronization induced by vagal nerve stimulation, Pompeiano & Swett found that low frequencies of stimulation activated Group II sensory afferents; rapid conducting axons likely coming from receptors in hair follicles and touch and pressure receptors. Higher frequency or more intense stimulation activated slower conducting, Group III sensory afferents. Since cortical

desynchronization was elicited by this type of stimulation, and in view of the conduction velocity of this group of fibers, it was concluded that this input was from nociceptors (Pompeiano & Swett, 1962b). In a subsequent acute experiment, it was found that neurons in the caudal pons and medullary regions received input from Group II afferents whereas stimulation of Group III afferents activated neurons in the rostral pons and mesencephalic reticular formation. Thus, it was suggested that EEG desynchronization elicited by stimulating Group III afferents was accomplished through ascending reticular activating system described by Moruzzi & Magoun while cortical synchronization produced by stimulating Group II afferents was obtained by activating the bulbar inhibitory system described by Magoun & Rhines (1946) and discussed by Bonvallet and others in relationship to cortical synchronization, desynchronization systems (Zanchetti, Wang & Moruzzi, 1952). Thus, the hypothesis has been advanced that repetitive stimulation activates the bulbar inhibitory system which, in turn, suppresses activity in the ascending reticular activating system (Moruzzi, 1960). This deactivation of the ARAS might allow synchronizing structures in the forebrain to come into play, with this effect being particularly evident immediately following the afferent volley.

Kleitman (1939), in a review of some of his early experiments, identified several other important conditions

which facilitate falling asleep. For example, an animal is more likely to go to sleep after having eaten a meal than before and to sleep longer when overfed than when underfed (Ruckebusch & Gaujoux, 1976). In fact, the instinctive-like searching for a place to sleep (Moruzzi, 1969) may be interpreted as an attempt on the part of the animal to optimize the conditions needed for sleep. Thus, a secure, warm place where the animal is not likely to be interrrupted is sought. Once such a place is found, the animal typically goes through a routine of behaviors such as grooming and adopting a characteristic posture prior to falling asleep. In the human, sexual intercourse has anecdotally been described by general practitioners as having soporific properties, and in animals has been observed to produce electrographic synchronization similar to that observed after other consummatory behaviors (Beyer et. al., 1971a; Boland & Dewsbury, 1971; Sawyer, 1969). In any case, none of these behaviors or preparations for sleep can be regarded as necessarily producing a reduction in sensory input; rather they seem to produce a particular type of input which facilitates sleep.

Some of these variables have come under experimental study. Clemente, Sterman & Wyrwicka (1964) reported that spindle-like slow waves occurred in the cortical EEG of cat after the animal received a food reward for pressing a lever. This electrographic synchronization was similar to that observed during ad lib milk consumption, grooming and drowsiness

(Roth, Sterman & Clemente, 1967; Sterman & Wyrwicka, 1967) and had the same frequency spectrum as sleep spindles. When the animal was allowed to work at the lever pressing task until satiation, it was noted that post-reinforcement synchronization observed during trials late in the session was longer in duration, the EEG waves were larger in amplitude and had a lower frequency than during trials early in the session. This progressive increase in the amount of cortical synchronization present throughout the course of an eating bout may be indicative of a gradual shift toward sleep. This interpretation is certainly borne out by observation of the behavioral sequences which typically lead to sleep in laboratory cat (unpublished personal observation). Whether the act of eating, some visceral stimuli or central state derived from having eaten is responsible for sleep onset remains an unanswered question. However, the fact that post-reinforcement synchronization is always suppressed in a very hungry animal (Sterman & Wyrwicka, 1967) and can be blocked by high frequency electrical stimulation of the ascending reticular activating system or by novel sensory stimuli (Clemente et. al., 1964) suggests an interplay between desynchronizing and synchronizing systems controls the overall state of the animal.

Sterman & Wyrwicka (1967) have suggested that voluntary suppression of movement is another important condition for falling asleep. They have identified an electrographic

correlate of behavioral inhibition, the sensorimotor rhythm which is optimally recorded from the sensorimotor cortex. They propose that this rhythm is generated by a thalamocortical system that is distinct from the system responsible for producing the post-reinforcement synchronization pattern, and that these systems are capable of operating in both the waking and sleep states (Howe & Sterman, 1972; 1973). Thus, in their view, the sleep that follows satiation would occur as a result of drive reduction and would be characterized by the presence of a post-reinforcement synchronization type pattern while sleep occurring following trained behavioral inhibition would be dominated by a sensorimotor type pattern.

Sidis (1908 - cited by Kleitman, 1939) and Kleitman (1927 - cited by Kleitman, 1939) showed that gentle physical restraint of movement was effective in inducing sleep. Whether such restraint increases cutaneous stimulation and facilitates sleep through a mechanism similar to that suggested by Pompeiano & Swett's experiments is unknown. It is known that forcefully restraining an animal on its back may induce a cataleptic-like state where the animal will remain immobile (tonic immobility), in often very awkward positions, for several minutes. Although many hypotheses have been advanced to account for this strange behavior (See Crawford & Prestude, 1977 for relevant papers) changes in somatosensory and proprioceptive input clearly play an important role in

eliciting this effect (Klemm, 1977). Despite the fact that EEG synchrony, decreased muscle tonus and suppression of spinal reflexes may accompany tonic immobility (Carli, 1977), it is not generally believed that tonic immobility is a sleep state. Nevertheless, it is likely that there is some overlap in the conditions which facilitate the occurrence of sleep and those which are prerequisite for tonic immobility.

(D) Discussion

It should be clear from the present review, that the concept of a brainstem activating system responsible for maintaining wakefulness, and of brainstem and forebrain synchronizing systems responsible for the production of sleep, have dominated research on sleep processes. Although the importance of sensory input in maintaining activity in these systems has never been denied, more emphasis has been placed on the interaction between synchronizing and desynchronizing systems. Thus, in the more recent theories of sleep, sleep has been regarded as an active process, where activity in synchronizing systems is considered to be necessary in order for sleep to occur, and a high level of tonic activity in the ascending reticular activating system is considered a prerequisite for wakefulness.

Since sleep time increases in human subjects during sensory deprivation, one is forced to consider the role of

sensory input in maintaining activity in brain synchronizing and desynchronizing systems. Thus, the question may be asked whether the additional sleep obtained during sensory deprivation is a stimulus-induced sleep, where stimuli present during deprivation enhance activity in synchronization systems or whether it is a deafferentation type sleep, where decreased afferent bombardment during deprivation results in a decrease in the tonic activity in the reticular activating system, thereby reducing its capacity to sustain wakefulness. Alternatively, the entire conceptual framework of synchronizing and desynchronizing systems in opposition to one another may be rejected and the effect of sensory deprivation on sleep might be simply regarded as a disruption of a regulatory process.

A choice between these alternative views of the effects of sensory deprivation on sleep cannot be made on the basis of data currently available. If similar phenomena could be demonstrated in cat, however, comparisons with other experimental treatments which are known to affect sleep, such as sensory deafferentation, brain lesions and electrical stimulation could be made. Although comparisons of this sort will not provide definitive answers to the questions outlined in this section, certain explanations will likely seem more plausible than others. A further reduction in the number of competing explanations for the effects of sensory deprivation on sleep

will be possible once the minimum conditions needed to produce the effect are known.

III. REM Sleep

(A) Brainstem Control of Phasic and Tonic Phenomena of REM Sleep

The sleep that is characterized by episodic twitching of the extremities and the occurrence of rapid eye movements has long been associated with dreaming (Moruzzi, 1964) but it took the work of Dement (1958) and Dement & Kleitman (1957a; b) to bring this phase of sleep into the realm of scientific investigation. For our purposes, the subsequent electrophysiological research aimed at determining what brain structures participate in the control of REM sleep is the most relevant since it appears from the human experiments that the function of this system is modified by sensory deprivation.

There is unanimous agreement in the literature that REM sleep is generated and controlled by neural processes located in the brainstem since removal of all structures rostral to the pontine reticular formation including the hypothalamus does not prevent the cyclic reoccurrence of total muscle atonia and REMs which characterize this phase of sleep (Hobson, 1965; Jouvet, 1962). The fact that the diencephalon and forebrain are not needed for occurrence of REM sleep is particularly clear in the chronic decerebrate cat (Villablanca; 1966a) where electrophysiological (pontine PGO waves) and behavioral signs (REMs, muscle atonia) of REM

sleep can be detected from behind the level of the transection while the forebrain can be either synchronized as in SWS or desynchronized as in waking or REM sleep. One should not conclude, however, that forebrain structures exert no influence on the REM sleep mechanism since there have been some clear demonstrations of effects of forebrain (Gadea-Ciria, 1976b; c; 1977a; b; Jeannerod, Mouret & Jouvet, 1965) and cerebellar lesions (Gadea-Ciria, 1976a) on REM sleep, and in particular on the oculomotor system involved in the generation of REMs.

The electrophysiological phenomena associated with REM sleep can be classified into six categories, along two major dimensions as in Table XXXV, Appendix A. This classification scheme rests on the assumption that CNS control of phasic and tonic phenomena occurring during REM sleep is carried out by structures in the pontine reticular formation. As has already been mentioned, the electrographic signs of REM sleep such as desynchronized EEG and PGO waves in the LGN and visual cortex no longer occur in the isolated forebrain of chronic decerebrate cat even though episodes of REM sleep reoccur in the brainstem (Villablanca, 1966a). This result suggests that forebrain manifestations of REM sleep are prevented in this preparation because pathways ascending from the brainstem have been cut. This interpretation is supported by experiments where localized lesions have been done to delineate the pathways involved (Candia et. al., 1967; Carli et. al.,

1965; Hobson et. al., 1969; Laurent, Cespuglio & Jouvet, 1974; Laurent, Guerrero & Jouvet, 1974; Laurent & Guerrero, 1975).

Localized lesions within the pontine reticular formation have also been done to determine more precisely the locus of the executive mechanisms controlling REM sleep phenomenon. Carli & Zanchetti (1965) have identified the middle and caudal portions of the nucleus reticularis pontis oralis as being critical for the occurrence of REM sleep since lesioning this structure was most consistently associated with a reduction in REM sleep. Jouvet (1962; 1969; 1972), however, has suggested that the executive mechanism controlling the ascending and descending components of REM sleep exist more caudally in middle and caudal portions of the nor-adrenergic locus coeruleus since selective lesions in these regions were capable of suppressing either forebrain or hindbrain manifestations of REM sleep.

Just as Jouvet has suggested that a dissociation of the control of ascending and descending components of REM sleep exists, Pompeiano and his collaborators have indicated other dimensions along which the control of REM sleep phenomenon may be divided. In the early description of REMs during REM sleep two types of eye movements were noted (Aserinsky & Kleitman, 1953; 1955); those that occurred singly and those that occurred in groups. The isolated REMs were similar to saccadic eye movements (Jeannerod, Mouret & Jouvet,

1965)-or perhaps a smooth pursuit eye movement (Fuchs & Ron, 1968; Salzarulo et. al., 1973) while the REM bursts were like the fast phase of nystagmus. These two classes of eye movements appear to be generated by different mechanisms since systemic injections of reserpine, which causes a release of stored monoamines (Brooks, Gershon & Simon, 1972), induce a regular repetitive occurrence of isolated PGO waves (See Section IIIB for details on the relationship between the occurrence of PGO waves and REMs) with the only evidence of a burst pattern being an occasional occurrence of pairs of PGO waves (Brooks & Gershon, 1972) whereas intravenous injections of eserine sulfate, an anticholinesterase, produce muscle atonia and the regular repetitive occurrence of bursts of REMs in acute decerebrate cat (Magherini, Pompeiano & Thoden, 1972; Seguin, Magherini & Pompeiano, 1973). In regard to the burst pattern, Thoden, Magherini & Pompeiano (1972) found neurons located mainly in the medial vestibular nuclei which either increased or decreased their firing rate prior to the occurrence of REM bursts with the direction of eye movements in the burst (i.e. either nasally or temporally) determining whether an increase or a decrease in firing rate was obtained. Since Bizzi, Pompeiano & Somogyi (1964) found similar relationships between changes in firing rate and the occurrence of REM bursts during REM sleep of unrestrained cat, it is possible that these neurons are involved in the genera-

tion of REM bursts both in the acute eserinized cat and during REM sleep. This inference is supported by the fact that medial vestibular neurons are known to project to motoneurons in the oculomotor nucleus through the medial longitudinal fasciculus (Lorente de Nó, 1933; Szentágothai, 1950). Furthermore, Pompeiano and his collaborators found that bilateral lesions of the medial and descending vestibular nuclei abolished the occurrence of REM bursts (Pompeiano & Morrison, 1965), the phasic increases in pyramidal tract discharge (Morrison & Pompeiano, 1966) and the phasic inhibition of spinal reflexes (Pompeiano & Morrison, 1966) associated with REM bursts during normal REM sleep of chronically prepared cat. The fact that single isolated REMs continued to occur after these lesions (But see Perenin & Jeannerod, 1971; Perenin et. al., 1972) and the fact that control lesions to the superior and lateral vestibular nuclei, total cerebellectomy and bilateral destruction of the VIII nerve did not prevent the occurrence of REM bursts during sleep, provide additional support for the hypothesis that REM bursts and isolated REMs are controlled by separate, specific brainstem systems. Since vestibular lesions similar to the ones employed by Pompeiano & Morrison also block the occurrence of REM bursts in eserinized decerebrate cat (Magherini et. al., 1972), one can tentatively conclude that this preparation may provide a useful model for study of the mechanisms responsible for

generating bursts of REM during paradoxical sleep (Hoshino & Pompeiano, 1976; Hoshino et. al., 1976; Karczmar et. al., 1970; Pompeiano & Hoshino, 1976; Pompeiano & Valentinuzzi, 1976).

(B) Relationship Between Unit Firing, PGO Waves and REMs: The Pontine Generator

Single units located in many different areas of the pontine reticular and bulbar reticular formations are known to increase their firing rate phasically in association with the occurrence of pontine PGO waves and REMs (Bizzi et. al., 1964; Chu & Bloom, 1974; Hobson et. al., 1974a; 1974b; McCarley & Hobson, 1975a; Netick et. al., 1977; Pivik et. al., 1977; Saito et. al., 1977). Although several models aimed at describing the brainstem mechanisms responsible for the genesis of the phasic phenomenon of REM sleep have postulated the existence of pacemaker and generator neurons (Pompeiano & Valentinuzzi, 1976; Ruch-Monachon et. al., 1976; Simon et. al., 1973), the exact identity of these elements remains unknown. It is generally believed that the output element of the brainstem reticular system, neurons which are presumed to be in the final common pathway to forebrain structures, oculomotor and hindbrain structures, are a population of cells located bilaterally in the paramedian pontine reticular formation (Ruch-Monachon et. al., 1976; Simon et. al., 1973). The individual neurons which constitute this population are

assumed to phasically increase their firing rate in unison by means of effective reciprocal excitatory connections. The summation of the EPSPs so generated both within this population of cells and in areas of the brain which receive input from these neurons constitutes the current sources of the PGO wave (Bizzi & Brooks, 1963; Brooks, 1967a; 1967b; 1968a; 1968b; Brooks & Bizzi, 1963; Calvet et. al., 1965; Jeannerod & Kiyono, 1969a).

A mechanism which may underlie the genesis of the PGO wave has been suggested from experiments where single, brief pulses of electrical stimulation were applied to the pontine reticular formation during REM sleep (Brooks & Bizzi, 1963; Malcolm, Watson & Burke, 1970). Under these conditions, it was found that PGO waves and REMs could be elicited in an all or nothing manner if a suprathreshold current pulse was applied at a time when a spontaneous PGO wave had not occurred for at least 200 msecs. If this same stimulation was applied during SWS, it was incapable of eliciting any response while during waking, it generated a saccadic eye movement and a PGO_W (See Brooks, 1968b and Jeannerod & Sakai, 1970 for the similarities and differences between PGO waves occurring during waking and REM sleep). In general, it appears that electrical stimulation of the brainstem is effective in eliciting PGO waves in any state where they are occurring spontaneously (Malcolm, Watson & Burke, 1970) with the provision

that the elicited PGO waves resemble the spontaneously occurring ones (Simon et. al., 1973). Subsequently, it has been reported that punctiform visual, auditory or cutaneous sensory stimulation are capable of eliciting PGO waves (Bowker & Morrison, 1976; Jeannerod & Kiyono, 1969b). The fact that sensory stimulation can activate the pontine generator is consistent with the observation that mesencephalic (Bell et. al., 1964; Groves et. al., 1973) and pontine reticular neurons (Baker et. al., 1976; Scheibel et. al., 1955) can typically be driven by sensory stimulation in one or more modalities. Since the latency to the onset of the PGO wave following electrical or sensory stimulation is much longer than would be expected if the response were simply an evoked potential and in view of the similarity between the spontaneous and elicited PGO waves, it is likely that the elicited PGO waves are generated through the normal physiological mechanisms. The fact that a threshold amount of stimulation is necessary for the elicitation of a PGO wave suggests that a certain amount of excitatory drive on the pontine generator is required before the population of generator neurons is activated. Since PGO waves are generated spontaneously during REM sleep, one could conclude that endogenously generated tonic activity is capable of producing the necessary excitatory input as well. Alternatively, the spontaneous generation of PGO could be due to some process intrinsic to the generator

neurons themselves. If this were the case, this process would have to be capable of being triggered by electrical or sensory stimulation.

The model of the brainstem system controlling REM sleep must take into account evidence which suggests that there are separate PGO wave generators on each side of the brainstem, which to some extent can function independent of each other. Brooks (1968b) reported that most PGO waves recorded from the LGN and visual cortex occur in bilateral synchrony indicating either that there are reciprocal connections between PGO wave generators on each side of the brainstem or that the generator neurons send axons (monosynaptically or through polysynaptic pathways) to both the ipsilateral and contralateral LGN and visual cortices. The fact that unilateral cooling of the brainstem at the level of the ponto-mesencephalic isthmus reversibly blocks the second component of biphasic PGO waves (Cespuglio et. al., 1975; Laurent & Guerrero, 1975) and the fact that unilateral ablation in this region decreases by half the number of PGO waves recorded from the LGN (Sakai et. al., 1976) and decouples the occurrence of PGO waves in one LGN from the occurrence of PGO waves in the other (Cespuglio et. al., 1976) indicates that the second alternative is likely true. The conclusion, however, seems to be contraindicated by the fact that bilateral synchrony of PGO waves also occurs in the pons

at the level of the abducens nucleus (Brooks & Gershon, 1971; Cespuglio, Laurent & Calvo, 1976). Cespuglio et. al. (1976) have suggested that this is due to reciprocal interconnection between abducens neurons on each side of the brainstem. This interpretation is supported by the fact that a midsaggital section of the brainstem extending from the level of the anterior border of the inferior colliculus caudally to the VIth nuclei eliminates the bilateral synchrony between the PGO waves recorded from the abducens nuclei without altering the synchrony between the PGO waves recorded in the LGN, while a midline section in the region of the supraoptic decussation eliminates bilateral synchrony of PGO waves in the LGN without interfering with synchrony in the abducens nuclei.

Brooks & Gershon (1971) noted that since there is bilateral synchrony of PGO waves in the abducens nuclei, the PGO wave must not reflect activity in motor neurons since this would mean that both lateral rectus muscles would contract simultaneously; an improbable result in view of the fact that the eyes typically move in bilateral coordination. The recent experiments of Cespuglio et. al. (1975; 1976) and Gadea-Ciria (1972) have clarified this problem by demonstrating that even though bilateral PGO waves are observed in the abducens nuclei, only one lateral rectus muscle contracts at a time. Furthermore, by making a mid-saggital section in the

region of the supraoptic decussation, thereby ensuring that PGO waves in the right and left LGN are being driven by the right and left pontine generator respectively, they have noted that the production of a PGO wave by the pontine generator on one side is always associated with a contraction of lateral rectus muscle on the contralateral side and lack of contraction (i.e. presumed inhibition) in the lateral rectus muscle on the ipsilateral side (Cespuglio et. al., 1976). Cespuglio et. al. (1975) have provided similar information for the participation of the oculomotor and trochlear nuclei in REMs, but the details have not been as well worked out. At the present, the best one can say is that lesions which block the occurrence of PGO waves in the LGN also eliminate them from the oculomotor and trochlear regions (Laurent, Cespuglio & Jouvet, 1974).

(C) The REM Sleep Cycle

Phenomena typically restricted to REM sleep, after specific pharmacological (Brooks & Gershon, 1972; Brooks, Gershon & Simon, 1972; Karczmar et. al., 1970) or surgical lesions (Jouvet, 1969; Simon et. al., 1973), can be observed to occur during waking and behavior which may be more typical of a waking animal can be seen during sleep. For example, Jouvet (1969; 1972) reported that destruction of the caudal portion of locus coeruleus caused a cat to display some most extraordinary behavior despite the fact that the animal was

apparently sleeping. Considerations of this sort have led to the hypothesis that REM sleep mechanisms are enabled (gated) by other systems (Jouvet, 1972; Simon et. al., 1973). The raphe nuclei, the locus coeruleus and the nucleus reticularis gigantocellularis are some structures which, on the basis of single unit data (Hobson, McCarley & Wyzinski, 1975; McCarley & Hobson, 1975b; McGinty & Harper, 1976; Sheu et. al., 1974; Siegel et. al., 1977; Vertes, 1977) and lesion studies (Jouvet, 1969; Puizillout et. al., 1974; Simon et. al., 1973) have been considered for a role in regulating the onset, duration and offset of REM sleep. This work is relevant to the present research in so far as the models that have been developed to explain the REM sleep cycle make predictions about REM sleep episode length in relationship to processes which occur during REM sleep. Since the human sensory deprivation experiments suggest that the control of events occurring during REM sleep may be modified independently of those processes controlling the sleep cycle, any theory which proposes an interaction between these systems would have to be reconsidered.

(D) Effects of Sensory Stimulation on REM Sleep

REM sleep has been suggested to play a critical role in many processes including memory consolidation (Gaarder, 1966; Oswald, 1969), the maintenance of homeostasis within the brain (Aserinsky, 1973; Stern & Morgane, 1974) and the

provision of a source of afferentation to the brain both during development (Roffwarg et. al., 1966) and in the mature state (Berger, 1969; Ephron & Carrington, 1966). Implicit in most of these theories about REM sleep is the assumption that processes which are critical for normal waking functions are carried out during REM sleep and that phenomena which occur during waking modulate REM sleep processes. In spite of this great emphasis, in theory, on an interaction between waking and sleep states, relatively little data has been obtained to indicate that, in fact, such interaction occurs.

It is a well known phenomenon to sleep researchers that when human subjects are required to sleep in a strange or novel environment, the amount of time spent in REM sleep decreases precipitously and the other stages of slow wave sleep become much shorter and sporadic in nature. Thus, the integrity of the sleep pattern may serve as a good index of the subject's reactivity to his environment, and in some instances, the well being of the subject (Baekland, 1970), but these effects on sleep cannot be associated with specific environmental features.

Attempts to determine what effect environmental complexity has on REM sleep have had limited success. Tagney (1973) reported that rats that were housed in groups, in large cages which contained many manipulanda such as ladders, tunnels and mazes, spent significantly more time in both SWS

and REM sleep over a twenty-three hour period than did control rats that were housed in isolation. If the isolated rats were transferred to the "sensory enriched environment" for sixteen days they significantly increased their SWS time, but not to the level set by rats that had been in the enriched environment for five weeks. Conversely, McGinty (1969b) reported that kittens reared in isolation from five weeks of age, to age two years, spent less time asleep than kittens reared in an enriched environment over a comparable period of time. Unfortunately, in these studies, the effects of environmental enrichment cannot be isolated from the effects of opportunity for exercise (Hobson, 1968; Horne & Porter, 1976) or the stress effects of isolation (Konrad & Bagshaw, 1970; Melzack, 1965). Control of these variables is important since increased exercise is known to increase SWS time while stress may decrease it. Horne & Walmsley (1976) in a similar study on human subjects, demonstrated that subjects who were exposed, during a seven hour period, to a variety of visually rich tasks prior to sleep, had a 21% to 31% average increase in stages III & IV of SWS relative to control nights which were preceded by limited visual stimulation. REM sleep duration was not different between these two conditions, but no measure of REM density was taken. Again, the results on SWS are open to question since there may have been a difference between the two conditions in terms of how much exercise the subjects got.

Berger (1969), on the basis of ontogenetic and phylogenetic data, proposed that eye movements during REM sleep serve to maintain tonus in the oculomotor system so that accurate coordinated eye movements can be made immediately upon waking. (See De La Pena et. al., 1973 for a similar theory.) Berger & Walker (1972) tested this theory by measuring the extent to which coordinated eye movements were made immediately prior to and after a REM sleep episode. They found that the incidence of ocular imbalance (i.e. the number of times that both eyes were not in alignment as subject tracked a target) and the magnitude of this imbalance was significantly greater prior to the onset of a REM sleep episode than after it. Thus, according to Berger's theory, a REM sleep episode is triggered so that a mechanism for initiating eye movements can be put into operation, thereby preventing further development of ocular misalignment. Since ocular alignment may also be maintained by oculomotor activity during waking, Berger (1969) predicted that intense oculomotor activity during waking would alleviate the need for eye movements during REM sleep. In an experiment designed to test this hypothesis, Berger (1968) found that monkeys trained to make many eye movements during waking had a lower REM density and a shorter duration REM sleep episode immediately following waking than when trained to make few eye movements. Linnstaedter (1971), in an analagous study where monkeys were trained to produce PGO_W

waves (PGO-like waves recorded from the LGN in the awake animal. These waves are typically associated with the occurrence of saccadic eye movements. See Brooks, 1968b; Jeannerod & Sakai, 1970 for details.) without the occurrence of eye movements was not able to find any effect on subsequent REM sleep. Thus, it appears that the actual occurrence of the eye movement and not just the field potential usually associated with the generation of an eye movement, is necessary in order to obtain the effect described by Berger.

Since the neural generation of REMs during sleep may partially be controlled by the vestibular nuclei (Pompeiano & Morrison, 1965), it is possible that changes in vestibular input might affect REM sleep processes. Studies which have been done to test this hypothesis, however, have produced conficting results. Jones & Sugie (1972) found that human subjects that slept on a platform oscillating about the horizontal axis (20° peak to peak oscillation; 0.25 to 0.50 Hz) never had any REM sleep during the course of a night's sleep. Ornitz et. al. (1973), however, in a study of the effects of vestibular stimulation (30° peak to peak oscillation at 0.17 cycles/second) on the sleep of four to five year old children found a tendency for the children to increase both total sleep time and REM sleep time on stimulation nights relative to control nights. In addition, REM density and other measures of eye movement frequency during sleep were significantly greater

during vestibular stimulation. In a study of newborn infants, Von Bernuth & Prechtl (1969) observed that vestibular stimulation during sleep reduced REM density. Since all of these studies used subjects of different ages, the differential effect of vestibular stimulation might be accounted for in these terms as Ornitz et. al. suggested, or it could be due to differences in the stimulus conditions.

In a somewhat related series of studies, Zimmerman, Stoyva & Metcalf (1970) and Zimmerman, Reite & Stoyva (1974) tested the effects of wearing spectacles which made the visual field appear upside down or tilted at a ninety degree angle. Since subjects initially feel very nauseated under these conditions (Allen et. al., 1972), one would expect that some change in vestibular input must be occurring. Thus, the large increase in REM sleep time found in human subjects (Zimmerman et. al., 1970) and in one of three cats run in an analagous animal experiment (Zimmerman et. al., 1974) is in line with the hypothesis that vestibular input has an important modulatory effect on REM sleep processes. No conclusive statement can be made though, since the critical variable responsible for the effects observed has not been worked out. The inconsistency found in the cat experiments and the failure of others to replicate the human experiments (Allen et. al., 1972) attest to the lack of understanding of what caused the effects described by Zimmerman and his colleagues.

(E) Discussion

The extensive research done on REM sleep processes in cat have provided a wealth of information about the neural systems controlling the phasic and tonic phenomena that occur during this phase of sleep. In addition, there are numerous theoretical papers that have attempted to use REM sleep phenomena as indicators of both normal (Aserinsky, 1973; Berger, 1969; Ephron & Carrington, 1966; Stern & Morgane, 1974) and abnormal processes (Clausen et. al., 1977; Feinberg, 1968; Ornitz et. al.,1969; Ornitz, 1972; Petre-Quadens & de Lee, 1970; Zarcone et. al., 1969). With this backlog of descriptive work and theoretical development, the sensory deprivation technique offers an unique opportunity to obtain new information about REM sleep mechanisms since it provides a way of directly manipulating REM sleep without grossly distorting the physiology of the organism, as pharmacological and surgical manipulations certainly must. Thus, the information obtained will further our understanding of the neural mechanisms controlling REM sleep under normal physiological conditions and will have direct bearing on theoretical issues in both the sensory deprivation and sleep literatures.

CHAPTER II

EXPERIMENT I

The objective of the first experiment was to obtain a description of the effects of sensory deprivation in cat. Since Potter & Heron (1972) had demonstrated a change in the percent time spent in different sleep stages and in REM density during sensory deprivation, both of these parameters were measured in the present experiment. Since an increase in the percent time spent asleep could be accomplished by increasing either the duration or the number of sleep episodes, these data were also tabulated for each sleep stage. In addition, the effects of sensory deprivation on the circadian rhythm of the sleep-wake cycle was determined since the circadian rhythm of activity cycles is known to be entrained by external cues (Aschoff, 1969; Mills, Minors & Waterhouse, 1974; Rusak & Zucker, 1975; Webb & Agnew, 1974). Thus, it seemed likely that under sensory deprivation conditions where external cues are minimal, the sleep-wake cycle might become free-running or possibly even totally desynchronized.

METHOD

Surgery

Six male adult cats under barbiturate anaesthesia were implanted with the following array of electrodes using standard stereotaxic and electrophysiological monitoring procedures: (1) one pair of transcortical visual cortex (area 17) electrodes, (2) one pair of transcortical frontal cortex electrodes (frontal pole), (3) one pair of bipolar lateral geniculate electrodes (AP = 5.0 mm., Lateral = 8.5 & 10.5 mm., Height = 3.5 & 5.0 mm.), (4) one pair of dorsal hippocampus electrodes (AP = 5.0 mm., Lateral = 4.0 mm., Height = 8.5 & 9.5 mm.), (5) EMG electrodes embedded in neck muscle, (6) EOG electrodes (one bone screw fastened to the back of the orbit of each eye). (7) One cat had bipolar electrodes implanted in the pontine reticular formation (Posterior = 6.0, Lateral = 2.0, Height = -7; Electrode lowered at an angle of 32^{o}, entering the cerebellum at Posterior = 19.0). (8) Three cats had YSI thermistors (part #44003) implanted in the intraperitoneal space near the midline in the region of the small intestine.

Apparatus and Material

The cage in which the animal was kept during the experiment was a large box (30" x 30" x 72") which was open at the top. Large plexiglas windows (14" x 24") on each side

of the box permitted a 360° view of the room. The box was located in the center of a sound-attenuated, shielded room (8' x 8'). Along one wall of the room were kept four cages housing other male cats. These animals were present in order to make the experimental chamber as much like a colony room as possible.

Lighting consisted of three 100 watt, diffuse lights located near the corners of the room. These lights were left on continuously (Intensity ranged from 1.5 to 7.5 foot-lamberts at the floor of the test chamber). Directly above the box containing the experimental animal was a fluorescent light (Intensity = 45 foot-lamberts at the floor of the cage). A speaker mounted above the fluorescent light was connected to a white noise generator. Mounted to the framework of the fluorescent light was a panel box which hung three feet above the floor of the cage. A shielded cable extended from the box and was connected to a 25 pin Amphenol plug mounted on the cat's head. This assembly gave the animal complete freedom of movement within the confines of its chamber. Some animals tended to twist the cable by turning in circles. This only happened occasionally though, and when it did the experimenter entered the test chamber to untangle the cat.

Bioelectric signals were led from the cat into a Model IV Grass polygraph located in an adjoining room. An

to of nd

video monitoring circuit allowed the experimenter to record the cat's behavior in a log book.

In one experiment, a measure of the total amount of time the cat spent moving about was obtained from a movement detection circuit. This circuit consisted of a Grass Model V integrator preamplifier whose inputs were connected to ground and to a loop of wire encircling the test cage. Movement by the cat changed its capacitance with respect to ground, thereby causing transients in the noise level being fed into the preamplifier. A movement detection threshold was obtained by feeding the integrated output of the preamplifier into a Schmidt gate. Whenever the cat made threshold size movements, the gate was opened and a circuit which generated current pulses at a constant frequency was allowed to function. These events were recorded by an event pen of a cumulative recorder. The threshold was set so that any gross body movements activated the timing circuit. Grooming and eating movements generally did not exceed threshold.

Procedure

In these experiments, each cat was allowed a three-week recovery following surgery in order to eliminate possible effects of barbiturate on REM sleep (Sharpless, 1970). Prior to beginning pre-deprivation recording, each cat had been living in the test chamber for at least a week, and had been connected to the cable for at least two days (Cat N06 was an

exception since in this animal pre-deprivation recording was begun with only one day prior habituation to the test chamber). Pre-deprivation records of EEG, EOG and EMG were taken continuously at a paper speed of 15 mm/second for three to nine days except for a 20 minute period each day when the animals were fed. Deprivation was initiated at the end of this period by suturing the cat's eyelids shut under nitrous oxide and halothane anaesthesia. This procedure required approximately 20 minutes to complete after which the animal was returned to the test chamber. A white noise (78 db) and the fluorescent light located above the test box were turned on at this time. Using the figures reported by Spiro & Kolbert (1974), it was estimated that the intensity of light reaching the cat's eye was reduced from 1.5 - 7.5 foot-lamberts to a uniform 0.23 to 0.36 foot-lamberts depending on eyelid pigmentation. Deprivation recording continued for six to fourteen days after which the cat was reanaesthetized to have its sutures removed. Pre-deprivation conditions were restored and post-deprivation recording was continued for another two to four days.

Data Analysis

Records were scored into sleep stages according to conventional electrographic criteria (Sterman et. al., 1965). Briefly, each 20 second epoch of EEG was classified into one of four categories; active waking, quiet waking, SWS or REM

sleep. Waking was distinguished from SWS by the presence of desynchronized cortical EEG and the absence of frontal cortical sleep spindles. SWS was distinguished from REM sleep by the presence of sleep spindles, slow waves and muscle tonus and the absence of hippocampal theta and REMs. Active waking and quiet waking were distinguished on the basis of the EOG record. It was noted that head movements which occur during eating, grooming, play or exploration induce slow eye movement which could be detected in the EOG record. Whenever such eye movements occupied more than 50% of the 20 second epoch, that period was classified as active waking. In one experiment, the amount of time the cat spent actively moving about was also determined from the movement-detection timing circuit for comparison.

In four sets of data, SWS was further subdivided into light slow wave sleep (LSWS) and deep slow wave sleep (DSWS) (Ursin, 1968). Records were classified as DSWS when large amplitude slow waves (1 to 4 Hz) were present at least 80% of the 20 second epoch on frontal cortical or occipital cortical channels.

For one representative pre-deprivation day and deprivation days one and three, the mean duration of each sleep stage was determined as was the average duration of the interval between successive occurrences of a particular

stage. A similar analysis was carried out for the sleep cycle which was defined as the period of time between the onset of a SWS episode and the end of a REM sleep episode which followed the SWS episode uninterrupted by periods of waking. Since SWS always precedes REM sleep in cat, the number of sleep cycles equals the number of REM episodes but sleep cycle duration and the interval between sleep cycles are different than for REM sleep.

The circadian rhythm in sleep and waking was analyzed by taking the percent time spent in each stage for three successive eight hour blocks and converting these percentages into standard scores. This was repeated for each recording day, thus eliminating the effect of daily fluctuations in the mean and variance by setting all means and standard deviations equal to zero and one respectively. The degree of correlation between individual days was determined by using the formula

$$r = \frac{\sum_{k=1}^{3} (Z_{\text{Day N}})(Z_{\text{Day N-1}})}{3}$$

Since each day in a given condition was correlated with every other day, $\frac{(N)(N-1)}{2}$ correlation coefficients were calculated. Thus for both the pre-deprivation and post-deprivation, three correlation coefficients were calculated

(N = 3), while for deprivation there were fifteen (N = 6). The mean degree of correlation within a given condition was then calculated and the group data was subjected to ANOVA to test the hypothesis $H_o: \mu_{\text{Pre-Dep.}} = \mu_{\text{Dep.}} = \mu_{\text{Post-Dep.}}$

The total number of REMs occurring during REM sleep for each 24 hour period were counted and expressed as an average number of REMs occurring per minute (i.e. REM density). The identification of REMs was facilitated by setting the low frequency filter on the polygraph so that one half amplitude attenuation occurred for a 5 Hz signal. The gain was then increased so that the largest amplitude REM was still within the limits of the available pen excursion. (The gain was usually set in the range of 10 to 20 μV/mm). Under these conditions, any signal producing a pen excursion of less than 1 to 2 mm was not counted. Since capacitance coupled recording was used, changes in the rate of charging and discharging the capacitor also generated "signals" which exceeded the threshold criteria. These, however, were easily identified and rejected since they occurred after an initial pen excursion with an amplitude proportional to the magnitude of the real signal and a rate of change typically slower than that generated by a REM. Intrascorer reliability was very good since repeated analysis produced identical results.

Repeated measures analysis of variance were done on

the mean time spent by each subject in the awake and sleep stages and on the mean REM density during pre-deprivation, deprivation and post-deprivation. Significant F tests were followed by multiple t tests using a procedure developed by Dunnett and outlined by Winer (1962, p80) where deprivation and post-deprivation group means were compared to the pre-deprivation group mean. (The critical value for this test is such that the probability of falsely rejecting the null hypothesis is less than or equal to 0.05 for each set of multiple comparisons.) After testing the significance of the differences between pre-deprivation group means and deprivation and post-deprivation values, a set of tests similar to those described above was performed to determine what day(s) of deprivation or post-deprivation differed from the mean of pre-deprivation. Within subject comparisons were done using a t test of the difference between correlated pairs of means (Guilford, 1965, p184) and tests of the significance of Pearson correlation coefficients were done by calculating a t statistic (Guilford, 1965, p163).

RESULTS

The literature contains several good descriptions of sleep in cats under laboratory conditions (Delorme et. al., 1964; Sterman et. al., 1965; Ursin, 1968) but no normative data have been reported for REM density. Comparing the sleep data with that collected during pre-deprivation of the present experiment (Table I) indicates that there exists a fairly large range in the mean percent time spent awake across studies. The results of the present study lay at the upper end of that range with the mean falling within one standard deviation of mean reported by Ursin (1968) and within two standard deviations of that reported by Delorme et. al. (1964). Comparing the percent time spent in SWS and REM sleep across studies reveals that both of these parameters were smaller in the present study. The proportion of SWS in relationship to REM sleep, however, was constant across studies (Table II). The means for LSWS and DSWS (Table IIIA) were within one standard deviation of those reported by Ursin (1968). The mean duration of SWS and REM sleep were shorter in the present study than has been reported by others (Table IIIB) but the mean duration of the intervals between sleep cycles and the number of episodes of REM sleep and SWS over a twenty-four hour period were comparable. Despite these differences between studies, both the amount

of time spent in different sleep stages and REM density fluctuated within narrow limits. Against this type of baseline the effects of deprivation were easy to resolve.

Figure 1 and Table IV give the average percent time for the six subjects spent in different waking and sleep states during pre-deprivation, deprivation and post-deprivation recording days. Two basic results are apparent from Figure 1. First, it is clear that on the average, the animals spent five to ten percent more time in SWS during deprivation than they did during either pre-deprivation or post-deprivation. Secondly, this additional sleep time was obtained at the expense of time spent in active waking and not by reducing time spent in quiet waking or in REM sleep. An analysis of variance performed on the group means for the different waking and sleep stages (Table V) substantiated these conclusions. Comparing deprivation and post-deprivation group means with pre-deprivation means demonstrated that pre-deprivation group means differed significantly from deprivation means for total waking ($t = 3.71$, $p<.005$), active waking ($t = 4.90$, $p<0.005$) and SWS ($t = 5.16$, $p<.005$). The comparable test made between pre-deprivation and post-deprivation group means failed to reach statistical significance ($t_{T.W.} = -0.06$, $t_{A.W.} = -1.48$, $t_{SWS} = -0.42$; all p's$>.05$) indicating that these parameters returned to baseline values during post-deprivation recording. Division of the deprivation

mean into six deprivation day means for total waking, active waking and SWS and comparing these with the overall group mean for pre-deprivation (Table VI) revealed that the amount of time spent in SWS and total waking during pre-deprivation was significantly different than during all six deprivation days (deprivation day 4 for total waking is an exception to this). For active waking, only the comparison between pre-deprivation and deprivation day 1 was significant although all the other tests bordered on statistical significance (Critical value $t = 2.43$, $p<.05$).

These general conclusions hold when individual subject data is viewed (Figures 2 to 6, Tables VII to XI). There are, however, a few additional features evident in the individual data that are not found in the group data. For example, Cat Q01 (Figure 4) reacted to deprivation not only by decreasing time spent in active waking but also by reducing its time in quiet waking, while Cat 411 (Figure 7) reduced its time spent in quiet waking without reducing the amount of time spent actively awake. Cat 403 (Figure 6), on the other hand, substantially increased its time spent in quiet waking on deprivation day 1 to complement a large reduction in active waking time and only a small increase in slow wave sleep time, while for Cat 007 (Figure 3), this occurred on deprivation day 4, only in this case, a reduction in slow wave sleep time complemented the increased time spent in quiet

waking. Cat Q01 showed a pattern similar to this on deprivation day 6.

Some subjects decreased the amount of time spent in REM sleep on the first few deprivation days (Cats N06, Q03; Figures 2 and 5) only to return to control levels (Figure 5) or perhaps slightly above control values (Figure 2) while for others (Cat 411, Figure 7) REM sleep time fluctuated slightly below pre- or post-deprivation day values throughout the entire deprivation period. One animal (Cat 007, Figure 3) did not change the amount of time spent in REM sleep during deprivation (Deprivation day 4 was an exception), while two cats (Cat Q01, 403; Figures 4 and 6) tended to have more REM sleep during deprivation than at other times. In the one animal where this was very pronounced (Cat Q01), the increase in REM sleep time was accompanied by the largest increase in slow wave sleep observed in these experiments.

Analysis of the activity index derived from electronic detection-timing circuit for Cat 007 (Figure 3) indicated that the mean time spent active during pre-deprivation was significantly greater than during deprivation ($t = 7.04$, $p<.01$). This was similar to the pattern that emerged from a test of the differences between the pre-deprivation and deprivation mean for active waking ($t = 9.74$, $p<.01$). This result is consistent with the fact that a significant positive correlation ($r = 0.70$, $t = 6.35$, $p<.01$) existed between the percent

time spent in active waking as determined from the classification of EEG records and percent time that the detection-timing circuit registered activity.

Since all subjects increased the amount of time spent in SWS during deprivation, the next step was to determine whether this additional SWS time was spent in LSWS or DSWS. Table XII presents the relevant data (The data of two cats was not analyzed since low frequency EEG was filtered out in these records and therefore, the distinction between LSWS and DSWS could not be made). A repeated measures ANOVA on this data showed that there was a significant treatment effect for LSWS ($F = 13.16$, $p<0.05$) but not for DSWS ($F = 0.48$, $p>.05$). Multiple t tests indicated that percent time spent in LSWS during deprivation was significantly greater than during pre-deprivation ($t = 3.75$, $p<.01$) while the pre-deprivation and post-deprivation group means did not differ from each other ($t = 1.16$, $p>.05$).

An analysis of the mean duration of each waking and sleep stage, and the average duration of time between successive occurrences of the same stage demonstrated that the increase in SWS time was due to a decrease in the duration of the interval between successive SWS episodes (Table XIV) and not to an increased episode duration (Table XIII; See Tables XV to XIX for individual subject data. No data from Cat N06 was available for analysis by this method since the

sequence and duration of waking and sleep stages was not retained when the raw records were first classified.). In fact, there was a tendency for SWS episodes to be shorter in duration during deprivation, but this effect failed to reach statistical significance. A similar analysis performed for active waking, quiet waking, REM sleep and the sleep cycle failed to demonstrate any significant difference between pre-deprivation and deprivation in terms of the mean episode duration or of mean duration of the interval between episodes. Again, there was a tendency for shorter duration episodes and in the case of active waking, the expected increase in the mean inter-episode duration failed to reach significance. Figures 8 and 9 which are records of raw data from two cats provide an illustration of these effects of deprivation. Cat Q01 (Figure 8) is exceptional in that this animal sustained an increase in REM sleep time during deprivation which appears in the raw data as an increase in the number of episodes of REM sleep. The large reduction in time spent actively awake is also evident in these figures, particularly for Cat Q01 on the first deprivation day. In addition, it appears that REM sleep is usually preceded by fairly long periods of SWS both during pre-deprivation and deprivation, but repeated interruption of SWS episodes by brief periods of quiet waking seem more typical during deprivation than during pre-deprivation.

From the raw data in Figures 8 and 9, it is also evident that the animals in this experiment spent more time asleep at one particular time of day than at another. Thus, for example, Cat Q03 slept more during the first eight hours of pre-deprivation day five than it did during the last eight hours. A similar pattern was present on deprivation day three suggesting that the circadian rhythm in the sleep-waking cycle may be unchanged by sensory deprivation. In order to evaluate this hypothesis, the percent time spent in SWS and REM sleep was plotted for successive eight hour blocks of time during pre-deprivation , deprivation and post-deprivation (Figures 10 and 11) as were the standard scores of this data (Figures 12 and 13). The correlation coefficients presented in Tables XX and XXIII and in Figures 12 and 13 above the portion of data from which they were derived by the autocorrelation technique described in the method section, provide an estimate of the degree of rhythmicity found in the plots of sleep-wake cycles over a number of days. Viewing these figures, it is obvious that a large positive correlation indicates that the circadian rhythm behaves much like a sine wave with regularly recurring peaks and troughs (See Cat 403). Zero and negative correlations are generated when peaks and troughs become phase shifted from day to day (See Cat Q03).

An analysis of variance of these correlation coeffic-

ients (Rows 1 to 4; Table XXIV) indicated that the rhythmicity of the sleep-waking cycle failed to change significantly across conditions for all measures except SWS. In the case of SWS, the pre-deprivation mean correlation was significantly different from the post-deprivation mean ($t = 3.31$, $p<.01$) but not from the mean correlation obtained during deprivation ($t = 1.47$, $p>.05$). This result suggests that the termination of deprivation caused a greater disruption in the circadian rhythm of SWS than did the initiation of deprivation.

Turning to the REM density data (Figure 14, Table XXV), a pattern very similar to that described for SWS emerges with REM density increasing significantly during deprivation and returning to pre-deprivation levels during post-deprivation recording ($F = 27.41$, $p<.01$; $H_o: \bar{X}_{Pre} = \bar{X}_{Dep}$, $t = 6.71$, $p<.005$; $H_o: \bar{X}_{Pre} = \bar{X}_{Post}$, $t = 0.63$, $p>.05$). Division of the deprivation group mean into six deprivation day means and comparing these with the overall group mean for pre-deprivation revealed that REM density on deprivation day one and on all subsequent deprivation days was significantly greater than during pre-deprivation (all p's$<.005$). Individual subject data (Figures 15 to 17) conformed to the average data except it should be noted that the REM density of subject N06 (Figure 15) was tending toward pre-deprivation levels by deprivation day fourteen.

When standard scores of REM density averaged over

successive eight hour blocks were plotted and an autocorrelational analysis similar to the one used for the sleep-waking cycle was employed, no circadian rhythm in REM density was found (Figure 18; Table XXVI). An analysis of variance performed on the group data (Row 5; Table XXIV) showed that the degree of rhythmicity in REM density did not significantly change across conditions. It will be noted in Table XXVI, however, that there was a tendency for REM density to be less arrhythmic during deprivation than during pre-deprivation. This was true for all animals except Cat 411.

DISCUSSION

A comparison of the percent time spent in different sleep stages under "standard laboratory conditions" reveals some startling differences across studies. The fact that cats in the present study slept only as much as those cited in other studies when they were in "sensory deprivation" suggests that what is commonly considered to be a standard laboratory environment is rather an impoverished sensory environment. This is particularly true in sleep studies, where precautions are taken to ensure that the animal is not disturbed by any external influence. Thus, the use of social isolation, sound attenuated chambers (Delorme et. al., 1964; Sterman et. al., 1965; Ursin, 1968) and masking white noise (Sterman et. al., 1965) may have produced a set of conditions which were actually as severe as those used in the present experiment during sensory deprivation. The fact that cats in Delorme et. al's. study slept a greater proportion of the day than those in any other study may have been due to the fact that these workers used a day-night cycle of 10 hours light; 14 hours dark. This interpretation is consistent with the experiment of Fishman & Roffwarg (1972) where it was found that the proportion of time that rat spent in SWS and REM sleep was different during a 12 hour light; 12 hour dark schedule than during either continuous darkness or continuous

illumination.

It is interesting to note that the additional time spent in REM and SWS in the studies by Delorme et. al., Sterman et. al. and Ursin, when compared to the present study, was due to a longer duration of SWS and REM sleep episodes and not to a greater number of episodes. This stands to contrast to the results obtained during sensory deprivation where additional sleep time was gained by the animal having a greater number of episodes of SWS accompanied by a slight reduction in the mean episode duration. Furthermore, the fact that the increase in SWS during sensory deprivation was not accompanied by an increase in REM sleep suggests it may be incorrect to assume that cats in this study simply attained a proportion of sleep during sensory deprivation that is more characteristic of laboratory kept animals. If this were true, one would have expected both SWS and REM sleep time to increase by an increase in episode duration.

The fact that the increase in SWS was due to an increase in light SWS is consistent with the increase in number of episodes of SWS during deprivation. Since light SWS is the first phase of sleep that the animal enters into when going to sleep, one would expect a number of transitions between quiet waking and sleep to occur before deep SWS was entered. Thus, if the proportion of this phase of sleep increased, as it did during deprivation, a greater number of

episodes of SWS would be generated.

The fact that the increase in light SWS was not accompanied by any change in deep SWS or REM sleep appears to conflict with what is known about the relationship between light SWS, deep SWS and REM sleep. Ursin (1968) reported that the percent time spent in deep SWS over a twenty-four period was positively correlated with the percent time spent in REM sleep. Light SWS was negatively correlated with deep SWS and also tended to be negatively correlated with REM sleep. According to these relationships, one would have expected the increase in light SWS to be accompanied by a decrease in both REM sleep and deep SWS had total sleep time remained constant. However, total sleep time increased and thus, the increase in light SWS was accompanied by a decrease in time spent awake.

Dividing waking time into active and quiet waking showed that active waking but not quiet waking was reduced by sensory deprivation. When active waking was further subdivided into different classes of behavior according to how much movement was involved in the activity by the activity detection-timing circuit, it was found that less time was spent in vigorous behaviors during deprivation than during pre-deprivation or post-deprivation. These results are consistent with behavioral observations where play and exploratory behaviors, behaviors which depended heavily on vision

for their execution, clearly occurred less during deprivation than during pre-deprivation. This was particularly evident in animals that had a high baseline level of activity.

The failure of sensory deprivation to cause a disruption of the sleep-wake cycle is perhaps, not surprising since there was only one identifiable zeitgeber in these experiments, the fixed feeding time, and this time marker remained constant across conditions. With such a salient cue marking the beginning of a new day, it seems likely that a twenty-four hour cycle would be adopted. This interpretation is consistent with experiments which show that feeding time can serve to entrain circadian rhythms (Edmonds & Adler, 1977a; b; Sulzman et. al., 1977). Even if the animal had a free running cycle though, it is unlikely that it would be detected since the longest period of uninterrupted recording was only six days, hardly long enough to cause phase shifting if the cycle was only marginally longer or shorter than twenty-four hours. In addition, the perturbance in the sleep-waking cycle caused by the beginning and particularly, the ending of deprivation, made interpretation of the cycle after these events difficult.

The changes observed in REM density during sensory deprivation were very large and reliably obtained. Further analysis of this phenomenon, however, will be presented in Experiment II, so discussion of this aspect of the first

experiment will be delayed till then. Nevertheless, it should be noted here that the change in REM density observed in these experiments, clearly parallels the effect of sensory deprivation on REM density in humans (Compare Figure 14 to Figure 19 and Figures 15, 16 & 17 to Figure 20). The two sets of results differed in that the average REM density for cat under baseline conditions was about five times greater than that found in humans. Thus, the human subjects showed a larger percent increase in REM density during deprivation while the cat subjects had a larger absolute increase. In addition, cat responded more rapidly to the onset and termination of deprivation than did the human subjects but both tended to reach plateau levels by the third or fourth deprivation day.

A comparison of the effect of sensory deprivation on the percent time spent in different sleep stages for cat (Figure 1) and human (Figure 21) reveals further similarities between the human and cat data. Thus, both responded to deprivation by decreasing time spent awake, with a significant reduction occurring on deprivation day one for cat and during the first two days of deprivation for human subjects. This additional sleep time was spent in light SWS by cat and stage I and stage II sleep by humans. In the case of the human subject, though, the increase in stage II time was significant only during the first two deprivation days while

the effect on stage I was significant on deprivation days one to six. The effect of sensory deprivation on REM sleep, stage III and stage IV sleep failed to reach significance.

The fact that sensory deprivation causes an increase in the amount of time spent in the light phases of sleep for both human and cat, but not in the sleep characterized by the presence of large amplitude slow waves (i.e. deep SWS and stage III & stage IV sleep) or REM sleep, is good evidence that the effects of sensory deprivation on sleep of human and cat are the same. The REM density data provide additional support for this hypothesis. Thus, on the basis of these results, one can tentatively conclude that the procedures employed in this experiment to produce sensory deprivation in cat provide a satisfactory model of human sensory deprivation experiments. It is interesting to note in this regard that Dallaire & Ruckebusch (1974) have recently reported that ponies deprived of visual and auditory input increased their time spent in SWS without altering REM sleep time as in the present experiment. In contrast to the present results though, REM sleep time increased above baseline levels during post-deprivation. No data on REM density was given so that it is not known whether this parameter changed during sensory deprivation.

Table I - Mean percent of recording time (± S.D.) spent in different sleep stages compared across studies.

Table I

	Delorme et. al. (1964) n = 9	Sterman et. al. (1965) n = 3	Ursin (1968) n = 12	Present Study n = 6
Total Time Awake	31.5 ± 7.0	42.3	44.9 ± 7.1	53.5 ± 4.9
Total Time Asleep	68.5 ± 6.5	57.7	55.1 ± 7.1	47.5 ± 4.9
SWS	53.5 ± 7.0	42.3 ± 6.1	41.7 ± 6.8	36.1 ± 5.5
REM Sleep	16.6 ± 2.0	15.5 ± 2.2	13.4 ± 3.3	11.0 ± 3.0

Table II - Mean percent of total sleep time (± S.D.) spent in different sleep stages compared across studies.

Table II

	Delorme et. al. (1964)	Sterman et. al. (1965)	Ursin (1968)	Present Study
SWS	77.0 ± 4.0	72.4 ± 4.2	75.9 ± 3.9	77.4 ± 5.5
REM Sleep	23.0 ± 4.0	27.5 ± 4.2	24.1 ± 3.9	22.6 ± 5.5

Table IIIA - Mean percent of recording time and total sleep time (± S.D.) spent in light and deep slow wave sleep compared across studies.

Table III A

	Percent of Recording Time		Percent of Sleep Time	
	Ursin (1968)	Present Study	Ursin (1968)	Present Study
LTS	15.9 ± 5.6	17.1 ± 4.0	29.0 ± 11.5	34.7 ± 5.6
DTS	25.3 ± 6.9	20.3 ± 2.1	46.5 ± 9.5	41.7 ± 3.4

Table IIIB - Mean duration and number of slow wave sleep and REM sleep episodes plus the mean duration of the interval between sleep cycles compared across studies (Mean ± S.D.). (In this Table and all subsequent Tables where time duration is reported, the units of time are given in minutes.)

Table IIIB

		Delorme et.al. (1964)	Sterman et.al. (1965)	Ursin (1968)	Present Study
SWS	Duration	------	5.6 ± 0.8	------	4.83 ± 1.04
	Number of Episodes	------	107.2 ± 12.9	------	113.00 ± 25.00
REM Sleep	Duration	5.8 ± 1.3	5.8 ± 1.3	5.7 ± 0.8	4.80 ± 1.09
	Number of Episodes	------	37.0 ± 7.2	34.[illegible] ± 8.[illegible]	35.20 ± 9.80
Sleep Cycle	Duation of the interval between sleep cycles	27.2	25.8 ± 7.1	23.5 ± [illegible]	25.20 ± 7.19

Table IV - Percent time spent in total waking, active waking, quiet waking, slow wave sleep and REM sleep during pre-deprivation, deprivation and post-deprivation (Mean ± S.E.).

Table IV

	Total Waking		Active Waking		Quiet Waking		Slow Wave Sleep		REM Sleep	
	$\bar{X}$	S.E.	$\bar{X}$	S.E.	$\bar{X}$	S.E.	$\bar{X}$	S.E.	$\bar{X}$	S.E.
Pre-Dep 1	54.7	1.9	23.2	4.5	31.5	4.1	34.8	1.9	10.5	1.1
Pre-Dep 2	46.4	4.6	22.9	4.3	27.7	3.5	37.1	1.8	11.2	1.5
Pre-Dep 3	50.9	3.2	22.6	4.1	28.2	3.5	37.8	3.0	11.3	1.1
Dep Day 1	44.2	3.7	12.9	3.1	30.4	4.8	45.2	3.2	10.1	2.3
Dep Day 2	44.3	2.7	18.0	3.9	26.0	3.8	44.3	2.9	11.2	1..
Dep Day 3	45.8	2.8	18.7	2.8	26.5	4.2	44.2	3.3	10.7	1.2
Dep Day 4	48.6	3.8	19.0	3.4	29.7	4.2	41.5	3.9	10.4	1.1
Dep Day 5	44.5	3.1	18.1	2.9	26.3	3.6	43.8	3.1	11.9	1.0
Dep Day 6	45.5	4.1	18.3	2.4	29.5	4.0	42.6	3.2	10.7	1.1
Post-Dep 1	53.3	3.0	21.3	4.2	33.4	2.3	35.1	2.8	11.6	1.0
Post-Dep 2	51.5	2.9	21.1	3.2	30.3	3.4	37.1	2.8	11.5	0.7
Post-Dep 3	52.0	2.3	18.6	1.5	33.4	2.5	36.6	2.5	11.4	1..

Table V - Repeated measures ANOVA on percent time spent in different stages of waking and sleep during pre-deprivation, deprivation and post-deprivation.

Table V

State	$MS_{treatment}$	MS_{error}	F	P
Total Waking	93.14	10.36	9.01	<.05
Active Waking	36.33	4.22	8.54	<.05
Quiet Waking	22.05	17.46	1.21	n.s.
Slow Wave Sleep	104.08	5.39	19.31	<.01
REM Sleep	8.77	2.62	3.33	n.s.

Table VI - Dunnett's multiple t test comparing all deprivation day group means with pre-deprivation means for total waking, active waking and slow wave sleep.

