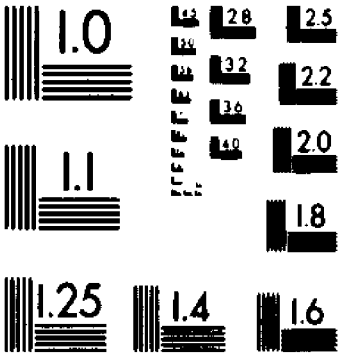

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THE EFFECTS OF SELECTIVE SLEEP-STAGE RESTRICTION ON DAYTIME SLEEPINESS IN YOUNG ADULTS

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THE EFFECTS OF SELECTIVE SLEEP-STAGE RESTRICTION ON DAYTIME SLEEPINESS IN YOUNG ADULTS

by

Paul Benjamin Glovinsky

A dissertation submitted to the Graduate Faculty
in Psychology in partial fulfillment of the requirements
for the degree of Doctor of Philosophy,
The City University of New York

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date

Steven J. Ellman
Chairman of Examining Committee

January 17, 1986
date

Executive Officer

Supervisory Committee

The City University of New York

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This doctoral thesis is dedicated to the memory of Arthur Arkin, M.D., my advisor when I began, and of my friend Andrew Mishkind, who remained interested in my work even as he realized that he would have to leave off his own.

TABLE OF CONTENTS

List of Tables

CHAPTER 1: REVIEW OF THE LITERATURE

A. THE DIFFERENTIATION OF SLEEP

The systematic study of sleep stages and their associations with aspects of waking behavior began in the mid 1930's, in the years following the development of the electroencephalograph. Workers such as H. Davis, A.L. Loomis, H. Blake and their colleagues provided the basis for much subsequent work on sleep.

Foremost in terms of influence was the differentiation of sleep into five states characterized by distinctive electric potential patterns (Loomis, 1936, Loomis, 1937). These five states, labeled A through E, roughly correspond to the present-day designation of relaxed wakefulness and Non-REM sleep stages 1-4. As soon as these states were distinguished, an effort was begun to correlate them to subjective and behavioral aspects of consciousness. It was clear to this group, for example, that sleep onset "begins somewhere in the latter part of the A state" (1937, p.136) although a series of experiments convinced them of the difficulties inherent in delineating a precise switchover from wakefulness to sleep, a situation which persists today.

A second line of inquiry involved establishing arousal thresholds for the various states. It was found that subjects in states D and E, corresponding to the current

designation of Stages 3 and 4, were the most difficult to arouse.

Even the question of whether dreams were associated with a particular sleep state, a vigorously contested topic thirty years later, was addressed in this initial group of studies. It was first thought that dreams were associated with "a peculiar wave of large amplitude lasting from two to four minutes at a time when no movement or change in heart beat or respiration occurred" but subsequent experiments suggested that "dreams are not likely to be associated with any unusual patterns of electrical potentials but with a state of sleep" (p.142). This state was observed to be the B state, which nearly two decades later would be further differentiated into NREM Stage 1 and REM sleep.

Before turning to this second major differentiation of sleep, it should be mentioned that Loomis and his coworkers presented graphs explicitly depicting the cyclical occurrence of the different sleep states (p.135). This groups interest in associating sleep-state changes with gross body movements perhaps caused them to overlook this feature of their data, although they did note that oftentimes sleep-state changes transpired without such movements. In any case, a prevailing hypothesis of the time, that of a "continuous curve of sleep", was amply refuted by these graphic presentations.

In the mid 1950's, Aserinsky and Kleitman described the occurrence of discrete periods of sleep characterized by rapid conjugate eye movements and a high incidence of dream recall (1955). This finding was amplified by Dement and Kleitman (1957), who reported that periods of rapid, conjugate eye movements appeared during every one of 126 undisturbed nights' sleep recorded from 33 subjects. Furthermore, subjects' sleep states showed "a regular cyclic variation with a period of about 90-100 minutes" (p.684).

Of special interest was a group of experiments drawing a clear distinction between the initial appearance of Stage 1 sleep (Loomis' B stage) and its subsequent appearances. Sleep-onset stage 1 was never accompanied by rapid eye movements, while later appearances were. The auditory thresholds necessary to elicit a response from subjects in the two states differed markedly, with more intense auditory stimuli necessary for arousal from later Stage 1 sleep (p.683). Finally, a distinction was drawn between the mental content associated with sleep-onset Stage 1 and later Stage 1 sleep. Wakened at sleep onset, subjects reported "hypnogogic" images and sensations which though "dreamlike" were "distinctly different from an actual dream". Subsequent Stage 1 awakenings tended to produce unequivocal dream reports.