Table VI

State	Dep 1	Dep 2	Dep 3	Dep 4	Dep 5	Dep 6
Total Waking	-3.36**	-3.30**	-2.69*	-1.56	-3.24**	-2.81*
Active Waking	-4.78***	-2.32	-2.00	-1.86	-2.26	-2.15
Slow Wave Sleep	5.02***	4.50***	4.45***	2.85*	4.23***	3.51***

*p < .05 **p < .01 ***p < .005

Table VII - Percent time spent awake by each subject during pre-deprivation, deprivation and post-deprivation.

Table VII

	Pre-Deprivation			Deprivation						Post-Deprivation		
	1	2	3	1	2	3	4	5	6	1	2	3
N06	--	57.0	51.6	51.8	47.3	45.2	45.0	43.7	40.8	46.4	51.4	--
007	57.4	47.9	55.1	44.0	48.9	50.2	64.9	53.0	49.7	63.4	51.8	51.4
Q01	55.0	52.5	54.6	32.0	38.9	38.5	42.7	36.4	41.5	56.0	57.6	52.6
Q03	47.4	45.9	35.5	34.4	33.5	36.7	37.8	34.8	30.0	44.5	38.0	44.1
403	57.6	57.4	55.9	53.4	46.8	51.9	51.7	49.6	58.2	58.2	53.4	58.0
411	56.0	49.7	53.0	49.5	50.6	52.5	49.5	49.2	53.1	51.2	56.5	53.9
$\bar{X}$	54.7	51.7	50.9	44.2	44.3	45.8	48.6	44.5	45.5	53.3	51.5	52.0
S.E.	1.9	4.6	3.2	3.7	2.7	2.8	3.8	3.1	4.1	3.0	2.9	2.3

Table VIII - Percent time spent in active waking by each subject during pre-deprivation, deprivation and post-deprivation.

Table VIII

	Pre-Deprivation			Deprivation						Post-Deprivation		
	1	2	3	1	2	3	4	5	6	1	2	3
N06	--	--	--	--	--	--	--	--	--	--	--	--
007	39.7	29.7	34.7	23.8	28.4	22.8	25.7	23.6	22.3	34.6	28.7	22.8
Q01	22.8	30.0	24.1	12.5	20.5	24.9	26.6	23.4	22.8	14.7	28.3	20.3
Q03	17.6	13.6	13.7	6.1	7.8	10.4	9.9	8.2	12.2	13.6	13.8	16.4
403	28.8	30.1	27.5	8.2	22.8	21.3	20.0	20.0	21.6	27.9	19.8	19.2
411	13.1	11.4	13.3	13.9	10.5	13.9	12.5	15.4	12.9	15.6	15.1	14.2
$\bar{X}$	23.2	22.9	22.6	12.9	18.0	18.7	19.0	18.1	18.3	21.3	21.1	18.6
S.E.	4.5	4.3	4.1	3.1	3.9	2.8	3.4	2.9	2.4	4.2	3.2	1.5

Table IX - Percent time spent in quiet waking by each subject during pre-deprivation, deprivation and post-deprivation.

Table IX

	Pre-Deprivation			Deprivation						Post-Deprivation		
	1	2	3	1	2	3	4	5	6	1	2	3
N06	--	--	--	--	--	--	--	--	--	--	--	--
007	17.7	18.3	20.4	20.2	20.5	27.4	39.2	29.5	27.4	28.8	23.1	28.6
Q01	32.2	22.5	30.4	19.5	18.5	13.6	16.1	13.0	18.7	41.3	29.3	32.3
Q03	29.8	32.3	21.9	31.7	27.0	22.2	24.5	25.4	24.4	30.9	24.2	27.7
403	34.8	27.3	28.4	45.2	24.0	30.6	31.7	29.6	36.6	30.3	33.6	38.7
411	43.0	38.3	39.7	35.6	40.1	38.6	37.0	33.9	40.3	35.7	41.4	39.7
$\bar{X}$	31.5	27.7	28.2	30.4	26.0	26.5	29.7	26.3	29.5	33.4	30.3	33.4
S.E.	4.1	3.5	3.5	4.8	3.8	4.2	4.2	3.6	4.0	2.3	3.4	2.5

Table X - Percent time spent in slow wave sleep by each subject during pre-deprivation, deprivation and post-deprivation.

Table X

	Pre-Deprivation			Deprivation						Post-Deprivation		
	1	2	3	1	2	3	4	5	6	1	2	3
N06	--	35.1	38.4	40.8	44.1	43.7	43.6	45.5	46.2	40.0	36.6	--
007	32.4	37.8	33.3	44.2	39.8	38.4	28.1	34.9	38.6	26.3	35.8	35.6
Q01	34.2	33.9	33.9	48.6	45.9	45.7	44.3	47.2	46.1	31.3	32.1	35.2
Q03	41.7	45.5	52.2	59.5	56.8	59.1	56.8	56.1	54.8	45.6	50.0	45.9
403	35.5	35.6	37.2	39.2	43.3	41.0	39.9	41.1	35.4	33.5	37.9	35.3
411	30.1	34.7	31.9	38.7	35.9	37.3	36.4	38.-	34.7	33.9	30.2	31.1
$\bar{X}$	34.8	37.1	37.8	45.2	44.3	44.2	41.5	43.8	42.6	35.1	37.1	36.6
S.E.	1.9	1.8	3.0	3.2	2.9	3.3	3.9	3.1	3.2	2.8	2.8	2.5

Table XI - Percent time spent in REM sleep by each subject during pre-deprivation, deprivation and post-deprivation.

Table XI

	Pre-Deprivation			Deprivation						Post-Deprivation		
	1	2	3	1	2	3	4	5	6	1	2	3
N06	--	7.9	10.0	7.4	8.6	11.1	11.4	10.8	13.0	13.5	12.1	--
007	10.2	14.2	11.7	11.8	11.3	11.4	7.1	12.1	11.7	10.3	12.4	12.9
Q01	10.8	13.6	11.5	19.4	15.2	15.8	13.0	16.4	12.4	12.7	10.3	12.3
Q03	11.0	8.7	12.3	2.8	8.4	8.3	8.8	10.4	8.6	10.0	12.0	10.0
403	6.9	7.0	6.9	7.4	9.9	7.1	8.3	9.3	6.4	8.2	8.7	6.7
411	13.9	15.6	15.2	11.8	13.6	10.2	14.1	12.4	12.2	14.7	13.3	15.0
$\bar{X}$	10.5	11.2	11.3	10.1	11.2	10.7	10.4	11.9	10.7	11.6	11.5	11.4
S.E.	1.1	1.5	1.1	2.3	1.1	1.2	1.1	1.0	1.1	1.0	0.7	1.4

Table XII - Percent time spent in light slow wave sleep and deep slow wave sleep during pre-deprivation, deprivation and post-deprivation.

Table XII

		Pre-Deprivation	Deprivation Day 3	Post-Deprivation
Light SWS	N06	15.5	20.9	16.3
	Q01	15.5	19.0	14.4
	Q03	23.8	32.8	18.4
	411	13.6	22.1	11.1
	$\bar{X}$	17.1	23.7	15.1
	S.E.	2.2	3.1	1.5
Deep SWS	N06	23.0	22.8	20.3
	Q01	18.4	26.7	16.8
	Q03	21.7	26.5	27.5
	411	18.3	15.2	20.0
	$\bar{X}$	20.3	22.8	21.1
	S.E.	1.2	2.7	2.3

Table XIII - Episode duration (Mean, S.E., F test) for active waking, quiet waking, slow wave sleep, REM sleep and the sleep cycle during pre-deprivation and deprivation days one and three.

Table XIII

	Pre-Dep.		Dep. 1		Dep. 3		
	$\bar{X}$	S.E.	$\bar{X}$	S.E.	$\bar{X}$	S.E.	F
Active Waking	4.03	0.36	3.32	0.87	5.20	1.00	2.86
Quiet Waking	2.72	0.52	2.35	0.36	2.22	0.28	2.19
Slow Wave Sleep	4.83	0.52	4.00	0.38	4.40	0.38	4.08
REM Sleep	4.80	0.54	3.96	0.52	3.94	0.45	1.70
Sleep Cycle	12.54	1.31	10.18	0.76	12.27	1.62	1.32

Table XIV - Duration of the interval between successive episodes (Mean, S.E., F test) of active waking, quiet waking, slow wave sleep, REM sleep and the sleep cycle during pre-deprivation and deprivation days one and three.

Table XIV

	Pre-Dep.		Dep. 1		Dep. 3		
	$\bar{X}$	S.E.	$\bar{X}$	S.E.	$\bar{X}$	S.E.	F
Active Waking	18.69	4.95	25.29	5.31	25.20	5.44	4.33
Quiet Waking	6.00	0.35	5.49	1.00	6.57	1.11	0.96
Slow Wave Sleep	7.65	1.09	4.38	0.49	5.28	0.53	12.58**
REM Sleep	32.88	3.96	36.86	9.91	33.85	7.90	0.12
Sleep Cycle	25.21	3.59	30.88	9.86	25.49	6.79	0.30

*p<.05 **p<.01

Table XV - Episode duration and the duration of the intervals between successive episodes of active waking (Mean ± S.E.) for five animals during pre-deprivation and deprivation days one and three.

Table XV

		Mean Episode Duration			Mean Duration of Interval Between Episodes		
		Pre-Dep.	Dep.1	Dep.3	Pre-Dep.	Dep.1	Dep.3
Active Waking	007	3.5	2.6	3.4	8.6	9.0	11.7
	Q01	3.5	3.6	8.3	11.7	28.2	24.6
	Q03	3.5	1.7	3.3	23.3	23.6	31.4
	403	4.2	2.2	4.2	13.9	23.6	16.1
	411	5.4	6.6	6.8	35.9	42.1	42.1
	$\bar{X}$	4.0	3.3	5.2	18.7	25.3	25.2
	S.E.	0.4	0.9	1.0	5.0	5.3	5.4

Table XVI - Episode duration and duration of the intervals between successive episodes of quiet waking (Mean ± S.E.) for five animals during pre-deprivation and deprivation days one and three.

Table XVI

		Mean Episode Duration			Mean Duration of Interval Between Episodes		
		Pre-Dep.	Dep.1	Dep.3	Pre-Dep.	Dep.1	Dep.3
Quiet Waking	007	1.6	1.4	2.1	6.8	5.2	5.4
	Q01	3.1	2.3	1.7	6.9	9.2	10.9
	Q03	2.4	1.8	1.7	5.5	3.7	6.1
	403	2.9	3.2	2.5	5.3	3.9	5.6
	411	3.6	3.1	3.2	5.5	5.5	4.8
	$\bar{X}$	2.7	2.4	2.2	6.0	5.5	6.6
	S.E.	0.3	0.4	0.3	0.4	1.0	1.1

Table XVII - Episode duration and duration of the intervals between successive episodes of slow wave sleep (Mean ± S.E.) for five animals during pre-deprivation and deprivation days one and three.

Table XVII

		Mean Episode Duration			Mean Duration of Interval Between Episodes		
		Pre-Dep.	Dep.1	Dep.3	Pre-Dep.	Dep.1	Dep.3
Slow Wave Sleep	007	4.4	3.6	3.6	6.8	4.3	5.6
	Q01	6.7	5.4	5.4	11.4	5.4	5.8
	Q03	4.5	3.8	4.9	5.1	2.6	3.3
	403	5.0	3.6	4.7	8.7	4.8	6.4
	411	3.6	3.7	3.4	6.4	4.8	5.5
	$\bar{X}$	4.8	4.0	4.4	7.7	4.4	5.3
	S.E.	0.5	0.4	0.4	1.1	0.5	0.5

Table XVIII - Episode duration and duration of the intervals between successive episodes of REM sleep (Mean ± S.E.) for five animals during pre-deprivation and deprivation days one and three.

Table XVIII

		Mean Episode Duration			Mean Duration of Interval Between Episodes		
		Pre-Dep.	Dep.1	Dep.3	Pre-Dep.	Dep.1	Dep.3
REM Sleep	007	6.4	4.3	4.9	31.2	26.0	31.1
	Q01	5.8	5.1	3.6	35.9	18.6	19.0
	Q03	3.8	2.1	3.3	31.9	71.4	31.3
	403	3.9	4.5	5.1	45.0	46.4	64.1
	411	4.1	3.8	2.8	20.4	21.9	23.8
	$\bar{X}$	4.8	4.0	3.9	32.9	36.9	33.9
	S.E.	0.5	0.5	0.5	4.0	9.9	7.9

Table XIX - Episode duration and duration of the intervals between successive episodes of the sleep cycle (Mean ± S.E.) for five animals during pre-deprivation and deprivation days one and three.

Table XIX - Episode duration and duration of the intervals between successive episodes of the sleep cycle (Mean ± S.E.) for five animals during pre-deprivation and deprivation days one and three.

Table XX - Autocorrelation coefficients derived from the circadian rhythm data for active waking during pre-deprivation, deprivation and post-deprivation.

Table XX

	Pre-Deprivation		Deprivation		Post-Deprivation	
	$\bar{r}$	S	$\bar{r}$	S	$\bar{r}$	S
007	0.42	0.50	0.85	0.15	0.93	0.06
Q01	0.76	0.20	0.87	0.17	-0.02	0.78
Q03	0.68	0.18	-0.05	0.66	-0.32	1.03
403	0.99	0.01	0.69	0.34	0.53	0.37
411	0.50	0.44	0.12	0.67	-0.35	0.86
$\bar{X}$	0.67		0.50		0.15	
S.E.	0.10		0.22		0.25	

Table XXI - Autocorrelation coefficients derived from the circadian rhythm data for quiet waking during pre-deprivation, deprivation and post-deprivation.

Table XXI

	Pre-Deprivation		Deprivation		Post-Deprivation	
	$\bar{r}$	S	$\bar{r}$	S	$\bar{r}$	S
007	0.62	0.31	0.72	0.29	-0.31	1.12
401	0.94	0.06	0.05	0.87	0.44	0.45
Q03	0.78	0.19	-0.09	0.67	0.37	0.52
403	0.53	0.41	0.85	0.17	0.04	0.03
411	0.72	0.23	0.80	0.19	-0.37	0.61
$\bar{X}$	0.72		0.47		0.03	
S.E.	0.07		0.20		0.17	

Table XXII - Autocorrelation coefficients derived from the circadian rhythm data for slow wave sleep during pre-deprivation, deprivation and post-deprivation.

Table XXII

	Pre-Deprivation		Deprivation		Post-Deprivation	
	$\bar{r}$	S	$\bar{r}$	S	$\bar{r}$	S
NQ6	0.87	0.12	0.59	0.39	-0.11	0.20
007	0.65	0.30	0.62	0.34	0.35	0.53
Q01	0.89	0.12	0.51	0.47	0.77	0.19
Q03	0.96	0.04	0.03	0.69	0.02	0.86
403	0.91	0.08	0.90	0.10	0.75	0.21
411	0.64	0.31	0.68	0.29	-0.45	0.55
$\bar{X}$	0.82		0.56		0.22	
S.E.	0.06		0.29		0.20	

Table XXIII - Autocorrelation coefficients derived from the circadian rhythm data for REM sleep during pre-deprivation, deprivation and post-deprivation.

Table XXIII

	Pre-Deprivation		Deprivation		Post-Deprivation	
	$\bar{r}$	S	$\bar{r}$	S	$\bar{r}$	S
N06	0.64	0.31	0.75	0.23	-0.12	--
007	-0.03	0.90	0.74	0.33	0.76	0.21
Q01	0.40	0.51	0.12	0.78	0.78	0.19
Q03	0.30	0.51	-0.17	0.74	-0.15	0.82
403	0.93	0.07	0.95	0.05	0.89	0.10
411	0.74	0.23	0.47	0.56	0.37	0.55
$\bar{X}$	0.50		0.48		0.42	
S.E.	0.14		0.18		0.19	

Table XXIV - Summary of ANOVA of autocorrelation coefficients derived from the circadian rhythm data for active waking, quiet waking, slow wave sleep, REM sleep and REM density.

Table XXIV

	Pre-Dep.		Deprivation		Post-Dep.		
	$\bar{r}$	S.E.	$\bar{r}$	S.E.	$\bar{r}$	S.E.	F
Active Waking	0.67	0.10	0.50	0.22	0.15	0.25	2.9~
Quiet Waking	0.72	0.07	0.47	0.20	0.03	0.17	3.39
Slow Wave Sleep	0.82	0.06	0.56	0.12	0.22	0.20	5.49*
REM Sleep	0.50	0.14	0.48	0.18	0.42	0.19	0.07
REM Density	-0.05	0.12	0.15	0.06	-0.20	0.12	2.06

*$p < .05$

Table XXV - REM density for six subjects during pre-deprivation, deprivation and post-deprivation.

Table XXV

	Pre-Deprivation			Deprivation						Post-Deprivation		
	1	2	3	1	2	3	4	5	6	1	2	3
N06	63.0	68.5	56.9	73.1	71.0	76.5	77.8	81.4	80.4	54.1	52.2	--
007	50.7	49.9	47.5	60.6	63.7	75.6	64.4	75.2	69.5	53.5	48.9	50.5
Q01	44.8	43.0	49.8	57.1	52.7	60.3	60.0	59.2	59.4	49.9	54.0	48.1
Q03	55.6	53.0	55.7	61.9	79.1	75.5	76.8	70.2	78.2	60.3	65.1	62.8
403	44.5	47.4	44.5	63.7	70.9	69.4	72.2	74.9	70.8	51.5	58.6	57.8
411	42.9	41.3	41.9	52.1	59.8	56.4	52.3	51.3	51.2	41.4	38.8	37.8
$\bar{X}$	50.2	50.5	49.4	61.4	66.2	68.9	67.2	68.7	68.3	51.8	52.9	51.4
S.E.	3.2	4.0	2.5	2.9	3.9	3.6	4.1	4.6	4.6	2.5	3.7	4.3

Table XXVI - Autocorrelation coefficients derived from the REM density data during pre-deprivation, deprivation and post-deprivation.

Table XXVI

	Pre-Deprivation		Deprivation		Post-Deprivation	
	$\bar{r}$	S	$\bar{r}$	S	$\bar{r}$	S
N06	-0.05	0.90	0.17	0.64	-0.31	0.53
007	-0.19	1.01	0.41	0.67	-0.42	0.76
Q01	-0.10	0.95	0.03	0.67	-0.37	0.94
Q03	-0.28	1.04	0.20	0.70	0.03	0.76
403	-0.18	1.02	0.05	0.71	0.27	0.58
411	0.53	0.41	0.01	0.67	-0.37	0.87
$\bar{X}$	-0.05		0.15		-0.20	
S.E.	0.12		0.06		0.12	

Figure 1 - Average percent time spent in active waking, quiet waking, slow wave sleep and REM sleep for six cats during pre-deprivation, deprivation and post-deprivation (Mean ± S.E.).

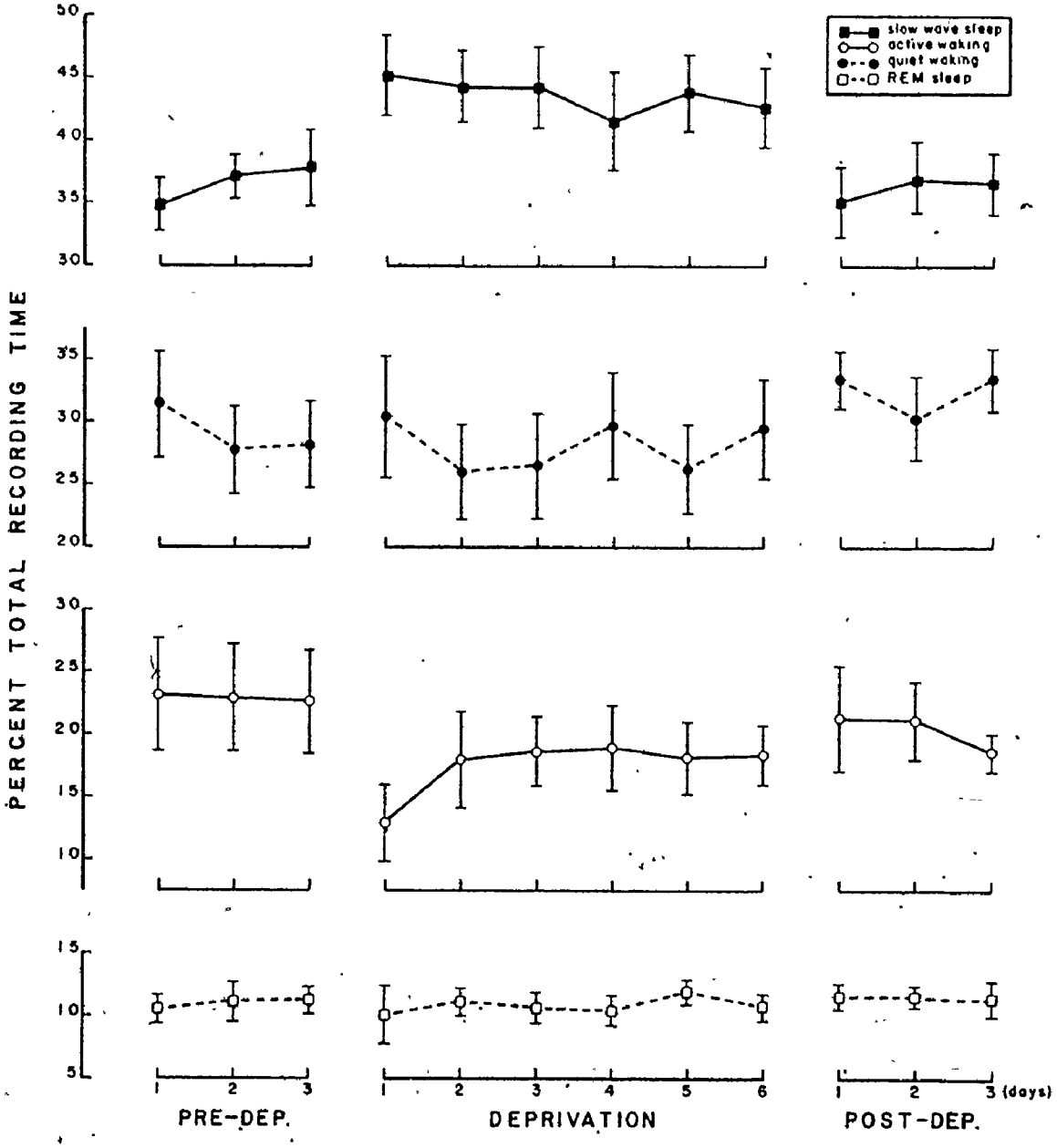

slow wave sleep
active waking
quiet waking
REM sleep
PERCENT TOTAL RECORDING TIME
50
45
40
35
30
35
30
25
20
30
25
20
15
10
15
10
5
1
2
3
1
2
3
4
5
6
1
2
3 (days)
PRE-DEP.
DEPRIVATION
POST-DEP.

Figure 2 - Percent time spent awake, in slow wave sleep and REM sleep for Cat N06 during pre-deprivation, deprivation and post-deprivation.

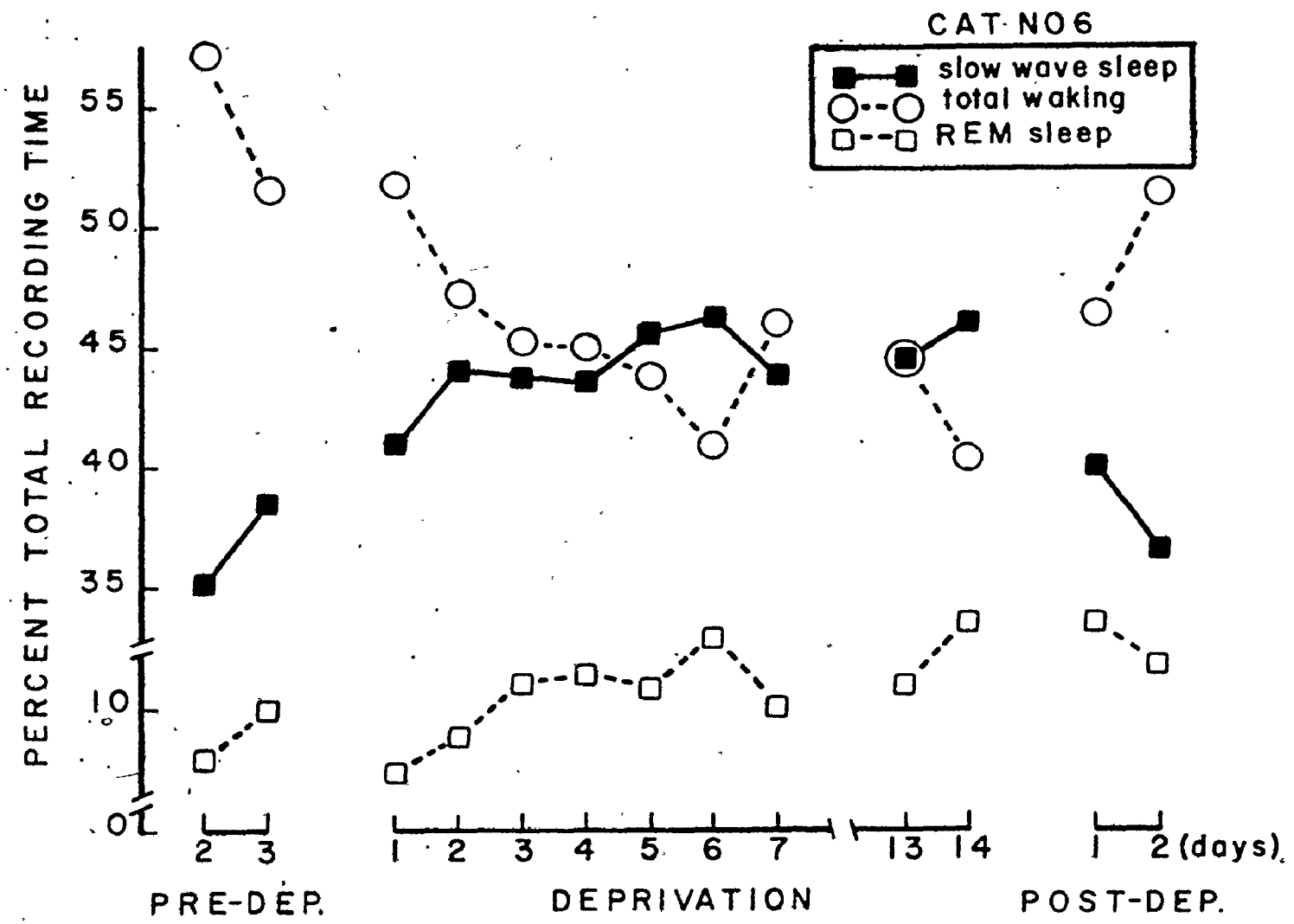

CAT NO6
slow wave sleep
total waking
REM sleep
PERCENT TOTAL RECORDING TIME
55
50
45
40
35
10
0
2 3
1 2 3 4 5 6 7
13 14
1 2 (days)
PRE-DEP.
DEPRIVATION
POST-DEP.

Figure 3 - Percent time spent in active waking, activity, quiet waking, slow wave sleep and REM sleep for Cat 007 during pre-deprivation, deprivation and post-deprivation.

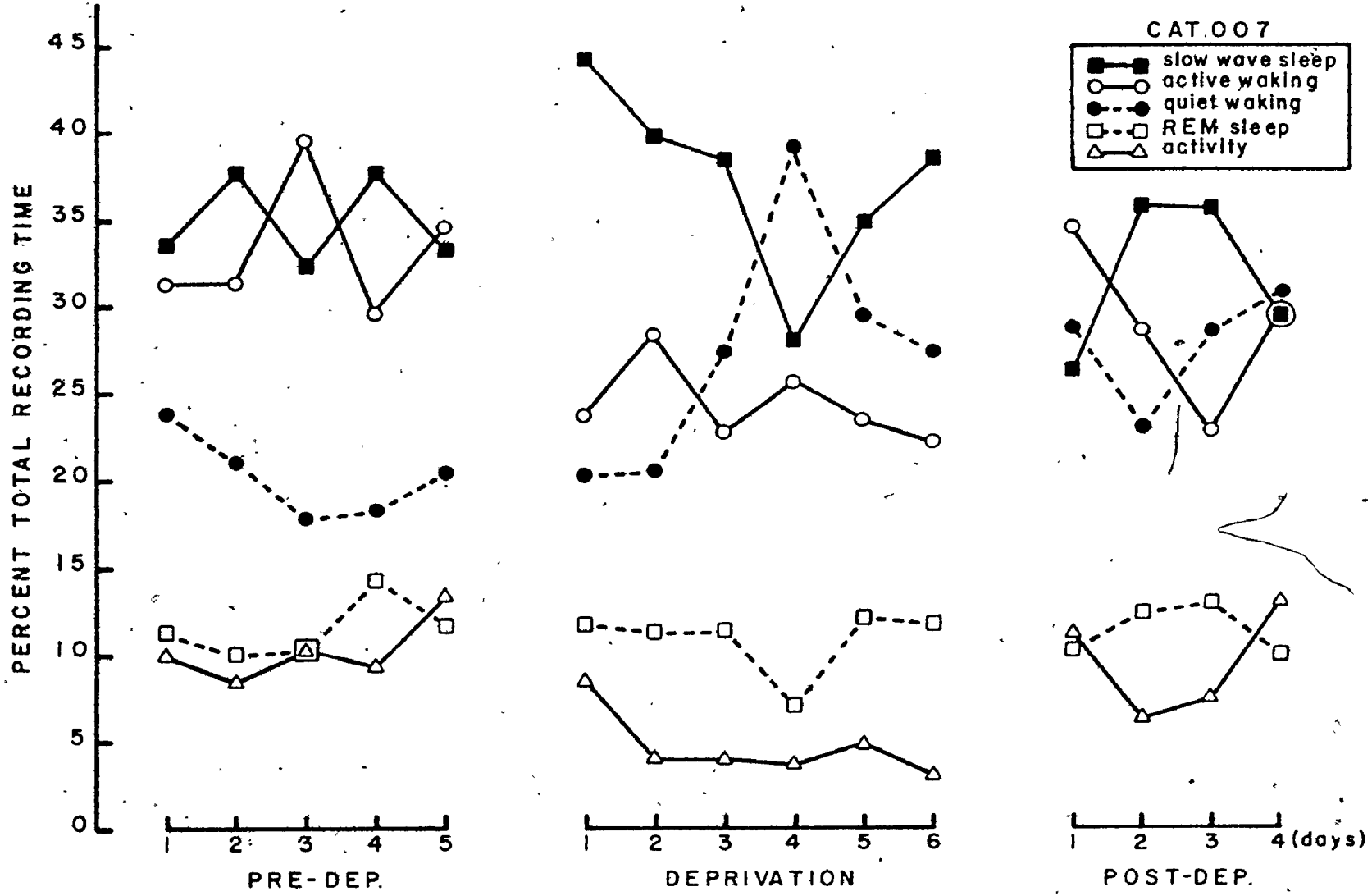

CAT.007
slow wave sleep
active waking
quiet waking
REM sleep
activity
PERCENT TOTAL RECORDING TIME
0
5
10
15
20
25
30
35
40
45
1 2 3 4 5
PRE-DEP.
1 2 3 4 5 6
DEPRIVATION
1 2 3 4 (days)
POST-DEP.

Figure 4 - Percent time spent in active waking, quiet waking, slow wave sleep and REM sleep for Cat Q01 during pre-deprivation, deprivation and post-deprivation.

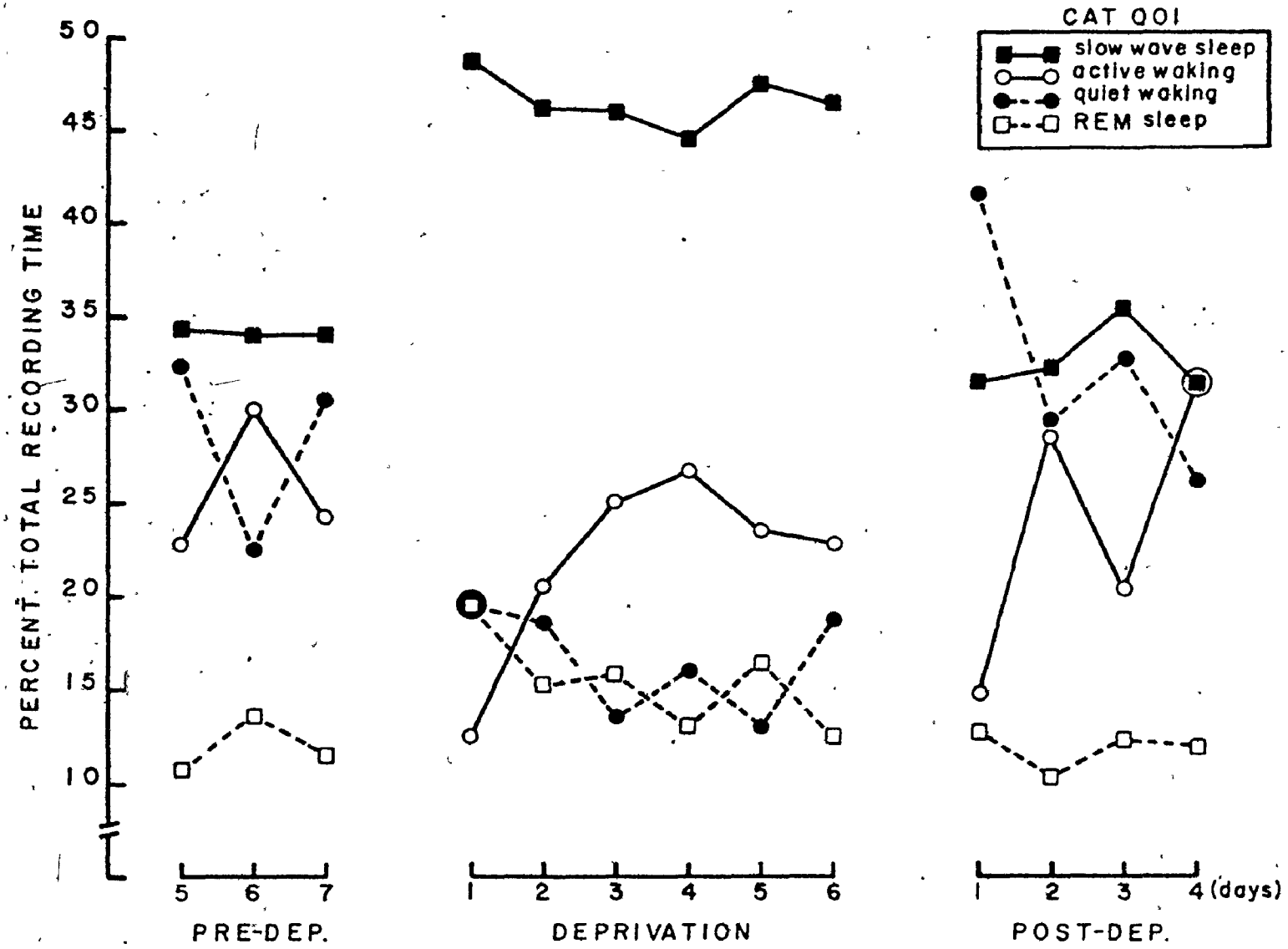

CAT 001
slow wave sleep
active waking
quiet waking
REM sleep
PERCENT TOTAL RECORDING TIME
50
45
40
35
30
25
20
15
10
5 6 7
PRE-DEP.
1 2 3 4 5 6
DEPRIVATION
1 2 3 4 (days)
POST-DEP.

Figure 5 - Percent time spent in active waking, quiet waking, slow wave sleep and REM sleep for Cat Q03 during pre-deprivation, deprivation and post-deprivation.

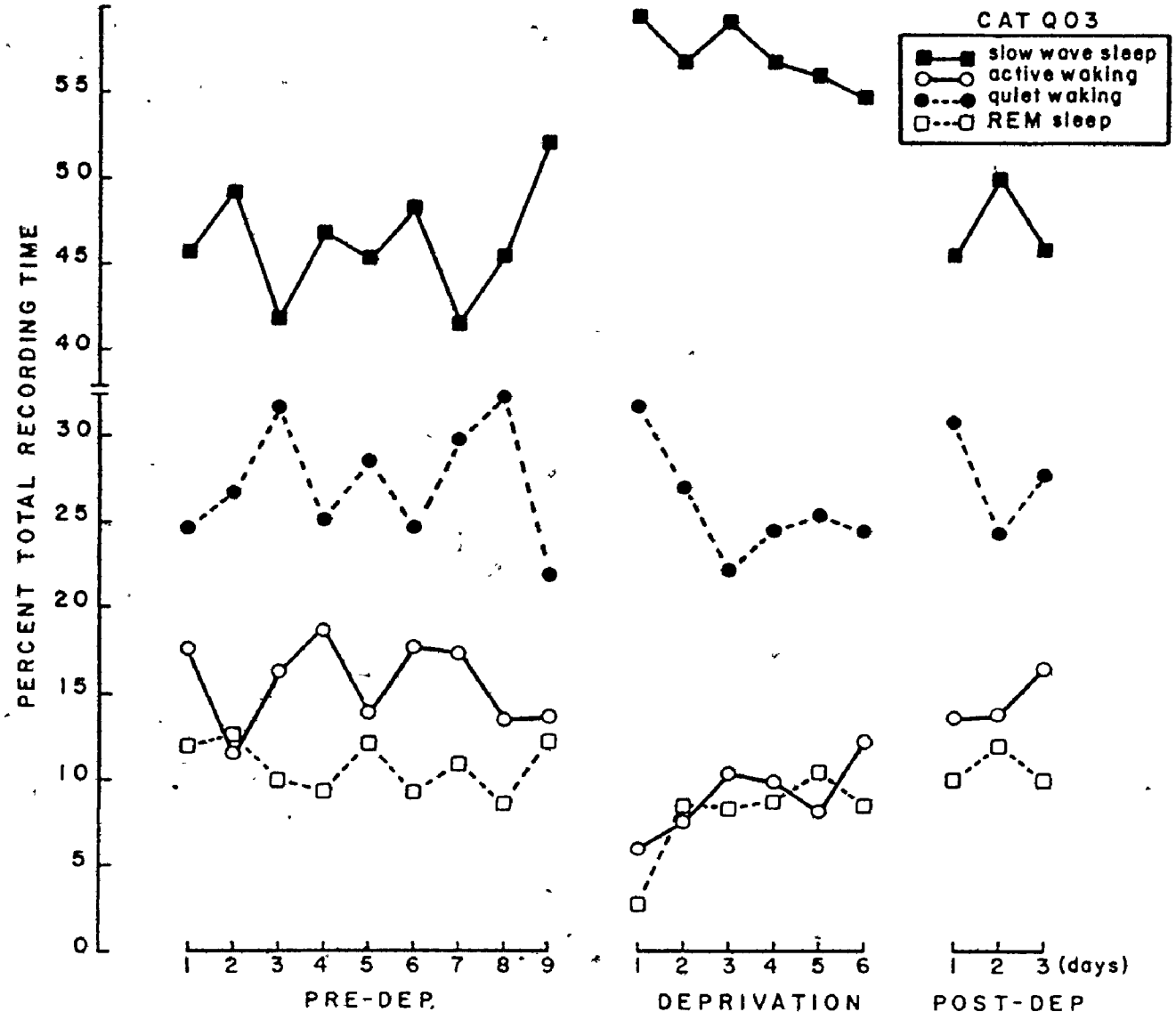

CAT Q03
slow wave sleep
active waking
quiet waking
REM sleep
PERCENT TOTAL RECORDING TIME
55
50
45
40
30
25
20
15
10
5
0
1 2 3 4 5 6 7 8 9
PRE-DEP.
1 2 3 4 5 6
DEPRIVATION
1 2 3 (days)
POST-DEP

Figure 6 - Percent time spent in active waking, quiet waking, slow wave sleep and REM sleep for Cat 403 during pre-deprivation, deprivation and post-deprivation.

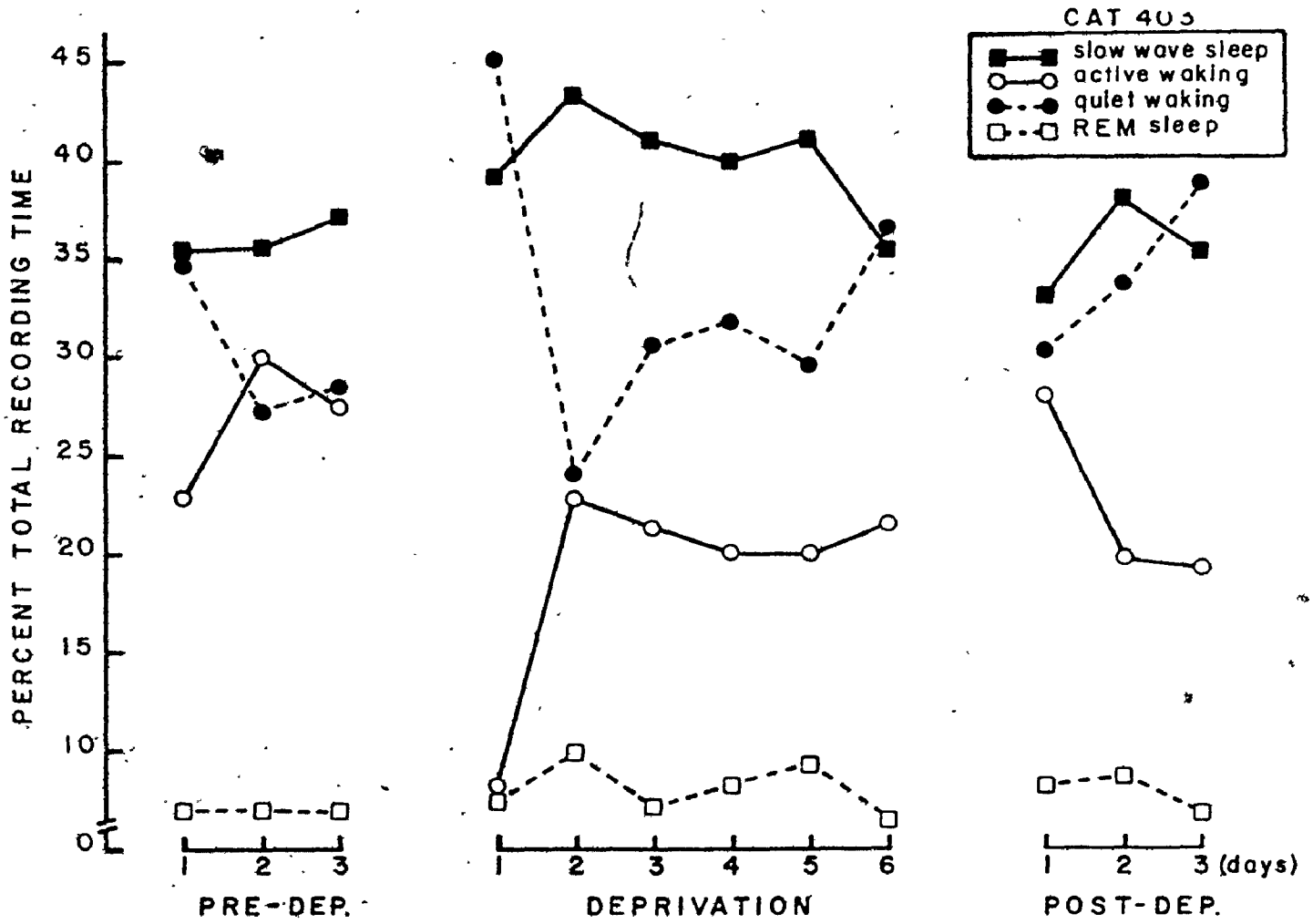
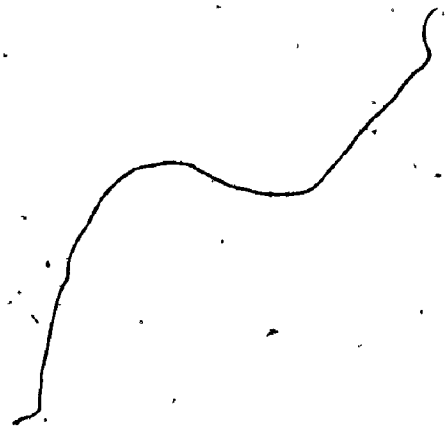

CAT 405
slow wave sleep
active waking
quiet waking
REM sleep
PERCENT TOTAL RECORDING TIME
45
40
35
30
25
20
15
10
0
1 2 3
PRE-DEP.
1 2 3 4 5 6
DEPRIVATION
1 2 3 (days)
POST-DEP.

Figure 7 - Percent time spent in active waking, quiet waking, slow wave sleep and REM sleep for Cat 411 during pre-deprivation, deprivation and post-deprivation.



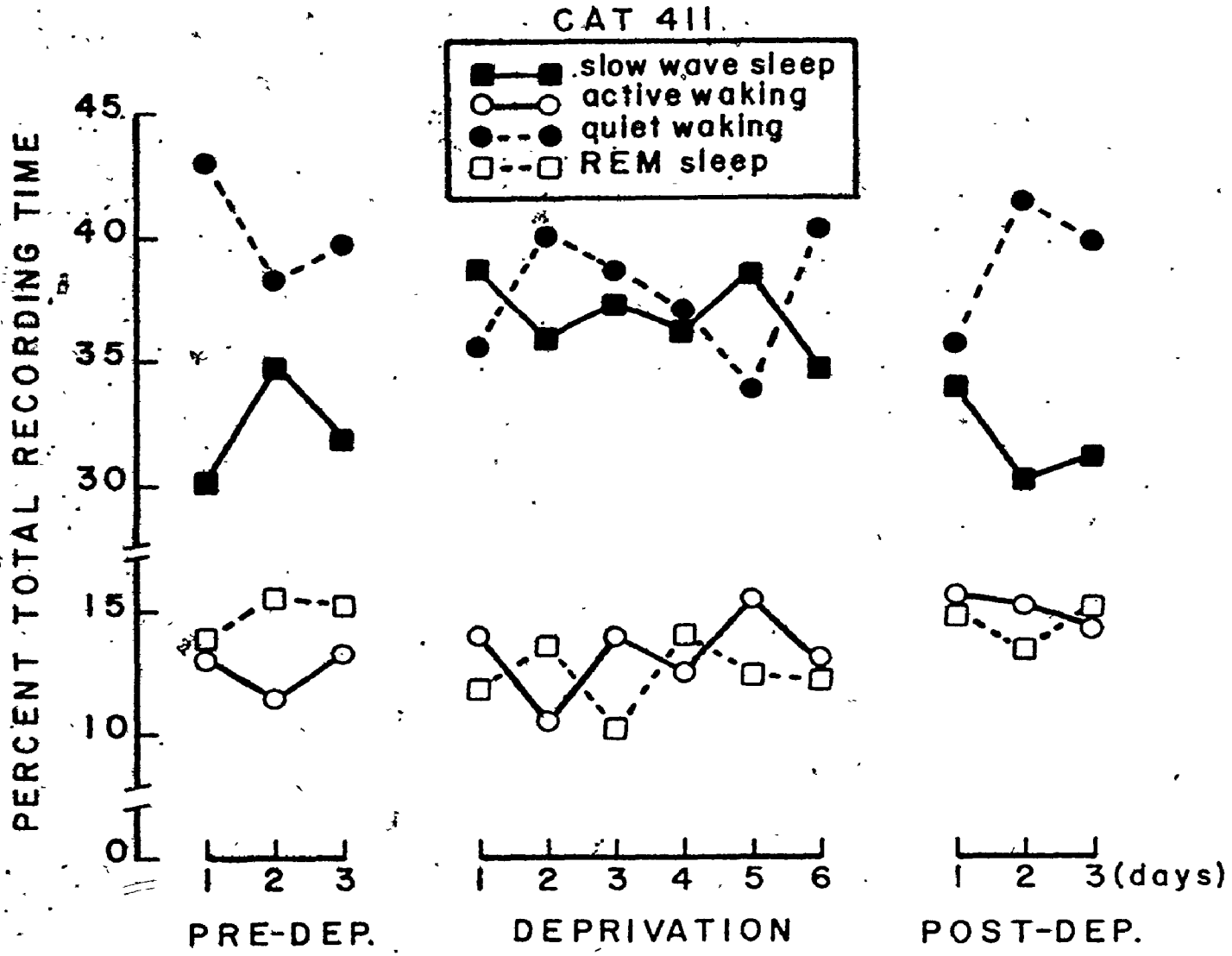

CAT 411
slow wave sleep
active waking
quiet waking
REM sleep
PERCENT TOTAL RECORDING TIME
45
40
35
30
15
10
0
1 2 3
1 2 3 4 5 6
1 2 3 (days)
PRE-DEP.
DEPRIVATION
POST-DEP.

Figure 8 - Sleep-waking pattern of Cat Q01 on pre-deprivation day seven and deprivation days one and three (A = Active waking, Q = Quiet waking, S = Slow wave sleep, R = REM sleep).

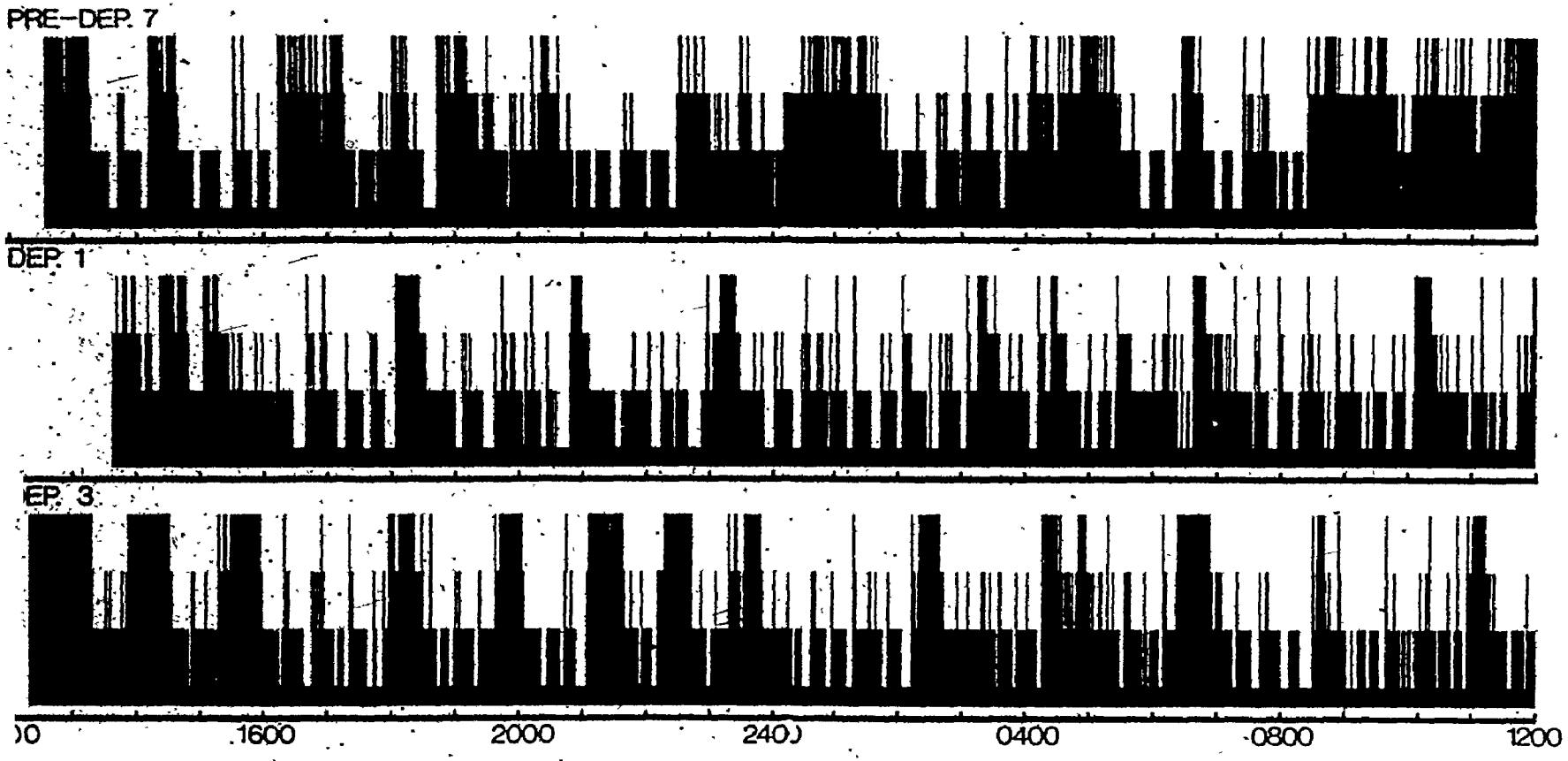

PRE-DEP. 7
DEP. 1
EP. 3
)0
1600
2000
240J
0400
0800
1200

Figure 9 - Sleep-waking pattern of Cat Q03 on pre-deprivation day five and deprivation day three (A = Active waking, Q = Quiet waking, S = Slow wave sleep, R = REM sleep).

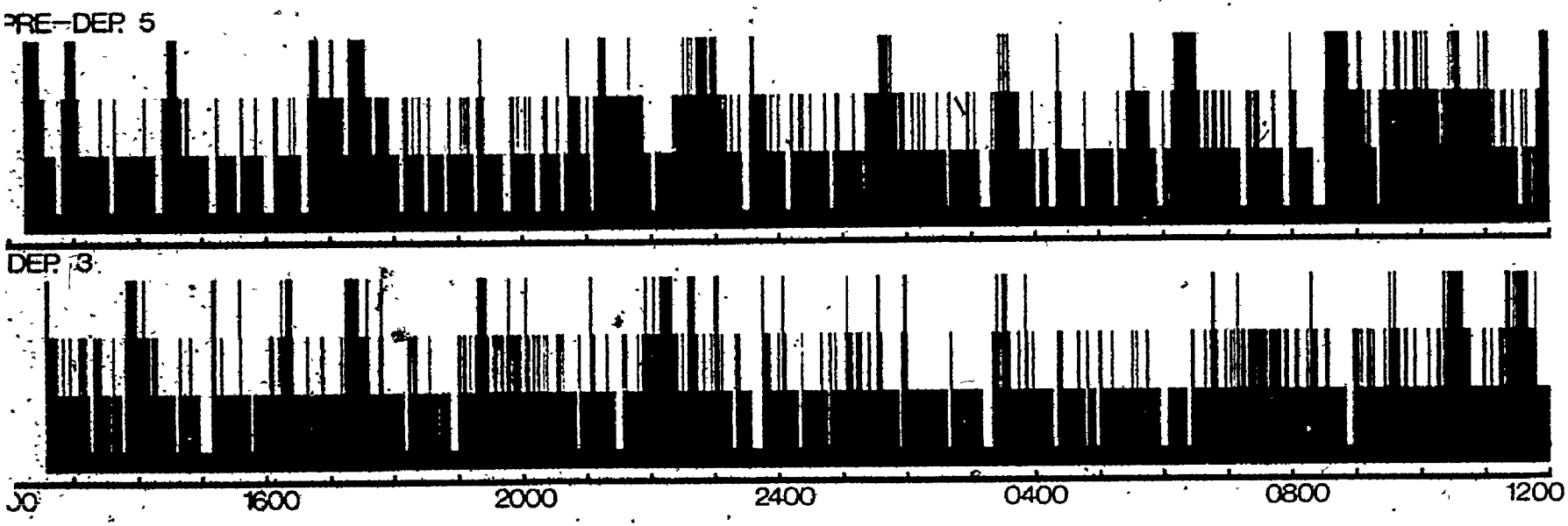

PRE-DEP. 5
DEP. 3
1600
2000
2400
0400
0800
1200

Figure 10 - Percent time spent in slow wave sleep for successive eight hour blocks of time during pre-deprivation, deprivation and post-deprivation.

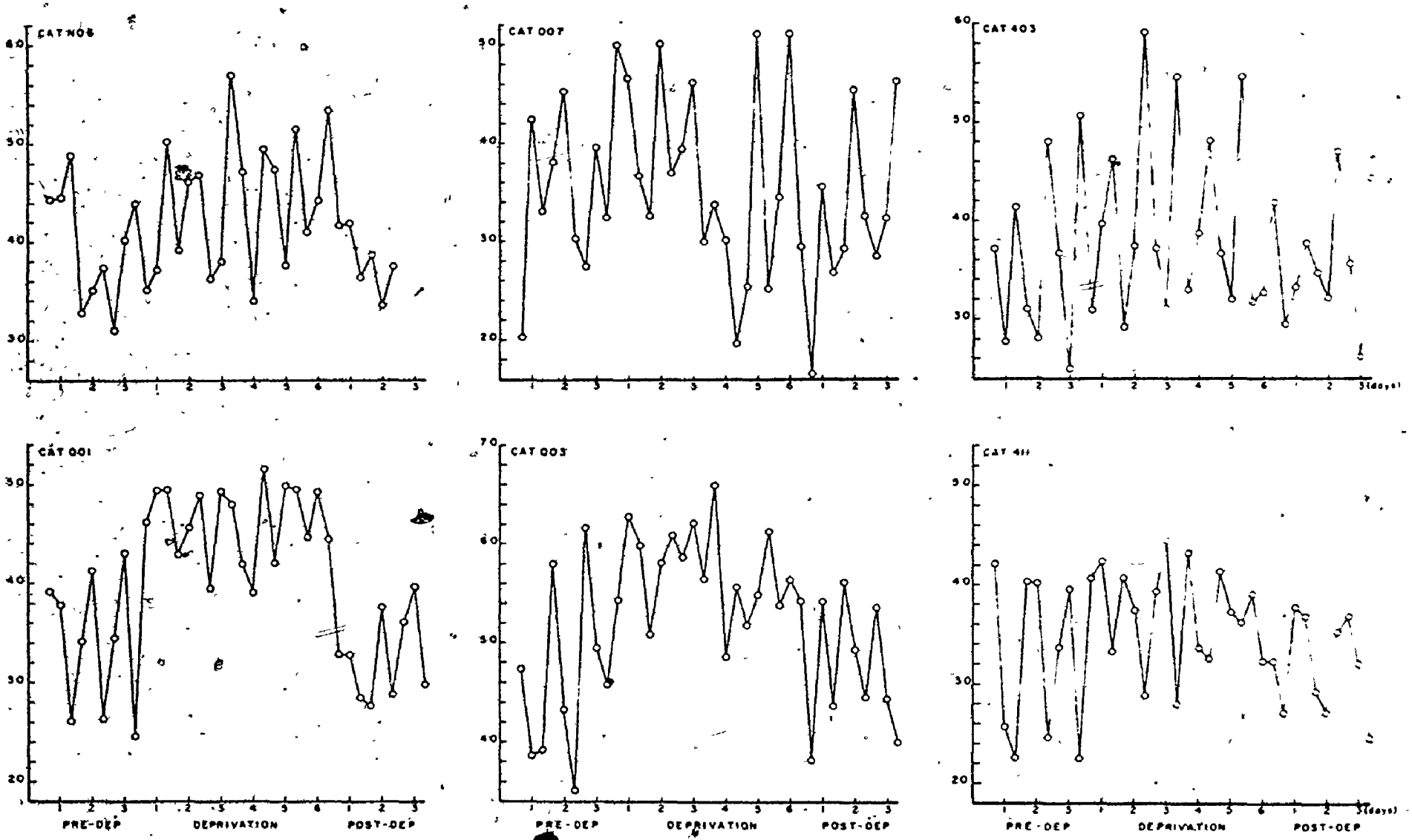
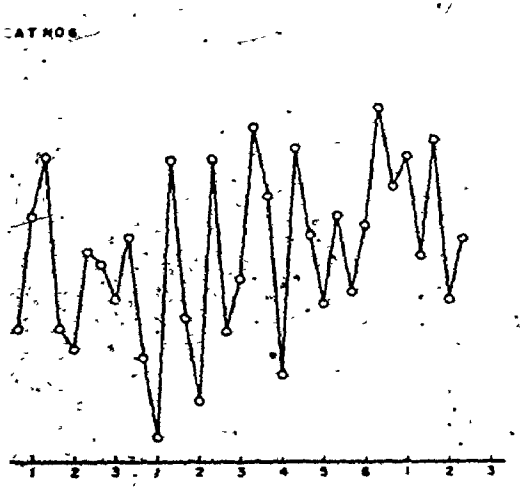
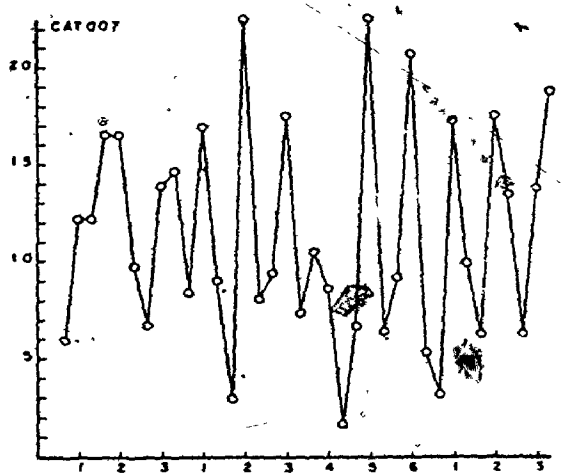
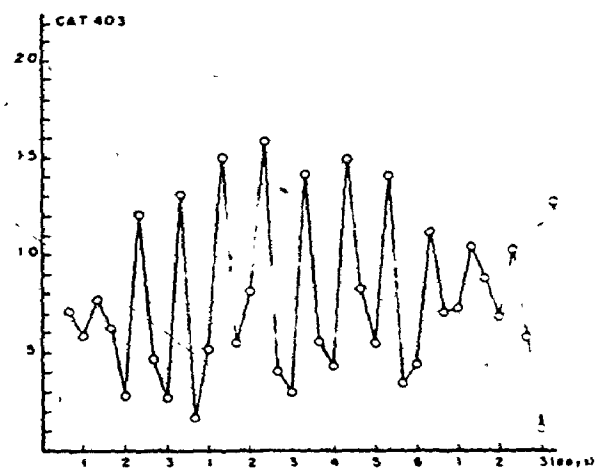
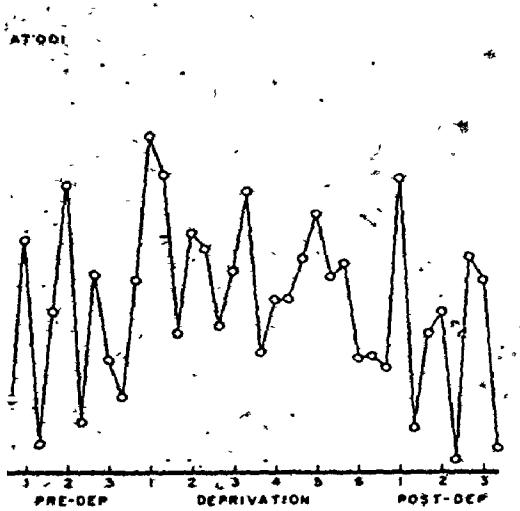

CAT 008
CAT 007
CAT 403
CAT 001
CAT 003
CAT 411
PRE-DEP
DEPRIVATION
POST-DEP
PRE-DEP
DEPRIVATION
POST-DEP
PRE-DEP
DEPRIVATION
POST-DEP

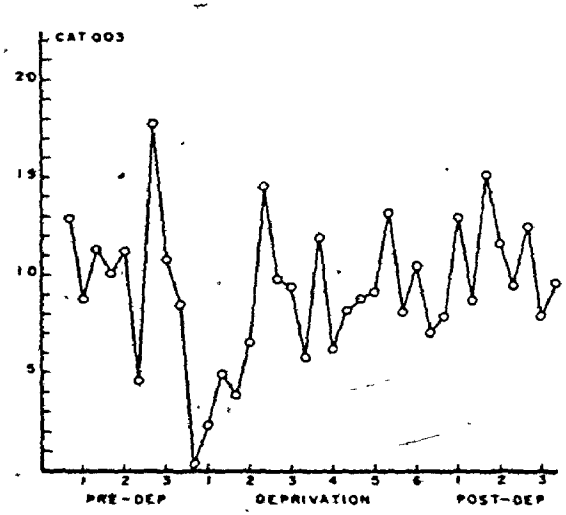
Figure 11 - Percent time spent in REM sleep for successive eight hour blocks of time during pre-deprivation, deprivation and post-deprivation.




CAT 007


CAT 403


PRE-DEP
DEPRIVATION
POST-DEP


CAT 003
PRE-DEP
DEPRIVATION
POST-DEP

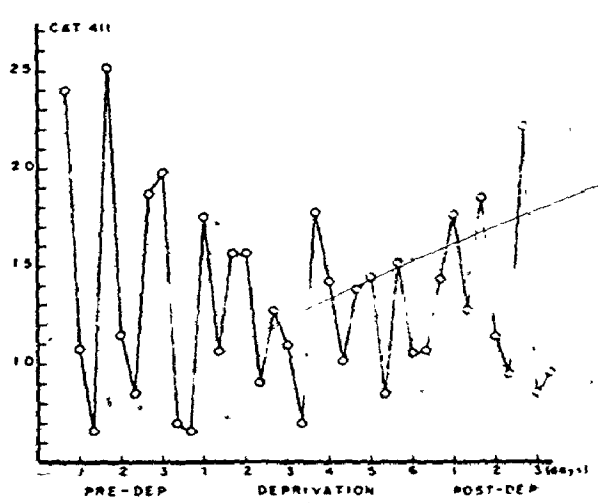

CAT 411
PRE-DEP
DEPRIVATION
POST-DEP

Figure 12 - Standard scores of percent time spent in slow wave sleep for successive eight hour blocks of time during pre-deprivation, deprivation and post-deprivation.

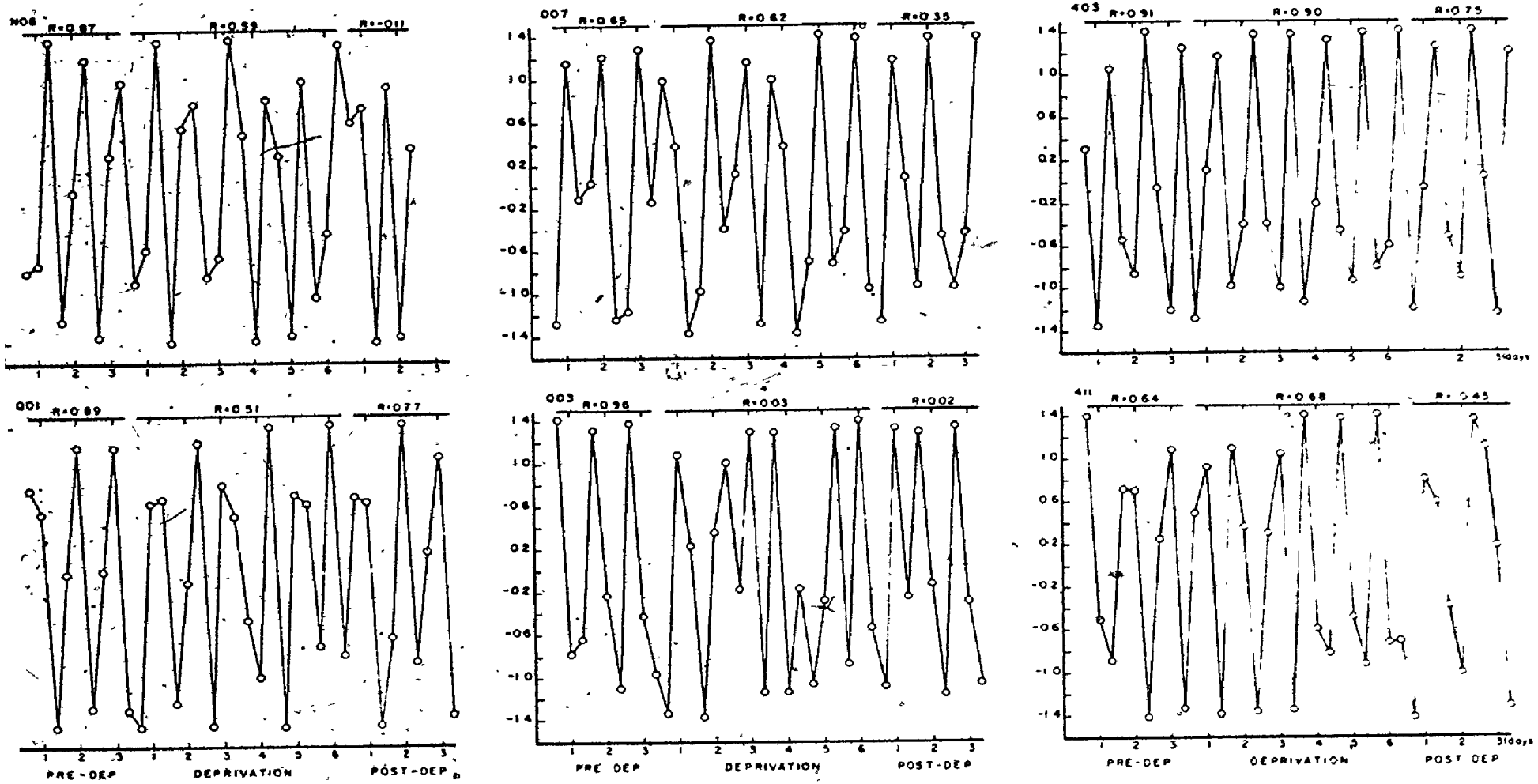

N08
R=0.87
R=0.52
R=-011
007
R=0.65
R=0.62
R=0.35
403
R=0.91
R=0.90
R=0.75
001
R=0.89
R=0.51
R=0.77
003
R=0.96
R=0.03
R=0.02
411
R=0.64
R=0.68
R=0.45
PRE-DEP
DEPRIVATION
POST-DEP
PRE DEP
DEPRIVATION
POST-DEP
PRE-DEP
DEPRIVATION
POST DEP

Figure 13 - Standard scores of percent time spent in REM sleep for successive eight hour blocks of time during pre-deprivation, deprivation and post-deprivation.

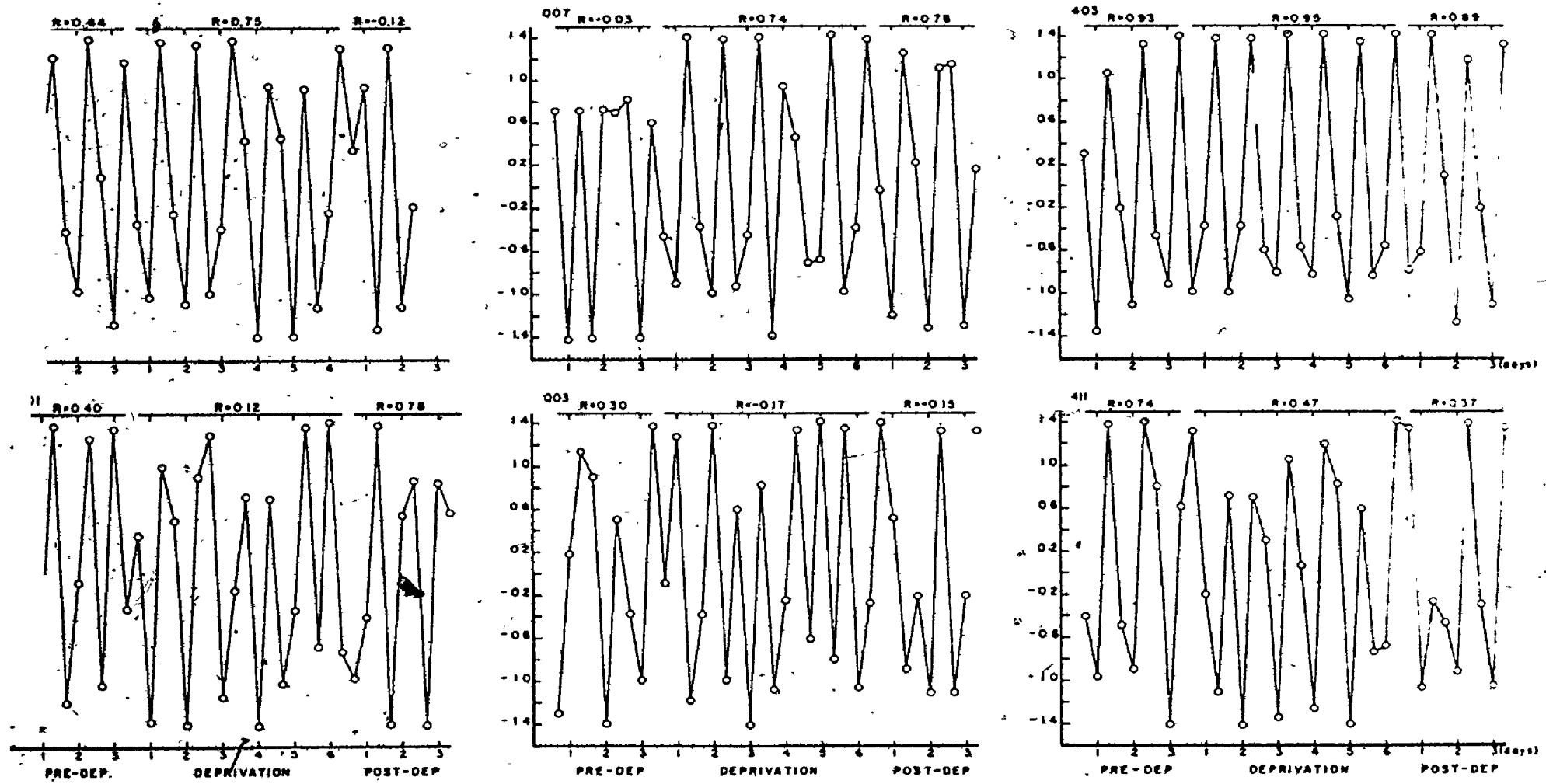

R=0.84
R=0.75
R=-0.12
007
R=-0.03
R=0.74
R=0.78
403
R=0.93
R=0.95
R=0.89
R=0.40
R=0.12
R=0.78
003
R=0.30
R=-0.17
R=-0.15
411
R=0.74
R=0.47
R=0.37
PRE-DEP.
DEPRIVATION
POST-DEP
PRE-DEP
DEPRIVATION
POST-DEP
PRE-DEP
DEPRIVATION
POST-DEP
(days)
(days)

Figure 14 - Average REM density for all subjects during pre-deprivation, deprivation and post-deprivation (Mean ± S.E.).

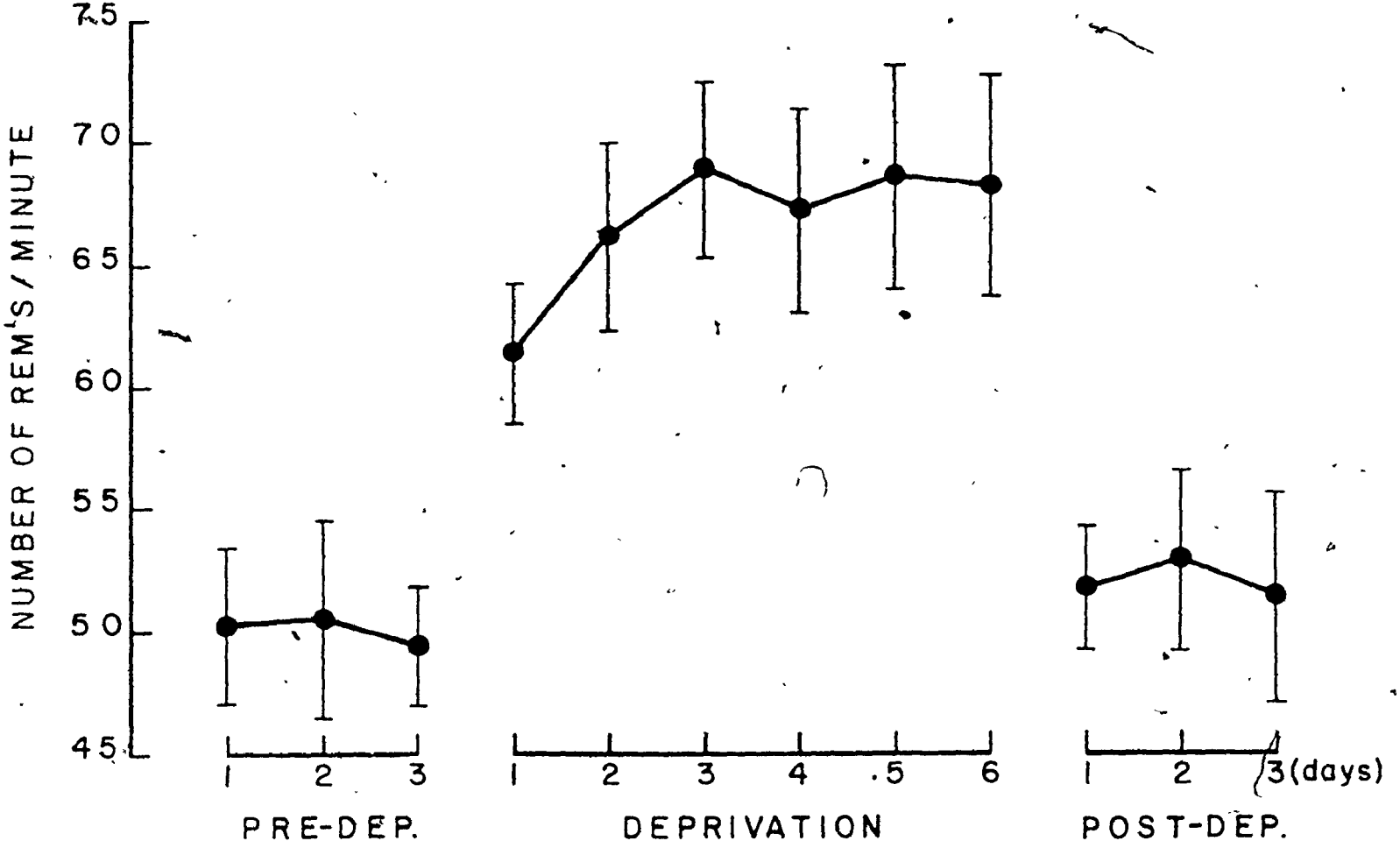

NUMBER OF REM'S / MINUTE
75
70
65
60
55
50
45
1
2
3
PRE-DEP.
1
2
3
4
.5
6
DEPRIVATION
1
2
3(days)
POST-DEP.

Figure 15 - Average REM density for Cats N06 and 007 during pre-deprivation, deprivation and post-deprivation.

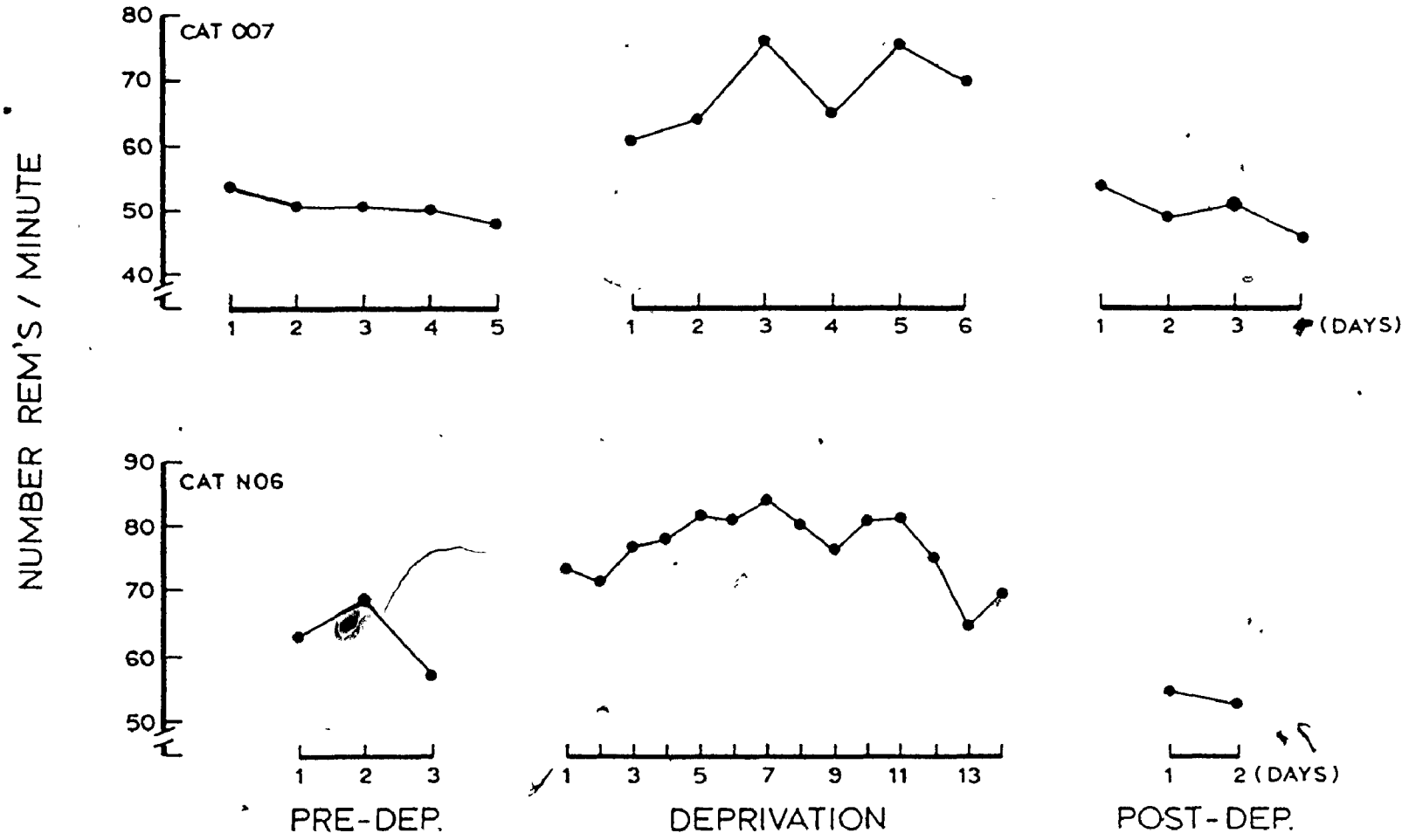

NUMBER REM'S / MINUTE
CAT 007
80
70
60
50
40
1 2 3 4 5
1 2 3 4 5 6
1 2 3
(DAYS)
CAT N06
90
80
70
60
50
1 2 3
1 3 5 7 9 11 13
1 2 (DAYS)
PRE-DEP.
DEPRIVATION
POST-DEP.

Figure 16 - Average REM density for Cats Q01 and Q03 during pre-deprivation, deprivation and post-deprivation.

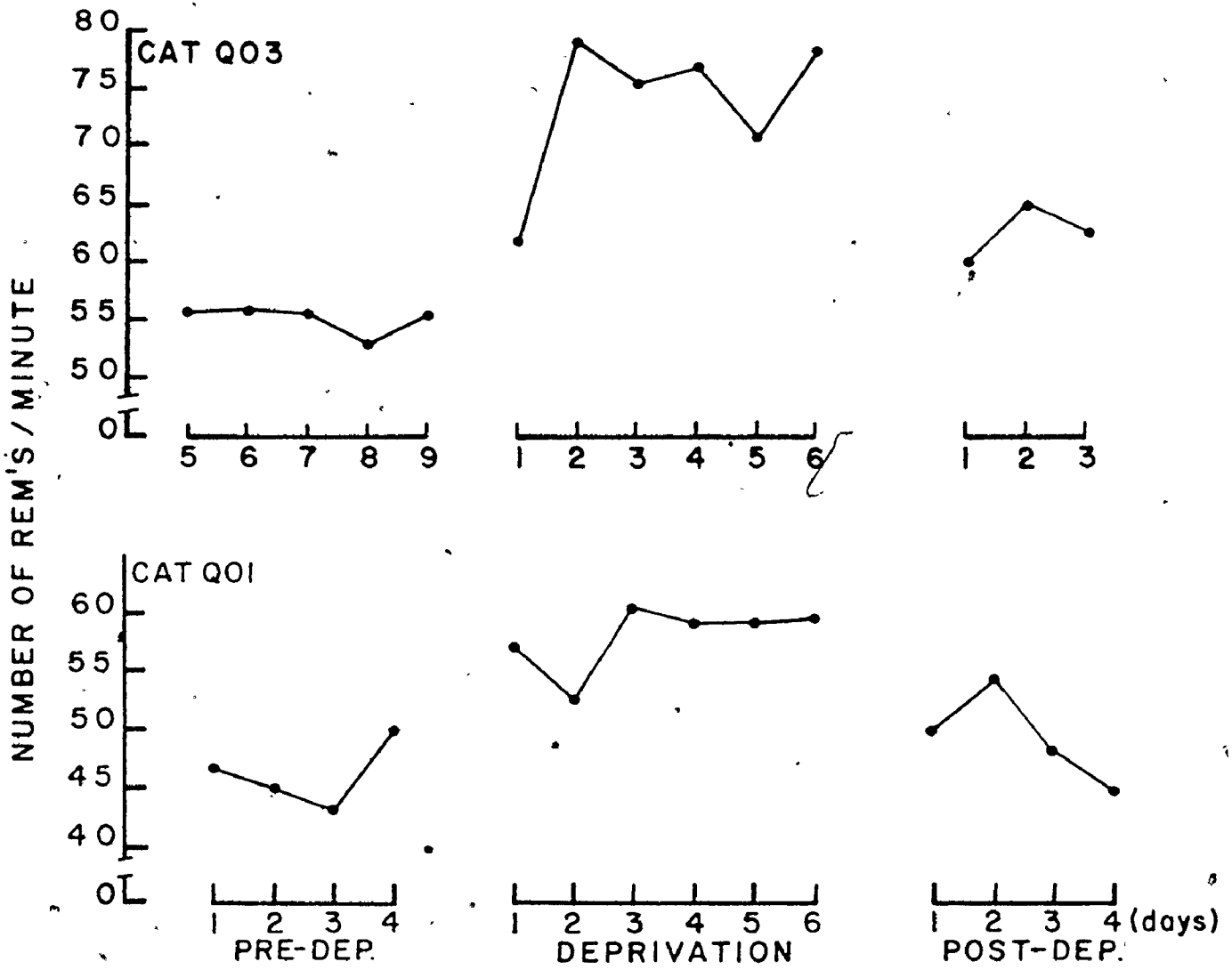

CAT Q03
CAT Q01
NUMBER OF REM'S / MINUTE
80
75
70
65
60
55
50
0
60
55
50
45
40
0
5 6 7 8 9
1 2 3 4 5 6
1 2 3
1 2 3 4
1 2 3 4 5 6
1 2 3 4 (days)
PRE-DEP.
DEPRIVATION
POST-DEP.

Figure 17 - Average REM density for Cats 403 and 411 during pre-deprivation, deprivation and post-deprivation.

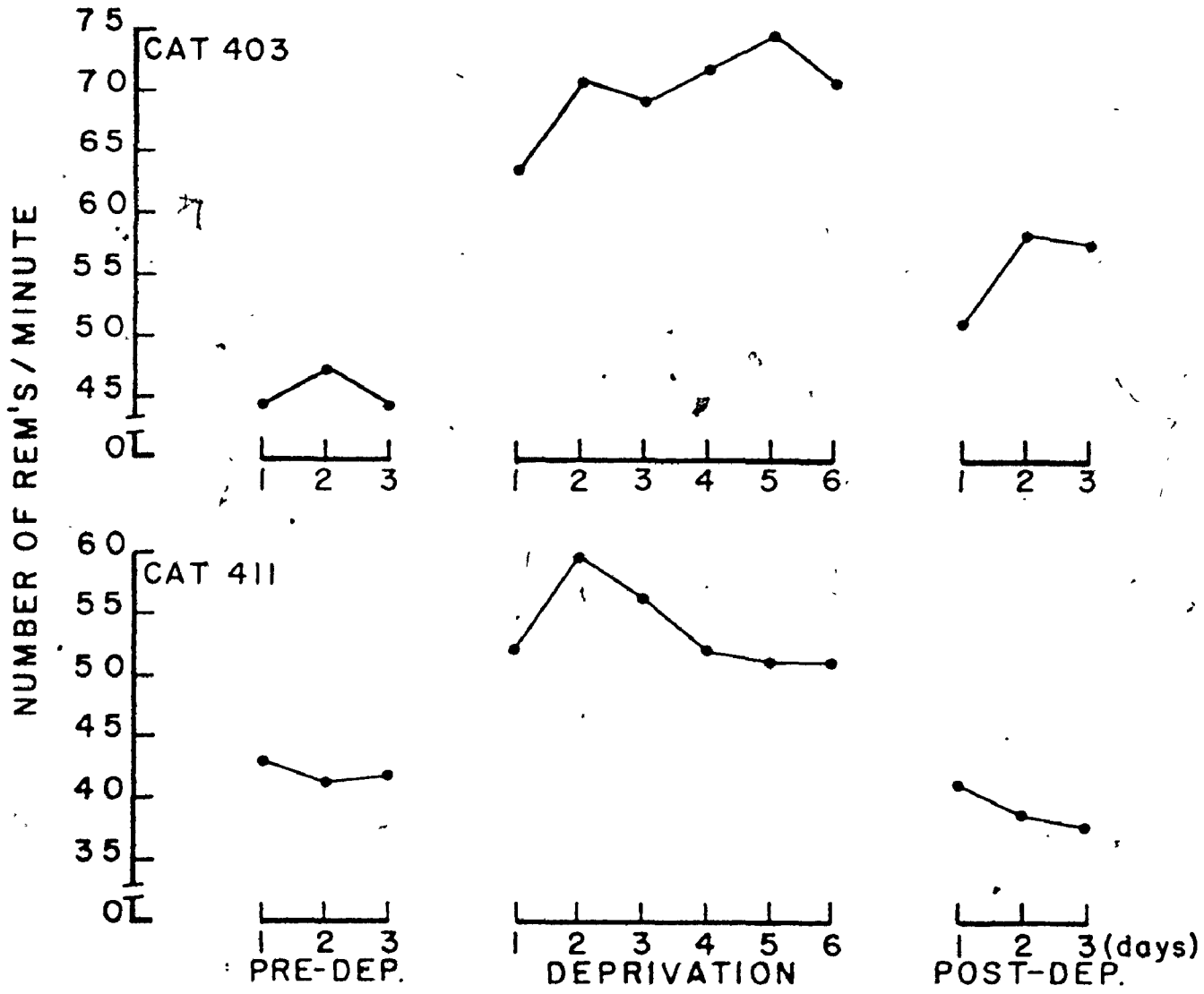

NUMBER OF REM'S / MINUTE
CAT 403
75
70
65
60
55
50
45
0
1 2 3
1 2 3 4 5 6
1 2 3
CAT 411
60
55
50
45
40
35
0
1 2 3
1 2 3 4 5 6
1 2 3 (days)
PRE-DEP.
DEPRIVATION
POST-DEP.

Figure 18 - Standard scores of REM density for successive eight hour blocks of time during pre-deprivation, deprivation and post-deprivation.

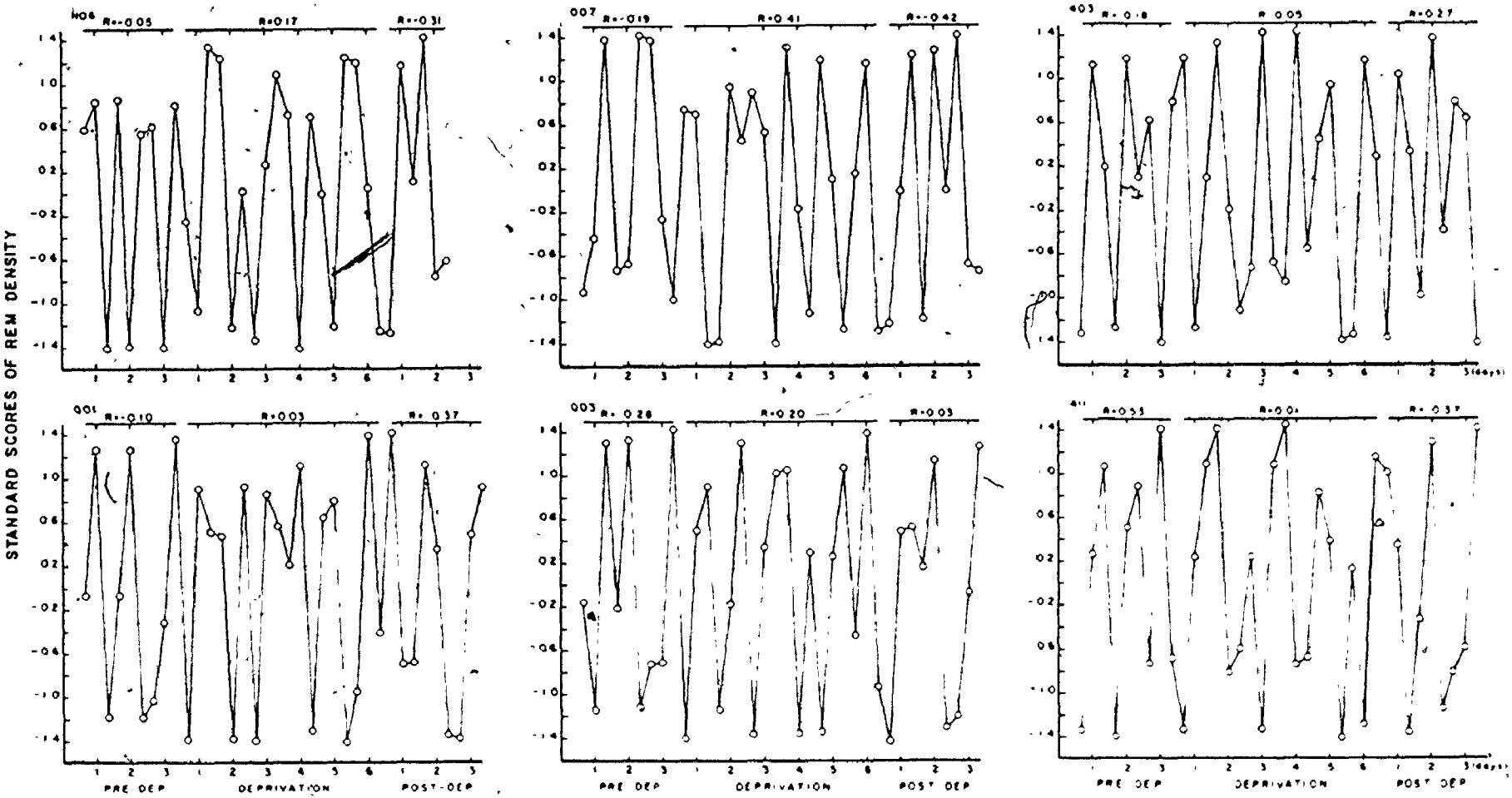

STANDARD SCORES OF REM DENSITY
PRE DEP
DEPRIVATION
POST-DEP
POST DEP
(days)

Figure 19 - Average REM density for eight human subjects during pre-deprivation, deprivation and post-deprivation (Mean ± S.E.).

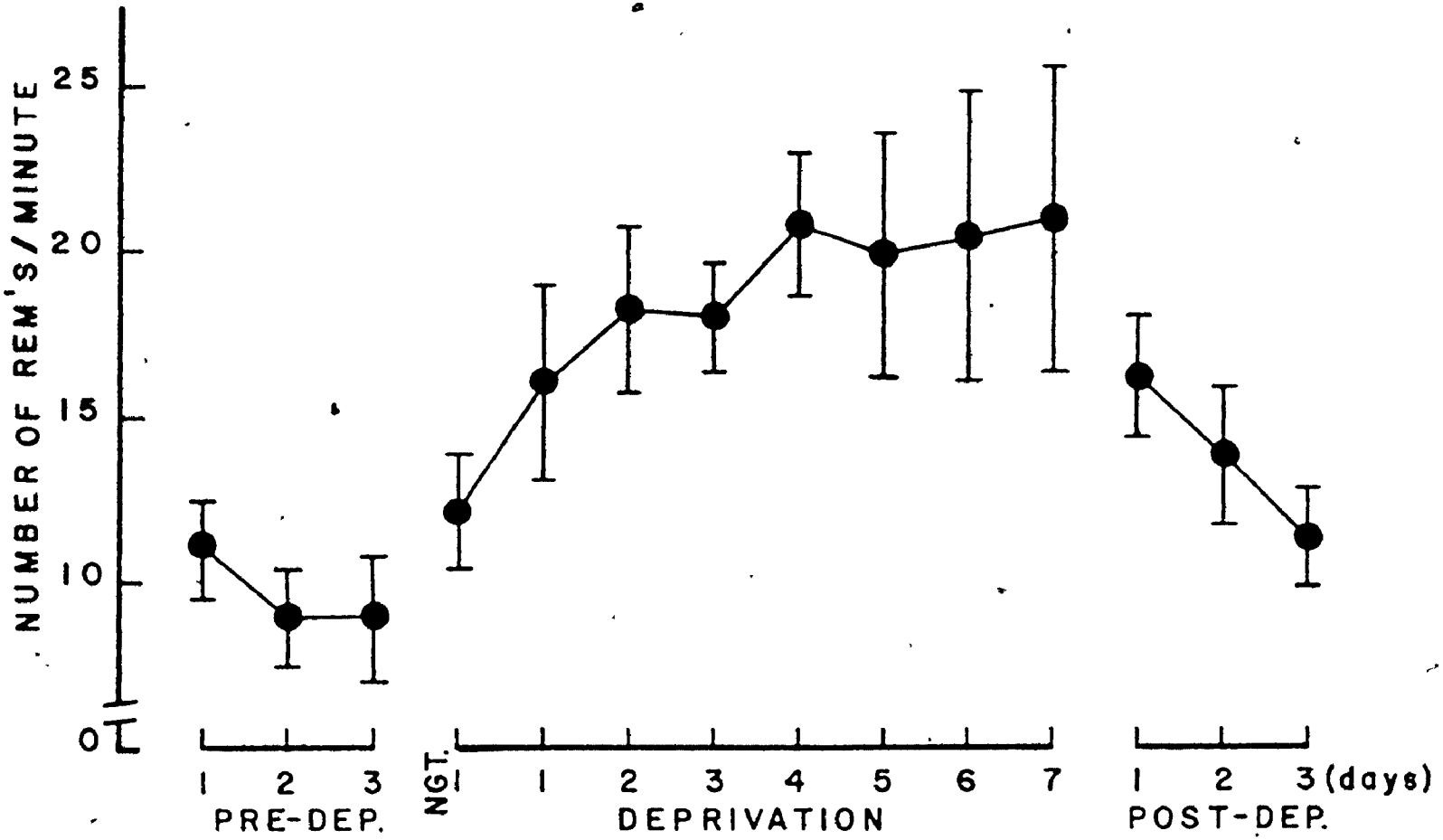
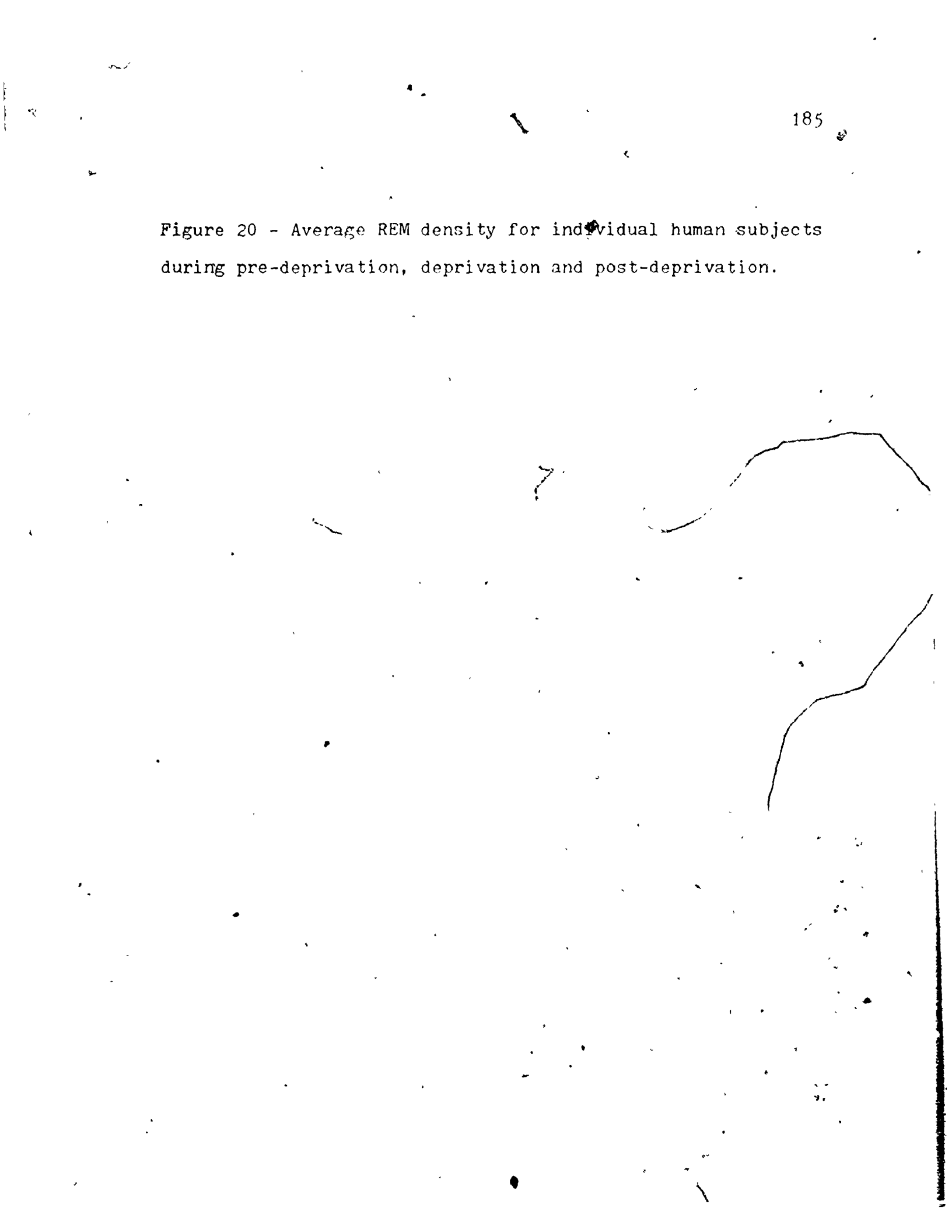

NUMBER OF REM'S/MINUTE
25
20
15
10
0
1 2 3
PRE-DEP.
NGT.
1 2 3 4 5 6 7
DEPRIVATION
1 2 3 (days)
POST-DEP.

Figure 20 - Average REM density for individual human subjects during pre-deprivation, deprivation and post-deprivation.



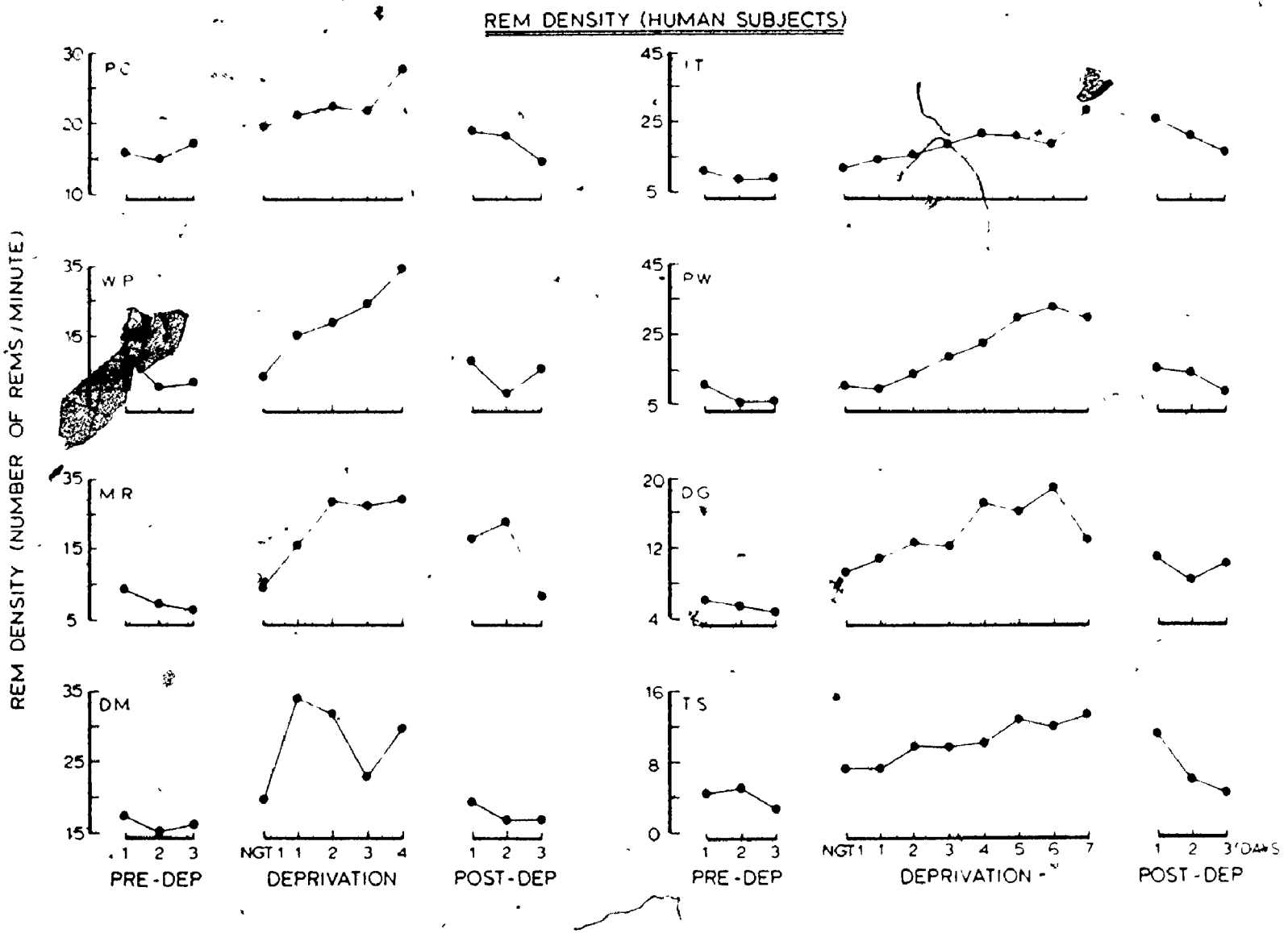
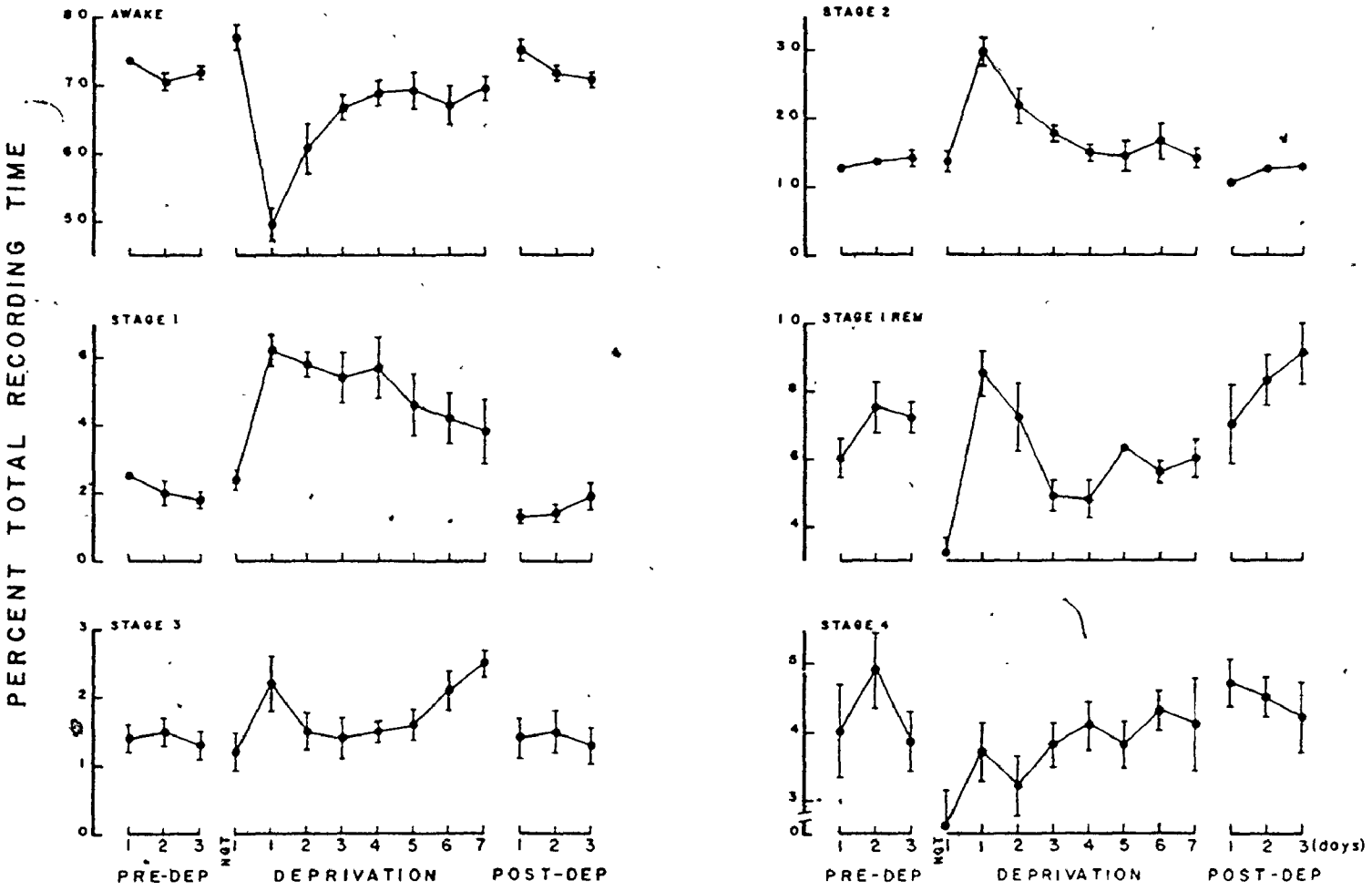

REM DENSITY (HUMAN SUBJECTS)
REM DENSITY (NUMBER OF REMS / MINUTE)
PC
IT
WP
PW
MR
DG
DM
TS
PRE-DEP
DEPRIVATION
POST-DEP
PRE-DEP
DEPRIVATION
POST-DEP
NGT1
DAYS

Figure 21 - Average percent time spent awake and in different sleep stages for eight human subjects during pre-deprivation, deprivation and post-deprivation (Mean ± S.E.).


PERCENT TOTAL RECORDING TIME
AWAKE
STAGE 1
STAGE 3
STAGE 2
STAGE 1 REM
STAGE 4
PRE-DEP
DEPRIVATION
POST-DEP
NOT
1 2 3 4 5 6 7
1 2 3 (days)

EXPERIMENT II

In experiment I, REM density was found to increase significantly above baseline levels during sensory deprivation. Since REMs are typically accompanied by PGO waves and are thought to be initiated as a result of activation of oculomotor neurons by the pontine generator, it is possible that the REM density effect is due to an increase in the frequency at which PGO waves are generated during sensory deprivation. This hypothesis was tested in the present experiment by calculating the average daily PGO density (i.e. mean number of PGO waves per minute of REM sleep) for two cats during pre-deprivation, deprivation and post-deprivation. Since other characteristics of the pontine generator might also have changed during sensory deprivation, a number of other measurements related to pontine generator function were made. Thus, time interval histograms of PGO wave occurrence and the temporal relationship between the occurrence of PGO waves on one side of the brain (left LGN) and the other (right LGN), between one structure (pontine reticular formation) and another (LGN), were described during pre-deprivation and deprivation.

An alternative mechanism that might underlie the increased REM density during sensory deprivation is suggested by the observation of Brooks (1968b) that under normal con-

ditions not every PGO wave is accompanied by a REM, at least, as measured by the electro-oculographic technique. The fact that the coupling between the occurrence of PGO waves and the electromyographic response of the extraocular muscle appears to be near perfect (Cespuglio et. al., 1976; Gadea-Ciria, 1972). however, suggests that a certain proportion of eye muscle contractions are normally too weak to elicit a detectable REM. Thus, the REM density effect observed in Experiment I may simply be a REM amplitude effect where previously undetectable eye movements during pre-deprivation become suprathreshold (i.e. threshold set by an amplitude criterion) countable eye movements during deprivation. In order to test this hypothesis, the average EOG amplitude was calculated on selected days during pre-deprivation and deprivation for four cats and the relationship between REM amplitude and REM density was determined.

Since several laboratories have reported that the frequency of hippocampal theta is more rapid during REM sleep when REMs (Sakai et. al., 1973; Sano et. al., 1973) or muscle twitches (Robinson, Kramis & Vanderwolf, 1977) are present than when they are absent, an analysis of the relationship between REM density, PGO density and hippocampal theta was made during pre-deprivation and deprivation. Sensory deprivation offers a unique opportunity to study these relationships since, if REM density and theta frequency

are positively correlated, as has been reported, then one would expect that the average frequency of theta during pre-deprivation would be less than during deprivation. Furthermore, previous studies have not taken into account the fact that the neural generator of REMs and gross motor activity during REM sleep can be active (as indicated by the occurrence of PGO waves) without the presence of detectable movement. In view of the recent emphasis on the relationship between hippocampal theta and movement (Vanderwolf, 1971), it would be informative to know whether theta frequency is higher during the occurrence of PGO and REMs than it is during the occurrence of PGO waves alone.

METHOD

PGO Wave Analysis

Two animals (Cat 403 and Cat 411) used in Experiment I, had electrodes in the LGN and pontine reticular formation which were optimally located to record clearly identifiable, easy to count PGO waves (Wave shape characteristics described by Bizzi & Brooks(1963) and Brooks (1967) were used to identify PGO waves). Since polygraphic records were taken continuously during Experiment I, it was possible to obtain a count of the total number of PGO waves generated during REM sleep on each pre-deprivation, deprivation and post-deprivation day. From this number the average daily PGO density was calculated.

Composite time interval histogram (TIH) distributions for PGO waves were obtained by using the PDP-8e time interval histogram program (DEC-LB-U40-B-D, Digital Equipment Corporation) to analyze magnetic tape records of PGO waves. The Schmidt gate level on the computer was set to minimize false detection by triggering on the first large amplitude component of the PGO wave. The gate level was evaluated by displaying the EEG signal being processed and the output of the Schmidt trigger on an oscilloscope. Using this procedure, a certain proportion of small amplitude PGO waves were not counted (See Figure 40 for some sample records). Since PGO

waves occurring in bursts tend to be smaller in amplitude than those occurring singly, the TIH distributions obtained would likely provide an underestimate of the proportion of short intervals and an overestimate of the proportion of longer intervals. However, since the signal/noise ratio appeared to remain quite constant over time, it is likely that the gate level selected for pre-deprivation and deprivation analysis was similar, thereby allowing a comparison between pre-deprivation and deprivation to be made. In any case, computer analysis agreed with the results obtained by manually counting the PGO waves, so it appears that no significant distortion of the data was produced as a result of using the TIH program.

Data selected for computer analysis was taken at the same time of day from representative eight hour periods during pre-deprivation and deprivation. The composite TIH distributions were constructed from four samples obtained under the two conditions. The individual TIH distributions that made up the composite distribution were very similar to each other.

In order to compare PGO to REM TIH distributions, a distribution of REM intervals for Cat 007 was obtained by manually measuring the duration of all inter-REM intervals occurring during REM sleep periods from comparable eight hour blocks of time during pre-deprivation and deprivation. Computer analysis was not feasible here since the detection

threshold required to discriminate signal from noise using capacity-coupled recording was so large as to prevent the detection of many of the REMs.

The same data that was used for calculating the PGO wave time interval histograms was reanalyzed using a cross-correlation program written for the PDP-8 computer. This program required two inputs, both of which were, in this case, gated on PGO waves recorded from different brain loci. The criteria for setting the Schmidt gate levels were identical to those used in the PIH analysis. The cross-correlation function was determined by calculating the time intervals between an input (as defined by the Schmidt trigger) on channel one and all subsequent inputs on channel two up to a maximum interval of 408 msecs. This calculation was done for every input occurring on channel one and a tally was made of the frequency of occurrence of all intervals up to 408 msecs. with the bin width being equal to 0.8 msecs. At the same time, an identical analysis was being performed on channel two input. Thus, the time interval between an event on channel two and all subsequent events on channel one were tallied, again up to a maximum of 408 msecs. From this analysis, two separate distributions were obtained and plotted back to back. Thus, in Figure 29, for example, time zero marks the occurrence of a PGO wave in the left PRF and the histogram appearing on the right hand side of the figure

is a frequency distribution of the intervals between time zero and the occurrence of PGO waves in the right PRF. Conversely, when considering the histogram on the left hand side of the figure, time zero now marks the occurrence of a PGO wave in the right PRF and the frequency distributions on the left side pertains to PGO waves occurring in the left PRF after time zero.

To guarantee that the relative gating levels between channels was constant from one analysis to the next, only those analyses where the proportion of counts in one channel with respect to the second were roughly the same across days were considered. Figure 28 provides an illustration of what happens to the cross-correlogram if the relative gating levels are changed. Here the gating level on the left pontine reticular formation channel was optimally set for PGO wave detection. The gating level on the right pontine reticular formation channel was systematically increased in sensitivity over repeated analysis of the same data. The results obtained at the least sensitive setting are given in the top graph of Figure 28, while those obtained at the greatest sensitivity appear in the bottom graph. The N values given in the corners of each graph are the number of PGO waves counted within the respective channels. The RA values are the ratios of the number of counts occurring in the channel labelled on the left hand side of the figure to the number of counts made in

the channel labelled on the right. The change in sensitivity in the right pontine channel is reflected by the increased number of counts made in this channel. The decreasing number of counts made in the left pontine channel are due to a change in the relative sensitivity; a parameter which is better described by the value of RA.

The systematic shift in the peak of the cross-correlogram at different values of RA in Figure 28 was due to the fact that as RA increased, PGO wave detection occurred lower down on the PGO wave form. The magnitude of the peak shift (about 8 to 10 msecs.), therefore, provides an estimate of the rise time of the PGO wave over the voltage range that the detection threshold was varied. The fact that a distribution of intervals around the peak value was obtained is likely due to variability in the amplitude of PGO waves (See Figure 40 for an example). Thus, for a given threshold, small amplitude PGO would be detected higher up on the rising phase of the wave than would large amplitude PGO waves.

EOG Amplitude Analysis

Data from four cats (Cats 007, Q03, 403 & 411) were selected for analysis on the basis of the magnitude of the REM density effect observed in Experiment I. Although an attempt was made to obtain a representative sample, there may have been a slight bias toward selecting cats that had a large increase in REM density during sensory deprivation since two

animals that had the largest increase in REM density (Cats Q03 & 403) were included while only one of two cats having intermediate effects (Cats 007 & N06) was selected. To counter this, one of two cats having the smallest effects (Cats Q01 & 411) was also included (See Figures 13 to 15 and Table XXV). The eight hour block of pre-deprivation data chosen for analysis in these four cats was selected so that the average REM density of this period was representative of the daily REM density but the deprivation data was taken from comparable eight hour blocks of time on deprivation days three and four when the REM density effect was maximal.