A separate study reported the relative confinement of

organized, vivid dreaming to periods of Stage 1 sleep with rapid eye movements (Dement and Kleitman, 1957b). This finding was contrary to the consensus of the time, which held that dreams occurred throughout sleep. It was followed by studies reporting that generally, dream mentation was not present following NREM awakenings (Wolpert and Trosman, 1958).The segregation of dreaming into REM sleep marked the extreme position in terms of the differentiation of sleep. A two state model of sleep, with each state characterized by confined processes, was proposed (Oswald, 1962). While researchers would eventually be compelled to retreat from this position, it did serve to generate much experimentation regarding the effects of "dream deprivation" and the functions of the various stages of sleep. These experiments will be reviewed in a subsequent section, with an emphasis on how they address the question of the relationship between the sleep stages and daytime sleepiness.

B. THE MEASUREMENT OF SLEEPINESS

Sleepiness refers to a transitional state between alertness and sleep. It is a "dynamic" state (Hobson, 1980) gradually changing in intensity and character. Incipient sleepiness may be just discernable amidst wakefulness; eventually the difficulty is one of differentiating sleepi-

ness from light sleep.

Sleepiness may be assessed through subjective, behavioral and physiological measures. Subjectively, we have all learned to identify the mental state which attends prolonged periods of wakefulness, although the particular aspects dwelt upon differs between individuals. Some common subjective indicators are a heaviness in the eyelids, lack of energy and motivation, inability to concentrate and a conscious desire for sleep (Hartmann, 1973; Dement, 1976; Pressman, 1982).

The ability to subjectively distinguish sleepiness provides the basis for the Stanford Sleepiness Scale (SSS), a seven point forced choice scale which has been demonstrated to distinguish between sleep deprived and non sleep deprived normals (Hoddes et al, 1972, 1973; Pressman, 1982).

Aspects of sleepiness are also open to behavioral observation, for example the eyelids of a sleepy person can be observed to droop, and the eyes themselves may take on a glazed appearance. Other aspects may be observed indirectly, as impaired concentration is inferred from lapses in performance.

Physiological measures of sleepiness include those based on EEG waveforms. Classification of EEG patterns associated with levels of sleepiness have ranged from fine discriminations based on varying mixes of alpha, theta and other

activity--discriminations so fine that they are difficult to agree upon (Itil, 1969)--to the standard scoring system, which in the interest of promoting inter-rater reliability jumps directly from wakefulness to sleep (Rechtshaffen and Kales, 1968). Computer analysis of waveforms has also been used to ascertain the ratio of theta to alpha activity (Walter, 1967; Matousek, 1967; Gevins, 1977). This method overcomes the difficulty inherent in manually classifying analog data, and is especially suited to monitoring sleepiness fluctuations over prolonged periods of wakefulness (Akerstadt et al, 1982).

The EEG may be used to assess sleepiness in other ways: A particular EEG criterion of sleep may be selected, and the latency before reaching this criterion under conditions conducive to sleep may be taken as a measure of sleepiness. This is the rationale behind the Multiple Sleep Latency Test (Richardson et al, 1978). The MSLT is a series of five daytime naps spaced every two hours throughout the day, usually beginning at 10:00 am. The average latency between lights out and polygraphic indications of any stage of sleep over the five naps constitutes the sleepiness score. This measure, which possesses face validity, is seen as reflecting a physiological sleep tendency, and less subject to the influence of moment-to-moment mood variations. It has been shown to be sensitive to differences between sleep-deprived and non-sleep deprived normals (Carskadon and Dement, 1979b; Pressman, 1982) and between

normals and patients with excessive daytime sleepiness (Richardson et al, 1978; Mitler, 1982; Reynolds et al, 1982; Pressman, 1982).Its reliability has been established in normal subjects retested over seven consecutive days after eight hour nocturnal bedtimes (Carskadon and Dement, 1982c).

The waking EEG may be examined for very brief periods of activity which would meet criteria for the earliest stage of sleep, had they lasted for ten or fifteen seconds (half of conventional scoring epochs). It has been found that these very brief sleep episodes, termed "microsleeps", are commonly seen during the subjective wakefulness of excessively sleepy patients (Guilleminault, 1975).

Yet another EEG-based measure which appears to be sensitive to changes in sleepiness is the auditory evoked response (AER). In a study comparing AERs recorded both prior to and after daytime naps in normals, sleep-deprived normals and narcoleptics, sleep deprived normals exhibited larger amplitudes on long-latency components N1P2 and P2N2 than non sleep-deprived normals (Pressman, 1982). Surprisingly, these components did not differ between non sleep-deprived normals and narcoleptics, even though these two groups were clearly distinguishable on other measures of sleepiness, including the MSLT. Post-hoc analysis revealed that this finding stemmed from the heterogenous nature of the narcoleptic AER sample: Waveforms recorded from narcoleptics

prior to naps containing only NREM sleep could indeed be distinguished from those of non sleep-deprived normals, but those preceding naps containing some REM sleep were not significantly different. An analogous situation had been known to exist with respect to AERs recorded during sleep--that is, responses recorded during REM sleep are indistinguishable from those recorded during wakefulness, while those taken from NREM sleep are of larger amplitude (Williams, 1962; Weitzman and Kremen, 1965). That such a REM/NREM dichotomy holds for sleepiness as well lends support to the idea that sleepiness is not a unitary phenomenon (Broughton, 1982). It may be unreasonable therefore to expect full concordance between sleepiness measures.

A number of sleepiness measures which are not EEG-based have been proposed. The body temperature rhythm has been shown to predict performance on a variety of tasks (Kleitman, 1963; Blake, 1967). More recently, subjective sleepiness ratings as well as sleep length and organization were also shown to correlate with the body temperature rhythm (Czeisler et al, 1980). These findings will be discussed within the context of circadian influences on sleepiness.

Pupillography provides an electrographic measure which is thought to reflect levels of central nervous system activation. In general, full alertness is associated with a

wide, stable pupil diameter, whereas decreased alertness is indicated by constricted, oscillating pupil size (Lowenstein and Lowenfield, 1963; Yoss et al, 1970). Pupillographic measurements taken throughout the day have shown fluctuations suggesting sensitivity to underlying ultradian arousal rhythms (Pressman et al, 1984; Lavie and Schulz, 1980).

C. DETERMINANTS OF SLEEPINESS

Sleepiness is influenced both by circadian and non-circadian factors. The latter, to be reviewed below, include sleep length and sleep quality. These non-circadian factors are often investigated via relatively straightforward experiments involving sleep restriction or selective sleep-stage deprivation. By contrast, circadian factors often require drastically altered sleep-wake schedules in order to be elucidated. Such investigations have demonstrated conclusively that sleepiness is not simply a function of time spent awake. Whether measured objectively by the MSLT, subjectively through scales or indirectly through performance measures, sleepiness sometimes decreases despite the passage of waking hours, and it can in fact increase after hours of sleep.

1. Circadian Determinants

It has been known for some time that human performance

fluctuates with a circadian periodicity (Freeman and Hoveland, 1934; Loveland and Williams, 1963; Blake, 1967; Colquhoun, 1981). From the earliest studies, it was apparent that different types of tasks followed different circadian patterns. For example, some tasks were performed better as the day wore on while performance on others worsened. In general, tasks involving a relatively large motor component or vigilance improve throughout the waking day (Blake, 1967; Hughs and Folkard, 1976) short-term memory reaches a mid-morning peak and declines through the evening, (Blake, 1967, Folkard and Monk, 1978) and long-term memory performance peaks in the afternoon (Folkard et al, 1977).

Perhaps because motor response provides the final common pathway for a variety of performance tests, the first pattern cited above, i.e. improvement throughout the day to an evening peak, is frequently observed. Kleitman, (1963) noting the parallel between this pattern and the circadian temperature cycle, hypothesized that performance depended simply on the overall level of arousal as indicated by the temperature rhythm.

While many important exceptions to this rule have been demonstrated, a cluster of abilities does appear tied to the temperature cycle. Even in the event of a dissociation between the sleep/wake cycle and temperature rhythm, as sometimes occurs under conditions of isolation from time

cues, these aspects of performance continue to follow the temperature cycle (Colquhoun, 1968).

A circadian rhythm in sleep tendency was demonstrated by Weitzman and colleagues, who ran subjects on a three hour "day" consisting of sixty minutes of bedtime followed by two hours of enforced wakefulness for 240 hours (1974). Despite this radically altered schedule, sleep continued to occur mainly in the late night and early morning bedtime hours. This temporal restriction of sleep was all the more remarkable in that subjects experienced a 35% sleep loss, and thus might have been expected to recover sleep whenever they could.

The circadian sleep-tendency rhythm was observed to be phase related to a body temperature rhythm which also persisted throughout the experiment, albeit with lowered amplitude and a phase delay relative to baseline conditions. The relationship between the two rhythms was such that sleep efficiency during the one hour nap opportunities was lowest between the hours of 1900 to 2200, when the temperature cycle was peaking.