The REM sleep periods occurring in the eight hours of time selected for analysis during pre-deprivation and deprivation were divided into twenty second epochs according to page divisions of the raw record. For each epoch, three measurements were made; a count of the total number of REMs occurring, a tally of the amplitude of each EOG deflection and a calculation of the average amplitude of all the EOGs. The number of REMs and their average amplitude were then entered into a scatterplot of REM density (number of REMs/ 20 seconds of REM sleep) versus the mean EOG amplitude (See Figure 34 for an example) where the number of data points equalled the number of twenty second epochs of REM sleep during the eight hour block of time being considered. Sample size was matched as closely as possible across conditions

for a given animal.

Linear regression analysis was performed using equations described by Guilford (1965) to obtain the least square lines needed to estimate variable X given Y and variable Y given X. The centroid point at the intersection of the two regression lines was tabulated as this represents the average value of both the X and Y variables. The degree of correlation between the two variables was also listed. Frequency distributions of EOG amplitudes during pre-deprivation and deprivation were also constructed (See Figure 38) and the average EOG amplitude under the two conditions was calculated. Statistical analysis of these parameters was made by using a single factor, repeated measures analysis of variance.

Hippocampal Theta Analysis

Two animals run in Experiment I (Cats Q03 & 411) had hippocampal electrode placements from which large amplitude, rhythmic slow waves in the four to seven Hertz frequency range could be recorded during REM sleep. For these cats, eight hour blocks of time during pre-deprivation and deprivation were selected on the basis of changes in REM density as in the REM amplitude analysis. In these eight hour periods, all REM sleep episodes greater than or equal to three minutes in duration were processed. Samples of record, one to two seconds in duration, were taken from the 3^{rd}, 10^{th}, 17^{th}, 24^{th} ... pages of record following the beginning of the REM

period (Page length = 20 seconds). If the desired sample could not be found on the selected sample page, then the preceeding and succeeding pages were searched. If the sample still was not obtained, then the preceeding and succeeding pages twice removed from the sample page were searched. The process was continued till the desired sample was obtained. Typically, samples were found on the target sample pages of on immediately adjacent pages.

Three different types of samples were searched for on each target sample page. The criteria for selecting Type 1 samples was the presence of a large number of REMs and PGO waves in a one or two second sample. Type 2 samples were selected on the basis of the presence of a large number of PGO waves without many REMs and Type 3 samples were chosen from record where there were neither REMs nor PGO waves (See Figure 40). Once all the desired samples were obtained, one experimenter went over the sampled time periods and measured the frequency of the hippocampal theta rhythm. A second experimenter then counted all the REMs and PGO waves in each sample and a measure of the REM and PGO density (number of events/second) in the samples was obtained. The mean and standard error of REM density, PGO density and theta frequency was tabulated for each sample type.

Histology

At the termination of the experiments, all cats were deeply anaesthetized with barbiturate and perfused through the heart with saline and 10% formal-saline. The brain was removed, fixed in formal-saline and then sectioned on a freeze microtome. The sections were floated onto glass slides and stained with thionin. Electrode tracks were traced and the locations of the tips of the deep electrodes were reconstructed on drawings taken from the atlas of Berman (1968). Only those electrodes from which signals such as PGO waves and hippocampal theta were recorded and analyzed were processed in this manner.

RESULTS

The daily average PGO density of Cats 403 and 411 remained relatively constant across conditions in this experiment, in contrast to the significant change in REM density that was found in Experiment I during deprivation (Figure 22). This lack of change in PGO density during deprivation is also evident in the time interval histogram for PGO waves recorded bilaterally from the LGN of Cat 403 (Figure 23) and from the pontine reticular formation (PRF) and LGN of Cat 411 (Figure 24). In contrast, the frequency histogram of inter-REM intervals for Cat 007 (Figure 25) showed a large increase in the proportion of short intervals found during deprivation. These contrasting effects of deprivation on PGO wave density and REM density can be seen in the raw records (Figures 26 & 27). In these figures, it is also clear that the single large peak occurring at short intervals in the TIH distributions for REMs and PGO waves is due to bursts of these phasic events while the long intervals between bursts and between isolated occurrences of PGO waves and REMs make up the long tail of the TIH distribution. The proportional increase of short inter-REM intervals is reflected in the appearance of more bursts of REMs in the raw record. Thus, when an arbitrary definition of a REM burst was made (Burst Criteria - (1) The occurrence of at least four inter-REM intervals of 200 msecs. or less in

succession. (2) If an inter-REM interval of 267 msecs. followed the last occurring 200 msec. interval described in point #1, then two additional 200 msec. intervals would have to occur in succession after the 267 msec. interval in order to be classified as part of the original burst. (3) If an inter-REM interval of 333 msecs. followed the last occurring 200 msec. interval described in point #1, then three additional 200 msec. intervals would have to occur in succession after the 333 msec. interval to be counted as part of the burst. (4) An interval between successive REMs longer than 400 msecs. terminates any burst.), it was found that for equivalent durations of REM sleep, mean burst density (Number of bursts/minute of REM sleep) and total burst duration were longer during deprivation than during pre-deprivation while the mean burst duration was slightly shorter (Table XXVII).

The analysis of the temporal relationship between the occurrence of a PGO wave in one brain structure relative to another during pre-deprivation and deprivation have uniformly shown that sensory deprivation is without effect. Thus, in Figure 29, PGO waves appear to originate on either side of the brainstem with about equal frequency both during pre-deprivation and deprivation. When considering PGO waves occurring in the pons versus those occurring in the LGN, however, it is clear that the pontine wave typically precedes the LGN wave by about 8 to 14 msecs. (Figures 30 & 31). Bilateral

occurrence of PGO waves in the LGN, on the other hand, appear to be delayed, one with respect to the other by about 8 to 10 msecs. even though one side does not predominantly precede the second (Figure 32). When the analysis time was extended to 408 msecs., the only additional piece of information obtained was a confirmation of the tendency of PGO waves to occur in bursts (Figure 33). This is indicated by the presence of multiple peaks in the cross-correlogram, a result that is particularly evident when the total number of events counted was increased (Bottom graph, Figure 33).

Thus, while two indices of PGO wave generation have remained invariant during sensory deprivation, REM parameters have changed dramatically. This contrast was further extended in the EOG amplitude analysis where a significant positive correlation between EOG amplitude and REM density was found during pre-deprivation for each set of data analyzed (Table XXVIIIc, column 1; Figures 34 to 37, Top graph). The fact that the magnitude of the correlation was unchanged by deprivation (Table XXVIIIc, column 2; Figures 34 to 37, bottom graph) means that the increase in REM density found in Experiment I must be accompanied by an increase in EOG amplitude. This is borne out in Figures 34 to 37 where the shift in the centroid point of the scatterplots, when comparing pre-deprivation to deprivation is indicative of the simultaneous increase in the mean EOG amplitude and REM density

(marked by arrows). Statistical comparison of EOG amplitude and REM density across conditions in this subset of four cats confirms these conclusions (Table XXVIIIA; B). Further evidence of the EOG amplitude effect is provided in the frequency distribution of EOG amplitudes (Figures 38 & 39), where larger amplitude REMs occur more frequently during deprivation. This is also noticeable in the raw records shown in Figure 27 and Figure 28.

The rather limited amount of data available on the effects of sensory deprivation on hippocampal theta frequency is provided in Table XXIX. Here one can see that the average theta frequency tended to be less in Type 2 samples than in Type 1 samples, and less in Type 3 samples than in Type 2 samples (See Figure 40 for an example). For Cat 411, the mean theta frequency of Type 1 samples was greater during deprivation than during pre-deprivation, while for Cat Q03, theta frequency was about the same under both conditions. Similar comparisons for Types 2 and 3 samples suggest that mean theta frequency during pre-deprivation is the same as during deprivation.

The location of electrode tips, represented in Figures 41 to 45 by small black dots, were in positions from which other workers have successfully recorded large amplitude signals (i.e. theta or PGO waves) in cat (Brooks & Bizzi, 1963; Brooks, 1967a; Brown, 1968). Thus, the hippocampal

electrodes from which the theta pattern shown in Figure 40 was recorded, were located directly above the CA_1 pyramidal cell layer for Cat Q03 (Figure 41) and above the subicular pyramidal cell layer for Cat 411 (Figure 42). The LGN electrodes in Cat 403 from which the records shown in Figure 27 were obtained, were located such that the deep lateral tip was at the border of the optic tract and layer C_2 while the shallow medial tip was located at borders of layers A_1 and C (Figure 43) using the terminology of Kaas, Guillery & Allman (1972). The LGN electrode in Cat 411 from which PGO waves shown in Figure 40 were recorded was similarly located (Figure 44). The pontine PGO waves shown for Cat 411 in Figure 40 were recorded from electrodes in the nucleus reticularis gigantocellularis, approximately two millimeters lateral from the midline and at the same depth and AP level as the nucleus olivaris superior lateralis, a prominent S-shaped nucleus appearing at this level of the neuroaxis (Figure 45).

DISCUSSION

These experiments clearly demonstrate that the large increase in REM density during sensory deprivation is not accompanied by a corresponding increase in PGO density. Similarly, the distribution of inter-REM intervals undergoes a large change during deprivation as do the distributions of EOG amplitudes while the TIH distribution for PGO waves and the cross-correlation between PGO waves occurring in two brain loci are not altered. Thus, it appears that parameters which characterize REMs undergo modification during deprivation while parameters which describe different aspects of PGO wave generation remain unchanged. These results appear to contradict the currently held belief that a brainstem generator is responsible for the production of both REMs and PGO waves. However, as can be seen in Figure 27, during pre-deprivation, many PGO waves occur unaccompanied by REMs, while this rarely happens during deprivation. Thus, it seems reasonable to suggest that a proportion of eye muscle contractions during pre-deprivation are too weak to elicit a detectable EOG response despite the fact that a PGO wave has been generated. During deprivation, though a greater proportion of REMs accompany PGO waves, perhaps because the oculomotor neurons are driven more intensely or because the extraocular muscles contract more vigorously to

to the same input. Thus, according to this hypothesis, one would expect that PGO density would limit the maximum increase in REM density that could be obtained. This expectation is borne out in the experiment on Cat 403 (Figure 22) where REM density reached an asymtotic level during deprivation which was roughly equivalent to the PGO density.

If REMs which are subthreshold for detection during pre-deprivation become suprathreshold during deprivation, then one would expect that the average amplitude of REMs during deprivation would be larger than REM recorded under pre-deprivation conditions if both subthreshold and suprathreshold REMs increase their amplitude. The present experiment provides evidence which is consistent with this hypothesis since the proportion of large amplitude EOG deflections increased during deprivation. However, an increase in the magnitude of the corneo-retinal potential or in the velocity of eye movements during deprivation could have a similar effect on the amplitude of the EOG response. These alternatives will be considered in the final discussion.

The fact that the temporal relationship between the occurrence of PGO in two brain loci does not change during sensory deprivation goes along with the lack of effect of deprivation on the frequency with which PGO waves are generated during REM sleep. In addition, the cross-correlational and TIH analyses provide data which agree with the description

of these REM sleep phenomena that have been given by other workers using different techniques. Thus, the cross-correlograms between PGO waves recorded simultaneously in the pontine reticular formation and the LGN (Figures 30 & 31) support the idea that the PGO wave is generated by a brainstem structure. The cross-correlation between PGO waves recorded on either side of the brainstem suggests that each side precedes the other in generating PGO about equally often (Figure 29). This is also true in the LGN, only here a PGO wave occurring on one side is followed by a PGO wave on the other side after about an eight msec. delay whereas in the pontine reticular formation, this delay is much briefer. Cespuglio, Laurent & Jouvet (1975) observed a similar delay (5 to 6 msecs.) between PGO waves recorded in the LGN after a brainstem hemisection was made in front of the pontine generator on one side. Under these conditions, the geniculate ipsilateral to the hemisection is driven only by the contralateral generator via a pathway crossing the midline in the region of the supraoptic decussation. The geniculate contralateral to the hemisection, on the other hand, receives a more direct projection from the generator on the same side of the brain. Therefore, the PGO wave recorded here always precedes the PGO wave recorded in the opposite LGN, a fact which can be demonstrated by averaging simultaneously recorded PGO waves.

The short delay between the occurrence of PGO waves recorded on one side of the pontine reticular formation with respect to those recorded on the other side, could be explained by the presence of an interconnection between sides. This interpretation is supported by the fact that a mid-saggital transection in the region of the abducens nucleus abolishes the bilateral synchrony between PGO waves recorded in this area of the brainstem (Cespuglio, Laurent & Calvo, 1976) and unilateral stimulation of the brainstem elicits bilaterally occurring PGO waves (Malcolm, Watson & Burke, 1970). In all of these cross-correlograms, however, one must bear in mind that the position of the peak of the distribution depends on the gating level selected. Thus, the bottom graph of Figure 28 could be taken as evidence that the pontine generator is unilaterally located in the right pontine reticular formation. Therefore, interpretation based on slight differences in the peak of the cross-correlogram can only be taken seriously if strong supporting evidence can be marshalled from other sources.

The time interval histogram data illustrate that the TIH distributions obtained for PGO waves recorded from the LGN and pontine reticular formation and for REMs are very similar in shape. This result agrees with the fact that REMs and PGO typically occur in close temporal contiguity with each other (Figure 27). In addition, the distinction between

the REM or PGO burst and the isolated occurrence of these phasic events is also evident in the TIH distributions, and provides further evidence for the idea that different mechanisms are responsible for the generation of these two types of patterns. Indeed, Chouvet & Gadea-Ciria (1974), on the basis of similar data, have empirically derived a semi-Markov model to represent the probability that one mechanism for generating PGO waves will become active (e.g. Burst generator) given the second PGO wave generating mechanism (isolated PGO wave generator) is already operative.

Further support for the hypothesis that separate mechanisms exist for the generation of isolated and bursts of PGO was obtained in a pilot study done on anaesthetized cats which had been pre-treated with reserpine (Serpasil, Ciba; 0.25 to 0.40 mg/kg ip.). PGO waves recorded from the pontine reticular formation and LGN under these conditions no longer occur in bursts. Rather, single PGO waves or at best, pairs of PGO waves, reoccur at fairly uniform intervals of time (Figure 46). This pattern is reflected in the TIH distributions where the large peak appearing in the long interval portion of the distribution corresponds to the regularly spaced occurrence of PGO waves and the smaller peak in the short interval "burst region" of the distribution corresponds to the occurrence of PGO wave couplets. Thus, reserpine seems to produce a state in which the pontine generator functions

quite differently from the way it does in REM sleep since the long intervals between isolated PGO become uniform in length, in contrast to REM sleep where no particular interval is favored although there is tendency for shorter intervals to be generated than for longer intervals (Figures 23 & 24). The burst pattern found in REM sleep clearly does not occur after reserpine, but it is possible that the couplets of PGO are the remnants of the burst pattern since the intervals between pairs of PGO fall within the "burst region" of the TIH distribution. These results agree well with those obtained by Brooks and his colleagues (Brooks & Gershon, 1971; 1972; 1977; Brooks, Gershon & Simon, 1972) in chronic cat which had been pre-treated with reserpine, and suggests that the systems responsible for producing the isolated and burst pattern of PGO may be differentially affected by reserpine.

Since the available evidence suggests that there are separate neural mechanisms for the generation of isolated REMs and REM bursts, one wonders whether sensory deprivation has a differential effect on these systems. The fact that in one cat, the proportion of short interval REMs, the number of REM bursts and the total burst duration increased during deprivation, is consistent with this idea. Jeannerod, Mouret & Jouvet (1965) have described another experimental treatment which alters the proportion of burst type REMs with respect to isolated REMs. They found that in normal cat, roughly

fifty percent of all eye movements occurring during REM sleep are the burst type while following frontal-cortical ablation, this proportion increased to greater than seventy percent. For comparison, the sum of the first five class intervals in Figure 25 equals 55.5% during pre-deprivation and 66.0% during deprivation. This rather gross similarity between the effects of sensory deprivation and frontal-cortical ablation is unlikely to be of any importance though, since Gadea-Ciria (1976b; c; 1977a; b) reported that the TIH distributions for PGO waves were greatly altered after frontal-cortical ablation. In contrast, during sensory deprivation, the PGO TIH distributions remained about the same across conditions despite the large change in the TIH distribution for REMs during deprivation.

The positive relationship between REM density and hippocampal theta frequency (Type 1 sample) found under pre-deprivation conditions of the present experiment agrees with the result obtained by Sakai et. al. (1973) under similar conditions. With the limited data available, it is impossible to decide whether theta frequency during PGO waves, but in the absence of REMs (Type 2 sample), is intermediate between the frequency obtained when REMs are present (Type 1 samples) and when neither REMs nor PGO waves are present (Type 3 samples), as is suggested by the pre-deprivation data of Cat 411 (Table XXIX), or whether theta frequency in the absence of

movement is the same irrespective of whether PGO waves are occurring (Type 2 vs Type 3 samples) as is suggested by the deprivation data for Cat 411 (Table XXIX).

The effects of deprivation on theta frequency are ambiguous in these experiments since in one animal (Cat 411) a large increase in theta frequency was obtained when comparing pre-deprivation to deprivation mean frequency for Type 1 samples, whereas theta frequency for the second animal (Cat Q03) was about the same under both conditions. In the case of Type 2 and 3 samples, the average theta frequency obtained during pre-deprivation was approximately the same as that found during deprivation.

Table XXVII - Mean burst density, mean burst duration and total burst duration on pre-deprivation day three and deprivation day five for Cat 007.

Table XXVII

	Burst Density (Bursts/min.)	Mean Burst Duration (secs.)	Total Burst Duration (secs.)
Pre-Deprivation Day 3	0.86	1.25	77.03
Deprivation Day 5	2.25	1.00	148.0

Table XXVIII - Average REM amplitude and REM density plus the degree of correlation between REM amplitude and REM density for four cats under pre-deprivation and deprivation conditions. (F statistic obtained from a repeated measures ANOVA.)

Table XXVIII

		Pre-Deprivation	Deprivation	F
REM Amplitude (μV)	007	49.24	66.10	29.56*
	Q03	92.63	121.72	
	403	77.32	98.67	
	411	84.47	96.40	
	$\bar{X}$	75.92	95.72	
	S.E.	9.43	11.41	
		B		
REM Density	007	54.38	87.70	37.74*
	Q03	61.25	80.26	
	403	47.90	78.24	
	411	46.15	63.16	
	$\bar{X}$	52.42	77.34	
	S.E.	3.43	5.15	
		C		
Correlation Coefficient	007	0.69	0.64	0.49
	Q03	0.54	0.61	
	403	0.51	0.53	
	411	0.44	0.23	
	$\bar{X}$	0.55	0.50	
	S.E.	0.05	0.09	

*$p < 0.05$

Table XXIX - Average PGO and REM density (Number/second) plus mean theta frequency obtained for two cats under pre-deprivation and deprivation conditions for sample Types 1, 2 and 3. (See text for differences between sample types).

Table XXIX

		Sample 1			Sample 2			Sample 3		
		PGO	REM	Theta	PGO	REM	Theta	PGO	REM	Theta
Cat 411										
Pre-Dep.	$\bar{X}$	5.36	4.65	4.79	3.82	0.61	4.58	0.05	0	4.20
	S.E.	0.14	0.21	0.14	0.15	0.14	0.19	0.02	0	0.10
Depri-vation	$\bar{X}$	5.52	5.44	5.59	3.60	0.80	4.60	0.05	0	4.47
	S.E.	0.16	0.22	0.12	0.13	0.11	0.14	0.03	0	0.10
Cat 203										
Pre-Dep.	$\bar{X}$	5.64	6.09	4.31				0.00	0	3.02
	S.E.	0.20	0.32	0.23				----	-	0.17
Depri-vation	$\bar{X}$	5.82	6.83	4.25				0.00	0	2.87
	S.E.	0.15	0.24	0.12				----	-	0.26

Figure 22 - Average REM and PGO density for Cats 403 & 411 during pre-deprivation, deprivation and post-deprivation.

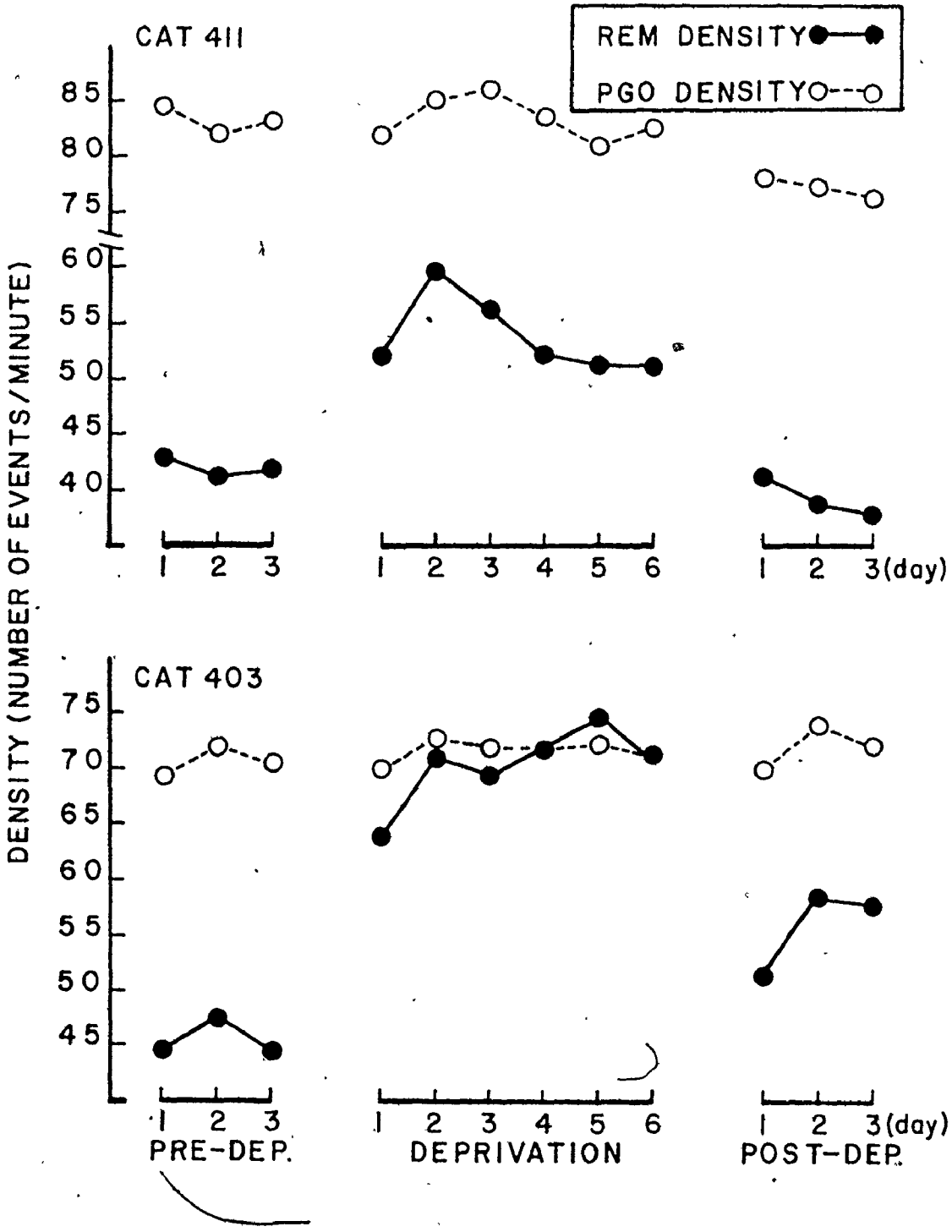

CAT 411
REM DENSITY
PGO DENSITY
DENSITY (NUMBER OF EVENTS/MINUTE)
85
80
75
60
55
50
45
40
1 2 3
1 2 3 4 5 6
1 2 3(day)
CAT 403
75
70
65
60
55
50
45
1 2 3
PRE-DEP.
1 2 3 4 5 6
DEPRIVATION
1 2 3(day)
POST-DEP.

Figure 23 - Time interval histograms of the interval between the successive occurrence of PGO waves recorded from the LGN of Cat 403 during pre-deprivation and deprivation.

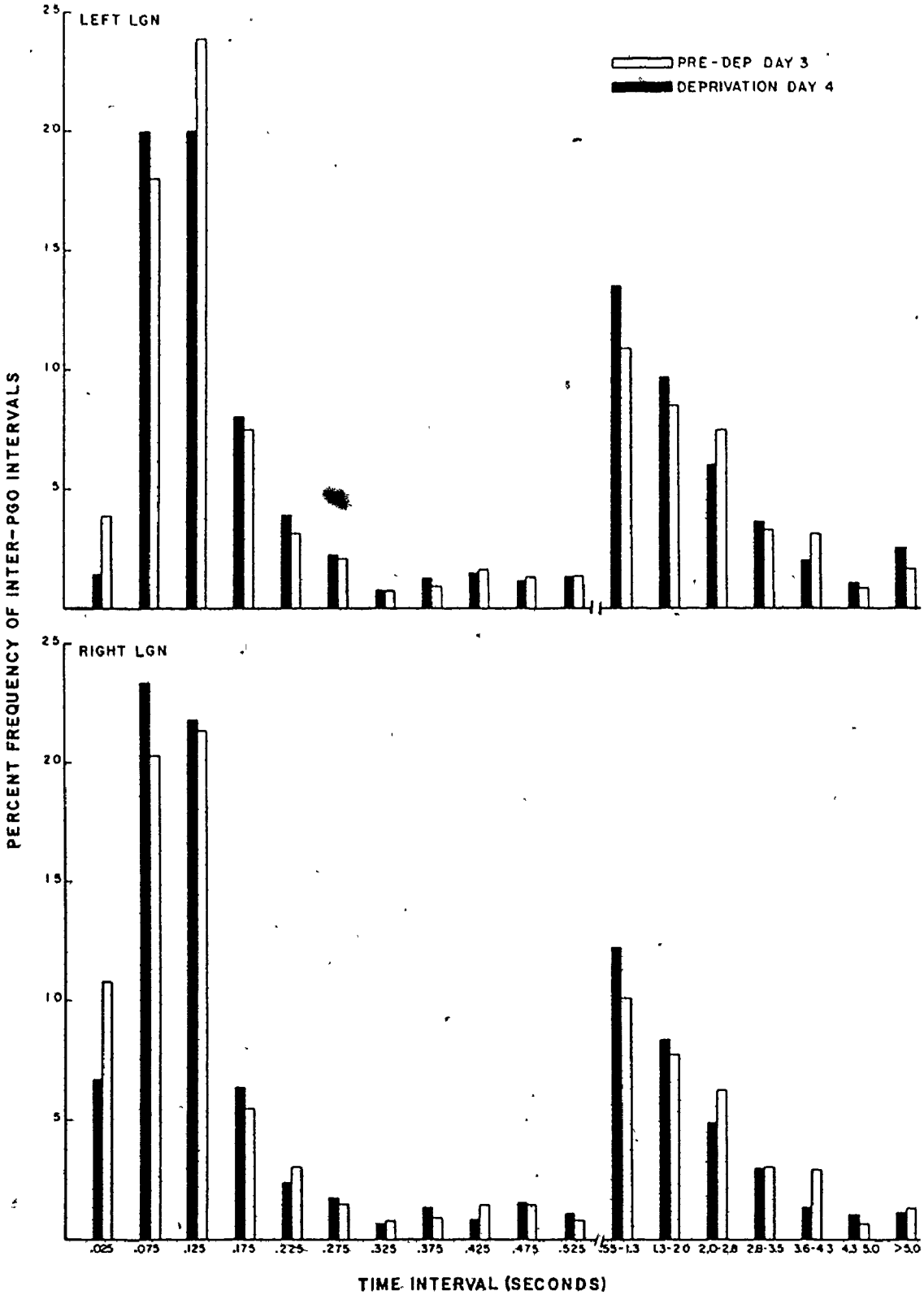

LEFT LGN
PRE-DEP DAY 3
DEPRIVATION DAY 4
RIGHT LGN
PERCENT FREQUENCY OF INTER-PGO INTERVALS
25
20
15
10
5
.025
.075
.125
.175
.225
.275
.325
.375
.425
.475
.525
.55-1.3
1.3-2.0
2.0-2.8
2.8-3.5
3.6-4.3
4.3
5.0
>5.0
TIME INTERVAL (SECONDS)

Figure 24 - Time interval histograms of the interval between successive occurrence of PGO waves recorded from the PRF and LGN of Cat 411 during pre-deprivation and deprivation.

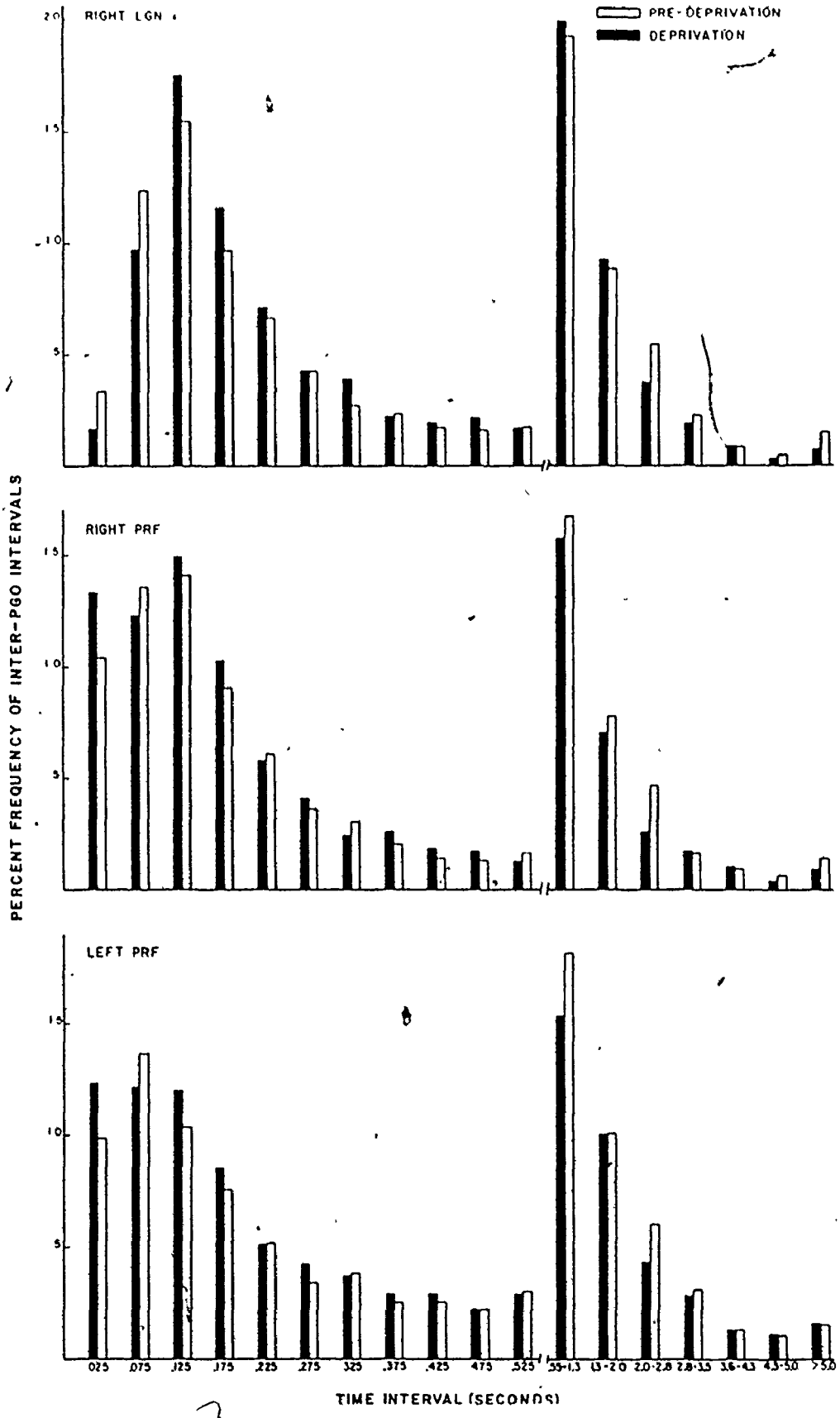

PRE-DEPRIVATION
DEPRIVATION
RIGHT LGN
RIGHT PRF
LEFT PRF
PERCENT FREQUENCY OF INTER-PGO INTERVALS
TIME INTERVAL (SECONDS)
.025
.075
.125
.175
.225
.275
.325
.375
.425
.475
.525
.55-1.3
1.3-2.0
2.0-2.8
2.8-3.5
3.5-4.3
4.5-5.0
>5.0

Figure 25 - Time interval histograms of the interval between the successive occurrence of REMs for Cat 007 during pre-deprivation and deprivation.

FREQUENCY DISTRIBUTION OF INTER-REM INTERVALS

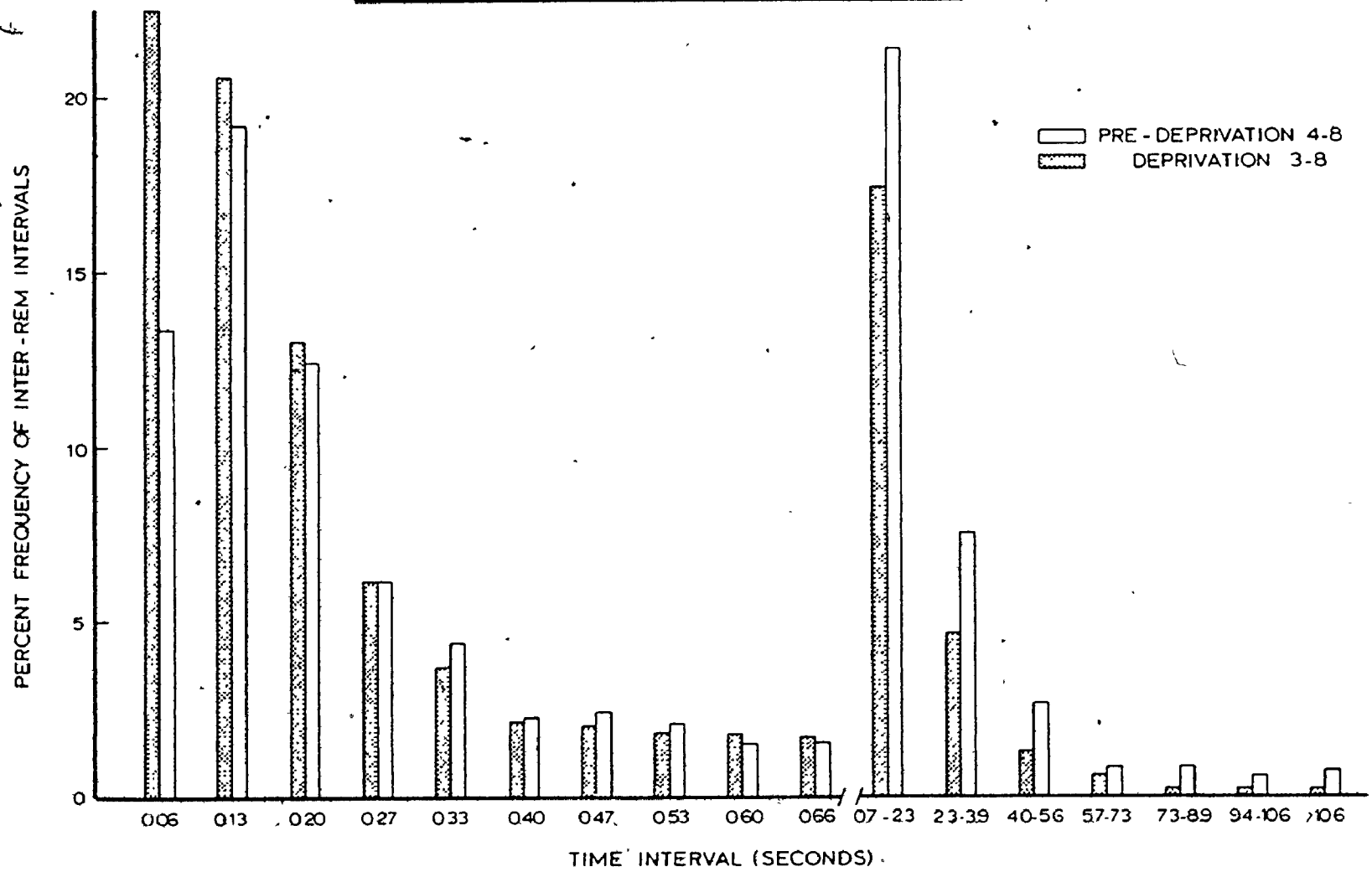


Figure 26 - EOG (top trace of pair) and EEG (visual cortex) records taken during REM sleep episodes of Cat 007 on pre-deprivation day three and deprivation day five. (The episodes were eight minutes long and each set of traces is the last twenty seconds of each minute.)

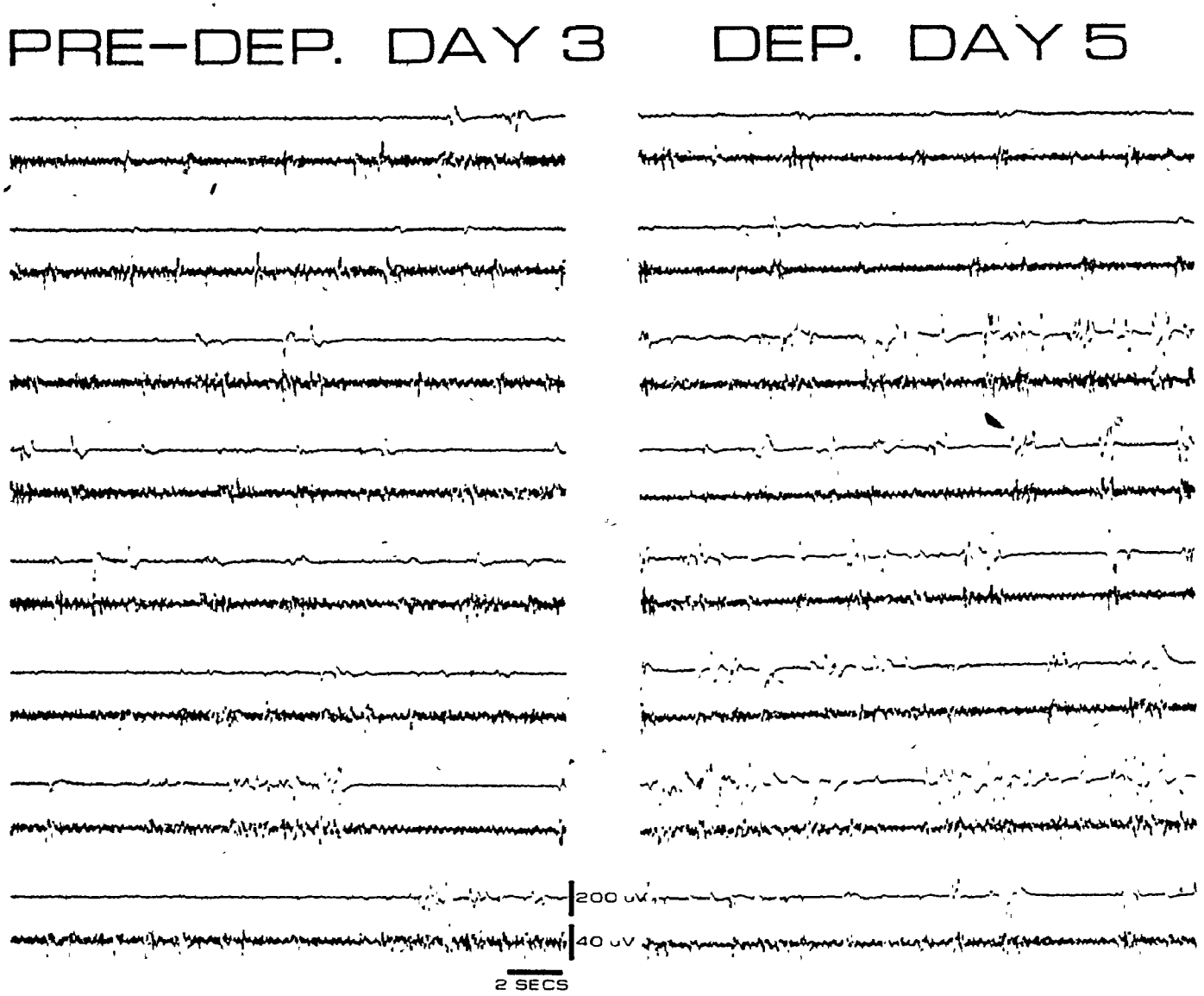

PRE-DEP. DAY 3
DEP. DAY 5
200 uV
40 uV
2 SECS

Figure 27 - EEG and EOG records taken during REM sleep episodes of Cat 403 on pre-deprivation day one and deprivation day three. (The episodes were seven minutes long and each set of traces is the last twenty seconds of each minute.)

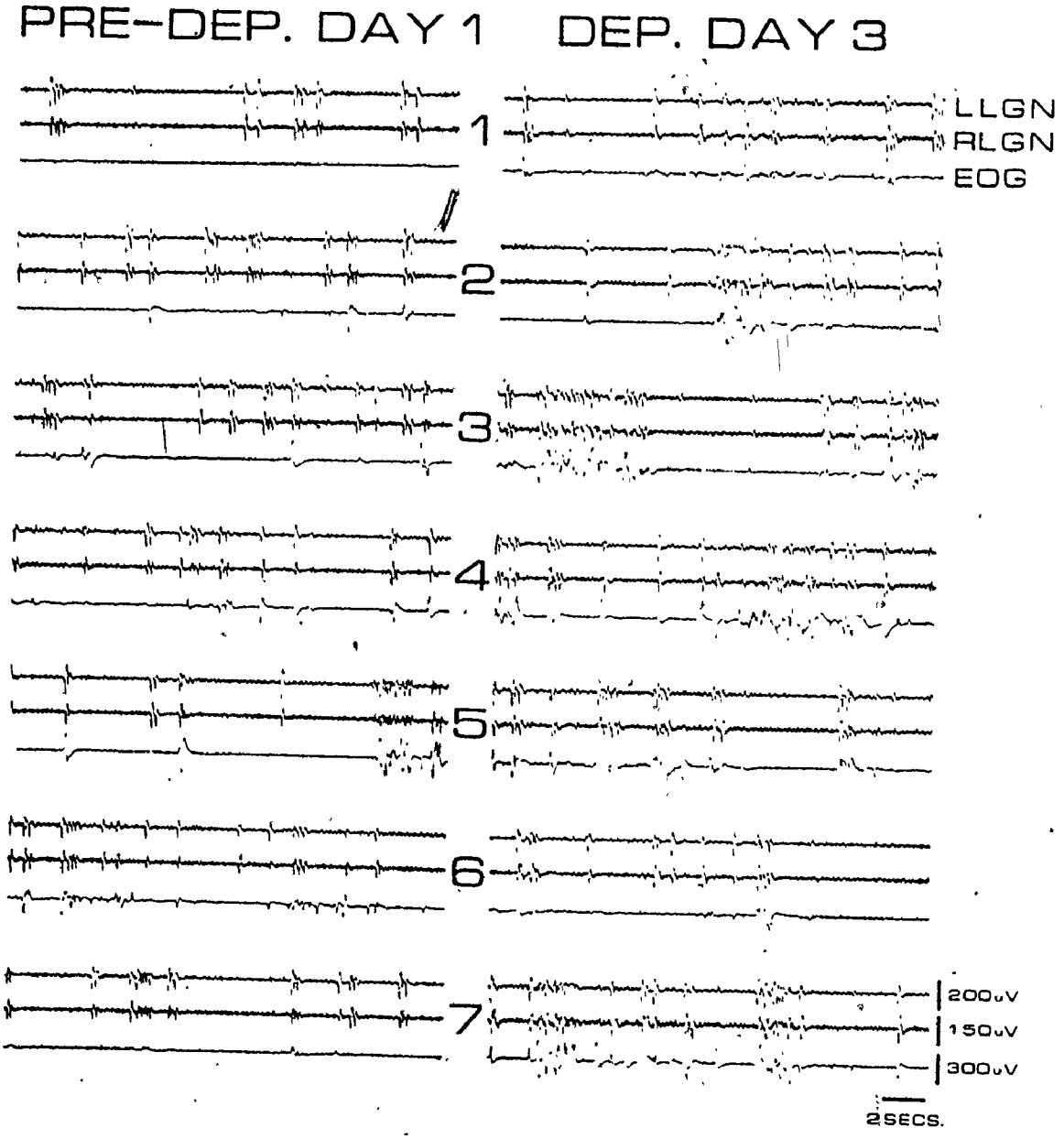

PRE-DEP. DAY 1
DEP. DAY 3
1
2
3
4
5
6
7
LLGN
RLGN
EOG
200uV
150uV
300uV
2SECS.

Figure 28 - Cross-correlation between the occurrence of PGO waves in the left and right PRF of Cat 411 as a function of changing the Schmidt gate level for detecting PGO in the right PRF.

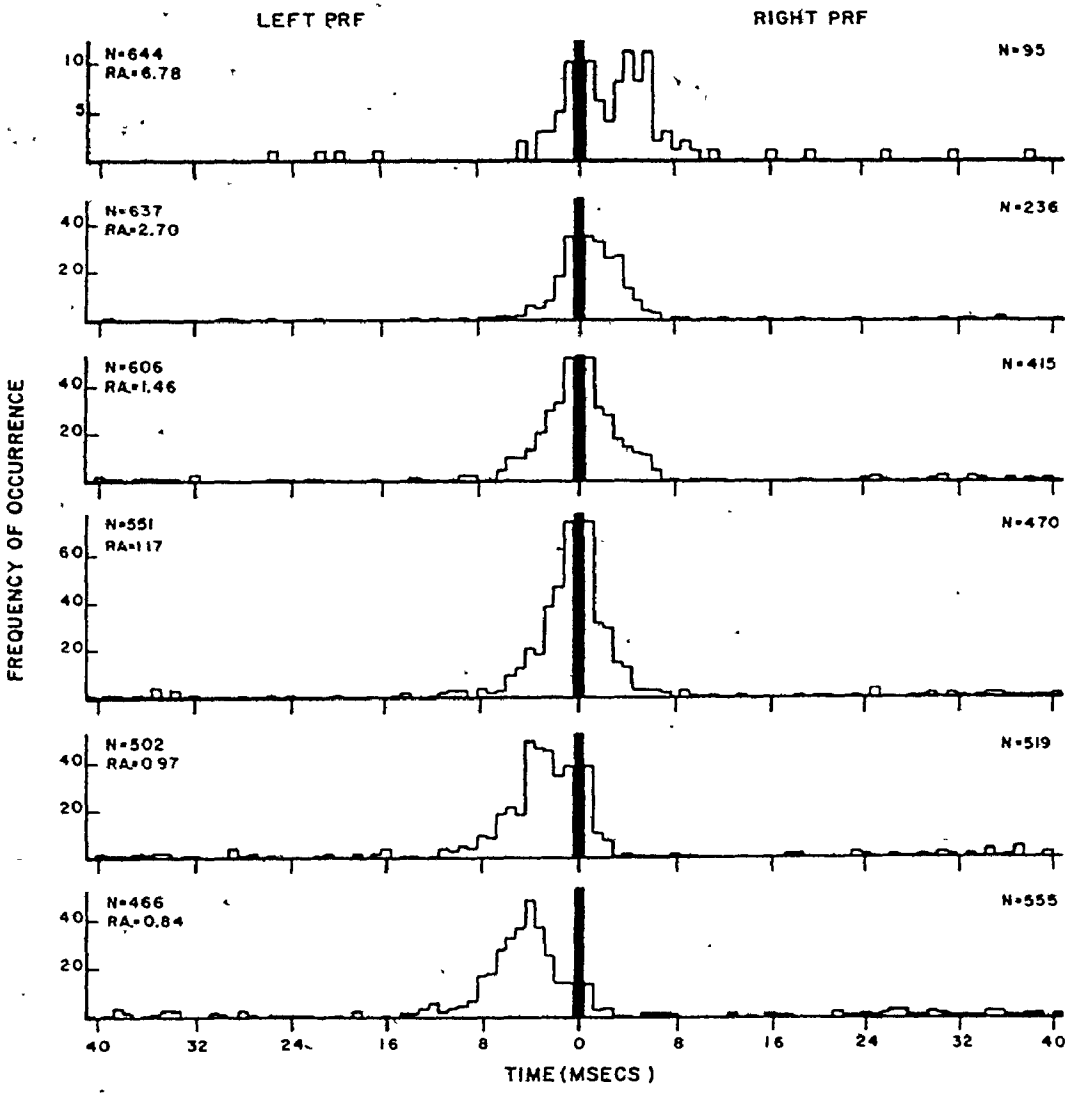

LEFT PRF
RIGHT PRF
N=644
RA=6.78
N=95
N=637
RA=2.70
N=236
N=606
RA=1.46
N=415
N=551
RA=1.17
N=470
N=502
RA=0.97
N=519
N=466
RA=0.84
N=555
FREQUENCY OF OCCURRENCE
TIME (MSECS)

Figure 29 - Cross-correlation between the occurrence of PGO waves in the left and right PRF of Cat 411 on pre-deprivation day two and deprivation day two.

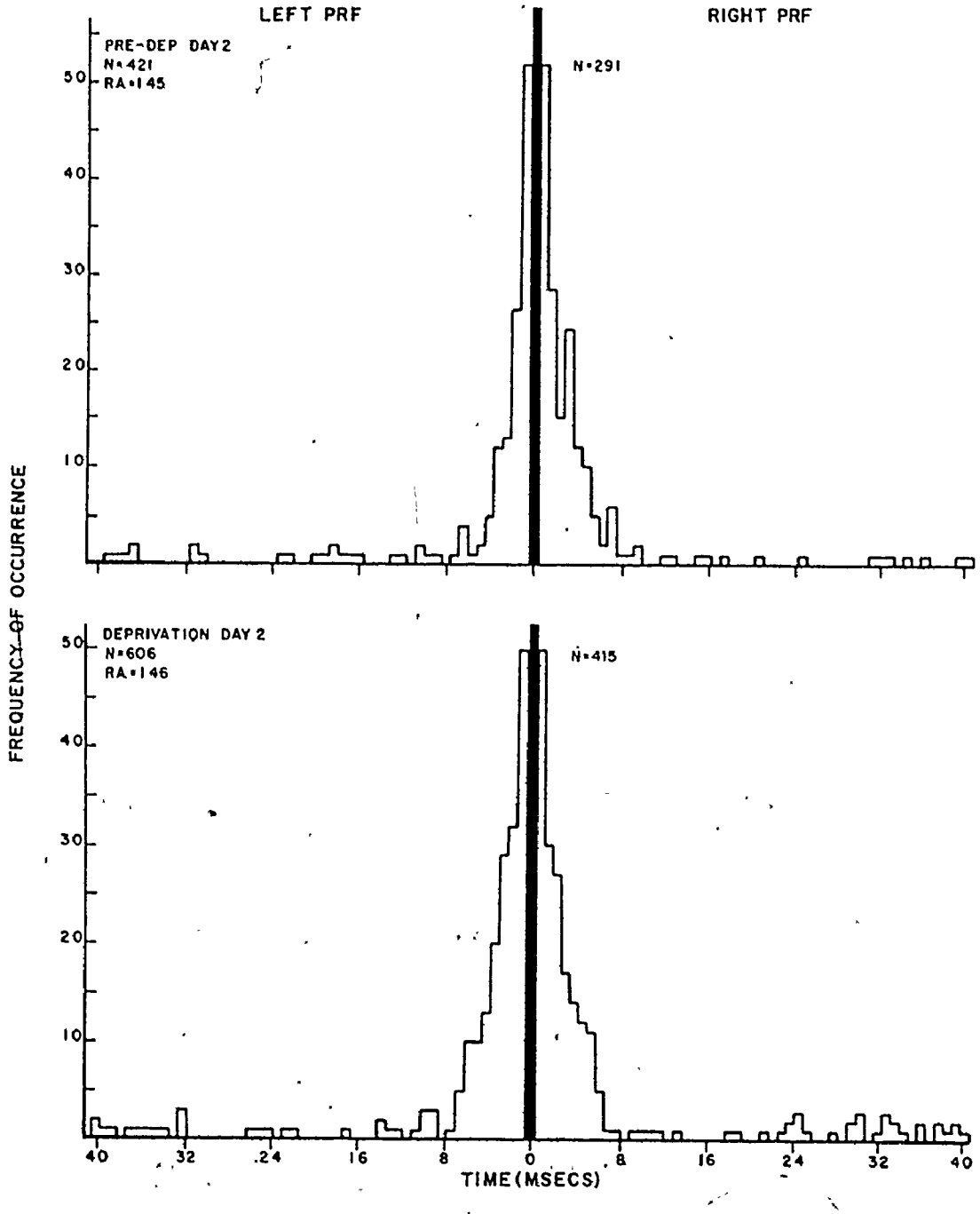

LEFT PRF
RIGHT PRF
PRE-DEP DAY 2
N=421
RA=145
N=291
DEPRIVATION DAY 2
N=606
RA=146
N=415
FREQUENCY OF OCCURRENCE
TIME (MSECS)
50
40
30
20
10
40
32
24
16
8
0
8
16
24
32
40

Figure 30 - Cross-correlation between the occurrence of PGO waves in the left PRF and right LGN of Cat 411 on pre-deprivation day two and deprivation day two.

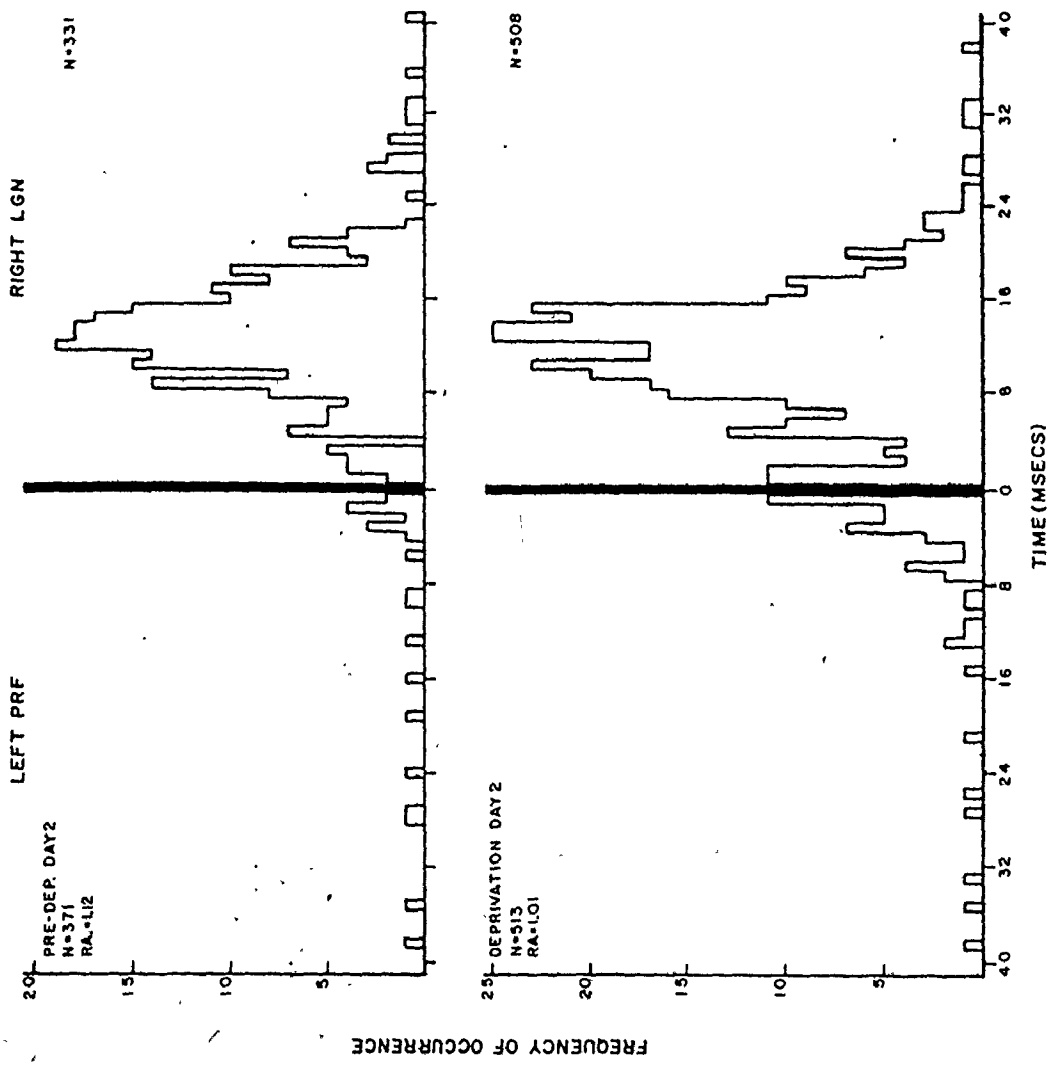

LEFT PRF
RIGHT LGN
PRE-DEP. DAY 2
N=371
RA=1.12
N=331
DEPRIVATION DAY 2
N=513
RA=1.01
N=508
FREQUENCY OF OCCURRENCE
TIME (MSECS)

Figure 31 - Cross-correlation between the occurrence of PGO waves in the right PRF and LGN of Cat 411 on pre-deprivation day two and deprivation day two.

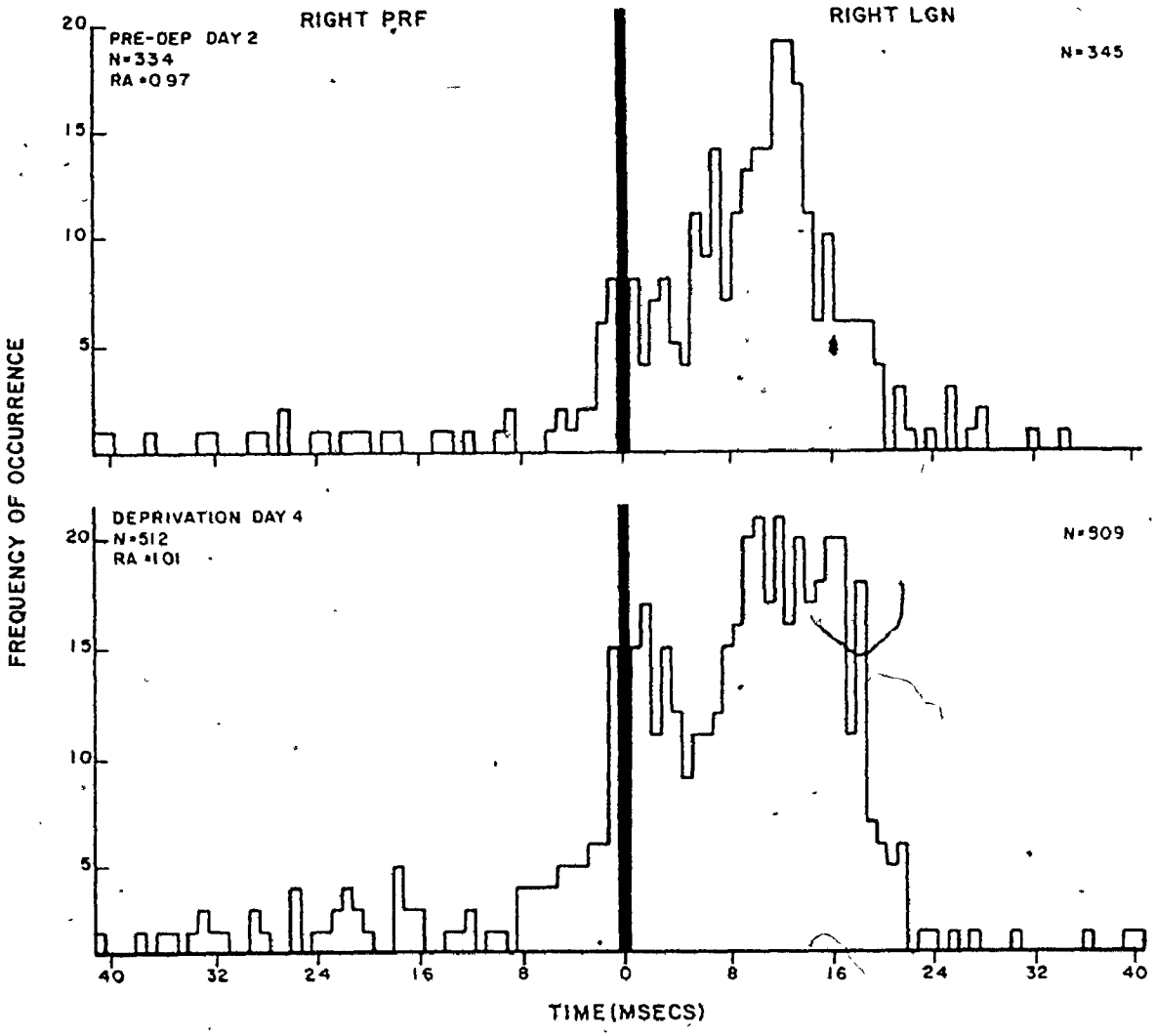

RIGHT PRF
RIGHT LGN
PRE-DEP DAY 2
N=334
RA =0.97
N=345
DEPRIVATION DAY 4
N=512
RA =1.01
N=909
FREQUENCY OF OCCURRENCE
TIME (MSECS)
20
15
10
5
40
32
24
16
8
0
8
16
24
32
40

Figure 32 - Cross-correlation between the occurrence of PGO waves in the left and right LGN of Cat 403 on pre-deprivation day three and deprivation day four (40 msec. correlogram).

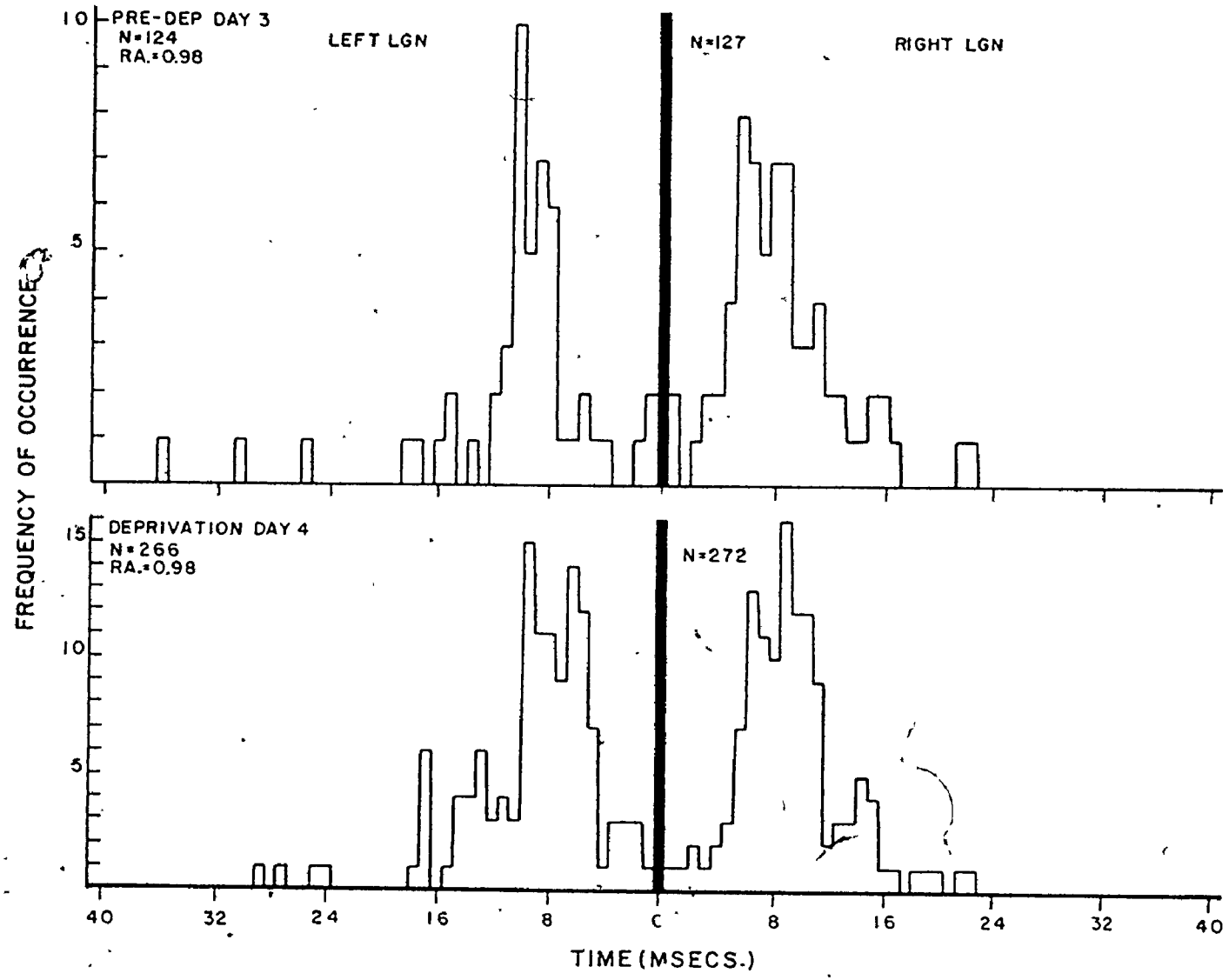

PRE-DEP DAY 3
N=124
RA.=0.98
LEFT LGN
N=127
RIGHT LGN
DEPRIVATION DAY 4
N=266
RA.=0.98
N=272
FREQUENCY OF OCCURRENCE
TIME (MSECS.)
40
32
24
16
8
0
8
16
24
32
40

Figure 33 - Cross-correlation between the occurrence of PGO waves in the left and right LGN of Cat 403 on pre-deprivation day three and deprivation day four (400 msec. correlogram).

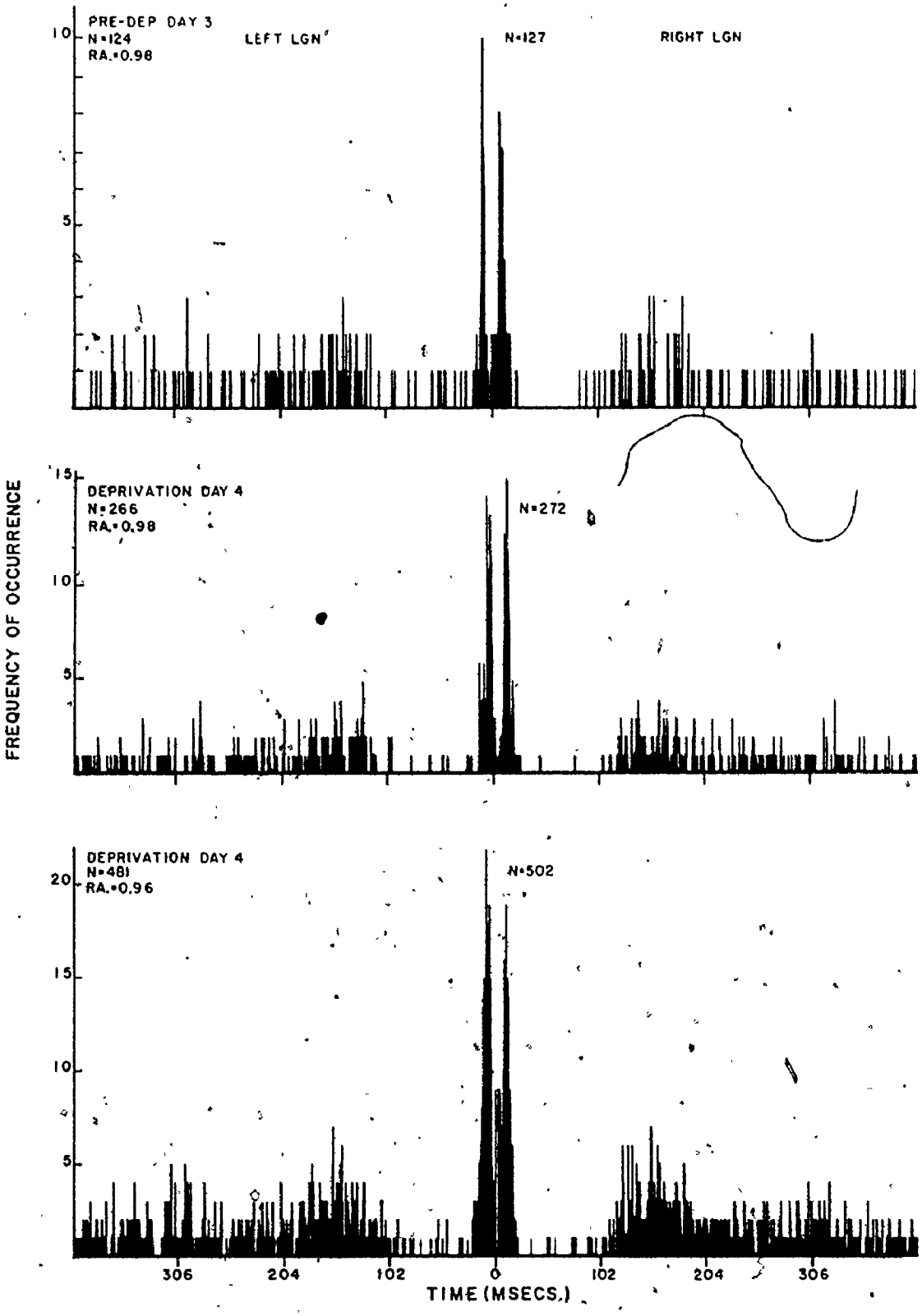

PRE-DEP DAY 3
N=124
RA.=0.98
LEFT LGN
N=127
RIGHT LGN
DEPRIVATION DAY 4
N=266
RA.=0.98
N=272
DEPRIVATION DAY 4
N=481
RA.=0.96
N=502
FREQUENCY OF OCCURRENCE
TIME (MSECS.)
306
204
102
0
102
204
306

Figure 34 - Scatterplot of the relationship between the number of REMs occurring in twenty second epochs of REM sleep and their mean amplitude on pre-deprivation day four and deprivation day three for Cat 007.

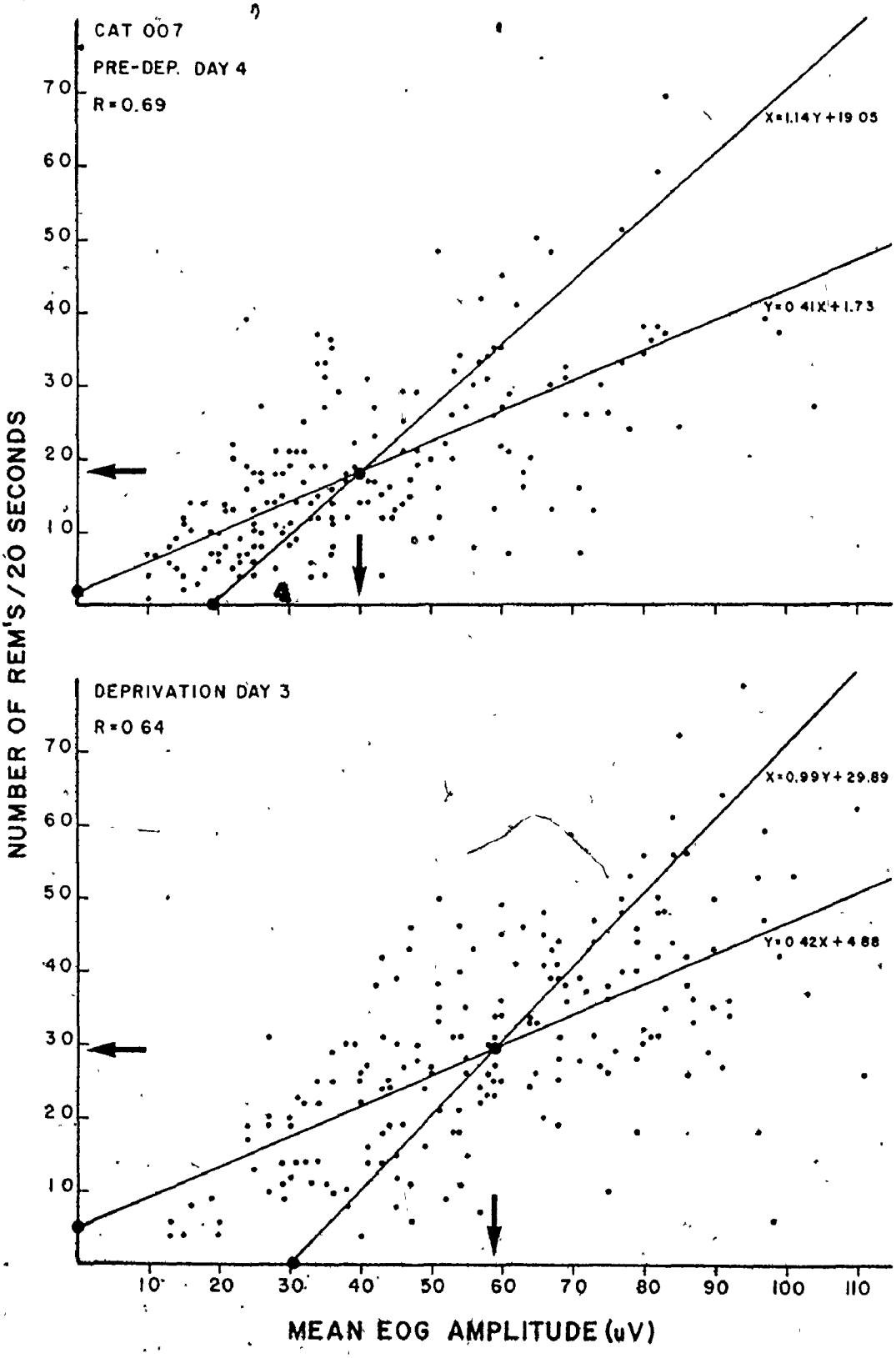

CAT 007
PRE-DEP. DAY 4
R=0.69
X=1.14Y+19.05
Y=0.41X+1.73
DEPRIVATION DAY 3
R=0.64
X=0.99Y+29.89
Y=0.42X+4.88
NUMBER OF REM'S/20 SECONDS
MEAN EOG AMPLITUDE (uV)
10
20
30
40
50
60
70
80
90
100
110

Figure 35 - Scatterplot of the relationship between the number of REMs occurring in twenty second epochs of REM sleep and their mean amplitude on pre-deprivation day nine and deprivation day four for Cat Q03.

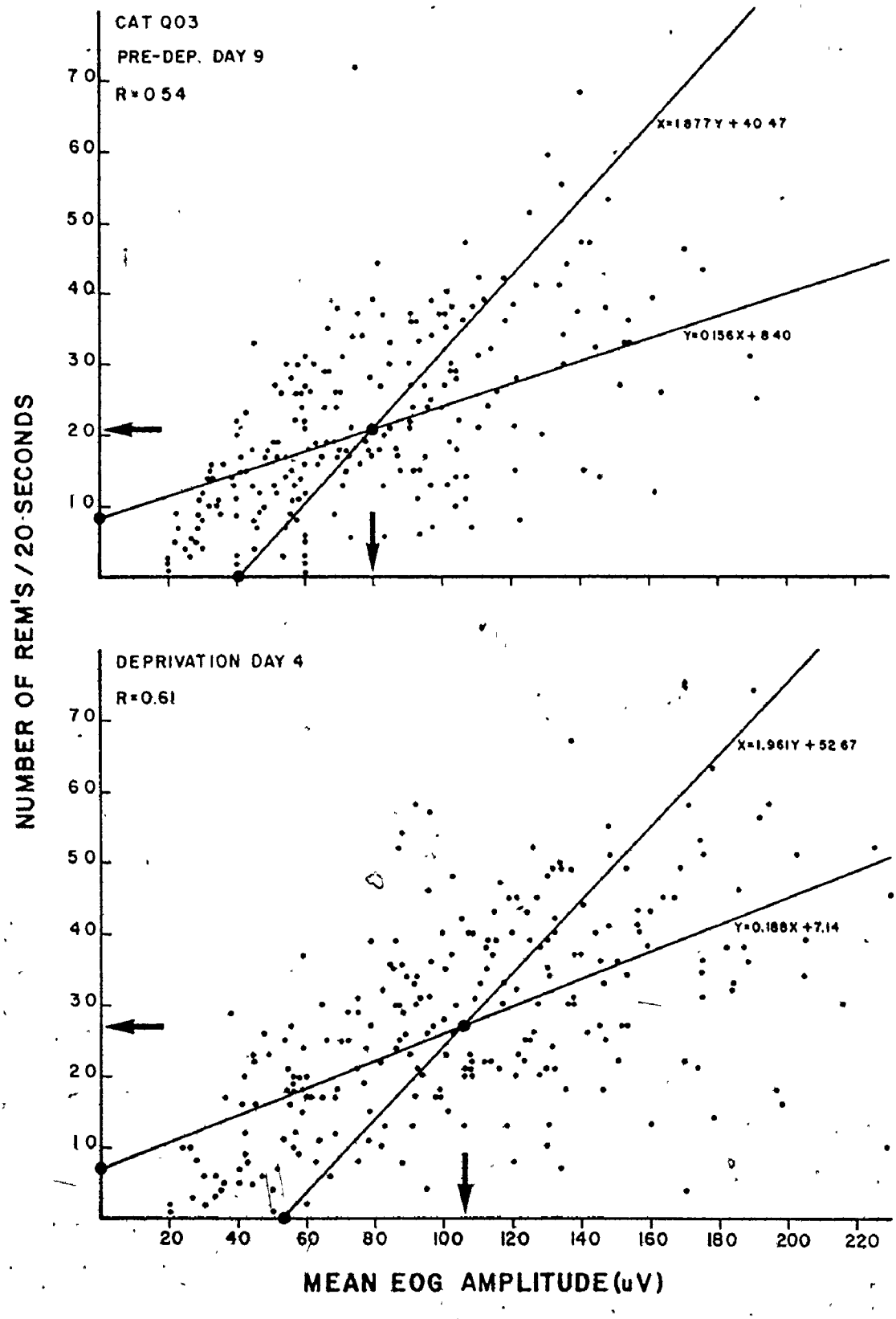

CAT Q03
PRE-DEP. DAY 9
R=0 54
X=1877Y + 40 47
Y=0156X + 8 40
DEPRIVATION DAY 4
R=0.61
X=1.961Y + 52 67
Y=0.188X + 7.14
NUMBER OF REM'S / 20-SECONDS
MEAN EOG AMPLITUDE (uV)
10
20
30
40
50
60
70
20
40
60
80
100
120
140
160
180
200
220

Figure 36 - Scatterplot of the relationship between the number of REMs occurring in twenty second epochs of REM sleep and their mean amplitude on pre-deprivation day three and deprivation day four for Cat 403.

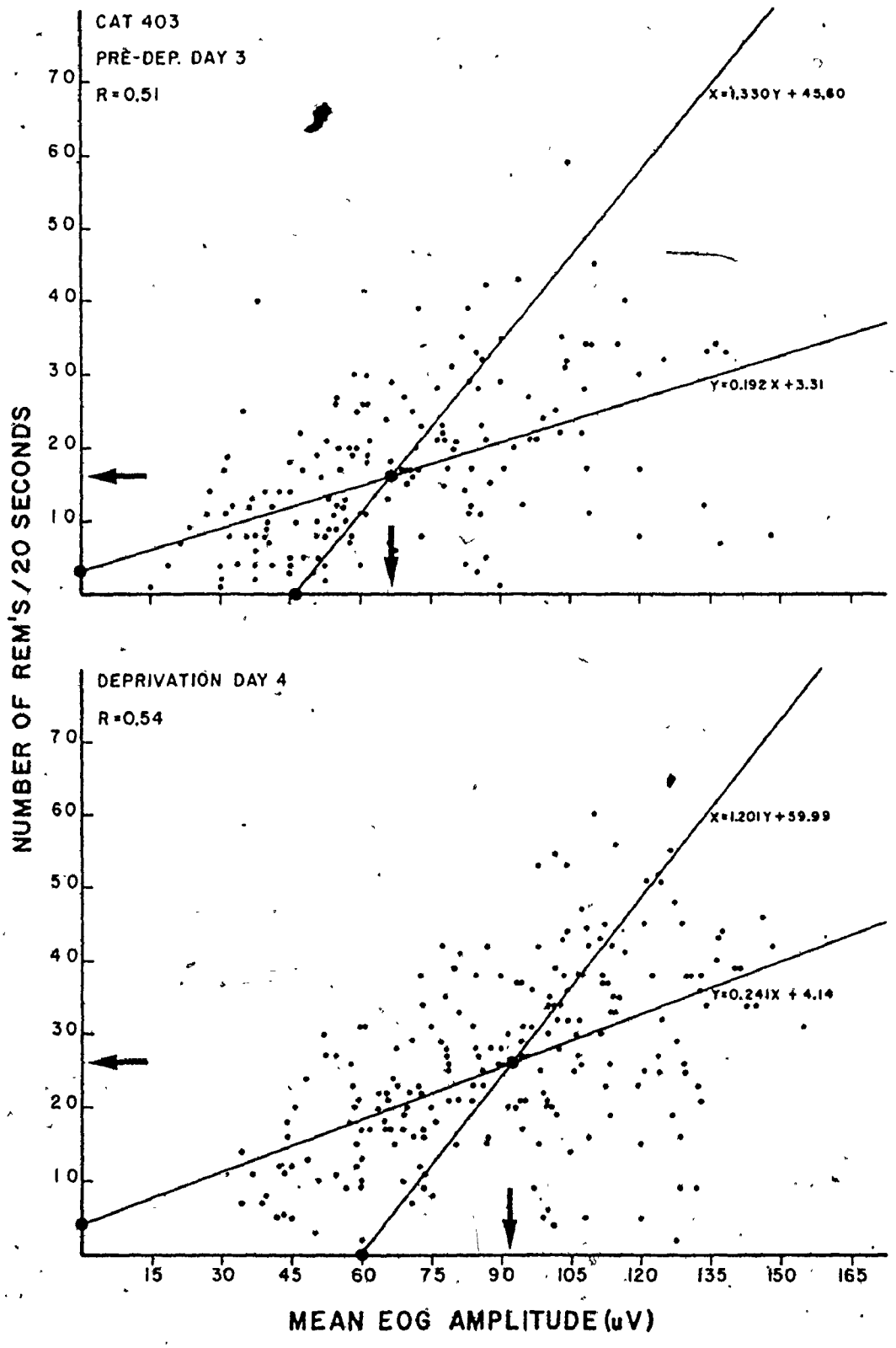

CAT 403
PRE-DEP. DAY 3
R=0.51
X=1.330Y + 45.60
Y=0.192X +3.31
DEPRIVATION DAY 4
R=0.54
X=1.201Y+59.99
Y=0.241X + 4.14
NUMBER OF REM'S/20 SECONDS
MEAN EOG AMPLITUDE (uV)
10
20
30
40
50
60
70
15
30
45
60
75
90
105
120
135
150
165

Figure 37 - Scatterplot of the relationship between the number of REMs occurring in twenty second epochs of REM sleep and their mean amplitude on pre-deprivation day three and deprivation day three for Cat 411.

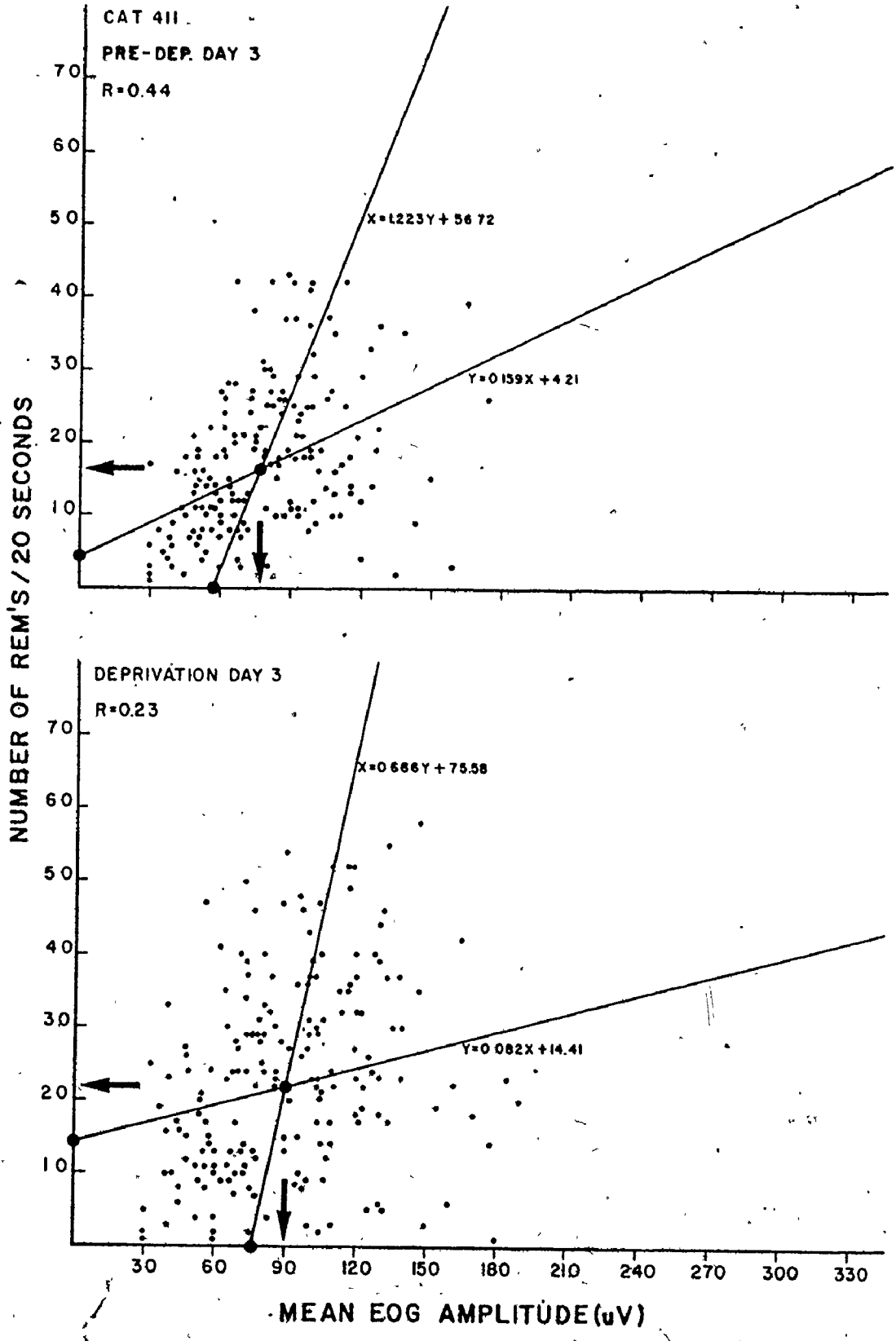

CAT 411
PRE-DEP. DAY 3
R=0.44
X=1.223Y+56.72
Y=0.159X+4.21
DEPRIVATION DAY 3
R=0.23
X=0.666Y+75.58
Y=0.082X+14.41
NUMBER OF REM'S / 20 SECONDS
MEAN EOG AMPLITUDE (uV)
10
20
30
40
50
60
70
30
60
90
120
150
180
210
240
270
300
330

Figure 38 - Frequency distribution of the amplitude of REMs occurring during an eight hour sample taken on pre-deprivation days three and nine and deprivation day four for Cats 403 and Q03 respectively.

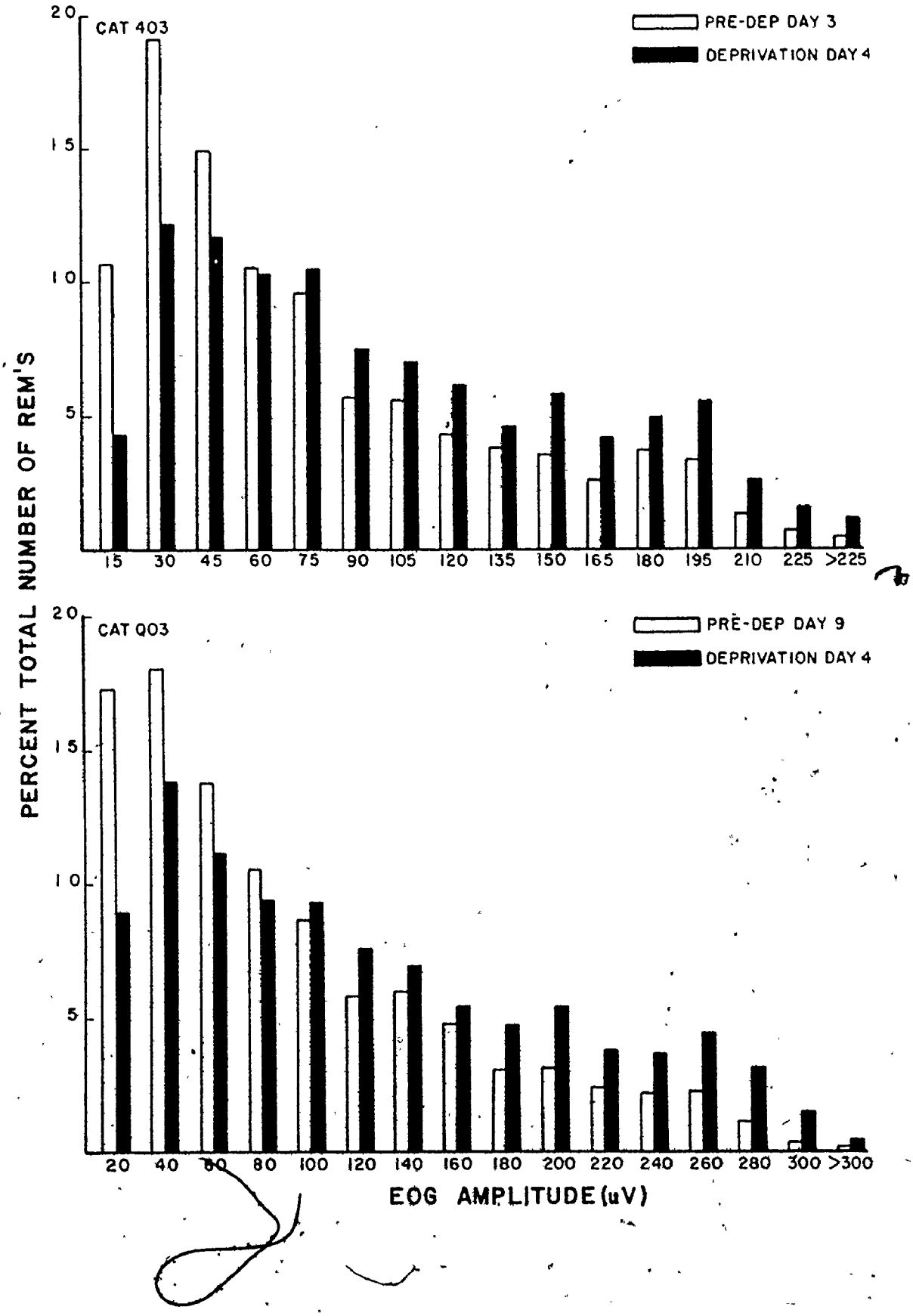

CAT 403
PRE-DEP DAY 3
DEPRIVATION DAY 4
CAT 003
PRE-DEP DAY 9
DEPRIVATION DAY 4
PERCENT TOTAL NUMBER OF REM'S
EOG AMPLITUDE (uV)
20
15
10
5
15 30 45 60 75 90 105 120 135 150 165 180 195 210 225 >225
20 40 60 80 100 120 140 160 180 200 220 240 260 280 300 >300

Figure 39 - Frequency distribution of the amplitude of REMs occurring during an eight hour sample taken on pre-deprivation days four and three and on deprivation day three for Cats 007 and 411 respectively.

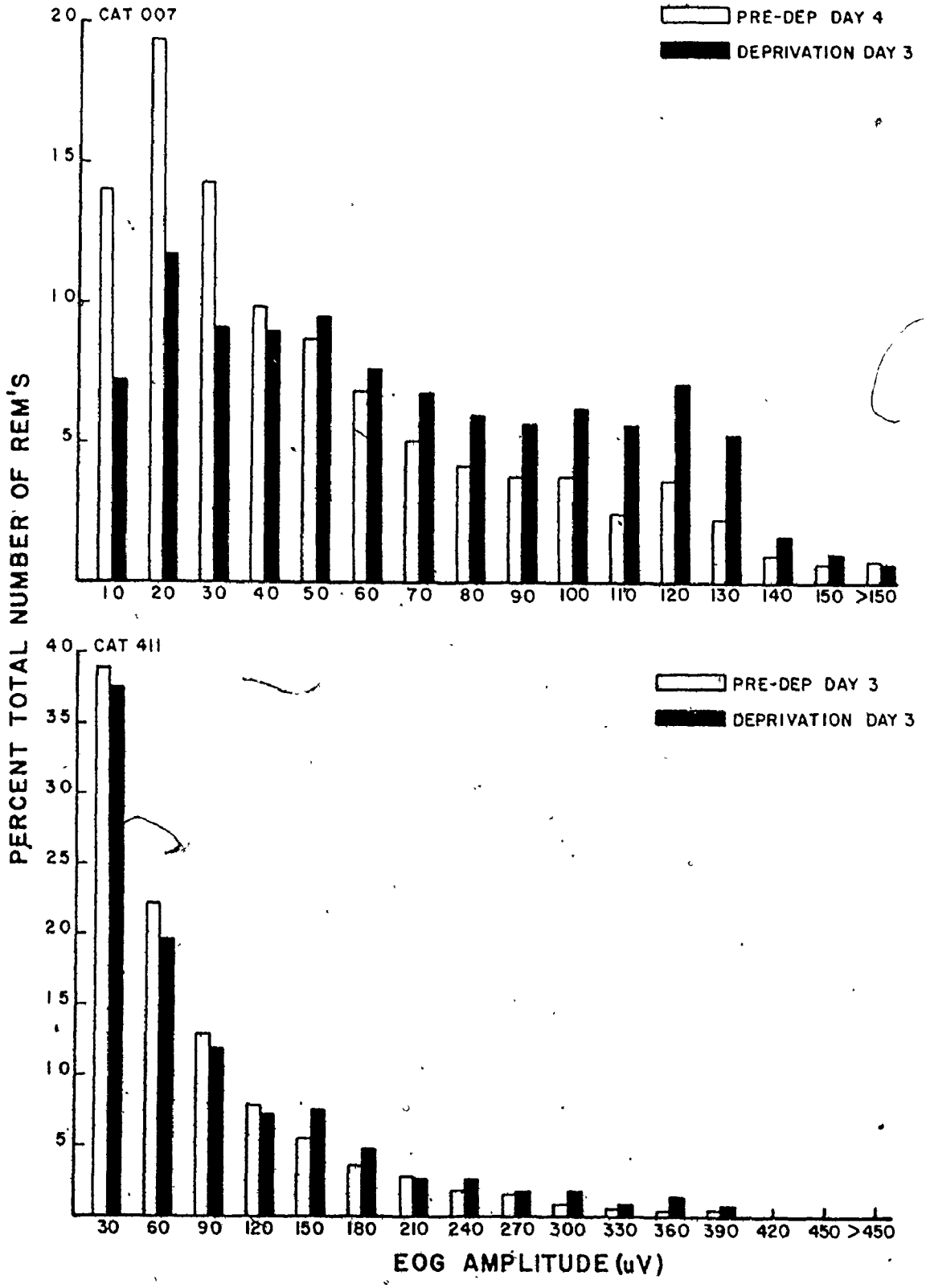

CAT 007
PRE-DEP DAY 4
DEPRIVATION DAY 3
20
15
10
5
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 >150
CAT 411
PRE-DEP DAY 3
DEPRIVATION DAY 3
40
35
30
25
20
15
10
5
30 60 90 120 150 180 210 240 270 300 330 360 390 420 450 >450
PERCENT TOTAL NUMBER OF REM'S
EOG AMPLITUDE (uV)

Figure 40 - Relationship between the frequency of hippocampal theta rhythm during REM sleep and the presence of both REMs and PGO waves (Type 1 sample), PGO waves alone (Type 2 sample) and neither REMs nor PGO waves (Type 3 sample). (All samples occurred within twenty seconds of each other under pre-deprivation conditions).

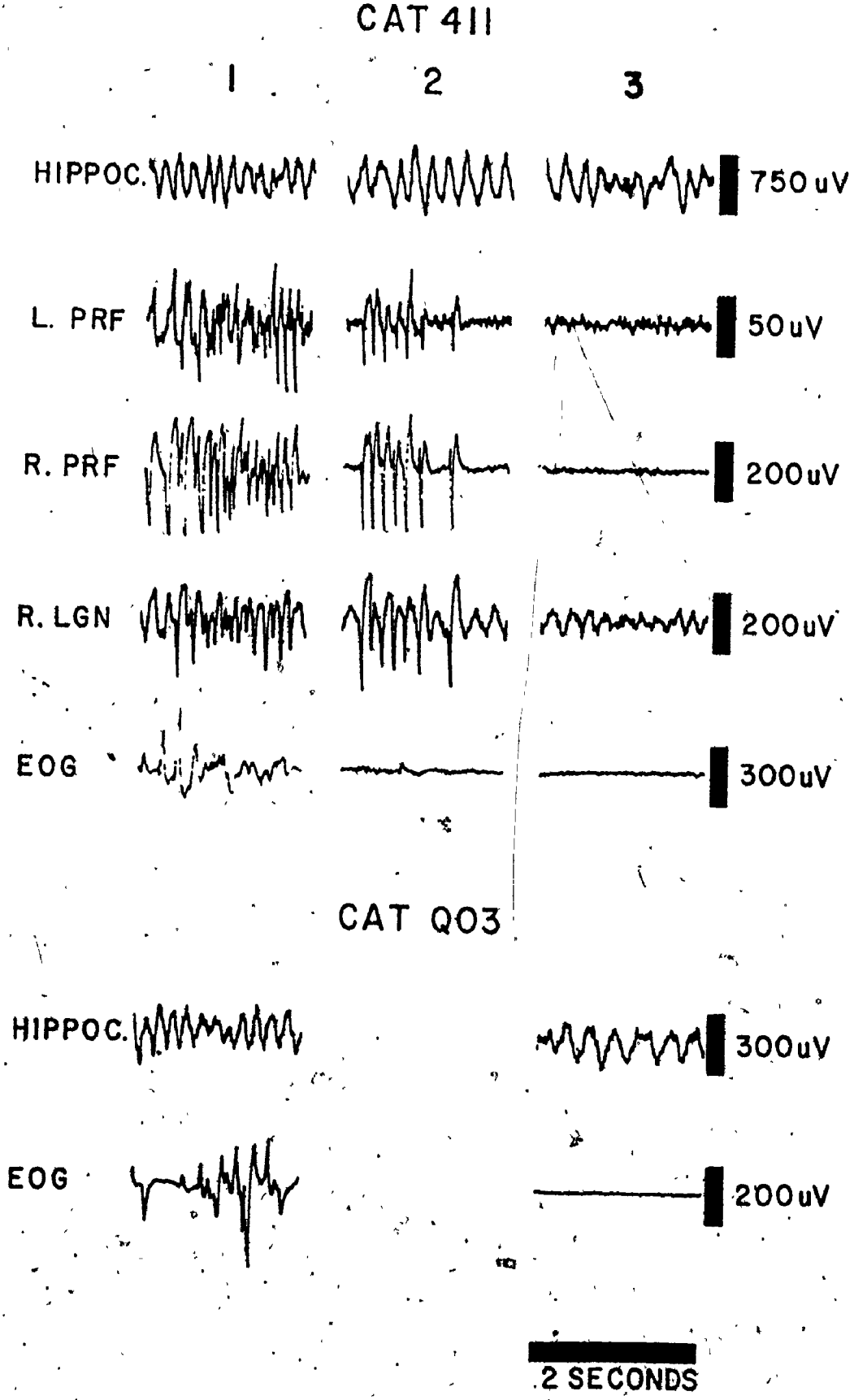

CAT 411
1
2
3
HIPPOC.
750 uV
L. PRF
50uV
R. PRF
200uV
R. LGN
200uV
EOG
300uV
CAT Q03
HIPPOC.
300uV
EOG
200uV
.2 SECONDS

Figure 41 - Location of the tip of the hippocampal electrode in Cat Q03 which was used in the theta frequency analysis. (Brain section redrawn from Berman, 1968.)

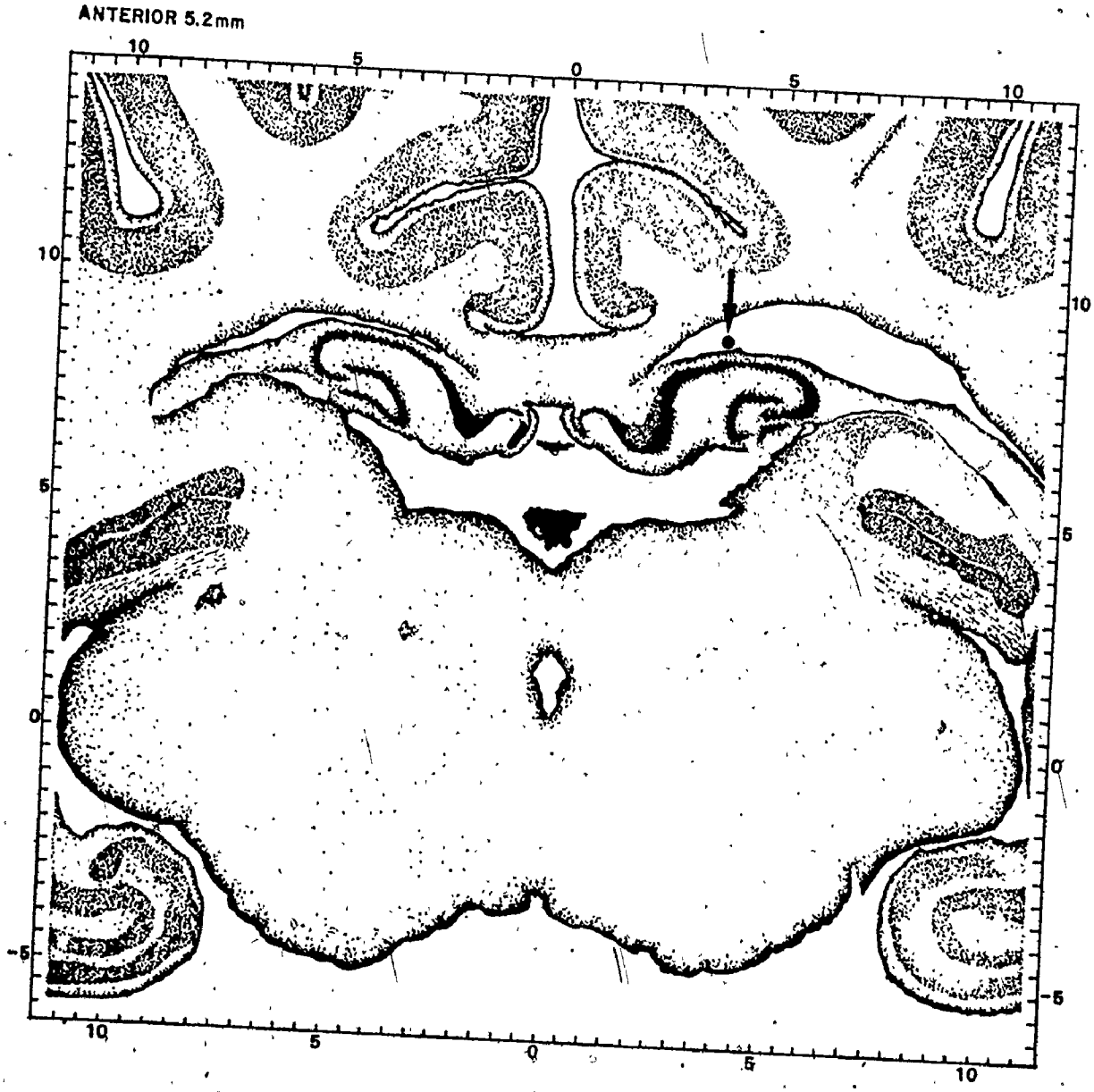

ANTERIOR 5.2mm
10
5
0
5
10
10
5
0
-5
10
5
0
5
10

Figure 42 - Location of the tip of the hippocampal electrode in Cat 411 which was used in the theta frequency analysis. (Brain section redrawn from Berman, 1968.)


ANTERIOR 5.2mm
10
5
0
5
10
10
5
0
-5
-5
10
5
0
5
10

Figure 43 - Bilateral location of the tips of the LGN electrodes in Cat 403 which were used to record PGO waves during REM sleep. (Brain section redrawn from Berman, 1968.)

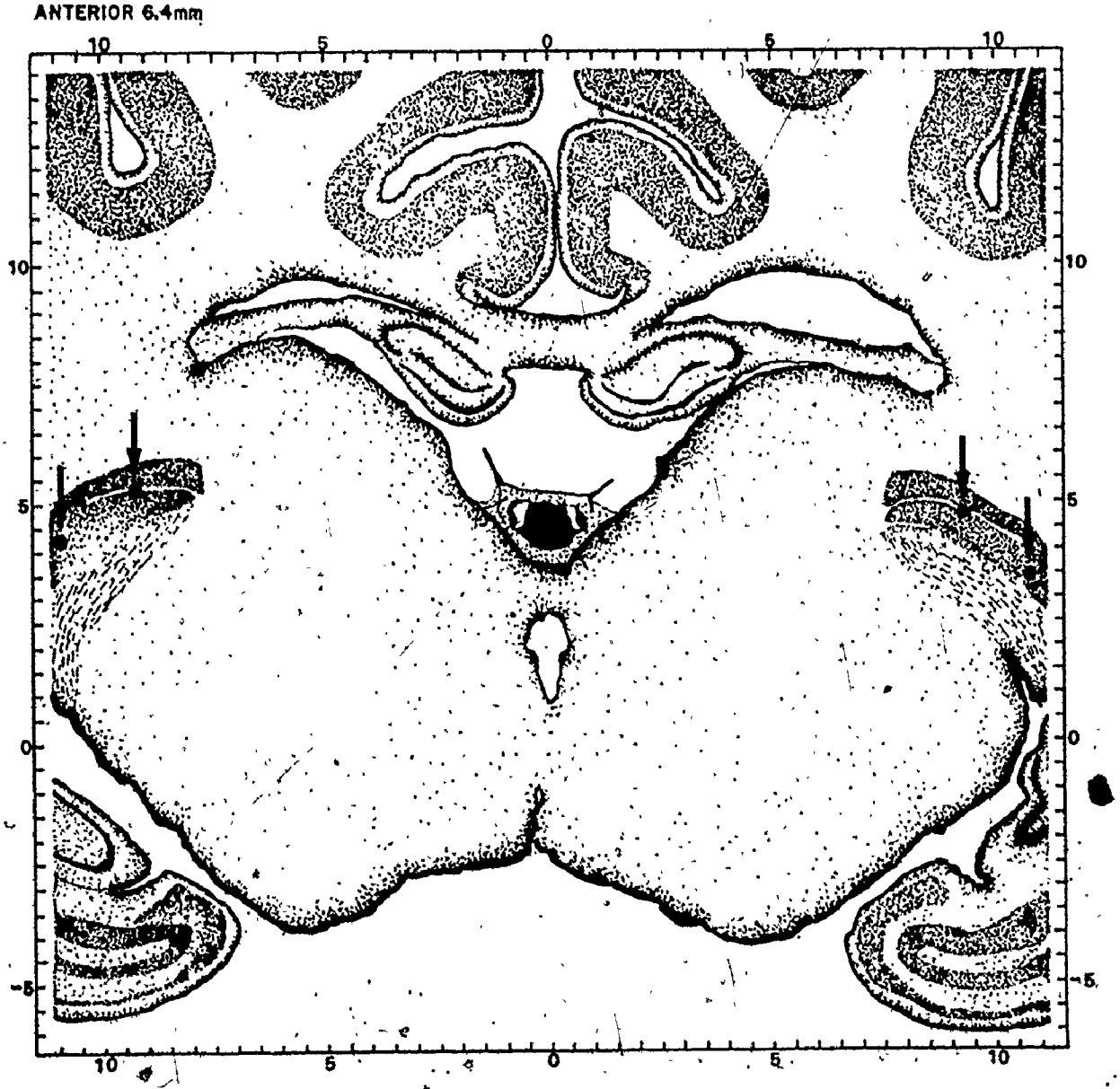

ANTERIOR 6.4mm
10
5
0
5
10
10
5
0
-5

Figure 44 - Location of the tips of the LGN electrodes in Cat 411 used to record PGO waves during REM sleep. (Brain section redrawn from Berman, 1968.)

ANTERIOR 6.4mm

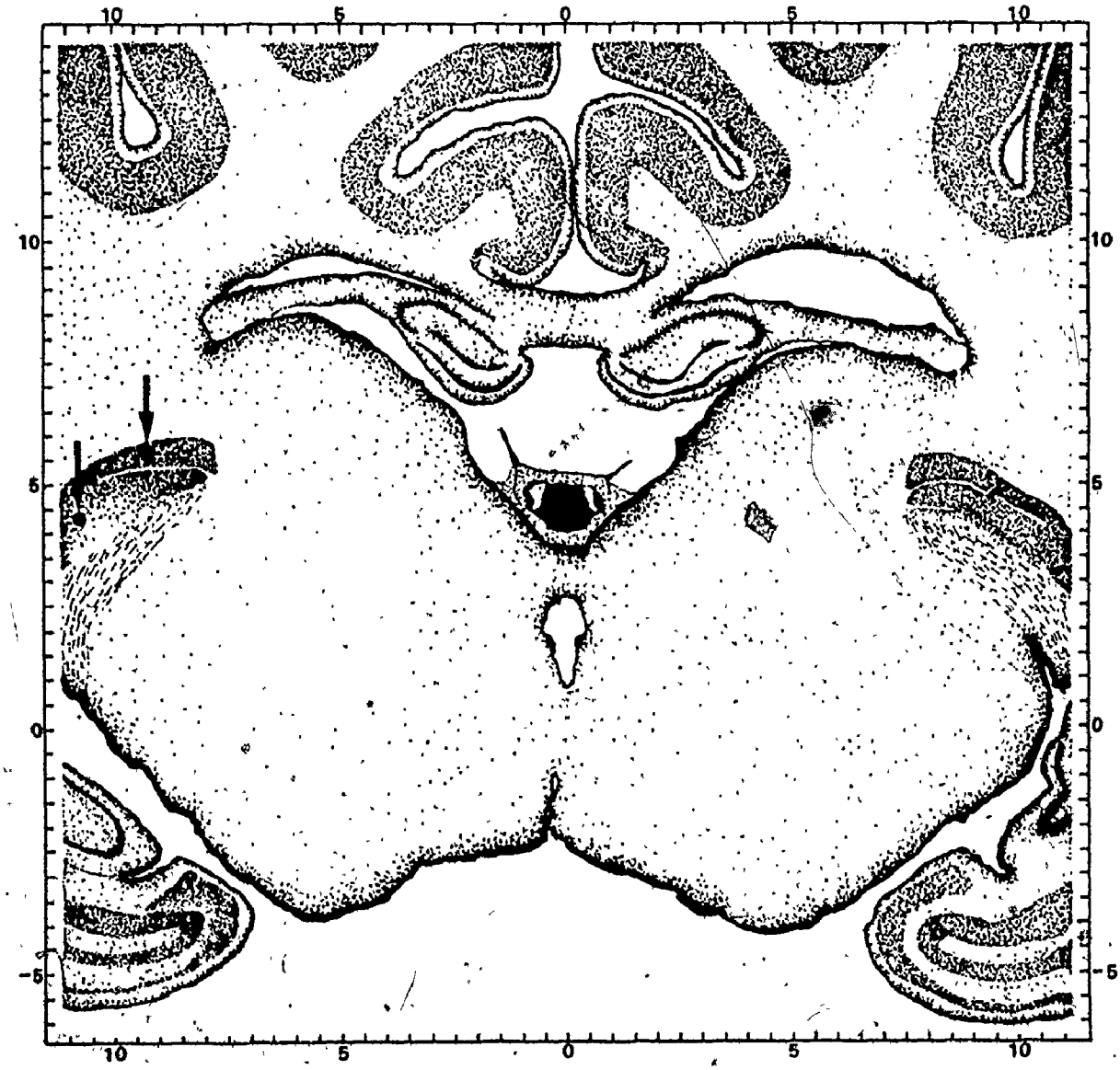


Figure 45 - Bilateral location of the tips of the PRF electrodes in Cat 411 used to record PGO waves during REM sleep. (Brain section redrawn from Berman, 1968.)

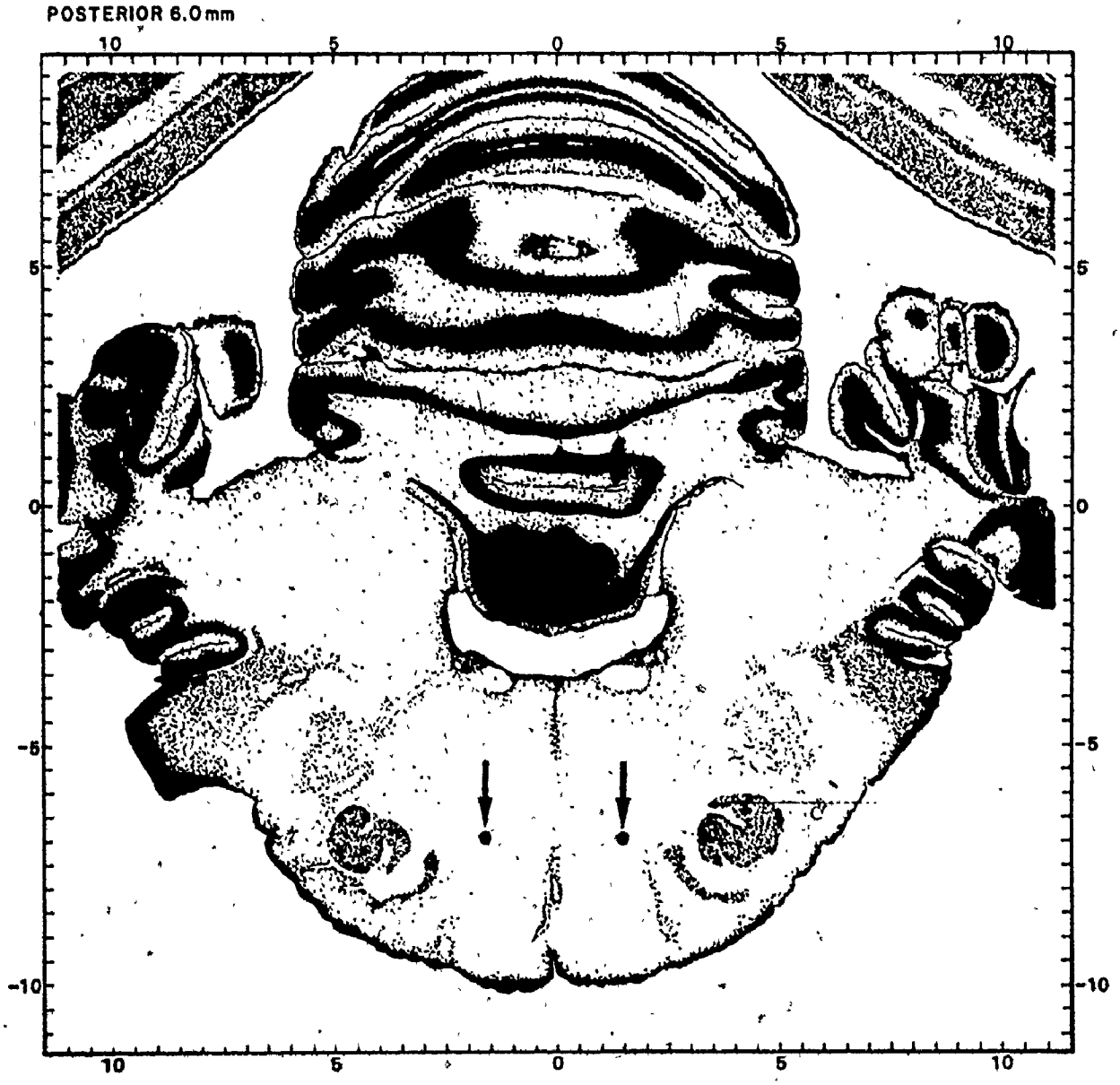

POSTERIOR 6.0mm
10
5
0
5
10
5
0
-5
-10

Figure 46 - PGO waves recorded from the LGN and pontine reticular formation of two cats after pre-treatment with reserpine.