The distribution of REM sleep was even more narrowly restricted than that of sleep in general, indicating the presence of a circadian rhythm in REM sleep propensity. 75% of all REM sleep occurred in the three hours beginning at 0600, 0900 and 1200. This morning peak in the REM propensity rhythm accorded with reports by Webb and colleagues

(1966, 1967) that REM dominated morning naps and was relatively absent from afternoon naps.

A circadian rhythmicity in sleep efficiency and REM sleep was also demonstrated during a series of naps spaced approximately every three and a half hours over a forty hour period (Moses, 1975). In accord with the findings of Weitzman et al. (1974) a distinct phase relationship between these parameters and the oral temperature cycle was observed, with greatest sleep efficiency and REM propensity at the trough of the temperature cycle.

Further evidence of the persistance of pre-experimental circadian patterns was provided by the finding that the nap opportunity corresponding to habitual bedtimes was essentially similar in terms of sleep staging to the first hour of baseline sleep.

The same peaks in sleep efficiency and REM propensity which had been observed in 3 and 3.5 hour "days" were also apparent in a study of five subjects undergoing 86 consecutive ninety minute "days", each consisting of thirty minutes of sleep opportunity followed by sixty minutes of enforced wakefulness (Carskadon and Dement, 1975). Under these conditions REM sleep appeared within ten minutes of sleep onset in all five subjects on 79 of the 110 occasions when it appeared at all. Most REM sleep was recorded between the hours of 0730 to 1400. No REM sleep was ever recorded on the midnight sleep period. It was found that REM sleep

tended to occur on alternating or at least intermittent naps; only 6 of the 110 naps containing REM sleep were consecutive, whereas 58 had one NREM sleep period intervening. The investigators suggested that these findings may be reflecting a refractory period for REM sleep of between 60 and 180 minutes in normal subjects, which would superimpose an ultradian pattern over the underlying circadian rhythm.

Moses et al. (1975) had found a significant correlation between the amount of REM sleep found in one nap and the amount of Stage 4 sleep found in the following nap, and vice versa. However, REM sleep was not observed to appear on alternate naps. Rather, its appearance on a particular nap was positively correlated with appearances on the immediately preceding or succeeding naps--indicative of a smooth circadian function. The two different schedules may have accounted for this discrepancy. The 90 minute "day" allowed for only thirty minutes of sleep, which had the effect of precluding the appearance of REM and slow wave sleep together--on fewer than 11 of the 225 naps containing SWS was REM present as well. One hour bedtimes as allotted under the 3.5 hour "day" allowed time for more transitions between REM and NREM sleep stages, so that manifestations of the circadian propensity of REM sleep were less impeded.

The finding that REM and SWS tended to be mutually exclusive under the ninety minute schedule prompted the use of this regimen as a "preparation" with which to focus on the

differential effects of REM and SWS. In particular, the relationship between these stages of sleep and proximate waking sleepiness was investigated (Carskadon and Dement, 1977).

Five additional subjects were run. The results in terms of sleep stage appearances within a ninety minute schedule confirmed the earlier findings. Data was therefore pooled over all ten subjects. Results of performance tests were not presented; it was noted that a "prolonged practice effect" was the most striking feature of the data.

The Stanford Sleepiness Scale (SSS) was administered both fifteen minutes before each nap opportunity and fifteen minutes afterwards. The sleepiest SSS ratings occurred in the late morning, and the most alert during the evening hours. This circadian cycle was 180 degrees out of phase with the normal temperature cycle, i.e. sleepiness was greatest over those hours corresponding to the temperature trough.

Pre-sleep SSS ratings were significantly correlated ($r=.35$) with the amount of REM sleep appearing in a particular nap, so that sleepier ratings tended to appear before naps containing greater amounts of REM sleep. In contrast, slow wave sleep was correlated ($r=.28$) with post-sleep SSS scores. In terms of change between pre and post nap sleepiness scores, subjects tended to become sleepier following naps containing SWS, whereas they became more alert

following naps containing REM sleep.

The authors suggested that "subjective reports of sleepiness may reflect a prodromal state for REM sleep in much the same manner that SWS or NREM sleep normally precedes REM sleep in nocturnal sleep periods." (p.132) They stopped short of attributing changes in sleepiness to the appearance of a particular sleep state, noting that both sleepiness changes and REM/SWS alternation may result from a single ultradian fluctuation. Nonetheless, the notion of sleepiness being selectively reversed by REM sleep was seen as "intriguing" and in line with earlier studies of reduced nocturnal sleep, which had showed that REM sleep is selectively deprived by this manipulation.