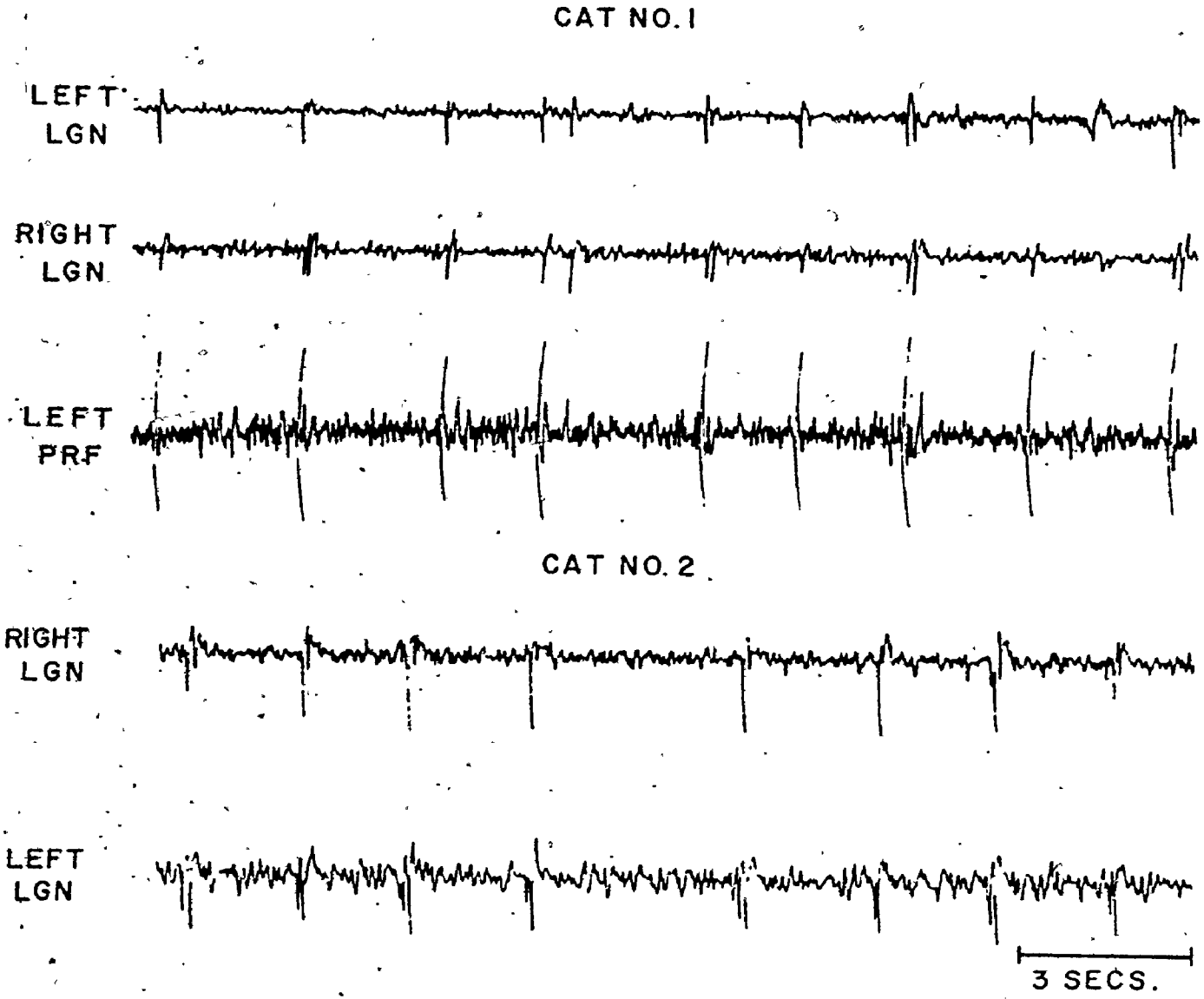
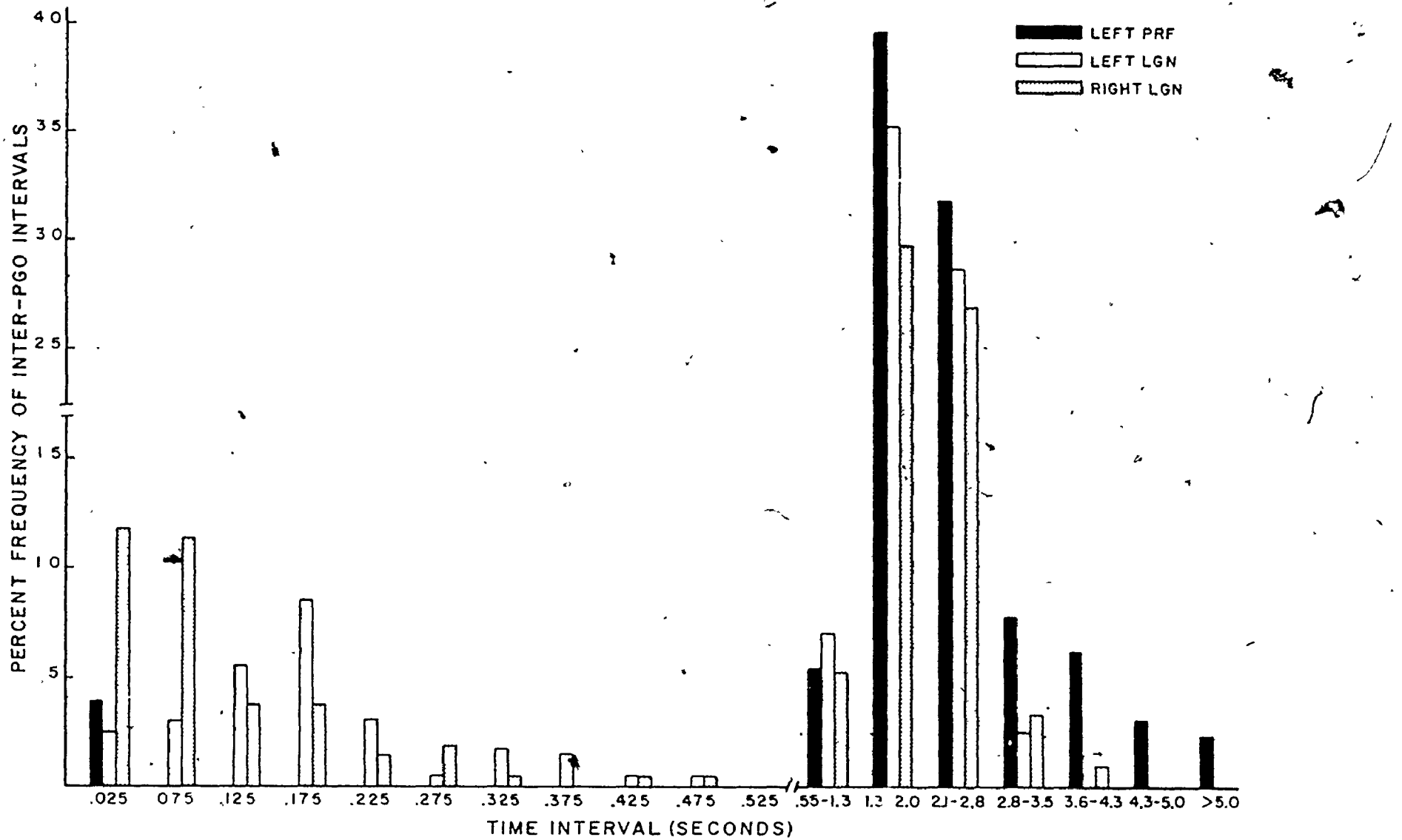

CAT NO. 1
LEFT LGN
RIGHT LGN
LEFT PRF
CAT NO. 2
RIGHT LGN
LEFT LGN
3 SECS.

Figure 47 - Time interval histograms of the interval between the successive occurrence of PGO waves recorded from the right LGN and the right and left PRF of an anaesthetized cat which had been pre-treated with reserpine (Data from Cat No. 1).


LEFT PRF
LEFT LGN
RIGHT LGN
PERCENT FREQUENCY OF INTER-PGO INTERVALS
40
35
30
25
15
10
5
.025
075
.125
.175
.225
.275
.325
.375
.425
.475
.525
.55-1.3
1.3
2.0
2.1-2.8
2.8-3.5
3.6-4.3
4.3-5.0
>5.0
TIME INTERVAL (SECONDS)

EXPERIMENT III

In Experiment I, some clear effects of sensory deprivation on the sleep of adult cat were demonstrated. In order to determine how sensory deprivation caused these changes to occur, it was first necessary to discover the minimal conditions needed to produce these effects. Experiment III was designed with this objective in mind. Since the deprivation procedure used in Experiment I not only prevented the animal from receiving any patterned visual or auditory input but also caused a reduction in the intensity of light reaching the eye and an increase in the amount of auditory stimulation, it was important to determine whether the effects of this procedure on sleep were due to (a) the loss of patterned visual and auditory input; (b) the change in the intensity of light reaching the eye; (c) the change in the amount of auditory stimulation; or (d) the fact that the eyelid was held closed over the eyeball.

The human experiments suggested that changes in light intensity was not an important variable since the light diffusing mask used in those experiments did not cause a large reduction in light intensity (Potter & Heron, 1972). In fact, there is some evidence indicating that total light deprivation may actually have less of an effect on some physiological parameters than does patterned visual deprivation (Zubek &

Welsh, 1963).

At the outset, it also seemed unlikely that the effect was due to eyelid suture per se since the masks worn in the human experiment did not interfere with eyelid movement. However, there was a tendency for human subjects to leave their eyes closed when wearing a mask of this type (personal observation).

Thus, by a process of elimination, one is left with the hypothesis that cats in Experiment I slept more during deprivation because patterned visual and auditory stimulation was not available to them. This hypothesis is more compelling if one views sleep as a passive phenomenon that occurs whenever the arousing effect of external or internal perturbances abate (See Moruzzi, 1964 for the historical development of this idea). Consistent with this hypothesis is the observation that deafferented animals, animals which are disturbed less frequently by external or internal sources of stimulation, sleep considerably more than normal animals (Hagamen, 1959; Vital-Durand & Michel, 1971). Since this additional sleep time is spent in LSWS without any increase in DSWS or REM sleep (Vital-Durand & Michel, 1971), an obvious parallel exists between the effects of sensory deprivation and chronic deafferentation. Therefore, in addition to trying to determine the sensory deprivation conditions needed to produce the sleep effects, a comparison of effect of visual

deprivation with the effect of chronic visual deafferentation was sought.

METHOD

Cats Q03, 403 and 411 from the lid suture experiment were subsequently run in other conditions. These conditions were similar to the lid suture experiment in that they consisted of a pre-deprivation, deprivation and post-deprivation period of continuous EEG recording. The deprivation period was different in that one of the following treatments was administered.

Condition A: Lid Suture

Experiment I.

Condition B: Light Deprivation

The animal was placed in total darkness and subjected to 78 db white noise during the entire deprivation period.

Condition C: Lid Suture and Light Deprivation

The animal was placed in total darkness with its eyelids sutured shut and exposed to 78 db white noise for the entire period.

Condition D: Goggles

The animal was habituated to wearing a set of goggles (Figure 48, Top photograph) which were mounted in front of the cat's eyes by means of two aluminum rods inserted into aluminum tubing that was embedded in dental cement on the cat's head (Figure 49). The goggles were left on continuously except for one hour a day at feeding time. During pre-

deprivation and post-deprivation, transparent, 3 mm. thick, lucite screens were in place (Figure 48, Top photograph) while during deprivation, white translucent teflon screens were used (Figure 48, Bottom photograph). This screen was illuminated at a fairly uniform intensity (Figure 50) by a light bulb inserted in a hole in the back of the goggles (Figure 51, Top photograph). A Ganzfeld was achieved by shining the point source of light emitted by the light bulb through a ping pong ball hemisphere (Figure 51, Bottom photograph) onto the teflon screen. This intensity was selected so as to match the intensity of light calculated to reach the eye through sutured eyelids in Experiment I. A regulated power supply was used to illuminate the light bulbs in order to eliminate the effects of fluctuation in line voltage and a precision ten turn potentiometer was used to rheostatically control the intensity of light emitted at the screen's surface. Light intensity was calibrated against the potentiometer setting by using a brightness spot meter (Model UB ½; Photo Research Corp., Hollywood, California) in reference to a standard source of known intensity.

During deprivation, the goggles were removed for a one hour period at feeding time, as during pre-deprivation, but the animal was left in total darkness. The 78 db white noise, however, was left on as usual.

Condtion E: White Noise Plus Blindness

The animal was blinded under general anaesthesia by making a slit in the cornea and removing the entire contents of the eyeball. The eyeball was packed with gelfoam and the eyelids were then sutured together. Three weeks later, the eyelid sutures were removed. By this time the cornea incision had closed and the eyeball appeared to be normally inflated. Pre-deprivation recording was begun a week later. Deprivation was initiated simply by turning on the 78 db white noise.

Condition F: Blindness Plus Eyelid Suture

Deprivation under this condition consisted of sewing the eyelids of the blind animal shut under gas anaesthesia and then subjecting the animal to a 78 db white noise.

These conditions were not run in the same order for every animal, and conditions B, C and E were only done for Cat 403. A minimum of two weeks elapsed between conditions, and in some cases, it was as long as a month (i.e. Condition E). Records were scored using the procedures described in Experiment I but statistical analysis was minimized since there were too few subjects in each condition. In the following result section, the effects of these conditions on SWS time and REM density were emphasized since these two parameters were the ones most influenced in the lid suture experiment.

RESULTS

Comparing the effects of lid suture and total light deprivation on the percent time spent in SWS (Figures 52, 53, 54; Conditions A & B) indicates that while light deprivation may cause a small increase in SWS time, it is not of the magnitude observed following lid suture (Table XXX). In the one experiment where eyelid suture was coupled with light deprivation (Figure 53C), the increase in SWS time was comparable to that observed following lid suture alone. In the goggle condition, where the intensity of light reaching the eye following lid suture was approximated, the percent time spent in SWS remained constant (Figure 53D). The effects of being exposed to white noise after having been blind for one month were inconsistent across animals. On deprivation day 1, Cat Q03 had the largest increase in SWS time observed in any of these experiments (Figure 52E; Figure 55), although there was a trend toward pre-deprivation levels on deprivation days 2 and 3. Cat 403, on the other hand, underwent a slight decrease in SWS time on the first few days of deprivation (Figure 53E).

The pattern of the results observed for REM density was similar to that found for the percent time spent in SWS. Thus, while the effect of lid suture on REM density was large and reliably obtained, the effect of total light deprivation

was negligible (Figure 56; Figure 57, Conditions A & B, Table XXXI). However, when eyelid suture was coupled with light deprivation, REM density increased in a fashion similar to that observed following eyelid suture alone (Compared Condition A & C of Figure 57). The goggle condition produced a result similar to that observed following light deprivation if the result of the first day of light deprivation was disregarded (Figure 57D). Exposing Cat 403 to white noise after it had been blind for one month had no effect on REM density (Figure 57E) but it should be noted that this animal did not change its sleep time under these conditions either (Figure 53E). Eyelid suture performed six weeks after the animal was blinded (Figure 57F) did not have as large, or as sustained an effect on REM density as was observed when eyelid suture was performed before blinding (Figure 57A & C).

Finally, it should be noted that the effect of lid suture on either REM density or the percent time spent in SWS did not depend on the order in which the conditions were run. Thus, Cat 411 had the usual increase in REM density (Figure 56A) and SWS time (Figure 54A) following lid suture despite the fact that this animal was subjected to the light deprivation condition prior to being run in the lid suture condition. Similarly, Cat 403 had an increase in REM density and SWS time following lid suture plus light deprivation equivalent to that obtained following lid suture alone (Figures

53 & 57; A & C) even though a light deprivation condition was interposed between these two experiments. Pre-deprivation baseline levels of REM density and sleep-waking parameters fluctuated sightly from one condition to the next, however, and some of the changes in baseline may have been due to the order in which the conditions were administered. As in Experiment I though, REM density changes occurred independent of changes in PGO density (Figure 58). This was true both within and between experimental conditions.

DISCUSSION

The total light deprivation procedure used in this experiment possessed most of the features found in lid suture techniques employed in Experiment I. Thus, patterned vision was prevented, light intensity was reduced and the animal was exposed to the same masking white noise. Despite these similarities, the effects of light deprivation on SWS time and REM density were marginal and would probably fail to reach statistical significance even if a large number of subjects had been run using a completed counterbalanced design. The fact that total light deprivation plus lid suture produced results nearly identical to lid suture alone indicates that the failure of light deprivation to have an effect cannot be attributed to differences between lid suture and light deprivation procedures in terms of how much the intensity of light was reduced during the experimental period. Furthermore, since the same intensity of white noise was used in both these conditions, one can conclude that white noise alone is not responsible for the sleep effects. Finally, the fact that PGO density was about the same during light deprivation alone as it was during lid suture plus light deprivation indicates that light deprivation by itself does not have an effect on the frequency of PGO wave generation. This result rules out the possibility that the effect of light deprivation

on REM density is masked by a simultaneous decrease in PGO density.

By a process of elimination, one is left with the conclusion that lid suture per se is the condition needed in order to obtain the sleep effects. However, this conclusion may be drawn prematurely since the loss of patterned vision achieved by subjecting the animal to total darkness may be fundamentally different from the loss of pattern vision obtained when the animal is forced to view a Ganzfeld through closed eyelids. Thus, the Goggle condition was run to see if the results from Experiment I could be replicated without sewing the cat's eyelids shut. Since this condition would have been critical in distinguishing between the two methods of producing a loss of pattern vision only if the Experiment I results were replicated, the failure to replicate the effect of lid suture may simply mean that the goggle condition did not adequately reproduce the field of view that the animal experienced when it had its eyelids sutured shut. One obvious difference between the field of view the animal had when it was looking at the illuminated screens of the goggles versus when it was seeing through closed eyelids was the fact that with the goggles on, the cat saw a circular disc of light surrounded by a metal ring which was in turn engulfed in darkness. However, if the animal brought its head close to a surface such as the wall or the floor of the cage, the light

reflected off the animal's face may have been bright enough to permit peripheral vision of things in its environment. It might be argued that had precautions been taken to eliminate this type of vision, the goggle condition may have worked.

Another difference between lid suture condition and the light deprivation and goggle conditions was the fact that gas anaesthesia was used only in the lid suture condition. However, the administration of anaesthesia cannot be considered the cause of the sleep effects since the animals in the lid suture experiment were also anaesthetized at the termination of deprivation, yet the sleep parameters rapidly returned to baseline levels. In addition, the human experiments, which produced effects similar to that observed in the cat experiments, used neither anaesthesia nor lid suture.

The fact that a substantial increase in sleep time was observed in the blindness plus white noise condition for Cat Q03 without the use of lid suture or anaesthesia suggests that the parallel between deafferentation and sensory deprivation may provide some insight into the mechanism underlying the sleep effects obtained during sensory deprivation. The failure of one animal to show this type of change was not considered to present a serious difficulty since others have reported that chronically deafferented cats show a large, reliable, sustained increase in SWS time (Hagamen, 1959;

Vital-Durand & Michel, 1971). These studies differ from the present work in that their animals were more completely deafferented (Hagamen's animals were blind, deaf and anosmic, while Vital-Durand & Michel's animals were capable of receiving only proprioceptive input from eye muscles and visceral input carried by the IXth to XIIth cranial nerves). Thus, it seems likely that the blind cats in the present study were able to adjust to partial deafferentation, perhaps by relying more on other sensory modalities. This interpretation is supported by the fact that after one month of visual deafferentation, blind animals were spending a normal proportion of their time in SWS. According to this view, the transient increase in SWS observed in Cat Q03 when white noise was coupled with blindness was due to a temporary failure in the animal's ability to compensate for loss of input.

Table XXX - Mean percent time spent in SWS during pre-deprivation, deprivation and post-deprivation of the lid suture and light deprivation conditions. (Mean ± S.E.).

TABLE XXX

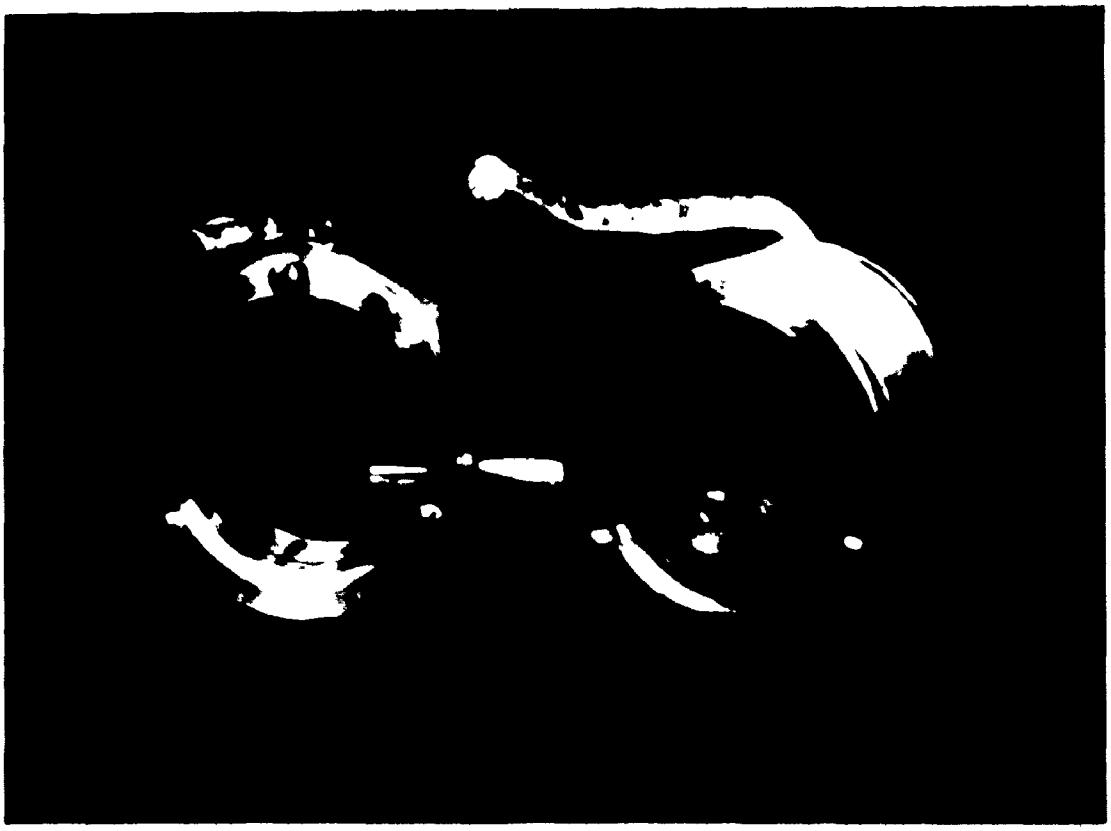
		Pre-Deprivation	Deprivation	Post-Deprivation
Lid Suture	Q03	46.5	57.2	47.2
	403	36.1	40.0	35.6
	411	32.2	36.9	31.7
	$\bar{X}$	38.3	44.7	38.2
	S.E.	4.3	6.3	4.7
Light Deprivation	Q03	42.5	45.9	41.8
	403	32.6	35.1	29.2
	411	33.5	33.9	29.9
	$\bar{X}$	36.2	38.3	33.6
	S.E.	3.2	3.8	4.1

Table XXXI - Average REM density during pre-deprivation, deprivation and post-deprivation of the lid suture and light deprivation conditions.

TABLE XXXI

		Pre-Deprivation	Deprivation	Post-Deprivation
Lid Suture	Q03	53.6	73.6	62.7
	403	45.5	70.3	56.0
	411	42.0	53.8	39.3
	$\bar{X}$	47.0	65.9	52.7
	S.E.	3.4	6.1	7.0
Light Deprivation	Q03	55.9	58.9	53.8
	403	51.9	46.2	47.1
	411	50.5	49.4	52.1
	$\bar{X}$	52.8	51.5	51.0
	S.E.	1.6	3.8	2.0

Figure 48 - Goggles worn by Cat 403 during pre-deprivation and post-deprivation (Top photograph) and during deprivation (Bottom photograph).



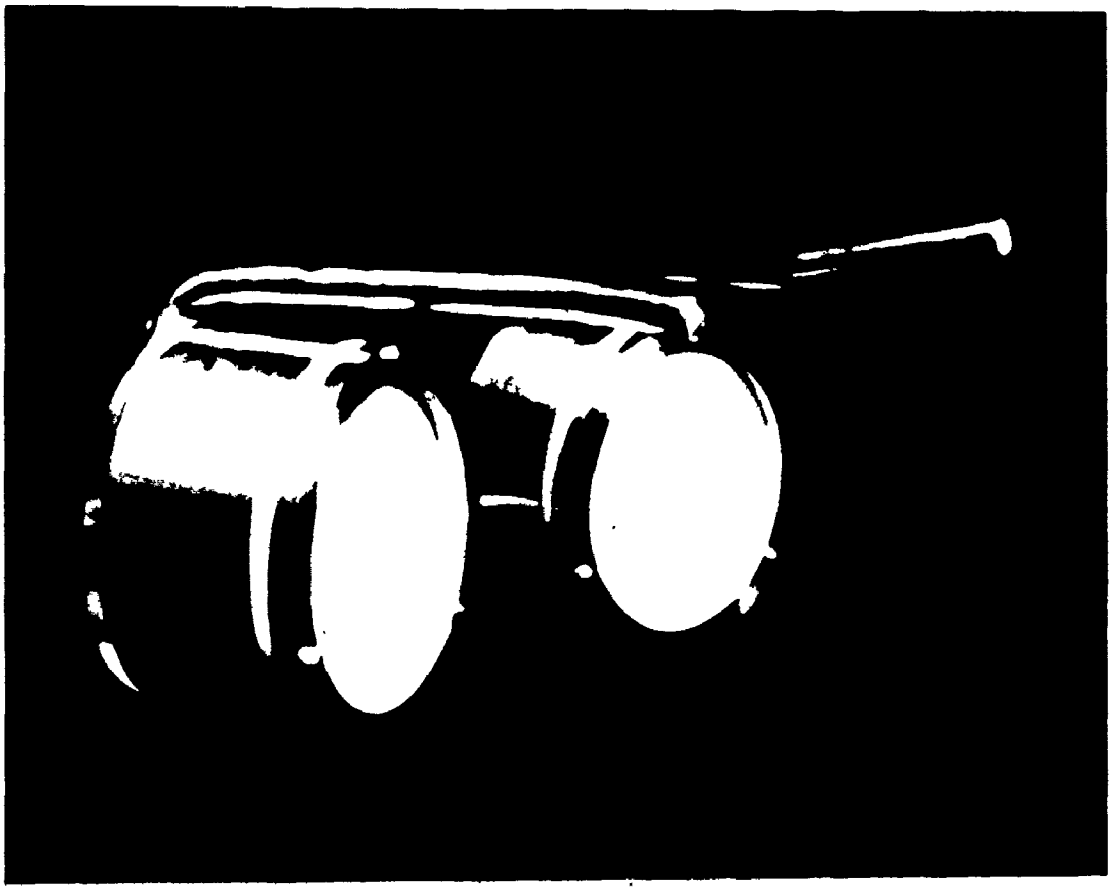


Figure 49 - Cat 403 wearing goggles under pre-deprivation conditions.

Figure 50 - Regional differences in the intensity of light (foot-lamberts) emitted by teflon screen when back illuminated through ping pong ball hemisphere.

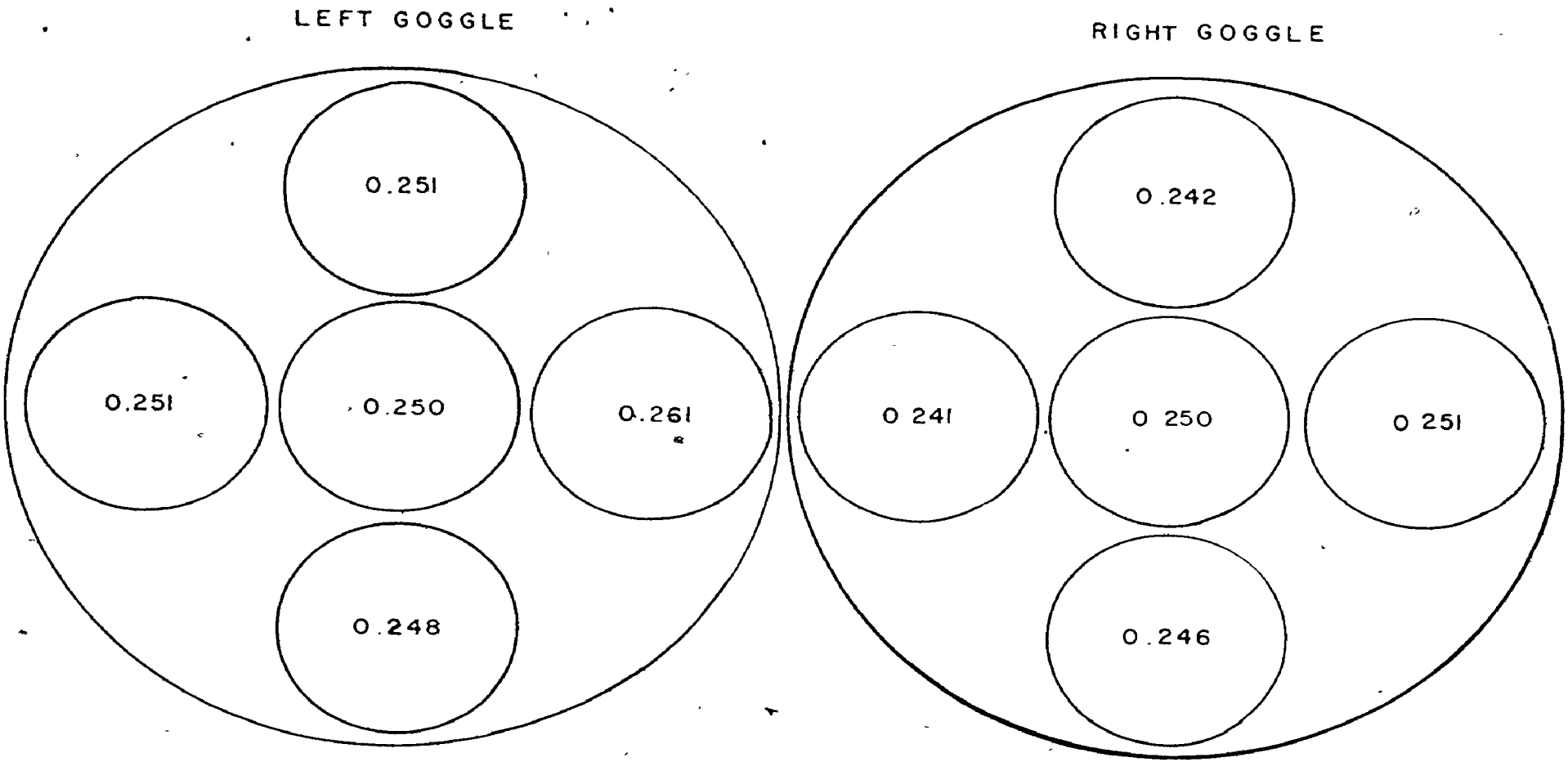

LEFT GOGGLE
0.251
0.251
0.250
0.261
0.248
RIGHT GOGGLE
0.242
0 241
0 250
0 251
0.246

Figure 51 - Internal structure of goggles worn by Cat 403 during deprivation.

Figure 52 - Percent time spent in different stages of wakefulness and sleep by Cat Q03 compared across conditions.

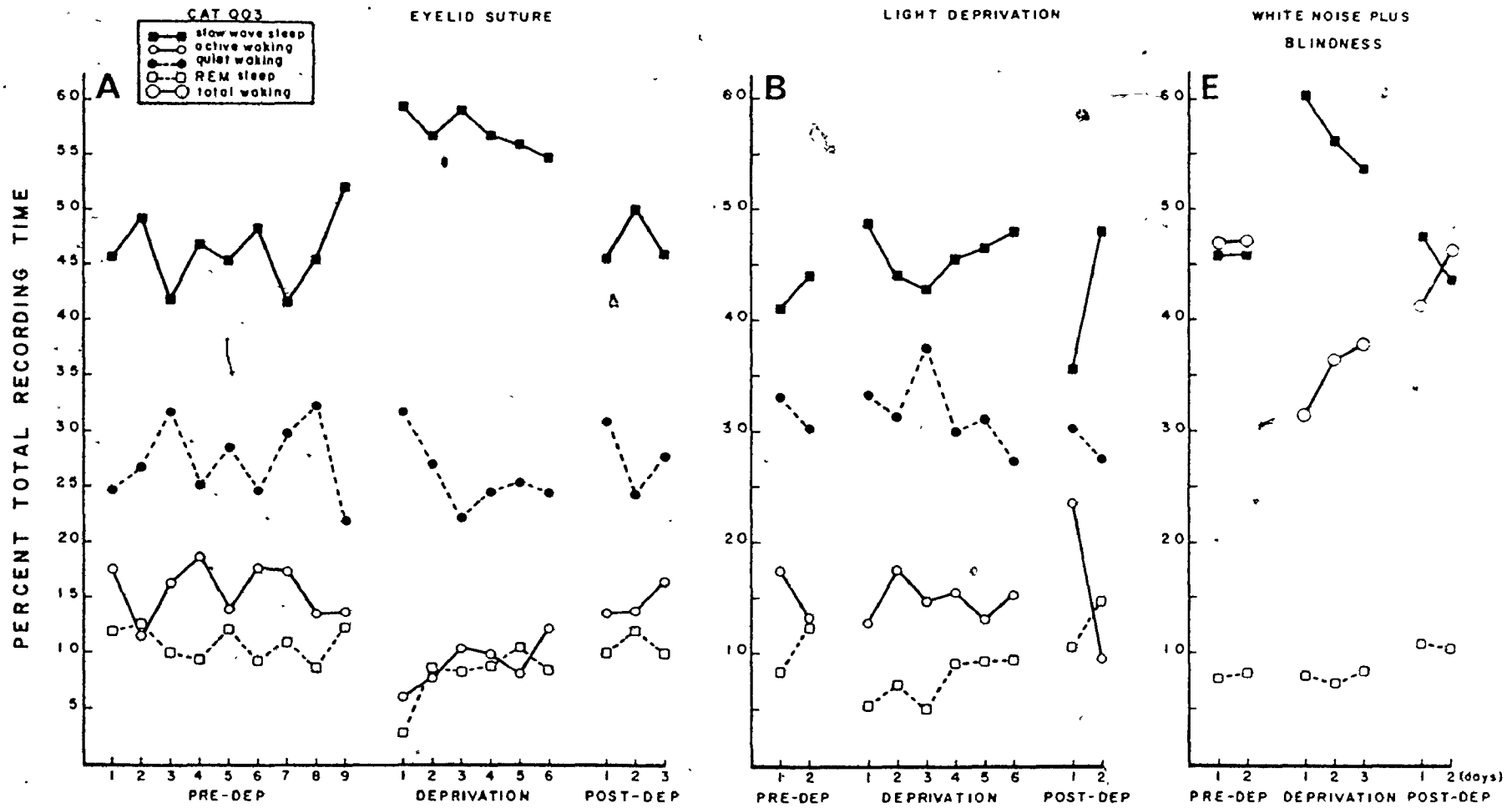

CAT 903
slow wave sleep
active waking
quiet waking
REM sleep
total waking
EYELID SUTURE
LIGHT DEPRIVATION
WHITE NOISE PLUS
BLINDNESS
A
B
E
PERCENT TOTAL RECORDING TIME
PRE-DEP
DEPRIVATION
POST-DEP
(days)

Figure 53 - Percent time spent in different stages of wakefulness and sleep by Cat 403 compared across conditions.

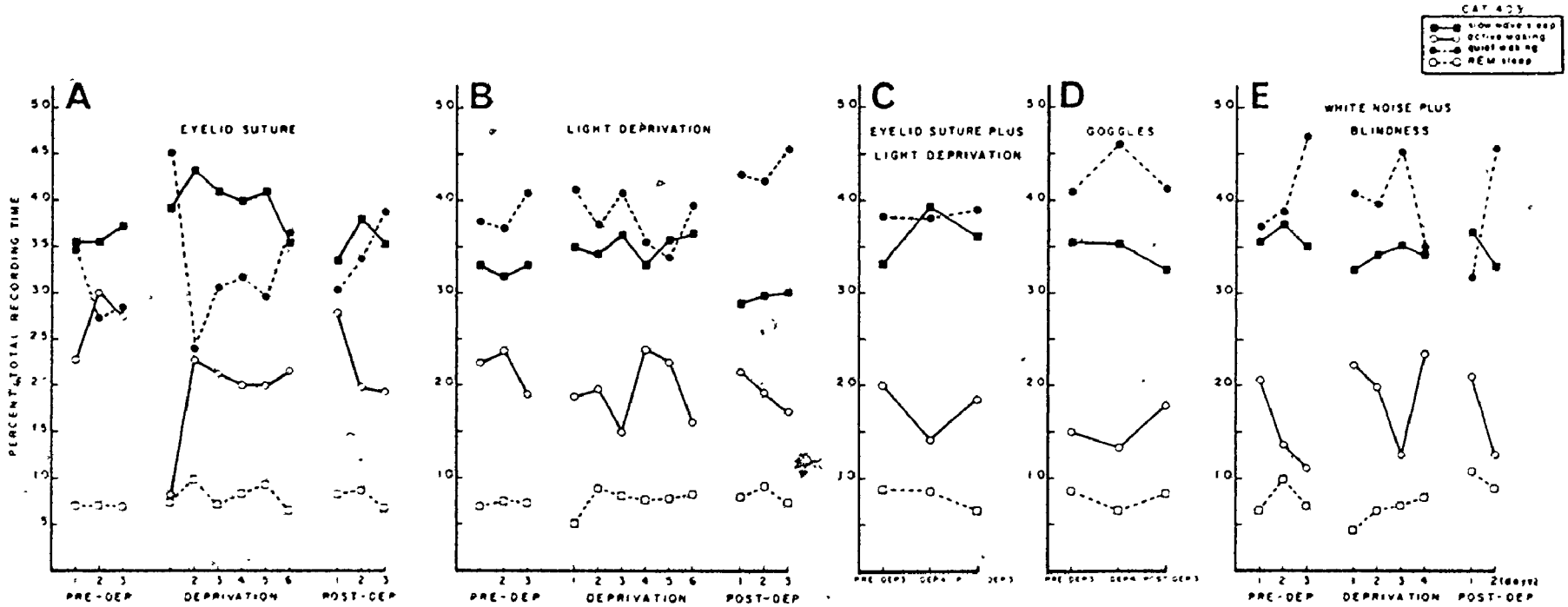

CAT 403
A
EYELID SUTURE
B
LIGHT DEPRIVATION
C
EYELID SUTURE PLUS
LIGHT DEPRIVATION
D
GOGGLES
E
WHITE NOISE PLUS
BLINDNESS
PERCENT TOTAL RECORDING TIME
PRE-DEP
DEPRIVATION
POST-DEP

Figure 54 - Percent time spent in different stages of wakefulness and sleep by Cat 411 compared across conditions.

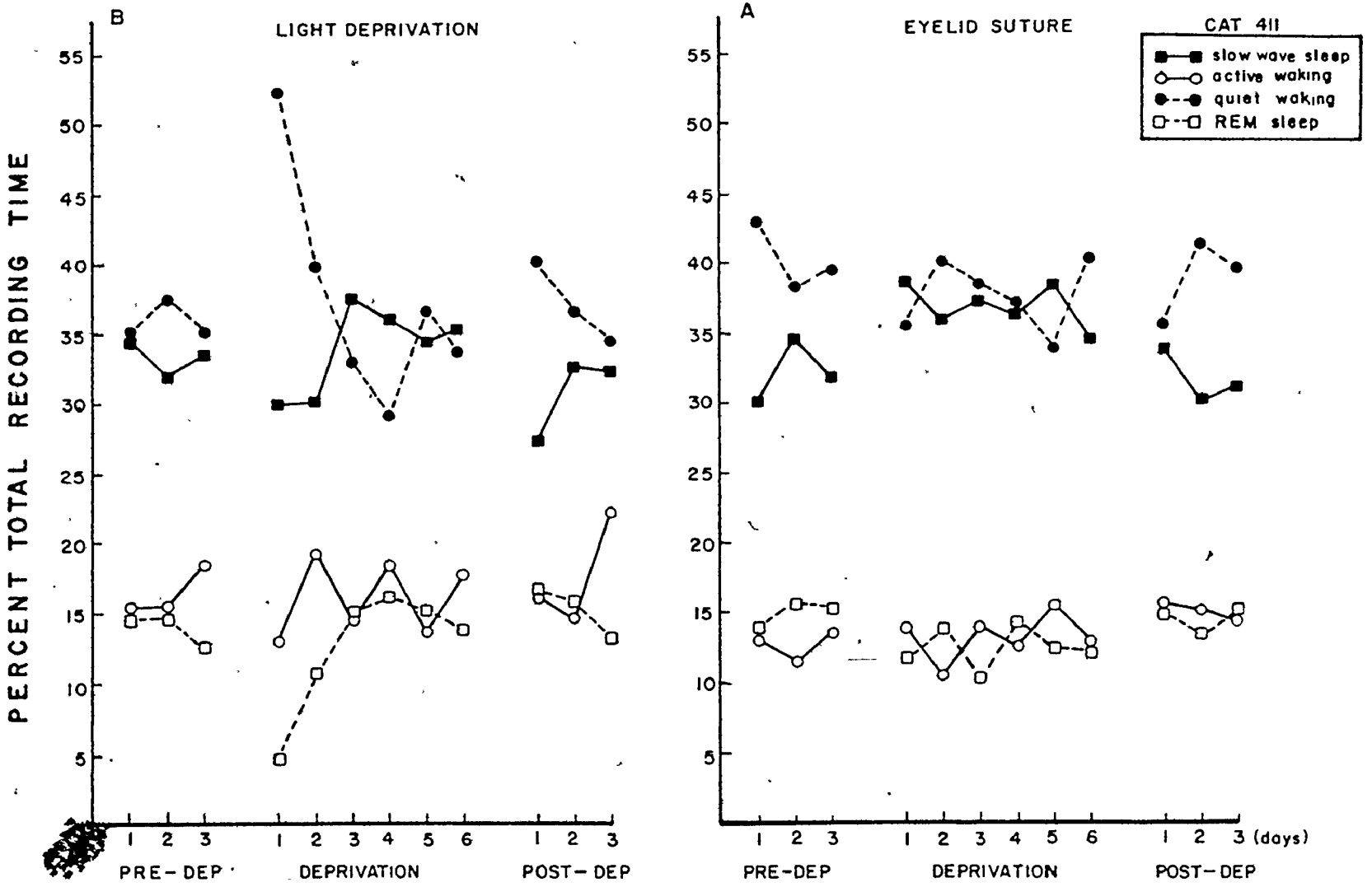

B
LIGHT DEPRIVATION
A
EYELID SUTURE
CAT 411
slow wave sleep
active waking
quiet waking
REM sleep
PERCENT TOTAL RECORDING TIME
55
50
45
40
35
30
25
20
15
10
5
1 2 3
1 2 3 4 5 6
1 2 3
PRE-DEP
DEPRIVATION
POST-DEP
(days)

Figure 55 - Sleep-waking pattern of Cat Q03 on pre-deprivation day two and deprivation days one and three (A = Active waking, Q = Quiet waking, S = Slow wave sleep, R = REM sleep).

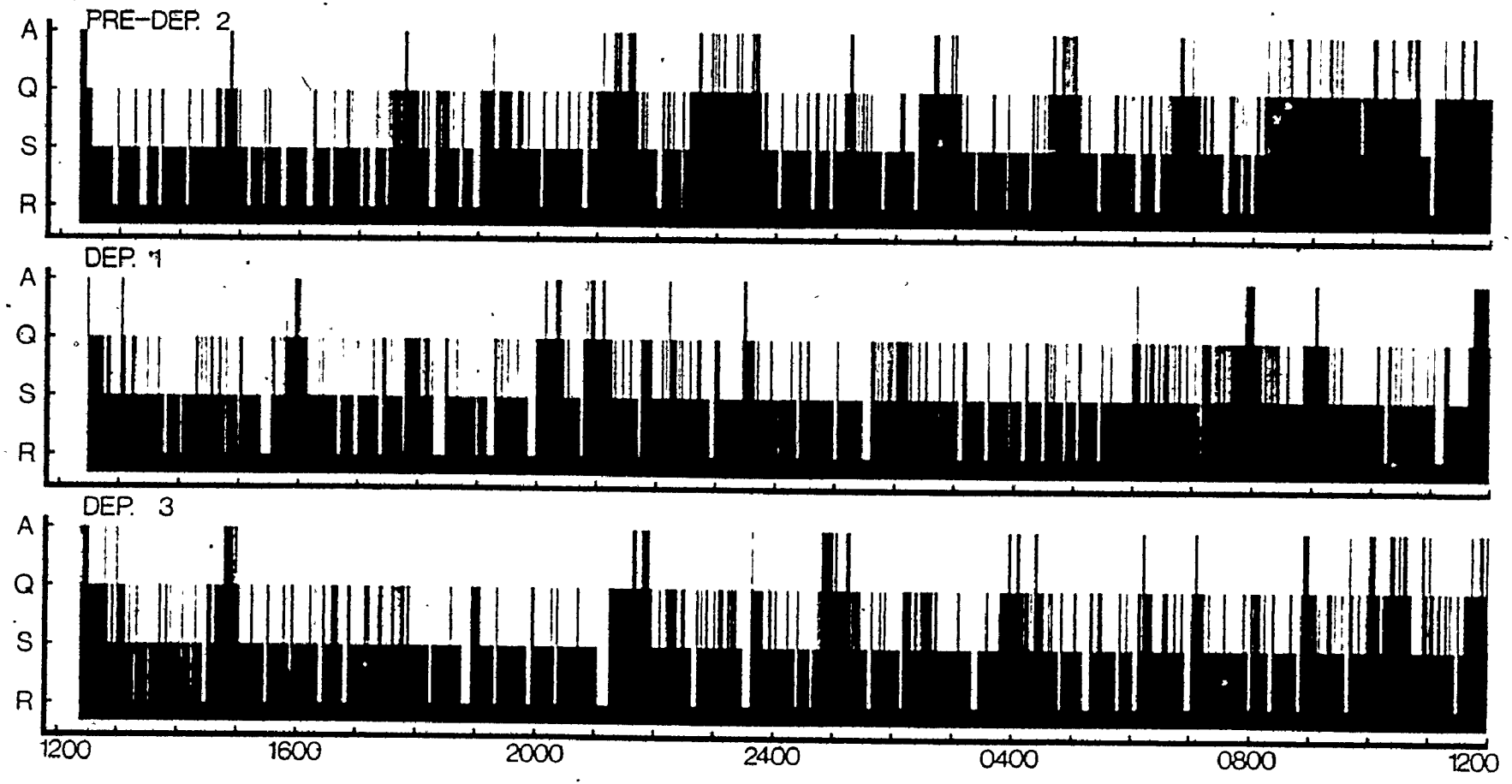

PRE-DEP. 2
A
Q
S
R
DEP. 1
A
Q
S
R
DEP. 3
A
Q
S
R
1200
1600
2000
2400
0400
0800
1200

Figure 56 - Average REM density of Cats Q03 (Top diagram) and 411 (Bottom diagram) compared across conditions.

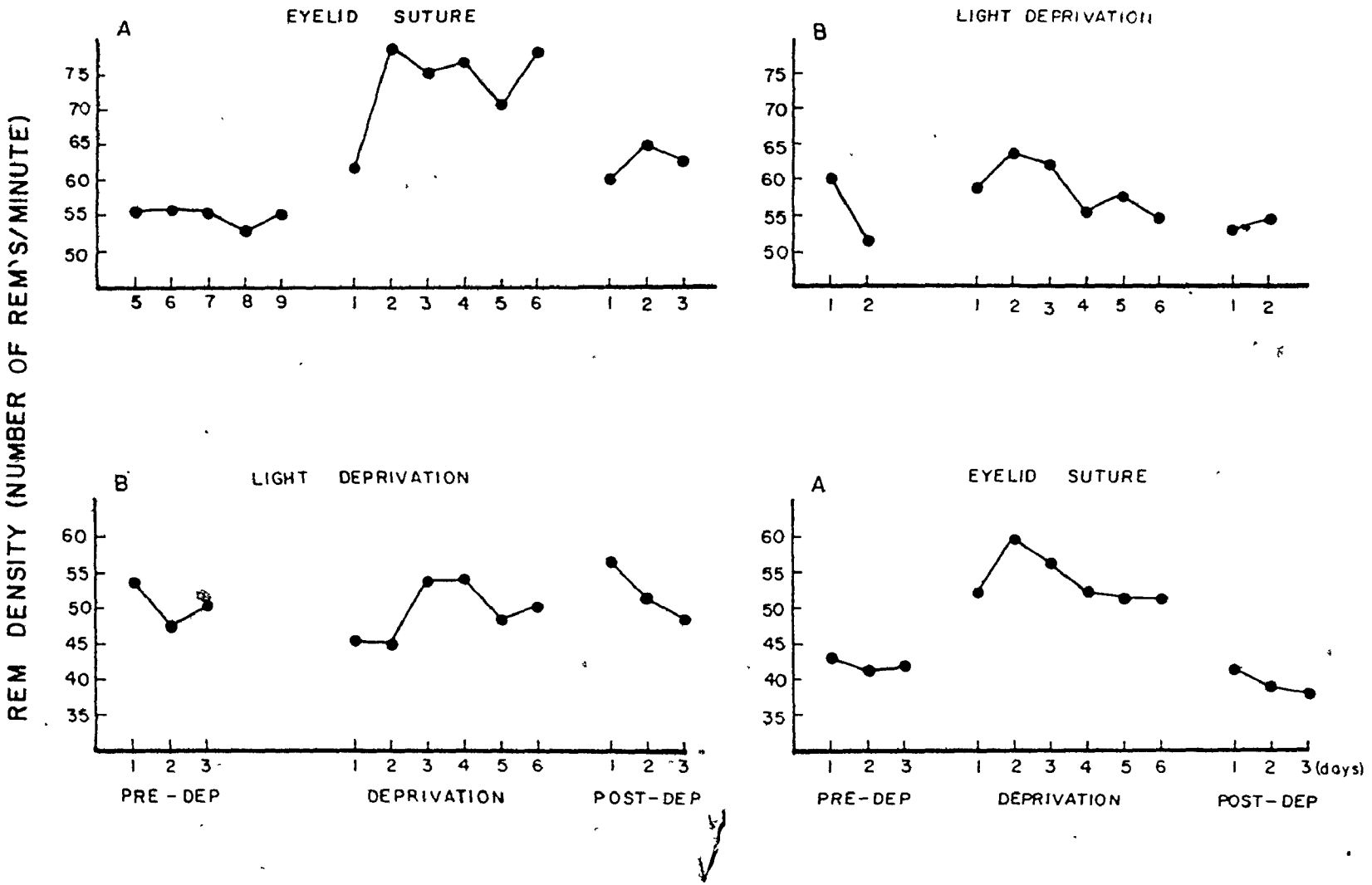

REM DENSITY (NUMBER OF REM'S/MINUTE)
A
EYELID SUTURE
B
LIGHT DEPRIVATION
B
LIGHT DEPRIVATION
A
EYELID SUTURE
PRE-DEP
DEPRIVATION
POST-DEP
PRE-DEP
DEPRIVATION
POST-DEP
(days)

Figure 57 - Average REM density of Cat 403 compared across conditions.

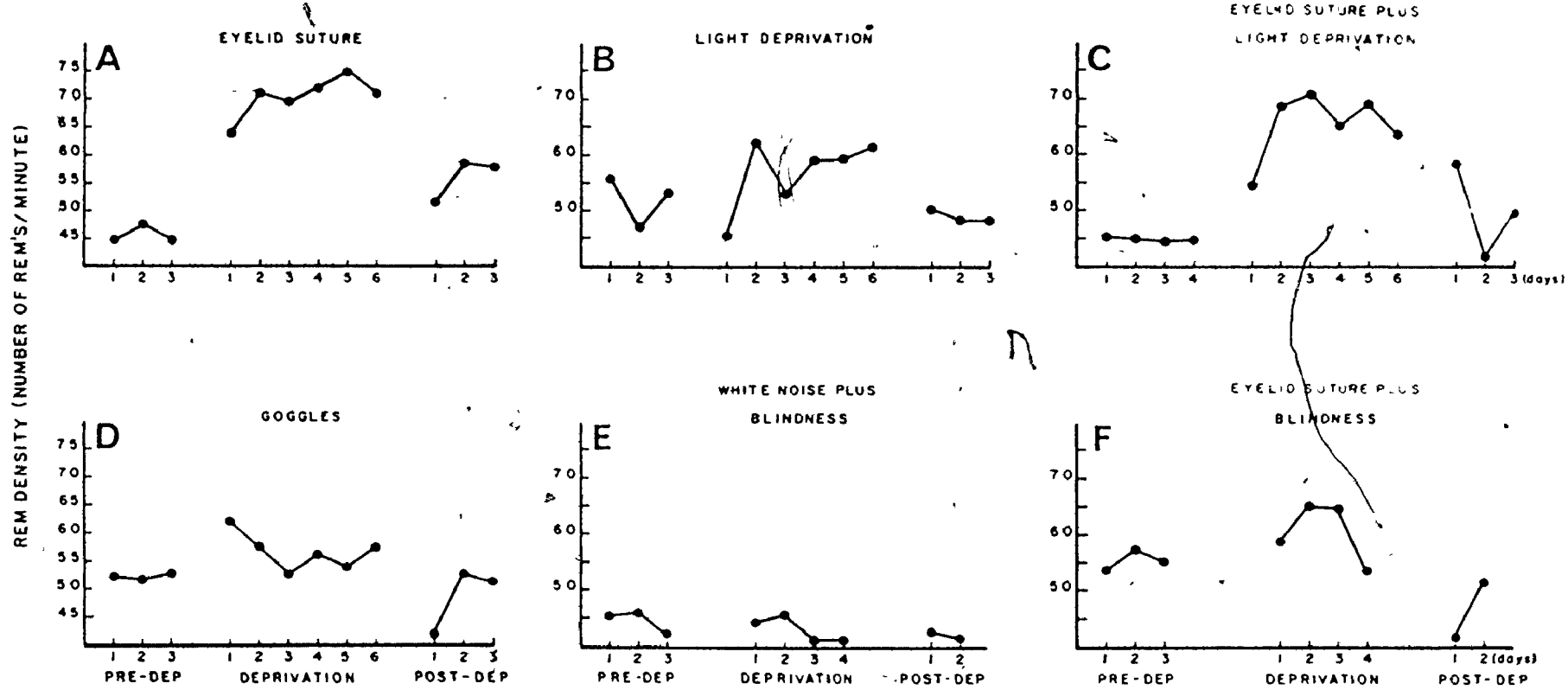
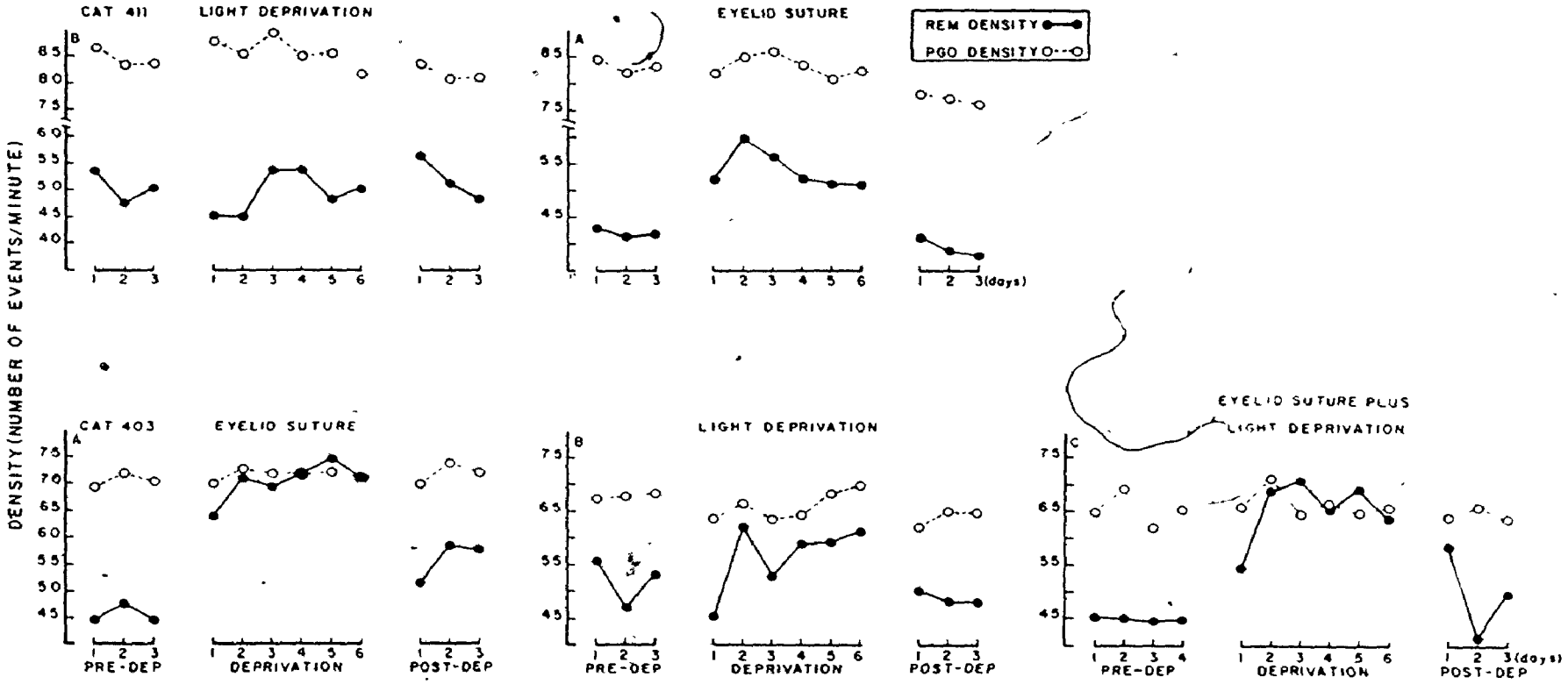

REM DENSITY (NUMBER OF REM'S/MINUTE)
A
EYELID SUTURE
B
LIGHT DEPRIVATION
C
EYELID SUTURE PLUS
LIGHT DEPRIVATION
D
GOGGLES
E
WHITE NOISE PLUS
BLINDNESS
F
EYELID SUTURE PLUS
BLINDNESS
PRE-DEP
DEPRIVATION
POST-DEP
(days)

Figure 58 - Average REM and PGO density of Cats 403 and 411 compared across conditions.


CAT 411
LIGHT DEPRIVATION
EYELID SUTURE
REM DENSITY
PGO DENSITY
DENSITY (NUMBER OF EVENTS/MINUTE)
(days)
CAT 403
EYELID SUTURE
LIGHT DEPRIVATION
EYELID SUTURE PLUS
LIGHT DEPRIVATION
PRE-DEP
DEPRIVATION
POST-DEP

EXPERIMENT IV

During brief periods of sensory deprivation, human subjects have been reported to lose up to a kilogram of body weight despite apparently normal or perhaps increased food consumption (Zubek, 1969b, p275). No comparable studies on the effects of sensory deprivation on body weight and food consumption of adult animal have been done, though Miller et. al. (1969, 1971) reported that adult rhesus monkeys, that had been reared in total isolation during the first year of life, ate and drank about thirty percent more than normally reared monkeys. Thus, on the basis of these data, one might expect that some change in food consumption and body weight might occur during sensory deprivation in cat. There are several other lines of evidence which lead to the same conclusion.

Schapiro et. al. (1970a; b; 1971; 1973) have found that destruction of the visual, auditory, vestibular or olfactory sensory modalities cause a permanent reduction in stomach acid and pepsin secretion in dog in response to injections of histamine or insulin. Since a temporary reduction in secretion in response to sham feeding or teasing with meat was also obtained in blindfolded animals (Schapiro et. al., 1973), and since parotid secretion is also known to be less in darkness than in light (Pangborn & Sharon, 1971; Shannon

et. al., 1972; 1975), it is possible that secretion of digestive juices might also be decreased during sensory deprivation. Such an effect might account for the reduction in body weight described by Zubek and perhaps, the increased food consumption reported by Miller et. al. In any case, these data on stomach and parotid secretion force one to consider the possibility that sensory deprivation has a much larger spectrum of effect than has been demonstrated so far in this thesis.

Another piece of evidence which suggests that sensory deprivation might be affecting a number of systems beside those controlling sleep comes from experiments dealing with the role of hypothalamic-midbrain structures in sleep processes. It is well known that hypothalamic lesions result in impairment in the regulation of food and water intake; effects which are usually attributed to damage of specific hypothalamic nuclei (Morgane, 1961). Conversely, the effects of hypothalamic lesions on sleep are usually considered to be due to damage of tracts passing through the hypothalamic area (Nauta, 1946; Ranson, 1939). Nevertheless, Nauta & Haymaker (1969) have suggested, on the basis of the extensive interconnections between the basal forebrain, hypothalamus and midbrain reticular regions, and because of the great similarity in neuronal cytoarchitecture in these areas, that control of specific function will not be sharply

delineated. According to this view, the fact that lesions of the brainstem reticular formation (Bellinger & Bernardis, 1976; Roussel, 1976) and hypothalamus (McGinty, 1969a; Nauta, 1946; Ranson, 1939) cause disturbances in sleep as well as disrupt the regulation of food consumption, body weight and body core temperature can be taken as evidence of a joint control of sleep and other regulatory processes by a hypothalamic-midbrain system. Further support for this idea comes from studies which demonstrate that the regulation of sleep, food consumption, body weight and body core temperature are jointly affected by other experimental treatments (Andersson & Larsson, 1961; Magoun, 1938; Parmeggiani & Rabini, 1970; Roberts et. al., 1969; Roberts & Robinson, 1969; Schmidek et. al., 1972) and often covary under normal conditions (Siegel, 1975). Therefore, it seems likely that sensory deprivation, which has large effects on sleep processes, might also have an effect on these other regulatory systems. Consequently, food consumption, body weight and body core temperature were measured in Experiment I to determine whether these parameters changed during sensory deprivation.

METHOD

Total food and milk consumption during each twenty-four hour period of pre-deprivation, deprivation and post-deprivation were determined by calculating to the nearest 0.1 gram (1 ml) the differences between the weight (volume) of the fresh food (milk) and that recovered at the end of the period. No correction was made for evaporative loss but this likely remained the same across conditions since the room temperature and the relative humidity fluctuated within narrow limits (21 ± 2°C; 50 ± 2% relative humidity). Body weight was measured daily to the nearest 0.2 kg.

Rectal temperature was measured at 1200 hours by inserting a thermometer eight centimeters into the rectum for three minutes. A continuous record of body core temperature was derived from a thermistor implanted in the intraperitoneal space near the midline in the region of the small intestine. Post-mortem examination of Cat Q01, the one animal where technically satisfactory results were obtained, revealed that the thermistor lay within four centimeters of the tip of the thermometer when the thermometer was inserted eight centimeters into the rectum. Insulated wires were run subcutaneously from the thermistor to the headcap where they were connected to pins in an amphenol plug.

A continuous measurement of body core temperature was

made by using a Wheatstone bridge circuit (Figure 59) which was balanced at the thermistor resistance equivalent to a temperature of 37.5°C. Imbalance in the circuit due to changes in the thermistor's resistance, R_t, caused a potential difference to develop between the inputs to the amplifier e_1 and e_2. The magnitude of this potential $(e_1 - e_2)$ was equal to $E_{in}\left[\frac{R_t}{R_t + R_2} - \frac{R_3}{R_3 + R_4}\right]$. The gain of the amplifier was set so that a one centimeter pen deflection was equivalent to a 1°C change in the thermistor temperature above its zero value of 37.5°C. Total current flowing through the thermistor at body temperature was approximately 30 μ amps, a value low enough so as not to significantly alter the thermistor's temperature.

A representation of changes in body core temperature over the twenty-four hour period was obtained from the continuous record by measuring the temperature at two minute intervals. Five successive two minute samples were averaged and these ten minute averages were plotted.

RESULTS

Table XXXII gives the average daily rectal temperature for two animals during pre-deprivation, deprivation and post-deprivation. Both of these cats tended to have a lower rectal temperature during deprivation than during either pre-deprivation or post-deprivation. These results were confirmed for Cat Q01 in Figure 60 where the twenty-four hour fluctuations in the thermistor derived core temperature are plotted. In addition to the overall reduction in temperature, there appears to be a flattening out of the circadian rhythm in body temperature during deprivation. The fact that a large positive correlation existed between rectal temperature and the thermistor derived temperature ($r = 0.94$, $t = 14.76$; $p < .001$) indicates that there was a good agreement between the two methods used for recording temperature. This result was expected since the thermistor was located in close proximity to the tip of the rectal thermometer when it was inserted a fixed distance into the rectum.

Table XXXIIIA presents the body weight and food consumption data for three cats that tended to increase their food consumption during sensory deprivation. When a test of the difference between the mean food consumption during six pre-deprivation days and six deprivation days was made for each of these animals, it was found that food consumption

was significantly greater during deprivation (Appendix A, Table XXXVI, Rows 1 to 3). Milk consumption also tended to be greater , but this effect was significant for only one animal (Cat 411). A fourth animal (Cat N06) also showed this pattern of increased food consumption during deprivation, but no quantitative data was obtained. However, it was noted that during pre-deprivation, a large portion of the animal's food ration was left at the end of the day while during deprivation, the animal ate all the food given to it.

Two out of the six animals run in Experiment I did not significantly alter their food or milk consumption during deprivation (Cats Q03 & 403, Table XXXVI) and when the data from all five cats was pooled (Table XXXIIIB) and subjected to analysis of variance, there was no significant treatment effect on food ($F = 1.70$, $p>.05$) or milk ($F = 0.62$, $p>.05$) consumption, but body weight did change ($F = 9.78$, $p<.05$). Multiple t tests indicated that mean body weight during pre-deprivation differed significantly from body weight during deprivation ($t = 3.26$, $p<.01$) and post-deprivation ($t = 4.22$, $p<.01$).

Food and milk consumption also tended to be greater on days when the animal was exposed to total light deprivation and white noise (Data collected in Experiment III) than on pre-deprivation days (Table XXXIV), but again, these effects were non-significant ($F = 2.10$, $p>.05$; $F = 3.15$, $p>.05$,

respectively). Within subject analysis of these data led to the same conclusions (Table XXXVI). The apparent effect of light deprivation on milk consumption in Cat Q03 can probably be disregarded since the absolute magnitude of the effect is extremely small (Table XXXIV). In fact, it is likely that most of the 15 to 20 ml of milk that this cat was recorded as consuming was actually lost in evaporation.

DISCUSSION

The results of the present experiment, while not conclusive, certainly suggest that the regulation of body weight, body core temperature and food and milk consumption may be affected by sensory deprivation since four out of six animals increased their food and milk consumption. (Cats listed in Table XXXIIIA plus Cat N06 where the effect of deprivation was first noticed), and two animals decreased their body core temperature. (Temperature data was not obtained from other cats because they either were recalcitrant when it came to taking rectal temperature measurements or else thermistors had not been implanted or they failed to function prior to the experiment.) These effects were reversible in that post-deprivation values tended to be similar to pre-deprivation levels. This was not the case for body weight, though, since the average body weight during post-deprivation was significantly greater than during pre-deprivation. Whether post-deprivation weight eventually returned to pre-deprivation levels is unknown since the cats in this experiment were not followed for a long enough period of time in post-deprivation. It seems unlikely that the increase in body weight from pre-deprivation to deprivation to post-deprivation was part of a growth curve since the animals used in this experiment were fully mature.

The reason for the failure of Cats Q03 and 403 to change their food and milk consumption is unclear. Both of these animals had as large an increase in SWS time and REM density as other cats that increased food consumption during deprivation (Compare Cat Q01 to Q03; Table XXXIV; Figure 4 to Figure 5; Figure 16; Compare Cat 403 to 411; Table XXXIV; Figure 6 to Figure 7; Figure 17). Although all animals tended to spend less time actively moving about during deprivation than in pre-deprivation, differences in the magnitude of this effect do not seem to be related to differences in food consumption patterns between animals since Cat Q01 (Figure 4) and Cat 403 (Figure 6) exhibited a similar change in active waking during deprivation, despite differential effects of deprivation on food and milk consumption. Similar comparisons in regard to REM sleep time (See Siegel, 1975; Compare Cat 403 to 411; Cat Q03 to 007) and body core temperature (Compare Cat Q01 to Q03; Table XXXII) suggest that the effect of deprivation on food and milk consumption may be unrelated to its effect on these other parameters. However, more research is required before any firm conclusion can be made.

There are many hypotheses which could be advanced to account for the apparent effects of deprivation on food consumption, body weight and body core temperature. For example, an increase in body weight could be due to an

increase in food consumption or a decrease in activity levels while an increase in food consumption might be used as mechanism for raising body temperature during deprivation. To come full circle, it may be that the initial drop in temperature occurs as a result of decreased activity levels. Alternatively, sensory deprivation may be having a more fundamental effect on regulatory systems, endocrine systems and metabolism.

In any case, more research is required to establish the validity of these effects of deprivation and determine why it is certain animals are not susceptible. Furthermore, the apparent discrepancy between the human and cat experiments in terms of the effect of deprivation on body weight needs to be resolved and the question of why animals would increase their food consumption in these experiments, when energy expenditure is likely to be less than normal as a result of decreased activity levels and perhaps lower body temperature, needs to be answered. Perhaps this latter effect is related to the reduction in gastric juice secretion reported by others after sensory deafferentation which may be only a small part of a more general effect of sensory deprivation on digestive processes.

Table XXXII - Average daily rectal temperature (^{o}C) measured at 1200 hours during pre-deprivation, deprivation and post-deprivation for Cats Q01 and Q03.

Table XXXII

		Pre-Deprivation	Deprivation	Post-Deprivation
CAT Q01	$\bar{X}$	38.9	38.4	38.5
	S.	0.8	0.4	0.4
CAT Q03	$\bar{X}$	38.3	38.2	38.4
	S.	0.1	0.1	0.2

Table XXXIIIA - Mean body weight (kg.) and average daily food (g.) and milk consumption (ml.) for Cats 007, Q01 and 411 during pre-deprivation, deprivation and post-deprivation.

Table XXXIIIA

		Body Weight	Food Consumed	Milk Consumed
Pre-Deprivation	007	3.48	178.6	109.2
	Q01	3.47	134.5	125.2
	411	----	178.1	63.3
	$\bar{X}$	3.48	163.7	99.2
	S.E.	0.01	14.6	18.6
Deprivation	007	3.78	246.3	135.0
	Q01	3.79	226.4	172.2
	411	----	205.3	118.0
	$\bar{X}$	3.79	226.0	141.7
	S.E.	0.01	11.8	15.9
Post-Deprivation	007	3.90	254.3	166.7
	Q01	3.78	158.2	109.6
	411	----	143.0	62.5
	$\bar{X}$	3.84	185.2	112.9
	S.E.	0.06	34.8	30.1

Table XXXIIIB - Mean body weight (kg.) and average daily food (g.) and milk consumption (ml.) during pre-deprivation, deprivation and post-deprivation for five of the cats run in Experiment I.

Table XXXIIIB

		Body Weight	Food Consumed	Milk Consumed
Pre-Deprivation	007	3.48	178.6	109.2
	Q01	3.47	134.5	125.2
	Q03	6.93	213.8	61.2
	403	3.92	180.1	81.0
	411	----	178.1	63.3
	$\bar{X}$	4.45	177.0	88.0
	S.E.	0.83	12.6	12.7
Deprivation	007	3.78	246.3	135.0
	Q01	3.79	226.4	172.2
	Q03	7.08	200.5	27.5
	403	3.96	182.7	79.2
	411	----	205.3	118.0
	$\bar{X}$	4.65	212.2	106.4
	S.E.	0.81	11.0	24.7
Post-Deprivation	007	3.90	254.3	166.7
	Q01	3.78	158.2	109.6
	Q03	7.20	228.0	38.0
	403	3.97	190.5	94.0
	411	----	143.0	62.5
	$\bar{X}$	4.71	194.8	94.2
	S.E.	0.83	20.8	22.0

Table XXXIV - Average daily food (g.) and milk consumption (ml.) for Cats Q03, 403 and 411 during pre-deprivation, deprivation and post-deprivation of the light deprivation experiment.

Table XXXIV

		Food Consumed	Milk Consumed
Pre-Deprivation	Q03	197.9	18.0
	403	190.5	94.0
	411	160.9	53.0
	$\bar{X}$	183.1	55.0
	S.E.	11.3	22.0
Deprivation	Q03	208.6	16.2
	403	203.8	133.5
	411	190.4	100.7
	$\bar{X}$	200.9	83.5
	S.E.	5.4	34.9
Post-Deprivation	Q03	198.4	21.0
	403	215.2	67.3
	411	162.3	41.0
	$\bar{X}$	192.0	43.1
	S.E.	15.6	13.4

Figure 59 - Wheatstone bridge circuit used to measure body core temperature.

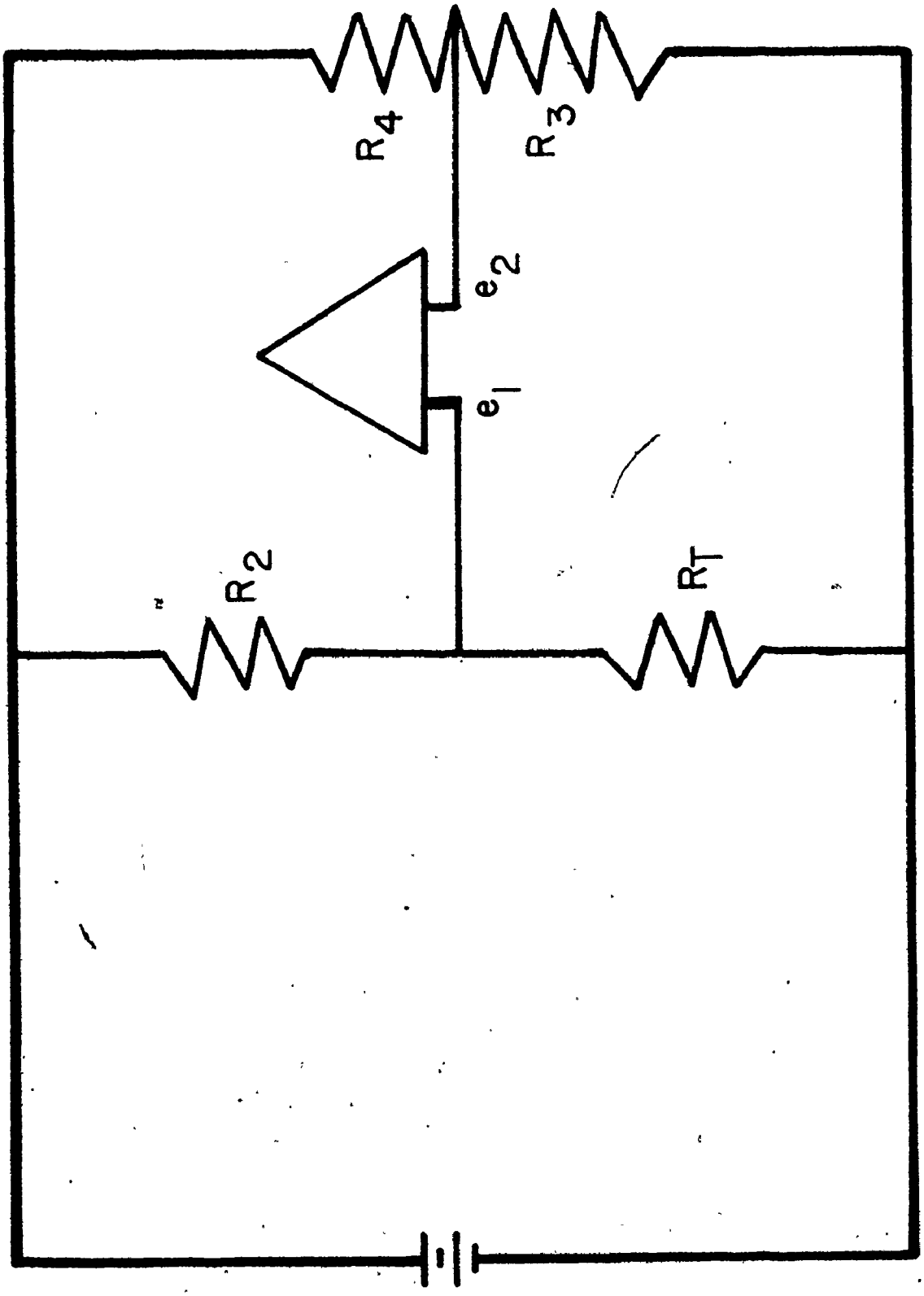
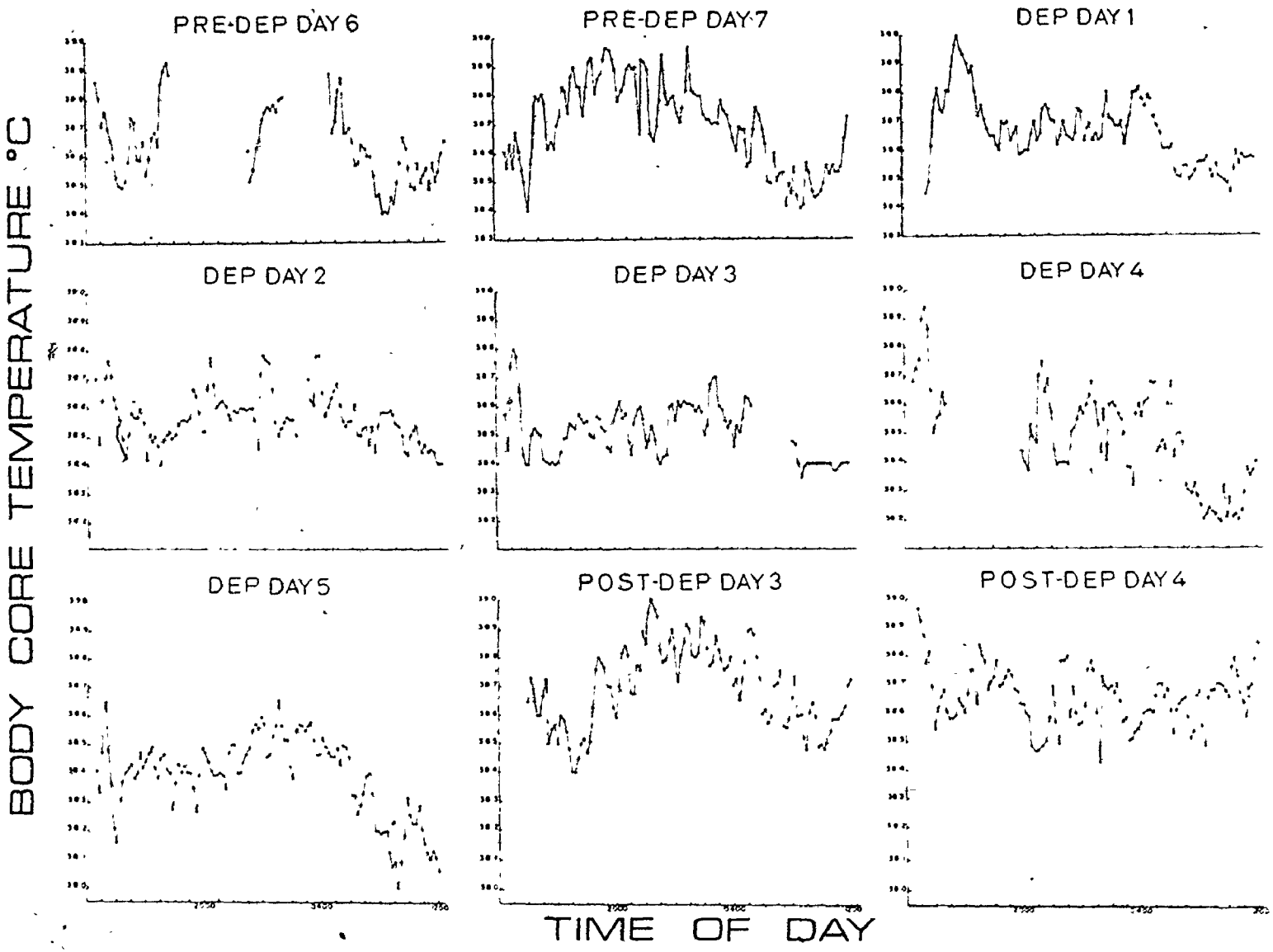

R4
R3
e2
e1
R2
RT

Figure 60 - Changes in body core temperature over a twenty-four hour period for Cat Q01 during pre-deprivation, deprivation and post-deprivation.


PRE-DEP DAY 6
PRE-DEP DAY 7
DEP DAY 1
DEP DAY 2
DEP DAY 3
DEP DAY 4
DEP DAY 5
POST-DEP DAY 3
POST-DEP DAY 4
BODY CORE TEMPERATURE °C
TIME OF DAY

CHAPTER III

I. Summary of Results

The main findings of Experiments I to IV will be listed here and their interpretation and relevance will be discussed in the next section.

(1) In Experiment I, it was demonstrated that during sensory deprivation adult cat significantly increased the proportion of time spent in LSWS without altering the amount of time spent in DSWS or REM sleep.

(2) The increase in LSWS was obtained by increasing the number of episodes of SWS and not by increasing episode duration. In fact, episode duration of SWS, REM sleep, active and quiet waking tended to be shorter during deprivation than in pre-deprivation.

(3) The increase in sleep time was accompanied by a corresponding decrease in time spent awake. When waking time was further divided into active and quiet waking, it was found that the proportion of time spent in active waking tended to be less during deprivation but only the deprivation day one result reached statistical significance. Time spent in quiet waking was also less during deprivation for some animals.

(4) REM density was significantly greater on all deprivation days than on pre-deprivation days.

(5) The effects of sensory deprivation on sleep in cat were similar to those obtained from human subjects since in the human studies, the light phases of sleep (Stages I & II) and REM density both increased during deprivation.

(6) In Experiment II, it was found that several characteristics of PGO wave generation such as PGO wave density, the distribution of time intervals between successive PGO waves and the temporal relationships between PGO waves occurring in two brain loci remained the same during deprivation as in pre-deprivation while REM density and the distribution of time intervals between successive REMs changes significantly.

(7) A significant positive correlation between REM density and the average EOG amplitude was discovered. Since the magnitude of this correlation was not affected by sensory deprivation, the increase in REM density was found to be accompanied by a significant increase in the average amplitude of REMs.

(8) The relationship between the occurrence of REMs and hippocampal theta frequency described by others, was confirmed and some new data on the relationship between theta frequency and the occurrence or absence of PGO waves was obtained. In addition, a preliminary account of the effects of sensory deprivation on these relationships was given.

(9) In Experiment III, the minimum condition needed to obtain the effects of sensory deprivation on sleep was found to be

the lid suture procedure used to prevent vision in these experiments. Thus, total light deprivation and white noise had only marginal effects while light deprivation and white noise coupled with lid suture had the same magnitude of effect as lid suture plus white noise.

(10) The increase in LSWS, described by others following nearly complete sensory deafferentation, was similar to that obtained during sensory deprivation though cats which had been blind for one month were found to spend a normal proportion of their time asleep. However, one blind cat, when exposed to the same intensity white noise as used during the lid suture experiment, had a large transient increase in SWS.

(11) In Experiment IV, some cats were found to significantly increase their food consumption and body weight during sensory deprivation and/or decrease their body core temperature. Other animals failed to show these effects.

II. General Discussion

(A) Validity of the Animal Model Approach to the Study of Sensory Deprivation Effects

In designing Experiment I, an attempt was made to replicate the conditions of the human sensory deprivation experiment. Perhaps the best indicator of the success of this attempt is the fact that the sleep of both human and cat was similarly affected by sensory deprivation. Thus, both groups increased the percent time spent in the light stages of SWS without changing time spent in deep SWS or REM sleep. In addition, both groups increased their REM density during deprivation, with the onset and offset of the effect being roughly the same in each group. Differences between these experiments were also present, both in terms of the way subjects responded to deprivation, and in terms of the experimental conditions. For example, the increase in sleep time in the human experiment lasted only a few days whereas it tended to persist throughout the entire deprivation period in the cat study and the absolute magnitude of the increase in REM density was larger in the cat experiment while the relative increase was greater in the human study. In regard to differences in experimental conditions, pattern vision was prevented by lid suture in cat whereas human subjects were required to wear a light diffusing mask which attenuated light

much less than did closed eyelids in cat. Furthermore, a greater degree of control over tactile input and general activity levels was obtained in the human experiment than in cat experiment because of the greater compliance of human subjects.

After considering the similarities and differences between the human sensory deprivation experiments and the present work, one could speculate on whether the effects observed in these two sets of experiments are being produced by a factor that is common to both situations and that is operating through similar mechanisms. This question is obviously important since its answer would determine whether or not conclusions reached in the animal studies are applicable to the human situation. The fact that the experimental conditions and the subjective responses are similar, at least in a gross way, suggests that the generalization across experiments may be possible, although the presence of these similarities certainly cannot be regarded as critical proof that we are indeed dealing with the same phenomena. Nevertheless, we are justified in adopting the working hypothesis that the sensory deprivation effects obtained in the human and cat experiments are being produced through similar mechanisms.

(B) Effect of Sensory Deprivation on SWS: Deafferentation or Stimulus-Induced Sleep

The increase in SWS time during sensory deprivation might be interpreted as either a stimulus-induced sleep or as a deafferentation-like sleep. According to the former hypothesis, the monotonous characteristic of stimulation during deprivation is responsible for the increased sleep. It will be recalled, however, that the critical variable needed to obtain stimulus-induced sleep was repetitive stimulation at low frequency. The continuous stimulation (e.g. white noise and continuous illumination) used in these experiments, while maybe monotonous, is certainly not repetitive. Thus, a parallel between sleep obtained during sensory deprivation and stimulus induced sleep cannot be drawn.

In the case of the deafferentation hypothesis, it is known that chronic deafferentation leads to an increase in the proportion of time spent in light SWS (Vital-Durand & Michel, 1971) without affecting other sleep stages, just as sensory deprivation does. In addition, acute deafferentation in brainstem transected preparations also results in cortical synchronization. Thus, a strong parallel exists between the effects of deafferentation and sensory deprivation. This does not necessarily mean that sensory deprivation produces a condition of decreased sensory input, as deafferentation obviously does. Rather, in the sense of Kleitman's and Bremer's

deafferentation theory of sleep, it is presumed that fewer stimuli capable of eliciting or sustaining wakefulness are present during sensory deprivation. This return to a passive theory of sleep induction, where the amount of sensory input available to the organism determines how much sleep is obtained, while maybe adequate for handling the increase in LSWS during sensory deprivation and following sensory deafferentation, cannot explain the results of sleep deprivation experiments which demonstrate that powerful, sleep-promoting influences arise from within the organism after long periods of sleeplessness. However, since only the amount of time spent in DSWS (Stages III & IV in humans) and REM sleep is compensated for after sleep deprivation, while LSWS (Stages I & II) is not well regulated, perhaps the amount of LSWS obtained is determined mainly by environmental conditions while processes within the brain are responsible for the regulation of DSWS and REM sleep.

It may be argued that total light deprivation used in Experiment III also imposes a condition where there are fewer stimuli available to awaken the animal, yet the cats in this experiment only marginally increased their sleep time, if at all. It is difficult to isolate the critical difference between the light deprivation and lid suture procedures, but it should be stated that in the human sensory deprivation experiments, light deprivation and silence typically is also less

effective in altering reponse measures than is unpatterned stimulation. Similarly, Claes (1939 - cited by Arduini & Hirao, 1959) found that eye enucleation led to a synchronization of cortical EEG in encephale isolé cat while dark adaptation did not. Arduini & Hirao (1959) obtained similar results in the midpontine pretrigeminal preparation, but also found that continuous illumination was capable of inducing synchrony in cortical EEG just as deafferentation did. Therefore, there may be something unique about optic nerve activity during unpatterned light stimulation which is capable of eliciting cortical synchronization. The fact that one cat subjected to total light deprivation and lid suture had as large an increase in sleep time as when exposed to lid suture alone, suggests, however, that continuous illumination is not critical for obtaining the sleep effects described in this thesis.

(C) Effects of Sensory Deprivation on REM Sleep

(i) REM Density

In Experiment II, it was suggested that the change in REM density observed during sensory deprivation, occurred as a result of an increase in the amplitude of REMs. Although this hypothesis is consistent with the fact that the average EOG amplitude of REMs occurring during deprivation was larger than those occurring in pre-deprivation, other interpretations are possible. Thus, a similar increase in EOG amplitude could be obtained if either the size of the corneo-retinal

potential (the potential difference that exists from the front to the back of the eyeball) or the velocity at which the eyes moved, increased, since the amplitude of the capacitance-couple EOG response is directly proportional to both of these variables as well as to the degree of eye rotation (Kris, 1960; Mowrer, 1936; Shackel, 1967). It is impossible to decide between these alternative mechanisms for increasing EOG amplitude without direct experimental evidence but they can be ranked in terms of which seem more likely on the basis of data collected in these experiments and on information available in the literature.

The source of the potential difference across the eyeball is not known (Marg, 1951), but it does not depend entirely on the presence of the retina since an EOG response can still be recorded following eye evisceration (Pasik et. al., 1965) even though the degree of dark adaptation influences the magnitude of the corneo-retinal potential in the intact eye (Arden & Kelsey, 1962; Gonshor & Malcolm, 1971; Kris, 1958). Exposing the completely dark adapted eye to normal room lighting causes a damped oscillation of the corneo-retinal potential which begins with a large increase followed by a smaller decrease and so on through two cycles which take about an hour before the potential is back to baseline levels (Gonshor & Malcolm, 1971; Homer et. al., 1967; Kris, 1958). In contrast, transition from a lighted environment to total

darkness causes an initial transient increase in the corneo-retinal potential which is followed by a sustained decrease in the potential at a value which is slightly less than the baseline level (Kris, 1958). Therefore, on the basis of these results, one would expect that, if anything, the corneo-retinal potential would be less following lid suture than before. The fact that total light deprivation without lid suture had little effect on REM density suggests that a light-induced change in the corneo-retinal potential is not responsible for the effect of lid suture on REM density.

The corneo-retinal potential has been reported to undergo diurnal variation (Kris, 1957) as well as systemic increase during the course of a night's sleep (Aserinsky, 1955; Jacobs et. al., 1973). Unfortunately, none of these experiments controlled for changes in the ability of the subject to shift fixation from one target to a second. Such a control is critical since the amplitude of the corneo-retinal potential is typically evaluated by measuring the size of potential change produced by eye rotation through a known angle. Since Berger & Walker (1972) have presented evidence which suggests that oculomotor function may be less efficient at some times than at others, effects which others have attributed to changes in corneo-retinal potential may, in fact, be due to changes in oculomotor ability. In spite of this weakness in the data, it is possible that eyelid suture

does cause an increase in the corneo-retinal potential by affecting the normal physiological variation in this potential. Perhaps the closing of the eyelid over the cornea exerts some pressure on the eyeball which leads to change in the potential or somehow changes volume conduction. Stepanik (1958 - cited by Shackel, 1967), however, found that increasing intraocular pressure had no immediate effect on the corneo-retinal potential although releasing the pressure did cause the potential to increase. Arden & Kelsey (1962) reported that an equivalent decrease in corneo-retinal potential was obtained following the elimination of light irrespective of whether the eye was left open and the light was simply turned off or the eye was closed and bandaged shut. Therefore, it appears that in the short term, eye closure and perhaps mild pressure exerted on the eyeball does not have an effect on the corneo-retinal potential.

Both the degree and the velocity of eye rotation are known to vary over a large range in the awake human (Robinson, 1964; Westheimer, 1954) and cat (Crommelinck & Roucoux, 1976; Stryker & Blakemore, 1972) with the large amplitude saccades usually occurring at a higher velocity than smaller amplitude saccades. Although the eye movements occurring during REM sleep have a velocity which is lower than that obtained during waking (Fuchs & Ron, 1968, Jeannerod et. al., 1965), the relationship between eye movement velocity and amplitude

has not been as carefully described. If one assumes that a positive correlation exists between these variables during REM sleep, as it does during waking, and that deprivation does not disrupt this relationship, then the EOG amplitude effect described in Experiment II likely is made up of both an amplitude and a velocity component.

On another level of explanation, REM density has been related to other variables. For example, Aserinsky (1969; 1971; 1973) reported a positive correlation between REM density and the accumulation of sleep in excess of normal. A similar relationship existed in the present study since REM density and the proportion of time spent in SWS tended to covary with each other. This was also true for REM density and stage I sleep in the human study, but not for total sleep time. Whether these variables are causally related to each other or to some other unknown factor remains to be determined.

Berger (1969) proposed that REM density is inversely proportional to the amount of oculomotor activity that occurs during waking. It is known that human subjects wearing black eye patches decrease the number of waking eye movements (Adams et. al., 1973) and there is a hint from observations made by subjects exposed to sensory deprivation that a similar effect may be obtained with a translucent mask, since

subjects seem to prefer leaving their eyes closed under such conditions. This seems reasonable since the alternatives are to attempt to focus on the inside surface of the mask, an impossibility since the near point of the eye is far exceeded, or to stare with the eyes focused at their maximum focal length. Therefore, according to Berger's hypothesis, human and cat subjects both increased their REM density during sensory deprivation because fewer waking eye movements were made during this period than normal. In the case of the human subjects, this may have occurred because of voluntary eye closure whereas in the cat experiments, this was achieved by enforced eye closure. Such an interpretation could also explain the lack of effect of total light deprivation on REM density since waking eye movements might occur normally under this condition. This is all highly speculative, though, and even if it was confirmed by direct measurement that the frequency of waking eye movements was different under these various conditions, a causal relationship between the occurrence of waking eye movements and REM sleep eye movements would then have to be established.

(ii) REM Sleep Time

Despite the fact that sensory deprivation had a large effect on rapid eye movements occurring during REM sleep, the proportion of time spent in this stage of sleep was not significantly altered. This result is consistent with the

hypothesis that the systems controlling the REM sleep cycle are different from those that control events occurring during REM sleep (Jouvet, 1972), and may call into question hypotheses which assume an interaction between these systems (Hobson et. al., 1975; McCarley & Hobson, 1975b). However, in view of the fact that several parameters characterizing PGO wave generation were unchanged by sensory deprivation, and the mechanism for the change in REM density was not worked out, it seems a little premature to address these larger issues.

(D) Effects of Sensory Deprivation on Regulatory Systems

Moruzzi (1969) proposed that sleep is a consummatory act with clearly defined appetitive behavior preceding it and satiation occurring after. The sleep deprivation studies have carried this idea one step further by showing that prolonged waking is followed by increased sleep. Thus, it has been proposed that some chemical factor progressively increases in concentration in the brain during waking and is gradually dissipated during sleep (Dement et. al., 1970; Jouvet, 1969; Stern & Morgane, 1974). Stated in another way, one could say that sleep is a regulatory mechanism which serves to maintain the internal mileau of the brain within set limits. Within this context, it seems reasonable to suggest that the hypothalamus should be involved in the control of sleep since it participates in the control of other regulatory behaviors

and that interaction between these regulatory systems might occur. The demonstration that sensory deprivation has an effect on both sleep and on food consumption, body weight and body core temperature may be evidence of such interaction.

It is realized that the evidence for an effect of deprivation on food consumption, body weight and body core temperature is weak, and that even if such effects were present, they could be secondary to some other effect of deprivation. Nevertheless, these results are potentially the most interesting of all those obtained in the present experiments since they provide the oppportunity for establishing sleep on par with other regulatory processes as well as providing a way of synthesizing some of the other effects of deprivation. For example, the frequency of the alpha rhythm is known to decrease significantly during sensory deprivation but there have been no good explanations of how this might occur (Tait, 1977). However, since alpha frequency is positively correlated with body core temperature (Hoagland, 1936; Jasper & Andrews, 1938) and basal metabolic rate (Ross & Schwab, 1939; Rubin et. al., 1937) it is possible that the hypothermia observed in cat during sensory deprivation may also occur in human subjects and underlie the change in alpha frequency.

(E) Conditions Needed to Produce Sensory Deprivation Effects

One of the objectives of this thesis was to determine the minimum conditions needed to obtain the effects of sensory deprivation on sleep. Some progress has been made in this regard since in Experiment III, the presence of white noise, the reduction of the intensity of light reaching the eye and the loss of vision per se have been ruled out as being critical. Lid suture, on the other hand, appeared to be necessary. A subset of effects that might occur as a result of lid suture such as the alteration of the corneo-retinal potential and changes in eye motility during waking have already been discussed, as have the possible interrelationships between the various effects of deprivation. Further research, both in the human and cat, should help to establish the causal sequence of events which underlie the changes seen during sensory deprivation.

(F) Effects of Sensory Deprivation During Development and in the Mature Animal

It is generally believed that there exists a critical period during development when the visual system is particularly susceptible to the effects of sensory deprivation (Berman & Daw, 1977; Daw & Wyatt, 1977; Hubel & Wiesel, 1970; Wiesel & Hubel, 1963a; b). Deprivation imposed outside of the critical period is thought to have no effect and recovery from deprivation effects produced during the critical period

is assumed to be impossible once the critical period has ended (Blakemore & van Sluyters, 1974; Cragg et al., 1976; Dursteler et. al., 1976; Movshon & Blakemore, 1974; Movshon, 1976a; b; Wiesel & Hubel, 1965b; Yinon, 1976). Recently, however, several laboratories have found evidence of plasticity in the visual system of adult cat which is analagous to that seen during development (Brown & Salinger, 1975; Creutzfeldt & Heggelund, 1975; Fiorentini & Maffei, 1974; Hoffman & Cynader, 1975; Kratz et. al., 1976; Salinger et. al., 1977a;b).

Functional plasticity also appears to be present in the vestibulo-oculomotor system of adult human and cat since the vestibulo-ocular reflex undergoes almost total phase inversion within two weeks of reversing visual input in the horizontal plane (Davies & Jones, 1976; Jones & Davies, 1976). The present results on the effects of sensory deprivation on oculomotor function during REM sleep provide additional evidence of plasticity in the oculomotor system of adult cat.

At first glance, it may seem that there can be no possible relationship between plasticity in the morphology and functional connectivity of the visual system and plasticity in oculomotor function. However, it is known that the development of ocular alignment occurs at about the time that the visual system becomes most susceptible to the effects of environmental factors (Sherman, 1972). Furthermore, experimental conditions which are known to affect the development

of the visual system also result in the pathological development of oculomotor function (Riesen, 1961a; b; Turkel, 1974) and experimentally produced ocular misalignment leads to functional changes in the visual system (Gordon & Gummow, 1975; Hubel & Wiesel, 1965; von Noorden et. al., 1970; von Noorden, 1973; Yinon et. al., 1975). With this type of interaction between the development of visual and oculomotor function, the question has naturally been raised as to whether deficiencies in ocular alignment cause the visual system to develop abnormally or whether abnormal development of the visual system causes the pathology found in the oculomotor system (Turkel, 1974). Maffei & Fiorentini (1976) present evidence from adult cat which supports the former hypothesis. They found that immobilization of one eye produced by cutting the lateral rectus muscle and sectioning the IIIrd and IVth cranial nerves caused a change in the ocular dominance of cortical neurons which occurred independent of whether or not the animal was permitted to see during the experimental period. Since immobilization of both eyes had no effect on ocular dominance, Maffei & Fiorentini (1976) concluded that asymmetry in eye motility was sufficient for producing a change in ocular dominance.

Although the mechanism responsible for the shift in ocular dominance in adult cat reported by Maffei & Fiorentini (1976) may not be the same as that underlying changes in

ocular dominance produced by sensory deprivation (Wiesel & Hubel, 1963b) or by experimentally produced ocular misalignment during development (Hubel & Wiesel, 1965; Yinon, 1976), it is reasonable to ask whether this is indeed true. Similarly, there may be no link between the effects produced by monocular eye immobilization in Maffei's studies and the REM density effect reported here, or between the REM density effect and the effects of sensory deprivation during development (See Fishbein et. al., 1966; Vital-Durand & Jeannerad, 1975). Nevertheless, in view of the almost dogmatic acceptance of the concept of the critical period, further work on plasticity in the adult and the developing animal is indicated.

III. Summary and Conclusions

The effects of sensory deprivation on sleep of human and cat were similar in that in both experiments, the proportion of time spent in the light phases of sleep, and the average REM density increased during deprivation and returned to pre-deprivation levels during post-deprivation recording. Since the increase in SWS obtained during sensory deprivation was similar to that found following sensory deafferentation, it was suggested that a common mechanism may underlie each of these phenomena. It was proposed that sensory deafferentation and sensory deprivation both produced a condition where there are fewer stimuli available which might keep the animal awake and consequently, sleep time increases. This interpretation is in accord with the deafferentation theory of sleep proposed by Kleitman.

The cause of the increase in REM density that occurs during sensory deprivation was not determined but some mechanisms seemed to be more likely than others. For example, the density effect did not appear to be due to an increase in the frequency with which the pontine generator initiated eye movements since the average PGO density remained constant across conditions. The fact that the coupling between the occurrence of PGO waves and detectable REMs was improved during sensory deprivation plus the fact that the average EOG

amplitude was larger during deprivation suggested that the increase in REM density occurred as a result of an increase in the size and likely the velocity of REMs during deprivation. Specifically, it was proposed that eye movements which were subthreshold for detection during pre-deprivation became suprathreshold in deprivation. This result may have occurred as a result of oculomotor neurons being driven more intensely by the pontine generator during deprivation. Alternatively, the responsivity of the oculomotor system may have increased during deprivation as a result of lack of use during waking or because of altered sensory input.

Lid suture appeared to be the necessary condition for producing both the increase in sleep time and the increase in REM density. Several alternative mechanisms whereby lid suture might cause these changes were considered. In the author's opinion, the following sequence of events following lid suture seems most plausible. The elimination of vision and the masking out of noises made by other cats in the experimental chamber insured that during deprivation there were fewer stimuli available which might awaken the animal when asleep or which might sustain wakefulness after the animal had been awakened by other causes (e.g. hunger, thirst, the need to urinate and defecate, the temination of a sleep cycle). The additional sleep time was spent in light phases of sleep, which appear to be largely a "filler" stage of

sleep, while deep SWS and REM sleep, which have been shown by sleep deprivation studies to be more precisely regulated, were left unchanged.

Since a decrease in activity levels accompanied the increase in sleep time, the resulting change in vestibular input may have had an effect on oculomotor function. This hypothesis is in accord with studies which have demonstrated an effect of vestibular stimulation on REM sleep and with Aserinsky's reported correlation between time spent in SWS and REM density. In addition, it is consistent with Pompeiano's work on the role of vestibular nuclei in the genesis of rapid eye movements during REM sleep.

A second, equally plausible explanation for the effects of deprivation on REM density derives from Berger's hypothesis that eye movements during REM sleep serve to maintain co-ordination in the oculomotor system. Berger's prediction that there is an inverse relationship between the density of eye movements during waking and REM density would obviously have to be modified in order to bring it into line with the fact that the increase in REM density during deprivation is probably due to an increase in the average REM amplitude and velocity. Nevertheless, the basic idea that oculomotor function during waking influences oculomotor activity during sleep is a novel and exciting one. If such reciprocal interaction between systems controlling REMs and

waking eye movements occurs, then one would expect that waking eye movements might be altered by the sensory deprivation procedure. Although there is little data available on this question, it certainly seems plausible since similar parts of the brainstem appear to be critical for the genesis of REMs and saccadic eye movements (Cohen, 1971). Furthermore, the field potential associated with the occurrence of REMs and saccadic eye movements (i.e. PGO waves recorded during waking and REM sleep) have many characteristics in common (Brooks, 1968b; Cohen & Feldman, 1968; Sakai, 1973; Sakai & Cespuglio, 1976; Sakai et. al, 1976).

The question of whether the changes observed in body weight, food consumption and body core temperature occur in response to some other effect of deprivation or occur as part of a general effect of deprivation on regulatory systems, including sleep, cannot be answered without further experimentation. Similarly, more information about the effects of sensory deprivation on the relationship between the occurrence of PGO waves, REMs and the frequency of hippocampal theta is required.

BIBLIOGRAPHY

Adams, G.L., Yee. R.D., Hahn, P.M. & Pearlman, J.T. Effect of binocular and monocular patching on eye movements. Archives of Ophthalmology, 1973, 90, 117-120.

Akert, K., Koella, W.P. & Hess, R. Jr. Sleep produced by electrical stimulation of the thalamus. American Journal of Physiology, 1952, 168, 260-267.

Allen, S.R., Oswald, I., Lewis, S. & Tagney, J. The effects of distorted visual input on sleep. Psychophysiology, 1972, 9, 498-504.

Andersson, B. & Larsson, B. Influence of local temperature changes in the preoptic area and rostral hypothalamus on the regulation of food and water intake. Acta Physiologica Scandinavica, 1961, 52, 75-89.

Angeleri, F., Marchesi, G.F. & Quattrini, A. Effects of chronic thalamic lesions on the electrical activity of the neocortex and on sleep. Archives italiennes de Biologie, 1969, 107, 633-667.

Arden, G.B. & Kelsey, J.H. Changes produced by light in the standing potential of the human eye. Journal of Physiology, 1962, 161, 189-204.

Arduini, A. & Hirao, T. On the mechanism of the EEG sleep patterns elicited by acute visual deafferentation. Archives italiennes de Biologie, 1959, 97, 140-155.

Arduini, A. & Hirao, T. EEG synchronization elicited by light. Archives italiennes de Biologie, 1960, 98, 275-292.

Arduini, A. & Pinneo, L.R. Properties of the retina in response to steady illumination. Archives italiennes de Biologie, 1962, 100, 425-448.

Aschoff, J. Desynchronization and resynchronization of human circadian rhythms. Aerospace Medicine, 1969, 40, 844-849.

Aserinsky, E. Effects of illumination and sleep upon amplitude of electro-oculogram. Archives of Ophthalmology, 1955, 53, 542-546.

Aserinsky, E. The maximum capacity for sleep: Rapid eye movement density as an index of sleep satiety. Biological Psychiatry, 1969, 1, 147-159.

Aserinsky, E. Rapid eye movement density and pattern in the sleep of normal young adults. Psychophysiology, 1971, 8, 361-375.

Aserinsky, E. Relationship of rapid eye movement density to the prior accumulation of sleep and wakefulness. Psychophysiology, 1973, 10, 545-558.

Aserinsky, E. & Kleitman, N. Regularly occurring periods of eye motility and concomitant phenomena during sleep. Science, 1953, 118, 273-274.

Aserinsky E. & Kleitman, N. Two types of ocular motility occurring during sleep. Journal of Applied Physiology, 1955, 8, 1-10.

Baekland, F. Exercise deprivation: Sleep and psychological reactions. Archives of General Psychiatry, 1970, 22, 365-369.

Baker, J., Gibson, A., Glickstein, M. & Stein, J. Visual cells in the pontine nuclei of the cat. Journal of Physiology, 1976, 255, 415-433.

Batini, C., Magni, F., Palestini, M., Rossi, G.F. & Zanchetti, A. Neural mechanisms underlying the enduring EEG and behavioral activation in the midpontine pretrigeminal cat. Archives italiennes de Biologie, 1959b, 13-25.

Batini, C., Moruzzi, G., Palestini, M., Rossi, G.F. & Zanchetti, A. Persistent patterns of wakefulness in the pretrigeminal midpontine prepartion. Science, 1958, 128, 30-32.

Batini, C., Moruzzi, G., Palestini, M., Rossi, G.F. & Zanchetti, A. Effects of complete pontine transections on the sleep-wakefulness rhythm: The midpontine pretrigeminal preparation. Archives italiennes de Biologie, 1959a, 97, 1-12.

Batini, C., Palestini, M., Rossi, G.F. & Zanchetti, A. EEG activation patterns in the midpontine pretrigeminal cat following sensory deafferentation. Archives italiennes de Biologie, 1959c, 97, 26-32.

Batsel, H.L. Electroencephalographic synchronization and desynchronization in the chronic "cerveau isole" of the dog. Electroencephalography & Clinical Neurophysiology, 1960, 12, 421-430.

Batsel, H.L. Spontaneous desynchronization in the chronic cat "cerveau isole". Archives italiennes de Biologie, 1964, 102, 547-565.

Baust, W. & Bohnert, B. The regulation of heart rate during sleep. Experimental Brain Research, 1969, 7, 169-180.

Bell, C., Sierra, G., Buendia, N. & Segundo, J.P. Sensory properties of neurons in the mesencephalic reticular formation. Journal of Neurophysiology, 1964, 27, 961-987.

Bellinger, L.L. & Bernardis, L.L. Mesencephalic lesions followed by normophagia but decreased body weight. Journal of Neuroscience Research, 1976, 2, 47-56.

Berger, R.J. Characteristics of REM sleep following different rates of waking eye movement in the monkey. Perceptual & Motor Skills, 1968, 27, 99-117.

Berger, R.J. Oculomotor control: A possible function of REM sleep. Psychological Review, 1969, 76, 144-164.

Berger, R.J. & Walker, J.M. Oculomotor coordination following REM and non-REM sleep periods. Journal of Experimental Psychology, 1972, 94, 216-224.

Berlucchi, G., Maffei, L., Moruzzi, G. & Strata, P. EEG and behavioral effects elicited by cooling of medulla and pons. Archives italiennes de Biologie, 1964, 102, 372-392.

Berlucchi, G., Maffei, L., Moruzzi, G. & Strata, P. Mécanismes hypnogènes du tronc de l'encephale antagonistes du système réticulaire activateur. In: M. Jouvet (ed.) Aspects anatomo-fonctionnels de la physiologie du sommeil, pp89-105. Centre National de la Recherche Scientifique, Paris, 1965.

Berman, A.L. The Brain Stem of the Cat. A Cytoarchitectonic Atlas with Stereotaxic Coordinates. The University of Wisconsin Press, Madison, Wisconsin, 1968.

Berman, N. & Daw, N.W. Comparison of the critical periods for monocular and directional deprivation in cats. Journal of Physiology, 1977, 265, 249-259.

Bexton, W.H., Heron, W. & Scott, T.H. Effects of decreased variation in the sensory environment. Canadian Journal of Psychology, 1954, 8, 70-76.

Beyer, C., Almanza, J., De La Torre, L. & Guzmán-Flores, C. Brain stem multi-unit activity during "relaxation" behavior in the female cat. Brain Research, 1971a, 29, 213-222.

Beyer, C., Almanza, J. De La Torre, L. & Guzmán-Flores, C. Effect of genital stimulation of the brain stem multi-unit activity of anestrus and estrous cat. Brain Research, 1971b, 32, 143-150.

Bizzi, E. & Brooks, D.C. Functional connections between pontine reticular formation and lateral geniculate nucleus during deep sleep. Archives italiennes de Biologie, 1963, 101, 666-680.

Bizzi, E., Pompeiano, O. & Somogyi, I. Spontaneous activity of single vestibular neurons of unrestrained cats during sleep and wakefulness. Archives italiennes de Biologie, 1964, 102, 308-330.

Bizzi, E. & Spencer, W.A. Enhancement of EEG synchrony in the acute "cerveau isolé". Archives italiennes de Biologie, 1962, 100, 234-247.

Blakemore, C. & Van Sluyters, R.C. Reversal of the physiological effects of monocular deprivation in kittens: Further evidence for a sensitive period. Journal of Physiology, 1974, 237, 195-216.

Bohlin, G. Monotonous stimulation, sleep onset and habituation of the orienting reaction. Electroencephalography & Clinical Neurophysiology, 1971, 31, 593-601.

Boland, B.D. & Dewsbury, D.A. Characteristics of sleep following sexual activity and wheel running in male rats. Physiology & Behavior, 1971, 6, 145-149.

Bonvallet, M. & Allen, M.B. Jr. Prolonged spontaneous and evoked reticular activation following discrete bulbar lesions. Electroencephalography & Clinical Neurophysiology, 1963, 15, 969-988.

Bonvallet, M. & Bloch, V. Bulbar control of cortical arousal. Science, 1961, 133, 1133-1134.

Bonvallet, M. & Dell, P. Contrôle bulbaire du système activateur. In: M. Jouvet (ed.) Aspects anatomo-fonctionnels de la physiologie du sommeil. Centre National de la Recherche Scientifique, Paris, 1965.

Bonvallet, M. & Sigg, B. Étude electrophysiologique des afférences vagales au niveau de leur pénétration dans le bulbe. Journal de Physiologie (Paris), 1958, 50, 63-74.

Bowker, R.M. & Morrison, A.R. The startle reflex and PGO spikes. Brain Research, 1976, 102, 185-190.

Bremer, F. Cerveau "isolé" et physiologie du sommeil. Comptes Rendue de Societe de Biologie (Paris), 1935, 118, 1235-1241.

Bremer, F. L'activité cérébrale au cours du sommeil et de la narcose. Contribution à l'étude du mécanisme du sommeil. Bull. Acad. Roy. Med. Belg., 1937, 4, 68-86.

Bremer, F. L'activité électrique de l'ecorce cérébrale et le problème physiologique du sommeil. Boll. Soc. Ital. Biol. Sper., 1938, 13, 271-290.

Bremer, F. Discussion of Hess' report p136: Hess, W.R. The diencphalic sleep centre, p117-136. In: E.D. Adrian, F. Bremer & H.H. Jasper (eds.) Brain Mechanisms and Cousciuousness. Blackwell, Oxford, 1954.

Bronzino, J., Morgane, P.J. & Stern, W.C. EEG synchronization following application of serotonin to area postrema. American Journal of Physiology, 1972, 223, 376-383.

Brooks, D.C. Localization of the lateral geniculate nucleus monophasic waves associated with paradoxical sleep in the cat. Electroencephalography & Clinical Neurophysiology, 1967a, 23, 123-133.

Brooks, D.C. Effect of bilateral optic nerve section on visual system monophasic wave activity in the cat. Electroencephalography & Clinical Neurophysiology, 1967b, 23, 134-141.

Brooks, D.C. Localization and characteristics of the cortical waves associated with eye movement in cat. Experimental Neurology, 1968a, 22, 603-613.

Brooks, D.C. Waves associated with eye movement in the awake and sleeping cat. Electroencephalography & Clinical Neurophysiology, 1968b, 24, 532-541.

Brooks, D.C. & Bizzi, E. Brain stem electrical activity during deep sleep. Archives italiennes de Biologie, 1963, 101, 648-665.

Brooks, D.C. & Gershon, M.D. Eye movement potentials in the oculomotor and visual systems of the cat: A comparison of reserpine induced waves with those present during wakefulness and rapid eye movement sleep. Brain Research, 1971, 27, 223-239.

Brooks, D.C. & Gershon, M.D. An analysis of the effect of reserpine upon ponto-geniculo-occipital wave activity in the cat. Neuropharmacology, 1972, 11, 499-510.

Brooks, D.C. & Gershon, M.D. Amine repletion in the reserpinized cat: Effect upon PGO waves and REM sleep. Electroencephalography & Clinical Neurophysiology, 1977, 42, 35-47.

Brooks, D.C., Gershon, M.D. & Simon, R.P. Brain stem serotonin depletion and ponto-geniculo-occipital waves activity in the cat treated with reserpine. Neuropharmacology, 1972, 11, 511-520.

Brown, B.B. Frequency and phase of hippocampal theta activity in the spontaneously behaving cat. Electroencephalography & Clinical Neurophysiology, 1968 24, 53-62.

Brown, D.L. & Salinger, W.L. Loss of X-cells in lateral geniculate nucleus with monocular paralysis: Neural plasticity in the adult cat. Science , 1975, 189, 1011-1012.

Buchtel, H.A., Berlucchi, G. & Mascetti, G.G. Modifications in visual perception and learning following immobilization of one eye in cats. Brain Research, 1972, 37, 355.

Buisseret, P. & Imbert, M. Visual cortical cells: Their developmental properties in normal and dark reared kittens. Journal of Physiology, 1976, 255, 511-525.

Burke, W. & Hayhow, W.R. Disuse in the lateral geniculate nucleus of the cat. Journal of Physiology, 1968, 194, 495-519.

Calvet, J., Calvet, M.C, & Langlois, J.M. Diffuse activation waves during so-called desynchronized EEG patterns. Journal of Neurophysiology, 1965, 28, 893-907.

Camacho-Evangelista, A. & Reinoso-Suarez, F. Activating and synchronizing centers in cat brain: Electroencephalograms after lesions. Science, 1964, 146, 268-270.

Candia, O., Rossi, G.F. & Sekino, T. Brain stem structures responsible for the electroencephalographic patterns of desynchronized sleep. Science, 1967, 155, 720-722.

Carli, G. Animal hypnosis in the rabbit. The Psychological Record, 1977, 27(1), 123-143.

Carli, G., Armengol, V. & Zanchetti, A. Brainstem-limbic connections, and the electrographic aspects of deep sleep in the cat. Archives italiennes de Biologie, 1965, 103, 725-750.

Carli, G. & Zanchetti, A. A study of pontine lesions suppressing deep sleep in the cat. Archives italiennes de Biologie, 1965, 103, 751-788.

Cespuglio, R., Laurent, J.P. & Calvo, J.M. Organisation anatomique des activités phasique provoquées par la réserpine au niveau du système oculo-moteur. Electroencephalography & Clinical Neurophysiology, 1976, 40, 12-24.

Cespuglio, R., Laurent, J.P. & Jouvet, M. Étude des relations entre l'activité ponto-geniculo-occipitale (PGO) et la motricité oculaire chez le chat sous réserpine. Brain Research, 1975, 83, 319-335.

Chase, M.H., Nakamura, Y., Clemente, C.D. & Sterman, M.B. Afferent vagal stimulation: Neurographic correlates of induced EEG synchronization and desynchronization. Brain Research, 1967, 5, 236-249.

Chase, M.H., Sterman, M.B. & Clemente, C.D. Cortical and subcortical patterns of response to afferent vagal stimulation. Experimental Neurology, 1966, 16, 36-49.

Chouvet, G. & Gadea-Ciria, M. Analyse sequentielle de l' activité PGO chez le chat. Electroencephalography & Clinical Neurophysiology, 1974, 36, 597-607.

Chow, K.L. & Stewart, D.L. Reversal of structural and functional effects of long-term visual deprivation in cats. Experimental Neurology, 1972, 34, 409-433.

Chu, N.-S. & Bloom, F.E. Activity patterns of catecholamine-containing pontine neurons in the dorso-lateral tegmentum of unrestrained cats. Journal of Neurobiology, 1974, 5, 527-544.

Claes, E. Contribution a l'étude physiologique de la fonction visuelle. I. Analyse oscillographique de l'activité spontanée et sensorielle de l'aire visuelle corticale chez le chat non anesthesié. Arch. Int. Physiol., 1939, 48, 181-237.

Clausen, J., Sersen, E.A. & Lidsky, A. Sleep patterns in mental retardation: Down's syndrome. Electroencephalography & Clinical Neurophysiology, 1977, 43, 183-191.

Clemente, C.D., Sterman, M.B. & Wyrwicka, W. Post-reinforcement EEG synchronization during alimentary behavior. Electroencephalography & Clinical Neurophysiology, 1964, 16, 355-365.

Cohen, B. Vestibulo-ocular relations, pp105-148. In: P. Bach-Y-Rita & C.C. Collins (eds.) The Control of Eye Movements. Academic Press, N.Y., 1971.

Cohen, B. & Feldman, M. Relationship of electrical activity in pontine reticular formation and lateral geniculate body to rapid eye movements. Journal of Neurophysiology, 1968, 31, 806-817.

Cohen, H.B., Dement, W.C. & Barchas, J.D. Effects of chlorpromazine on sleep in cats pretreated with parachlorophenylalanine. Brain Research, 1973, 53, 363-371.

Cragg, B., Anker, R. & Wan, Y.K. The effect of age on reversibility of cellular atrophy in the LGN of the cat following monocular deprivation: A test of two hypotheses about cell growth. Journal of Comparative Neurology, 1976, 168, 345-354.

Crawford, F.T. & Prestrude, A.M. (eds.) Animal Hypnosis: Research and Theory. Psychological Record, 1977, 27 special issue (1), 1-218.

Creutzfeldt, O.D. & Heggelund, P. Neural plasticity in visual cortex of adult cats after exposure to visual patterns. Science, 1975, 188, 1025-1027.

Crommelinck, M. & Roucoux, A. Characteristics of cat's eye saccades in different states of alertness. Brain Research, 1976, 103, 574-578.

Cynader, M., Berman, N. & Hein, A. Recovery of function in cat visual cortex following prolonged deprivation. Experimental Brain Research, 1976, 25, 139-156.

Dallaire, A. & Ruckebusch, Y. Sleep patterns in the pony with observations on partial perceptual deprivation. Physiology & Behavior, 1974, 12, 789-796.

Davies, P. & Jones, G.M. An adaptive neual model compatible with plastic changes induced in the human vestibulo-ocular reflex by prolonged optical reversal of vision. Brain Research, 1976, 103, 546-550.

Daw, N.W. & Wyatt, H.J. Kittens reared in an unidirectional environment: Evidence for a critical period. Journal of Physiology, 1976, 257, 155-170.

De La Peña, A., Zarcone, V. & Dement, W.C. Correlation between measures of the rapid eye movements of wakefulness and sleep. Psychophysiology, 1973, 10, 488-500.

Dell, P. & Padel, Y. Rapid falling asleep provoked by selective stimulation of vagal afferents in the cat. Electroencephalography & Clinical Neurophsyiology, 1965, 18, 720-726.

Delorme, F., Froment, J.L. & Jouvet, M. Suppression du sommeil par la p-chloromethamphetamine et p-chlorophenylalanine. Comptes Rendus des Seances de la Societe de Biologie (Paris), 1966, 160, 2347-2351.

Delorme, F., Vimont, P. & Jouvet, D. Etude statistique du cycle veille-sommeils chez le chat. Comptes Rendus Des Seances de la Societe de Biologie (Paris), 1964, 158, 2128-2130.

Dement, W.C. The occurrence of low voltage, fast, electroencephalogram patterns during behavioral sleep in the cat. Electroencephalography & Clinical Neurophysiology, 1958, 10, 291-296.

Dement, W. The effect of dream deprivation. Science, 1960, 131, 1705-1707.

Dement, W., Ferguson, J., Cohen, H. & Barchas, J. Non-chemical methods and data using a biochemical model: The REM quanta, pp275-325. In: A. Mandel (ed.) Some Current Issues in Psychochemical Research Strategies in Man. Academic Press, New York, 1970.

Dement, W.C. & Kleitman, N. Cyclic variations in EEG during sleep and their relation to eye movements, body motility and dreaming. Electroencephalography & Clinical Neurophysiology, 1957a, 9, 673-690.

Dement, W.C. & Kleitman, N. The relation of eye movements during sleep to dream activity: An objective method for the study of dreaming. Journal of Experimental Psychology, 1957b, 53, 339-346.

Dempsey, E.W. & Morison, R.S. The production of rhythmically recurrent cortical potentials after localized thalamic stimulation. American Journal of Physiology, 1942, 135, 293-300.

Dews, P.B. & Wiesel, T.N. Consequences of monocular deprivation on visual behavior in kittens. Journal of Physiology, 1970, 206, 437-455.

Doane, B.K. Changes in Visual Function with Perceptual Isolation. Doctoral Thesis, McGill University, 1956.

Doane, B.K., Mahatoo, W., Heron, W. & Scott, T.H. Changes in perceptual function after isolation. Canadian Journal of Psychology, 1959, 13, 210-219.

Doty, R.W. Potentials evoked in cat cerebral cortex by diffuse and by punctiform photic stimuli. Journal of Neurophysiology, 1958, 21, 437-464.

Dürsteler, M.R., Garey, L.J. & Movshon, J.A. Reversal of the morphological effects of monocular deprivation in the kitten's lateral geniculate nucleus. Journal of Physiology, 1976, 261, 189-210.

Edmonds, S.C. & Adler, N.T. Food and light as entrainers of circadian running activity in the rat. Physiology & Behavior, 1977a, 18, 915-919.

Edmonds, S.C. & Adler, N.T. The multiplicity of biological oscillators in the control of circadian running activity in the rat. Physiology & Behavior, 1977b, 18, 921-930.

Ephron, H.S. & Carrington, P. Rapid eye movement sleep and cortical homeostasis. Psychological Review, 1966, 73, 500-526.

Favale, E., Loeb, C., Rossi, G.F. & Sacco, G. EEG synchronization and behavioral signs of sleep following low frequency stimulation of the brain stem reticular formation. Archives italiennes de Biologie, 1961, 99, 1-22.

Feinberg, I. Eye movement activity during sleep and intellectual function in mental retardation. Science, 1968, 159, 1256.

Ferguson, J. & Dement, W. The effect of variations in total sleep time on the occurrence of rapid eye movement sleep in cats. Electroencephalography & Clinical Neurophysiology, 1967, 22, 2-10.

Fiorentini, A. & Maffei, L. Change of biocular properties of the simple cells of the cortex in adult cats following immobilization of one eye. Vision Research, 1974, 14, 217-218.

Fishbein, W.M Schaumberg, H. & Weitzman, E.D. Rapid eye movements during sleep in dark-reared kittens. Journal of Nervous & Mental Disease, 1966, 143, 281-283.

Fishman, R. & Roffwarg, H.P. REM sleep inhibition by light in the albino rat. Experimental Neurology, 1972, 36, 166-178.

Fleming, T.C. & Evarts, E.V. Multiple response to photic stimulation in cats. American Journal of Physiology, 1959, 197, 1233-1236.

Foutz, A.S., Ternaux, J.P. & Puizillout, J.J. Les stades du sommeil de la preparation encephale isole: II Phases paradoxales. Leur declenchement par la stimulation des afferences baroceptives. Electro-encephalography & Clinical Neurophysiology, 1974, 37, 577-588.

Fuchs, A.F. & Ron, S. An analysis of rapid eye movements of sleep in the monkey. Electroencephalography & Clinical Neurophysiology, 1968, 25, 244-251.

Gaarder, K. A conceptual model of sleep. Archives of General Psychiatry, 1966, 14, 253-260.

Gadea-Ciria, M. Etude des latences entre les activites ponto-geniculo-occipitales au niveau du pont des noyaux genicules lateraux et des muscles droits externes chez le chat. Comptes Rendus des Sceances de la Societe de Biologie (Paris), 1972, 166, 634-639.

Gadea-Ciria, M. Cerebellar control of activity of the feline oculomotor system during paradoxical sleep. Experimental Neurology, 1976a, 51, 263-265.

Gadea-Ciria, M. Plasticity of ponto-geniculo-occipital waves during paradoxical sleep after frontal lobe lesions in the cat. Experimental Neurology, 1976b, 53, 328-338.

Gadea-Ciria, M. Sequential discharges of phasic activities (PGO waves) during paradoxical sleep after selective cortical lesions in the cat. Archives Italiennes de Biologie, 1976c, 114, 399-408.

Gadea-Ciria, M. Contribution of the frontal lobes to modulation of phasic activities in the oculomotor system during paradoxical sleep in the cat. Brain Research, 1977a, 120, 347-353.

Gadea-Ciria, M. Sequential discharges of phasic waves in the pontine oculomotor system during paradoxical sleep in chronic mesencephalic and decorticate cats. Electroencephalography & Clinical Neurophysiology, 1977b, 42, 709-712.

Ganz, L. & Fitch, M. The effect of visual deprivation on perceptual behavior. Experimental Neurology, 1968, 22, 638-660.

Ganz, L., Fitch, M. & Satterberg, J.A. The selective effect of visual deprivation on receptive field shape determined neurophysiologically. Experimental Neurology, 1968, 22, 614-637.

Gastaut, H. & Bert, J. Electroencephalographic detection of sleep induced by repetitive sensory stimuli, pp260-283. In: G.E.W. Wolstenholme & M. O'Connor (eds.) The Nature of Sleep. J. & A. Churchill Ltd., London, 1961.

Gonshor, A. & Malcolm, R. Effect of changes in illumination level on electro-oculography (EOG). Aerospace Medicine, 1971, 42, 138-140.

Gordon, B. & Gummow, L. Effects of extraocular muscle section on receptive fields in cat superior colliculus. Vision Research, 1975, 15, 1011-1019.

Groves, P.M., Miller, S.W., Parker, M.V. & Rebec, G.V. Organization by sensory modality in the reticular formation of the rat. Brain Research, 1973, 54, 207-224.

Guilford, J.P. Fundamental Statistics in Psychology and Education. McGraw Hill Book Company, Toronto, 1965.

Hagamen, W.D. Responses of cats to tactile and noxious stimuli. Archives of Neurology, 1959, 1, 203-215.

Harrison, F. An attempt to produce sleep by diencephalic stimulation. Journal of Neurophysiology, 1940, 3, 156-165.

Headon, M.P. & Powell, T.P.S. Cellular changes in the lateral geniculate nucleus of infant monkey after suture of the eyelids. Journal of Anatomy, 1973, 116, 135-145.

Hebb, D.O. The Organization of Behavior. A Neurophysiological Theory. John Wiley & Sons Inc., New York, 1949.

Hernández Peon, R. & Chávez Ibarra, G. Sleep induced by electrical or chemical stimulation of the forebrain. Electroencephalography & Clinical Neurophysiology, 1963, Supplement 24, 188-198.

Heron, W. The pathology of boredom. Scientific American. 1957, 196, 52-56.

Heron, W. Cognitive and physiological effects of perceptual isolation, pp6-33. In: P. Solomon et. al. (eds.) Sensory Deprivation. Harvard Universiry Press, Cambridge, 1961.

Heron, W., Doane, B.K. & Scott, T.H. Visual disturbances after prolonged perceptual isolation. Canadian Journal of Psychology, 1956, 10, 13-18.

Heron, W., Tait, G. & Smith, G.K. Effects of prolonged perceptual deprivation on the human electroencephalogram. Brain Research, 1972, 43, 280-284.

Hess, E.H. Imprinting. Science, 1959, 130, 133-141.

Hess, W.R. The diencephalic sleep centre, pp117-136. In: E.D. Adrian, F. Bremer & H.H. Jasper (eds.) Brain Mechanisms and Consciousness. Blackwell, Oxford, 1954.

Hickey, T.L., Spear, P.D. & Kratz, K.E. Quantitative studies of cell size in the cat's dorsal lateral geniculate nucleus following visual deprivation. Journal of Comparative Neurology, 1977, 172, 265-282.

Hoagland, H. Temperature characteristics of the "Berger rhythm" in man. Science, 1936, 83, 84[illegible]5.

Hobson, J.A. The effects of chronic brainstem lesions on cortical and muscular activity during sleep and waking in the cat. Electroencephalography & Glinical Neurophysiology, 1965, 19, 41-62.

Hobson, J.A. Sleep after exercise. Science, 1968, 162, 1503-1505.

Hobson, J.A., Alexander, J. & Frederickson, C.J. The effect of lateral geniculate lesions on phasic electrical activity of the cortex during desynchronized sleep in the cat. Brain Research, 1969, 14, 607-621.

Hobson, J.A., McCarley, R.W., Freedman, R. & Pivik, R.T. Time course of discharge rate changes by cat pontine brainstem neurons during sleep cycle. Journal of Neurophysiology, 1974b, 37, 1297-1309.

Hobson, J.A., McCarley, R.W., Pivik, R.T. & Freedman, R. Selective firing by cat pontine brain stem neurons in desynchronized sleep. Journal of Neurophysiology, 1974a, 37, 497-511.

Hobson, J.A., McCarley, R.W, & Wyzinski, P.W. Sleep cycle oscillation: Reciprocal discharge by two brainstem neuronal groups. Science, 1975, 159, 55-58.

Hodes, R. Electrocortical synchronization resulting from reduced proprioceptive drive caused by neuromuscular blocking agents. Electroencephalography & Clinical Neurophysiology, 1962, 14, 220-232.

Hoffman, K.P. & Cynader, M. Recovery in the LGN of the cat after early visual deprivation. Brain Research, 1975, 85, 179.

Homer, L.D., Kolder, H. & Benson, D.W., Jr. Parameter variations of a model of the oscillation of the human corneoretinal potential. Pflugers Archives , 1967, 294, 103-112.

Horne, J.A. & Porter, J.M. Time of day effects with standardized exercise upon subsequent sleep. Electroencephalography & Clinical Neurophysiology, 1976, 40, 178-184.

Horne, J.A. & Walmsley, B. Daytime visual load and the effects upon human sleep. Psychophysiology, 1976, 13, 115-120.

Hoshino, K. & Pompeiano, O. Selective discharge of pontine neurons during the postural atonia produced by an anticholinesterase in the decerebrate cat. Archives italiennes de Biologie, 1976, 114, 244-277.

Hoshino, K., Pompeiano, O., Magherini, P.C. & Mergner, T. The oscillatory system responsible for the oculomotor activity during the bursts of REMs. Archives italiennes de Biologie, 1976, 114, 278-309.

Howe, R.C. & Sterman, M.B. Cortical-subcortical EEG correlates of suppressed motor behavior during sleep and waking in the cat. Electroencephalography & Clinical Neurophysiology, 1972, 32, 681-695.

Howe, R.C. & Sterman, M.B. Somatosensory system evoked potentials during waking behavior and sleep in the cat. Electroencephalography & Clinical Neurophysiology, 1973, 34, 605-618.

Hubel, D.H. & Wiesel, T.N. The period of susceptibility to the physiological effects of unilateral eye closure in kittens. Journal of Physiology, 1970, 206, 419-436.

Ingram, W.R., Barris, R.W. & Ranson, S.W. Catalepsy: An experimental study. Archives of Neurology & Psychiatry, 1936, 35, 1175-1197.

Iwamura, Y., Uchino, Y. & Kidokoro, Y. Blood pressure and heart rate changes during para-sleep in vagotomized and atropinized cats. Brain Research, 1968, 7, 182-190.

Jacobs, L., Feldman, M., Rabinowitz, M. & Bender, M.B. Alterations of the corneo-fundal potential of the eye during sleep. Electroencephalography & Clinical Neurophysiology, 1973, 34, 579-586.

Jasper, H.H. Diffuse projection system. The integrative action of the thalamic reticular systems. Electroencephalography & Clinical Neurophysiology, 1949, 1, 405-420.

Jasper, H.H. & Andrews, H.L. Electro-encephalography III. Normal differentiation of occipital and precentral regions in man. Archives of Neurology & Psychiatry, 1938, 39, 96-115.

Jeannerod, M. & Kiyono, S. Décharge unitaire de la formation réticulée pontique et activite phasique ponto-geniculo-occipitale chez le chat sous réserpine. Brain Research, 1969a, 12, 112-128.

Jeannerod, M. & Kiyono, S. Effets de la réserpine sur la reponse reticulaire aux stimulations sensorielles. Brain Research, 1969b, 12, 129-137.

Jeannerod, M., Mouret, J. & Jouvet, M. Étude de la motricité oculaire au cours de la phase paradoxale du sommeil chez le chat. Electroencephalography & Clinical Neurophysiology, 1965, 18, 554-566.

Jeannerod, M. & Sakai, K. Occipital and geniculate potentials related to eye movements in the unanaesthetized cat. Brain Research, 1970, 19, 361-377.

Jones, G. & Pasnak, R. Light deprivation and visual-cliff performance in the adult cat. Psychonomic Science, 1970, 21, 278-279.

Jones, G.M. & Davies, P. Adaptation of cat vestibulo-ocular reflex to 200 days of optically reversed vision. Brain Research, 1976, 103, 551-554.

Jones, G.M. & Sugie, N. Vestibulo-ocular responses in man during sleep. Electroencephalography & Clinical Neurophysiology, 1972, 32, 43-53.

Jouvet, M. Recherches sur le structure nerveuses et les mécanisms responsables des differentes phases du sommeil physiologique. Archives italiennes de Biologie, 1962, 100, 125-206.

Jouvet, M. Neurophysiology of the states of sleep. Physiological Reviews, 1967, 47, 117-177.

Jouvet, M. Biogenic amines and the states of sleep. Science, 1969, 163, 32-41.

Jouvet, M. The role of monoamines and acetylcholine-containing neurons in the regulation of the sleep-waking cycle. Ergebnisse der Physiologie, 1972, 64, 166-307.

Jouvet, M. & Michel, F. Corrélations electrmyographic du sommeil chez le chat decortique et mesencephalique chronique. Comptes Rendus des Sceances de la Societe de Biologie (Paris), 1959, 153, 422-425.

Jouvet, M. & Renault, J. Insomnie persistante apres lesions des noyaux du raphe chez le chat. Comptes Rendus des Sceances de la Societe de Biologie, 1966, 160, 1461-1465.

Kaas, J.H., Guillery, R.W. & Allman, J.M. Some principles of organization in the dorsal lateral geniculate nucleus. Brain, Behavior and Evolution, 1972, 6, 253-299.

Karczmar, A.G., Longo, V.G. & Scotti de Carolis, A. A pharmacological model of paradoxical sleep: The role of cholinergic and monoamine systems. Physiology & Behavior, 1970, 5, 175-182.

Kawamura, H. & Yamamoto, K. Specific activation and induced spindle bursts in the auditory system. Japanese Journal of Physiology, 1961, 11, 260-269.

Khananashvili, M.M. & Bogoslovskii, M.M. Electrographic manifestations of sleep in the neuronally isolated cat cortex. Neurofiziologiya, 1976, 8, 559-567. (translated from Russian)

Kim, C., Choi, H., Kim, C.C., Kim, J.K., Kim, M.S., Park, H.J. & Ahn, B.T. Effect of hippocampectomy on sleep patterns in cats. Electroencephalography & Clinical Neurophysiology, 1975, 38, 235-243.

Kimura, D. Multiple response of visual cortex of the rat to photic stimulation. Electroencephalography & Clinical Neurophysiology, 1962, 14, 115-122.

Kiyono, S., Kawamoto, T., Sakakura, H. & Iwama, K. Effects of sleep deprivation upon the paradoxical phase of sleep in cats. Electroencephalography & Clinical Neurophysiology, 1965, 19, 34-40.

Kleitman, N. Studies on the physiology of sleep. V. Some experiments on puppies. American Journal of Physiology, 1927, 84, 386-395.

Kleitman, N. Sleep and Wakefulness. University of Chicago Press, Chicago, 1939.

Kleitman, N. & Camille, N. Studies on the physiology of sleep. VI. The behavior of decorticated dogs. American Journal of Physiology, 1932, 100, 474-480.

Klemm W.R. Identity of sensory and motor systems that are critical to the immobility reflex (Animal hypnosis). The Psychological Record, 1977, 27(1), 145-159.

Koella, W.P., Feldstein, A. & Czicman, J.S. The effect of para-chlorophenylalanine on the sleep of cats. Electroencephalography & Clinical Neurophysiology, 1968, 25, 481-490.

Konorski, J. Conditioned Reflexes and Neuron Organization. Cambridge University Press, Cambridge, 1948.

Konrad, K.W. & Bagshaw, M. Effects of novel stimuli on cats reared in a restricted environment. Journal of Comparative & Physiological Psychology, 1970, 70, 157-164.

Kratz, K. & Spear, P.D. Effects of visual deprivation and alterations in binocular competition on responses of striate cortex neurons in the cat. Journal of Comparative Neurology, 1976, 170, 141-151.

Kratz, K.E., Spear, P.D. & Smith, D.C. Postcritical-period reversal of effects of monocular deprivation on striate cortex cells in the cat. Journal of Neurophysiology, 1976, 39, 501-511.

Kris, C. Diurnal variation in periorbitally measured eye potential level. Electroencephalography & Clinical Neurophysiology, 1957, 9, 382.

Kris, C. Corneo-fundal potential variations during light and dark adaptation. Nature, 1958, 182, 1027-1028.

Kris, C. Vision: Electro-oculography, pp692-700. In: O. Glasser (ed.) Medical Physics, Volume 3. Year Book Publisher Inc., Chicago, 1960.

Kubzansky, P.E. The effects of reduced environmental stimulation on human behavior: A review, pp51-95. In: A.D. Biderman & H. Zimmer (eds.) The Manipulation of Human Behavior. John Wiley & Sons, New York, 1961.

Kuffler, S.W., Fitzhugh, R. & Barlow. H.B. Maintained activity in the cat's retina in light and darkness. Journal of General Physiology, 1957, 40, 683-702.

Laurent, J.P., Cespuglio, R. & Jouvet, M. Delimitation des voies ascendantes de l'activité ponto-géniculo-occipitales chez le chat. Brain Research, 1974, 65, 29-52.

Laurent, J.-P. & Guerrero, F.A. Reversible suppression of ponto-geniculo-occipital waves by localized cooling during paradoxical sleep in cats. Experimental Neurology, 1975, 49, 356-369.

Laurent, J.-P., Guerrero, A. & Jouvet, M. Reversible suppression of the geniculate PGO waves and of the concomitant increase of excitability of the intrageniculate optic nerve terminals in cats. Brain Research, 1974, 81, 558-563.

Lindsley, D.B. Common factors in sensory deprivation, sensory distortion and sensory overload. pp174-194. In: P. Solomon et. al. (eds.) Sensory Deprivation. Harvard University Press, Cambridge, 1961.

Lindsley, D.B., Bowden, J.W. & Magoun, H.W. Effect upon the EEG of acute injury to the brain stem activating system. Electroencephalography & Clinical Neurophysiology, 1949, 1, 475-486.

Lindsley, D.B., Schreiner, L.H., Knowles, W.B. & Magoun, H.W. Behavioral and EEG changes following chronic brain stem lesions in the cat. Electroencephalography & Clinical Neurophysiology, 1950, 2, 483-498.

Linnstaedter, L. The Effects of Operantly Conditioning Lateral Geniculate Spiking During Wakefulness on Paradoxical Sleep in the Owl Monkey (Aotus Trivergatus) PhD Thesis, Emory University, 1971.

LoPiccolo, M. Behavioral and Neuronal Effects of EEG Synchronizing Stimuli in the Cat. Unpublished PhD Thesis, McMaster University, 1977.

Lorente de No, R. Vestibulo-ocular reflex arc. Archives of Neurology & Psychiatry, 1933, 30, 245-291.

Lorenz, K. Uber die Bildung des Instinkbegriffes. Naturwiss, 1937, 25, 289-300.

Maffei, L. & Fiorentini, A. Asymmetry of motility of the eyes and changes of binocular properties of cortical cells in adult cats. Brain Research, 1976, 105, 73-78.

Magherini, P.C., Pompeiano, O. & Thoden, U. Cholinergic mechanisms related to REM sleep. I. Rhythmic activity of the vestibulo-oculomotor system induced by an anticholinesterase in the decerebrate cat. Archives italiennes de Biologie, 1972, 110, 234-259.

Magnes, J., Moruzzi, G. & Pompeiano, O. Synchronization of the EEG produced by low-frequency electrical stimulation of the region of the solitary tract. Archives italiennes de Biologie, 1961, 99, 33-67.

Magni, F., Moruzzi, G., Rossi, G.F. & Zanchetti, A. EEG arousal following inactivation of the lower brain stem by selective injection of barbiturate into the vertebral circulation. Archives italiennes de Biologie, 1959, 97, 33-46.

Magoun, H.W., Harrison, F., Brobeck, J.R. & Ranson, S.W. Activation of heat loss mechanism by local heating of the brain. Journal of Neurophysiology, 1938, 1, 101-114.

Magoun, H.W. & Rhines, R. An inhibitory mechanism in the bulbar reticular formation. Journal of Neurophysiology, 1946, 9, 165-171.

Malcolm, L.J., Watson, J.A. & Burke, W. PGO waves as unitary events. Brain Research, 1970, 24, 130-133.

Marczynski, T.J. & Sherry, C.J. Induction of sleep and "reward contingent positive variation" by photic stimuli in cat. Experimental Neurology, 1972, 34, 103-114.

Marg, E. Development of electro-oculography. Archives of Ophthalmology, 1951, 45, 169-185.

Mazzella, H., Garcia-Austt, E. & Garcia-Mullin, R. Carotid sinus and EEG. Electroencephalography & Clinical Neurophysiology, 1956, 8, 155.

McCarley, R.W. & Hobson, J.A. Discharge patterns of cat pontine brain stem neurons during desynchronized sleep. Journal of Neurophysiology, 1975a, 38, 751-766.

McCarley, R.W. & Hobson, J.A. Neuronal excitability modulation over the sleep cycle: A structural and mathematical model. Science, 1975b, 159, 58-60.

McGinty, D.J. Somnolence, recovery and hyposomnia following ventro-medial diencephalic lesions in the cat. Electroencephalography & Clinical Neurophysiology, 1969a, 26, 70-79.

McGinty, D.J. Effects of prolonged isolation and subsequent enrichment on sleep patterns in kittens. Electroencephalography & Clinical Neurophysiology, 1969b, 26, 332.

McGinty, D.J. & Harper, R.M. Dorsal raphe neurons: Depression of firing during sleep in cats. Brain Research, 1976, 101, 569-575.

McGinty, D.J. & Sterman, M.B. Sleep suppression after basal forebrain lesions in the cat. Science, 1968, 160, 1253-1255.

Melzack, R. Effects of early experience on behavior: Experimental and conceptual considerations. pp271-299. In: P.H. Hoch & J. Zubin (eds.) Psychopathology of Perception. Grune & Stratton, New York, 1965.

Mendelson, J., Kubzansky, P., Leiderman, P.H., Wexler, D., DuToit, C. & Solomon, P. Catecholamine excretion and behavior during sensory deprivation. Archives of General Psychiatry, 1960, 2, 147-155.

Miller, R.E., Caul, W.F. & Mirsky, I.A. Patterns of eating and drinking in socially-isolated rhesus monkeys. Physiology & Behavior, 1971, 7, 127-134.

Miller, R.E., Mirsky, I.A., Caul, W.F. & Sakata, T. Hyperphagia and polydipsia in socially isolated rhesus monkeys. Science, 1969, 165, 1027-1028.

Mills, J.N., Minors, D.S. & Waterhouse, J.M. The circadian rhythms of human subjects without timepieces or indications of the alternation of day and night. Journal of Physiology, 1974, 240, 567-594.

Morgane, P.J. Medial forebrain bundle and "feeding centers" of the hypothalamus. Journal of Comparative Neurology, 1961, 117, 1-25.

Morison, R.S. & Bassett, D.L. Electrical activity of the thalamus and basal ganglia in decorticate cats. Journal of Neurophysiology, 1945, 8, 309-314.

Morison, R.S. & Dempsey, E.W. A study of thalamo-cortical relations. Journal of Physiology, 1942, 135, 281-292.

Morrison, A.R. & Pompeiano, O. Vestibular influences during sleep. II. Effects of vestibular lesions on the pyramidal discharge during desynchronized sleep. Archives italiennes de Biologie, 1966, 104, 214-230.

Moruzzi, G. Synchronizing influences of the brain stem and the inhibitory mechanisms underlying the production of sleep by sensory stimulation. In: H.H. Jasper & G.D. Smirnov (eds.) Moscow Colloquium, 1958. Electroencephalography & Clinical Neurophysiology, 1960, Supplement 13, 231-256.

Moruzzi, G. Active processes in the brain stem during sleep. Harvey Lectures, 1963, 58, 233-297.

Moruzzi, G. The historical developemnt of the deafferentation hypothesis of sleep. Proceedings of the American Philosophical Society, 1964, 108, 19-28.

Moruzzi, G. Sleep and instinctive behavior. Archives italiennes de Biologie, 1969, 107, 175-216.

Moruzzi, G. The sleep-waking cycle. Ergebnisse der Physiologie, 1972, 64, 1-165.

Moruzzi, G. & Magoun, H.W. Brain stem reticular formation and activation of the EEG. Electroencephalography & Clinical Neurophsiology, 1949, 1, 455-473.

Movshon, J.A. Reversal of the physiological effects of monocular deprivation in the kitten's visual cortex. Journal of Physiology, 1976a, 261, 125-174.

Movshon, J.A. Reversal of the behavioral effects of monocular deprivation in the kitten. Journal of Physiology, 1976b, 261, 175-187.

Movshon, J.A. & Blakemore, C. Functional reinnervation in kitten visual cortex. Nature, 1974, 251, 504-505.

Mowrer, O., Ruch, T.C. & Miller, N.E. The corneo-retinal potential difference as the basis of the galvanometric method of recording eye movements. American Journal of Physiology, 1936, 114, 423-428.

Murphy, C.W., Kurlents, E., Cleghorn, R.A. & Hebb, D.O. Absence of increased corticoid excretion with the stress of perceptual deprivation. Canadian Journal of Biochemical Physiology, 1955, 33, 1062-1063.

Nakayama, T., Hammel, H.T., Hardy, J.D. & Eisenman, J.S. Thermal stimulation of electrical activity of single units of the preoptic region. American Journal of Physiology, 1963, 204, 1122-1126.

Nauta, W.J.H. Hypothalamic regulation of sleep in rats. An experimental study. Journal of Neurophysiology, 1946, 9, 285-316.

Nauta, W.J.H. & Haymaker, W. Hypothalamic nuclei and fiber connections, pp136-209. In: W. Haymaker, E. Anderson & W.J.H. Nauta (eds.) The Hypothalamus. Charles C. Thomas, Springfield, 1969.

Netick, A., Orem, J. & Dement, W. Neuronal activity specific to REM sleep and its relationship to breathing. Brain Research, 1977, 120, 197-207.

Ornitz, E.M. Development of sleep patterns in autistic children, pp363-381. In: C.D. Clemente, D.P. Purpura & F.E. Mayer (eds.) Sleep and the Maturing Nervous System. Academic Press, New York, 1972.

Ornitz, E.M., Forsythe, A.B. & De La Pena, A. The effect of vestibular and auditory stimulation on the rapid eye movements of REM sleep in normal children. Electroencephalography & Clinical Neurophysiology, 1973, 34, 379-390.

Ornitz, E.M., Ritvo, E.R., Brown, M.B., La Franchi, S., Parmelee, T. & Walter, R.D. The EEG and rapid eye movements during REM sleep in normal and autistic children. Electroencephalography & Clinical Neurophysiology, 1969, 26, 167-175.

Oswald, I. Experimental studies of rhythm, anxiety and cerebral vigilance. Journal of Mental Science, 1959, 105, 269-294.

Oswald, I. Falling asleep open-eyed during intense rhythmic stimulation. British Medical Journal, 1960, 1, 1450-1455.

Oswald, I. Human brain protein, drugs and dreams. Nature, 1969, 223, 893-897.

Pangborn, R.M. & Sharon, I.M. Visual deprivation and parotid response to cigarette smoking. Physiology & Behavior, 1971, 6, 559-561.

Parmeggiani, P.L. Sleep behavior elicited by electrical stimulation of cortical and subcortical structures in the cat. Helvica Physiologica Acta, 1962, 20, 347-367.

Parmeggiani, P.L., Agnati, L.F., Zamboni, G. & Cianci, T. Hypothalamic temperature during the sleep cycle at different ambient temperatures. Electroencephalography & Clinical Neurophysiology, 1975, 38, 589-596.

Parmeggiani, P.L., Franzini, C., Lenzi, P. & Zamboni, G. Threshold of respiratory responses to preoptic heating during sleep in freely moving cats. Brain Research, 1973, 52, 189-201.

Parmeggiani, P.L. & Rabini, C. Sleep and environmental temperature. Archives italiennes de Biologie, 1970, 108, 369-387.

Parmeggiani, P.L. & Sabattini, L. Electromyographic aspects of postural, respiratory and thermoregulatory mechanisms in sleeping cats. Electroencephalography & Clinical Neurophysiology, 1972, 33, 1-13.

Pasik, P., Pasik, T. & Bender, M.B. Recovery of the electro-oculogram after total ablation of the retina in monkeys. Electroencephalography & Clinical Neurophysiology, 1965, 19, 291-297.

Pavlov, I.P. Conditioned Reflexes. An Investigation of the Physiological Activity of the Cerebral Cortex. Translated by G.V. Anrep. Dover Publications Inc., New York, 1927.

Perenin, M.T. & Jeannerod, M. Lesions internucleaires: Effets sur la motricite oculaire pendant l'eveil et le sommeil paradoxal chez le chat. Brain Research, 1971, 32, 299-310.

Perenin, M.T., Maeda, T. & Jeannerod, M. Are vestibular nuclei responsible for rapid eye movements of paradoxical sleep? Brain Research, 1972, 43, 617-621.

Petre-Quadens, O. & de Lee, C. Eye-movements during sleep: A common criterion of learning capacities and endocrine activity. Developmental Medicine & Child Neurology, 1970, 12, 730-740.

Pettigrew, J.D. The effect of visual experience on the development of stimulus specificity by kitten cortical neurones. Journal of Physiology, 1974, 237, 49-74.

Pivik, R.T., McCarley, R.W. & Hobson, J.A. Eye movement - associated discharge in brain stem neurons during desynchronized sleep. Brain Research, 1977, 121, 59-76.

Pompeiano, O. Mechanisms of sensorimotor integration during sleep, pp1-179. In: E. Stellar & J.M. Sprague (eds.) Progress in Physiological Psychology (Vol.3). Academic Press, New York, 1970.

Pompeiano, O. & Hoshino, K. Tonic inhibition of dorsal pontine neurons during the postural atonia produced by an anticholinesterase in the decerebrate cat. Archives italiennes de Biologie, 1976, 114, 310-340.

Pompeiano, O. & Morrison, A.R. Vestibular influence during sleep. I. Abolition of the rapid eye movements of desynchronized sleep following vestibular lesions. Archives italiennes de Biologie, 1965, 103, 569-595.

Pompeiano, O. & Morrison, A.R. Vestibular influences during sleep. III. Dissociation of the tonic and phasic inhibition of spinal reflexes during desynchronized sleep following vestibular lesions. Archives italiennes de Biologie, 1966, 104, 231-246.

Pompeiano, O. & Swett, J.E. EEG and behavioral manifestations of sleep induced by cutaneous nerve stimulation in normal cats. Archives italiennes de Biologie, 1962a, 100, 311-342.

Pompeiano, O. & Swett, J.E. Identification of cutaneous and muscular afferent fibers producing EEG synchronization or arousal in normal cat. Archives italiennes de Biologie, 1962b, 100, 343-380.

Pompeiano, O. & Swett, J.E. Actions of graded cutaneous and muscular afferent volleys on brain stem units in the decerebrate cerebellectomized cat. Archives italiennes de Biologie, 1963, 101, 553-583.

Pompeiano, O. & Valentinuzzi, M. A mathematical model for the mechanism of rapid eye movements induced by an anticholinesterase in the decerebrate cat. Archives italiennes de Biologie, 1976, 114, 103-154.

Potter, W. & Heron, W. Sleep during perceptual deprivation. Brain Research, 1972, 40, 534-539.

Puizillout, J.J. & Foutz, A.S. Vago-aortic nerves stimulation and REM sleep: Evidence for a REM-triggering and a REM-maintenance factor. Brain Research, 1976, 111, 181-184.

Puizillout, J.J. & Ternaux, J.P. Endormement vago-aortique apres section saggittale médiane du tronc cérébral et après administration de p-chlorophenylalanine, ou destruction des noyaux du Raphé. Brain Research, 1974, 70, 19-42.

Pujol, J.-F., Buguet, A., Froment, J.-L., Jones, B. & Jouvet, M. The central metabolism of serotonin in the cat during insomnia. A neurophysiological and biochemical study after administration of p-chlorophenylalanine or destruction of the raphe system. Brain Research, 1971, 29, 195-212.

Ranson, S.W. Somnolence caused by hypothalamic lesions in the monkey. Archives of Neurology & Psychiatry, 1939, 41, 1-23.

Riesen, A.H. The development of visual perception in man and chimpanzee. Science, 1947, 106, 107-108.

Riesen, A.H. Excessive arousal effects of stimulation after early sensory deprivation. pp34-40. In: P. Solomon et. al. (eds.) Sensory Deprivation. Harvard University Press, Cambridge, 1961a.

Riesen, A.H. Stimulation as a requirement for growth and function in behavioral development. pp57-80. In: D.W. Fiske & S.R. Maddi (eds.) Function of Varied Experience. The Dorsey Press Inc., Homewood, Ill., 1961b.

Riesen, A.H. Effects of visual deprivation on perceptual function and the neural substrate. pp47-66. In: J. de Ajuriaguerra (ed.) Desafferentation Expérimentale et Clinique. Georg & Cie S.A., Geneve, Switzerland, 1965.

Riesen, A.H. Sensory deprivation pp117-147. In: E. Stellar & J.M. Sprague (eds.) Progress in Physiological Psychology. Academic Press, New York, 1966.

Rizzolatti, G. & Tradardi, V. Pattern discrimination in monocularly reared cats. Experimental Neurology, 1971, 33, 181-194.

Roberts, W.W., Berquist, E.H. & Robinson, T.C.L. Thermoregulatory grooming and sleep-like relaxation induced by local warming of preoptic area and anterior hypothalamus in opossum. Journal of Comparative & Physiological Psychology, 1969, 67, 182-188.

Roberts, W.W. & Robinson, T.C.L. Relaxation and sleep induced by warming of preoptic region and anterior hypothalamus in cats. Experimental Neurology, 1969, 25, 282-294.

Robinson, D.A. The mechanics of human saccadic eye movement. Journal of Physiology, 1964, 174, 245-264.

Robinson, T.E., Kramis, R.C. & Vanderwolf, C.H. Two types of cerebral activation during sleep: relations to behavior. Brain Research, 1977, 124, 544-549.

Rodieck, R.W. Maintained activity of cat retinal ganglion cells. Journal of Neurophysiology, 1967, 30, 1043-1071.

Roelofs, G.A., Van Den Hoofdakker, R.H. & Prechtl, H.F.R. Sleep effects of subliminal brain stimulation in cats. Experimental Neurology, 1963, 8, 84-92.

Roffwarg, H.P., Muzio, J.N. & Dement, W.C. Ontogenetic development of the human sleep cycle. Science, 1966, 152, 604- 619.

Roger, A., Rossi, G.F. & Zirondoli, A. Le rôle des nerfs crâniens dans le maintien de l'etat vigile de la preparation "encephale isole". Electroencephalography & Clinical Neurophysiology, 1956, 8, 1-13.

Roitbak, A.I. Electrical phenomena in the cerebral cortex during the extinction of orientation and conditioned reflexes. Electroencephalography & Clinical Neurophysiology, 1960, supplement 13, 91-100.

Ross, D.A. & Schwab, R.S. The cortical alpha rhythm in thyroid disorders. Endocrinology, 1939, 25, 75-79.

Roth, G.I., Walton, P.L. & Yamamoto, W.S. Area postrema: Abrupt EEG synchronization following close intra-arterial perfusion with serotonin. Brain Research, 1970, 23, 223-233.

Roth, S.R., Sterman, M.B. & Clemente, C.D. Comparison of EEG correlates of reinforcement, internal inhibition and sleep. Electroencephalography & Clinical Neurophysiology, 1967, 23, 509-520.

Roussel, B., Pujol, J.F. & Jouvet, M. Effets des lesions du tegmentum pontique sur les etats de sommeil chez le rat. Archives italiennes de Biologie, 1976, 114, 188-209.

Roux, W. Einleitung. Arch. Entwicklungsmech. Organ. 1895, 1, 1-42.

Rubin, M.A., Cohen, L.H. & Hoagland, H. The effect of artificially raised metabolic rate on the electroencephalogram of schizophrenic patients. Endocrinology, 1937, 21, 536-540.

Ruch-Monachon, M.A., Jalfre, M. & Haefely, W. Drugs and PGO waves in the lateral geniculate body of the curarized cat. V. Miscellaneous compounds. Synopsis of the role of central neurotransmitters on PGO wave activity. Archives Internationales de Pharmacodynamie et de Therapie, 1976, 219, 326-346.

Ruckebusch, Y. & Gaujoux, M. Sleep patterns of the laboratory cat. Electroencephalography & Clinical Neurophysiology, 1976, 41, 483-490.

Rusak, B. & Zucker, I. Biological rhythms and animal behavior. Annual Review of Psychology, 1975, 26, 137-171.

Saito, H., Sakai, K. & Jouvet, M. Discharge patterns of the nucleus parabrachialis lateralis neurons of the cat during sleep and waking. Brain Research, 1977, 134, 59-72.

Sakai, K. & Cespuglio, R. Evidence for the presence of eye movement potentials during paradoxical sleep in cats. Electroencephalography & Clinical Neurophysiology, 1976, 41, 37-48.

Sakai, K., Petitjean, F, & Jouvet, M. Effects of pontomesencephalic lesions and electrical stimulation upon PGO waves and EMPs in unanaesthetized cats. Electroencephalography & Clinical Neurophysiology, 1976, 41, 49-63.

Sakai, K., Sano, K. & Iwahara, S. Eye movements and hippocampal theta activity in cats. Electroencephalography & Clinical Neurophysiology, 1973, 34, 547-549.

Salinger, W.L., Schwartz, M.A. & Wilkerson, P.R. Selective loss of lateral geniculate cells in the adult cat after chronic monocular paralysis. Brain Research, 1977a, 125, 257-263.

Salinger, W.L., Schwartz, M.A. & Wilkerson, P.R. Selective cell loss in the lateral geniculate nucleus of adult cats following binocular lid suture. Brain Research, 1977b, 130, 81-88.

Salzarulo, P., Pêcheux, M.-G. & Lairy, G.C. A vecto-oculographic approach to fast sleep eye movements in man. Electroencephalography & Clinical Neurophysiology, 1973, 34, 539-542.

Sano, K., Iwahara, S., Senba, K., Sano, A. & Yamazaki, S. Eye movements and hippocampal theta activity in rats. Electroencephalography & Clinical Neurophysiology, 1973, 35, 621-625.

Sawyer, C.H. Some effects of hormones on sleep. Experimental Medicine & Surgery, 1969, 27, 177-186.

Schapiro, H., Britt, L.G. & Dohrn, O.D. Sensory deprivation on visceral activity. IV. The effect of temporary visual deprivation on canine gastric secretion. American Journal of Digestive Diseases, 1973, 18, 573-575.

Schapiro, H., Britt, L.G., Gross, C.W. & Gaines, K.J. Sensory deprivation on visceral activity. III. The effect of olfactory deprivation on canine gastric secretion. Psychosomatic Medicine, 1971, 33, 429-435.

Schapiro, H., Gross, C.W., Nakamura, T., Wruble, L.D. & Britt, L.G. Sensory deprivation on visceral activity. II. The effect of auditory and vestibular deprivation on canine gastric secretion. Psychosomatic Medicine, 1970a, 32, 515-521.

Schapiro, H., Wruble, L.D., Britt, L.G. & Bell, T.A. Sensory deprivation on visceral activity. I. The effect of visual deprivation on canine gastric secretion. Psychsomatic Medicine, 1970b, 32, 379-395.

Scheibel, M., Scheibel, A., Mollica, A. & Moruzzi, G. Convergence and interaction of afferent impulses on single units of reticular formation. Journal of Neurophysiology, 1955, 18, 309-331.

Schmidek, W.R., Hoshino, K., Schmidek, M. & Timo-Iaria, C. Influence of environmental temperature on the sleep-wakefulness cycle in the rat. Physiology & Behavior, 1972, 8, 363-371.

Schultz, D.P. Sensory Restriction: Effects on Behavior. Academic Press, New York, 1965.

Scott, T.H., Bexton, W.H., Heron, W. & Doane, B.K. Cognitive effects of perceptual isolation. Canadian Journal of Psychology, 1959, 13, 200-209.

Seguin, J.J., Magherini, P.C. & Pompeiano, O. Cholinergic mechanisms related to REM sleep. III. Tonic and phasic inhibition of monosynaptic reflexes induced by an anticholinesterase in the decerebrate cat. Archives italiennes de Biologie, 1973, 111, 1-23.

Senden, M. von. Raum-und Gestaltauffassung bei operierten Blindgeborenen vor und nach der Operation. J. Barth (ed.) Leipzig, 1932. English translation. Space and Sight. Free Press, Glencoe, Illinois, 1960.

Shackel, B. Eye movement recording by electro-oculography. pp299-334. In: P.H. Venables & I. Martin (eds.) A Manual of Psychophysiological Methods. North-Holland Publishing Co., Amsterdam, 1967.

Shannon, I.L., Feller, R.P. & Suddick, R.P. Light deprivation and parotid flow in the human. Journal of Dental Research, 1972, 51, 1642-1645.

Shannon, I.L., Feller, R.P. & Wescott, W.B. Environmental lighting and human salivary gland function. Proceedings of the Society for Experimental Biology & Medicine, 1975, 148, 758-761.

Shapiro, C.M., Moore, A.T., Mitchell, D. & Yodaiken, M.L. How well does man thermoregulate during sleep? Experientia, 1974, 30, 1279-1280.

Sharpless, S.K. Hypnotics and sedatives. I. The barbiturates. In: L.S. Goodman & A. Gilman (eds.) The Pharmacological Basis of Therapeutics. Collier-MacMillan Canada Ltd., Toronto, 1970, pp98-120.

Sharpless, S.K. & Halpern, L.M. The electrical excitability of chronically isolated cortex studied by means of permanently implanted electrodes. Electroencephalography & Clinical Neurophysiology, 1962, 14, 244-255.

Sharpless, S. & Jasper, H. Habituation of the arousal reaction. Brain, 1956, 79, 655-680.

Sherk, H. & Stryker, M.P. Quantitative study of cortical orientation selectively in visually inexperienced kitten. Journal of Neurophysiology, 1976, 39, 63-70.

Sherman, S.M. Development of interocular alignment in cats. Brain Research, 1972, 37, 187-203.

Sherman, S.M. Visual field defects in monocular and binocularly deprived cats. Brain Research, 1973, 49, 25-45.

Sherman, S.M. Permanence of visual perimetry deficits in monocularly and binocularly deprived cats. Brain Research, 1974, 73, 491-501.

Sherman, S.M. & Wilson, J.R. Behavioral and morphological evidence for binocular competition in the postnatal development of the dog visual system. Journal of Comparative Neurology, 1975, 161, 183-195.

Sheu, Y.-S., Nelson, J.P. & Bloom, F.E. Discharge patterns of cat raphe neurons during sleep and waking. Brain Research, 1974, 73, 263-276.

Sidis, B. An experimental study of sleep. Journal of Abnormal Psychology, 1908, 3, 1-32.

Siegel, J.M. REM sleep predicts subsequent food intake. Physiology & Behavior, 1975, 15, 399-403.

Siegel, J.M., McGinty, D.J. & Breedlove, S.M. Sleep and waking activity of pontine gigantocellular field neurons. Experimental Neurology, 1977, 56, 553-573.

Simon, R.P., Gershon, M.D. & Brooks, D.C. The role of the raphe nuclei in the regulation of ponto-geniculo-occipital wave activity. Brain Research, 1973, 58, 313-330.

Singer, W. & Tretter, F. Unusually large receptive fields in cats with restricted visual experience. Experimental Brain Research, 1976, 26, 171-184.

Slomin, V. Jr. & Pasnak, R. The effect of visual deprivation on the depth perception of adult and infant rats and adult squirrel monkeys (Saimiri Sciurea). Vision Research, 1972, 12, 623-626.

Slosarska, M. & Zernicki, B. Wakefulness and sleep in the isolated cerebrum of the pretrigeminal cat. Archives italiennes de Biologie, 1971, 109, 287-304.

Snyder, F., Hobson, J.A., Morrison, D.F. & Goldfrank, F. Changes in respiration, heart rate and systolic blood pressure in human sleep. Journal of Applied Physiology, 1964, 19, 417-422.

Spinelli, D.N., Hirsch, H.V.B., Phelps, R.W. & Metzler, J. Visual experience as a determinant of the response characteristics of cortical receptive fields in cats. Experimental Brain Research, 1972, 15, 289-304.

Spiro, R.H. & Kolbert, G.S. A new technique for functional visual deprivation. Electroencephalography & Clinical Neurophysiology, 1974, 37, 654-656.

Stepanik, J. Das bestand potential des auges und die experimentelle steigerung des intraocularen druckes bein menschen. Graefes Archives of Ophthalmology, 1958, 160, 226-235.

Sterman, M.B. & Clemente, C.D. Forebrain inhibitory mechanisms: Cortical synchronization induced by basal forebrain stimulation. Experimental Neurology, 1962a, 6, 91-102.

Sterman, M.B. & Clemente, C.D. Forebrain inhibitory mechanisms: Sleep patterns induced by basal forebrain stimulation in the behaving cat. Experimental Neurology, 1962b, 6, 103-117.

Sterman, M.B. & Clemente, C.D. Forebrain mechanisms for the onset of sleep. pp83-97. In: O. Petre-Quadens & J.D. Schlag (eds.) Basic Sleep Mechanisms. Academic Press, New York, 1974.

Sterman, M.B., Knauss, T., Lehman, D. & Clemente, C.D. Circadian sleep and waking patterns in the laboratory cat. Electroencephalography & Clinical Neurophysiology, 1965, 19, 509-517.

Sterman, M.B. & Wyrwicka, W. EEG correlates of sleep: Evidence for separate forebrain substrates. Brain Research, 1967, 6, 143-163.

Stern, W.C. & Morgane, P.J. Theoretical view of REM sleep function: Maintenance of catecholamine systems in the central nervous system. Behavioral Biology, 1974, 11, 1-32.

Stryker, M. & Blakemore, C. Saccadic and disjunctive eye movements in cats. Vision Research, 1972, 12, 2005-2013.

Suedfeld, P. Changes in intellectual performance and in susceptibility to influence. pp126-166. In: J.P. Zubek (ed.) Sensory Deprivation: Fifteen Years of Research. Appleton-Century-Crofts, New York, 1969.

Suedfeld, P. The benefits of boredom: Sensory deprivation reconsidered. American Scientist, 1975, 63, 60-69.

Sulzman, F.M., Fuller, C.A. & Moore-Ede, M.C. Feeding time synchronizes primate circadian rhythms. Physiology & Behavior, 1977, 18, 775-779.

Szentagothai, J. The elementary vestibulo-ocular reflex arc. Journal of Neurophysiology, 1950, 13, 395-407.

Tagney, J. Sleep patterns related to rearing rats in enriched and impoverished environments. Brain Research, 1973, 53, 353-361.

Tait, G. The Effect of Perceptual Deprivation on the Human EEG. Unpublished PhD thesis. McMaster University, 1977.

Thoden, U., Magherini, P.C. & Pompeiano, O. Cholinergic mechanisms related to REM sleep. II. Effects of an anticholinesterase on the discharge of central vestibular neurons in the decerebrate cat. Archives italiennes de Biologie, 1972; 110, 260-283.

Thompson, W.R. & Schaefer, T.Jr. Early environmental stimulation. pp81-105. In: D.W. Fiske & S.R. Maddi (eds.) Function of Varied Experience. The Dorsey Press, Inc., Homewood, 1961.

Tizard, B. Repetitive auditory stimuli and the development of sleep. Electroencephalography & Clinical Neurophysiology, 1966, 20, 112-121.

Turkel, J. Perceptual Environment and Development of Cat Visual System. Unpublished PhD thesis. McMaster University, 1974.

Ursin, R. Two stages of slow wave sleep in the cat and their relation to REM sleep. Brain Research, 1968, 11, 347-356.

Vanderwolf, C.H. Limbic-diencephalic mechanisms of voluntary movement. Psychological Review, 1971, 78, 83-113.

Van Hof-Van Duin, J. Development of visuomotor behavior in normal and dark reared cats. Brain Research, 1976a, 104, 233-241.

Van Hof-Van Duin, J. Early and permanent effects of monocular deprivation on pattern discrimination and visuomotor behavior in cats. Brain Research, 1976b, 111, 261-276.

Vernikos-Danellis, J., Leach, C.S., Winget, C.M., Rambaut, P. C. & Mack, P.B. Thyroid and adrenal cortical rhythmicity during bed rest. Journal of Applied Physiology, 1972, 33, 644-648.

Vernon, J.A., McGill, T.E., Gulick, W.L. & Candland, D.K. The effect of human isolation upon some perceptual and motor skills. pp41-57. In: Solomon et. al. (eds.) Sensory Deprivation. Harvard University Press, Cambridge, 1961.

Vertes, R.P. Selective firing of rat pontine gigantocellular neurons during movement and REM sleep. Brain Research, 1977, 128, 146-152.

Villablanca, J. The electrocorticogram in the chronic "cerveau isolé" cat. Electroencephalography & Clinical Neurophysiology, 1965, 19, 576-586.

Villablanca, J. Behavioral and polygraphic study of "sleep" and "wakefulness" in chronic decerebrate cats. Electroencephalography & Clinical Neurophysiology, 1966a, 21, 562-577.

Villablanca, J. Ocular behavior in the chronic "cerveau isole" cat. Brain Research, 1966b, 2, 99-102.

Villablanca, J. Electrocorticogram in the chronic "isolated hemisphere" of the cat. Effect of atropine and eserine. Brain Research, 1967, 3, 287-291.

Villablanca, J. Role of the thalamus in sleep control: Sleep-wakefulness studies in the chronic diencephalic and athalamic cats. pp51-81. In: O. Petre-Quadens & J.D. Schlag (eds.) Basic Sleep Mechanisms. Academic Press, New York, 1974.

Villablanca, J. & Marcus, R. Sleep-wakefulness, EEG and behavioral studies of chronic cats without the neocortex and striatum: The diencephalic cat. Archives italiennes de Biologie, 1972, 110, 348-382.

Villablanca, J.R., Marcus, R.J. & Olmstead, C.E. Effects of caudate nuclei or frontal cortex ablations in cats. II. Sleep-wakefulness, EEG and motor activity. Experimental Neurology, 1976, 53, 31-50.

Villablanca, J. & Salinas-Zeballos, M.E. Sleep-wakefulness, EEG and behavioral studies of chronic cats without the thalamus: The athalamic cat. Archives italiennes de Biologie, 1972, 110, 383-411.

Villablanca, J. & Schlag, J. Cortical control of thalamic spindle waves. Experimental Neurology, 1968, 20, 432-442.

Vital-Durand, F. & Jeannerod, M. Eye movement related activity in the visual cortex of dark-reared kittens. Electroencephalography & Clinical Neurophysiology, 1975, 38, 295-301.

Vital-Durand, F. & Michel, F. Effets de la desafferentation peripherique sur le cycle veille-sommeil chez le chat. Archives italiennes de Biologie, 1971, 109, 166-186.

Vital-Durand, F., Putkonen, P.T.S. & Jeannerod, M. Motion detection and optokinetic responses in dark-reared kittens. Vision Research, 1974, 14, 141-142.

Von Bernuth, H. & Prechtl, H.F.R. Vestibulo-ocular response and its state dependency in newborn infants. Neuropadiatrie, 1969, 1, 11-24.

von Noorden, G.K. Experimental amblyopia in monkeys. Further behavioral observations and clinical correlations. Investigative Ophthalmology, 1973, 12, 721-726.

von Noorden, G.K., Dowling, J.E. & Ferguson, D.C. Experimental amblyopia in monkeys. I. Behavioral studies of stimulus deprivation amblyopia. Archives of Ophthalmology, 1970, 84, 206-214.

Webb, W.B. & Agnew, H.W., Jr. Sleep and waking in a time-free environment. Aerospace Medicine, 1974, 45, 617-622.

Westheimer, C. Mechanism of saccadic eye movements. Archives of Ophthalmology, 1954, 52, 710-724.

Wiesel, T.N. & Hubel, D.H. Effects of visual deprivation on morphology and physiology of cells in the cat's lateral geniculate body. Journal of Neurophysiology, 1963a, 26, 978-993.

Wiesel, T.N. & Hubel, D.H. Single-cell responses in striate cortex of kittens deprived of vision in one eye. Journal of Neurophysiology, 1963b, 26, 1003-1017.

Wiesel, T.N. & Hubel D.H. Comparison of the effects of unilateral and bilateral eye closure on cortical unit responses in kittens. Journal of Neurophysiology, 1965a, 28, 1029-1040.

Wiesel, T.N. & Hubel, D.H. Extent of recovery from the effects of visual deprivation in kittens. Journal of Neurophysiology, 1965b, 28, 1060-1072.

Winer, B.J. Statistical Principles in Experimental Design. McGraw Hill Book Company, Toronto, 1962.

Winget, C.M., Vernikos-Danellis, J., Cronin, S.E., Leach, C. S., Rambaut, P.C. & Mack, P.B. Circadian rhythm asynchrony in man during hypokinesis. Journal of Applied Physiology, 1972, 33, 640-643.

Wit, A. & Wang, S.C. Temperature-sensitive neurons in preoptic/anterior hypothalamic region: effects of increasing ambient temperature. American Journal of Physiology, 1968, 215, 1151-1159.

Yinon, U. Age dependence of the effect of squint on cells in kitten's visual cortex. Experimental Brain Research, 1976, 26, 151-157.

Yinon, U., Auerbach, E., Blank, M. & Friesenhausen, J. The ocular dominance of cortical neurons in cats developed with divergent and convergent squint. Vision Research, 1975, 15, 1251-1256.

Zanchetti, A., Wang, S.C. & Moruzzi, G. The effect of vagal afferent stimulation on the EEG pattern of the cat. Electroencephalography & Clinical Neurophysiology, 1952, 4, 357-361.

Zarcone, V., Gulevich, G., Pivik, T., Azumi, K & Dement, W. REM deprivation and schizophrenia. Biological Psychiatry, 1969, 1, 179-184.

Zernicki, B. Pretrigeminal cat. Brain Research, 1968, 9, 1-14.

Zimmerman, J., Reite, M. & Stoyva, J. Inverted vision and REM sleep: Experiments with adult cats. Sleep Research, 1974, 3, 112.

Zimmerman, J., Stoyva, J. & Metcalf, D. Distorted visual feedback and augmented REM sleep. Psychophysiology, 1970, 7, 298.

Zubek, J.P. Counteracting effects of physical exercise performed during prolonged perceptual deprivation. Science, 1963, 142, 504-506.

Zubek, J.P. Sensory and perceptual-motor effects. pp 207-253. In: J.P. Zubek (ed.) Sensory Deprivation: Fifteen Years of Research. Appleton-Century-Crofts, New York, 1969a.

Zubek, J.P. Physiological and biochemical effects. pp254-288. In: J.P. Zubek (ed.) Sensory Deprivation: Fifteen Years of Research. Appleton-Century-Crofts, New York, 1969b.

Zubek, J.P., Aftanas, M., Hasek, J., Samson, W., Schludermann, E., Wilgosh, L. & Winocur, G. Intellectual and perceptual changes during prolonged perceptual deprivation: Low illumination and noise level. Perceptual and Motor Skills, 1962, 15, 171-198.

Zubek, J.P & MacNeill, M. Effects of immobilization: Behavioral and EEG changes. Canadian Journal of Psychology, 1966, 20, 326-336.

Zubek, J.P. & Schutte, W. Urinary excretion of adrenaline and noradrenaline during prolonged perceptual deprivation. Journal of Abnormal Psychology, 1966, 71, 328-334.

Zubek, J.P. & Welch, G. Electroencephalographic changes after prolonged sensory and perceptual deprivation. Science, 1963, 139, 1209-1210.

Zuckerman, M. Perceptual isolation as a stress situation. Archives of General Psychiatry, 1964, 11, 255-276.

Zuckerman, M. Variables affecting deprivation results. pp47-84. In: J.P. Zubek (ed.) Sensory Deprivation: Fifteen Years of Research. Appleton-Century-Crofts, New York, 1969.

Table XXXVI - Test of the difference between the mean consumption of food and milk during pre-deprivation and deprivation of the lid suture and light deprivation experiments (matched pair t test for correlated means, Guilford, 1965, p184).

Table XXXVI - Test of the difference between the mean consumption of food and milk during pre-deprivation and deprivation of the lid suture and light deprivation experiments (matched pair t test for correlated means, Guilford, 1965, p184).