In studies reviewed thus far, circadian rhythmicity in sleepiness was measured in terms of sleep efficiency during naps spaced at regular intervals throughout both day and night. These schedules typically caused substantial sleep deprivation besides the circadian disruption they entailed. With the development of the MSLT as a measure of sleep tendency, sleepiness could be construed in terms of latency rather than efficiency. This enabled measurement of sleepiness without allowing significant amounts of sleep to accumulate. Richardson and colleagues (1982) made use of this ability in designing a study assessing the circadian rhythmicity of sleepiness under a relatively natural sleep/wake schedule. Ten subjects underwent a modified

MSLT over three days, along with standard polysomnographic testing on the first two nights. On the third night, subjects went to sleep at their usual bedtimes, but were only allowed to sleep until 0115.
After fifteen minutes of enforced wakefulness, they were allowed to resume sleep, and the latency to sleep onset was recorded. This procedure was repeated at two hour intervals throughout the night, yielding a measure of sleep tendency during normal sleeping hours with only a moderate amount of sleep loss.

The circadian sleep latency rhythm was demonstrated to be distinctly bimodal, with maximal latencies (alertness) near 0930 and 1930, and minimal latencies near 1530 and 0530.

Subjective sleepiness measures yielded a monophasic rhythm, with no clear midday trough. The oral temperature rhythm did contain a midday trough, but it was much less pronounced than had been observed in the MSLT rhythm. The authors noted that earlier investigations of the circadian temperature rhythm failed to consistently show even a modest midday trough, and surmised that the one they had observed may have resulted from the regular daytime nap schedule or as a consequence of increased sleep tendency, rather than being an independent fluctuation. In fact investigations which have employed sophisticated monitors of core temperature rather than relying on oral readings have not demonstrated the existance of a midday temperature trough

(Weitzman et al, 1981).

It should be remembered that sleep efficiency had followed a monophasic cycle, with greatest values in the late night and early morning hours, at the trough of the temperature cycle. The discrepency between the biphasic sleep tendency cycle and the monophasic sleep efficiency cycle may indicate that low temperature values are more crucial to sleep maintenance than sleep initiation. Thus subjects can fall asleep readily in the mid-afternoon but generally not stay asleep for long.

An explicit relationship between sleep maintenance and body temperature was demonstrated in subjects who selected their own sleep/wake schedules without awareness of clock time, light/dark cycles or other zeitgebers (Czeisler et al, 1980). The investigation focussed on those subjects who had become "internally desynchronized"--those whose sleep/wake cycles had assumed a periodicity varying up to more than twice as long as that of their temperature rhythms.

Under these conditions, it was found that sleep length averaged close to 14.5 hours when bedtimes coincided with the temperature maxima, whereas the average length was 7.8 hours if sleep began at the trough of the cycle.

Subjective sleepiness measurements taken during self-selected waking periods showed a clear relationship to the

temperature cycle. Subjects rated themselves sleepier during periods corresponding to the trough of the temperature rhythm, and not surprisingly, they tended to begin more of their sleep periods then. These sleep periods begun at the height of sleepiness tended to be shorter than those begun near the peak of the temperature cycle, since in the latter case, sleep persisted until the next upslope of the temperature cycle.

Sleep periods begun near the trough of the temperature cycle were also characterized by more rapid accumulation of REM sleep. It took just over three hours to accumulate fifty minutes of REM sleep under these conditions, whereas it took over five hours to accumulate the same amount in periods starting near the temperature maxima.

If sleep periods were begun just after the trough of the temperature cycle, they were likely to exhibit REM sleep within ten minutes. It is relevant to the present discussion to note that this circadian phase of peak SOREMP probability was precisely the phase of maximum subjective sleepiness.

No such explicit relationship was observed between subjective sleepiness and the timing of slow wave sleep. The occurrence of slow wave sleep was in fact not synchronized to the body temperature rhythm at all, but appeared instead to be sleep-dependent, that is, linked to the sleep-wake cycle.

2. Non-Circadian Determinants

a. Sleep Fragmentation

Relatively few studies have examined the systematic, frequent disruption of sleep on sleep architecture, performance, mood or sleepiness. Originally, investigators were concerned with the effects that environmental stimuli had on the quality of sleep (Roth et al, 1972) but with the emergence of interest in clinical sleep disorders, attention has focused on the effects of endogenously produced disruption. Roth et al (1980) reported that the number of changes to Stage 1 sleep over the course of a night, an index of sleep disruption, is associated with daytime sleep tendency. Subsequent studies have modelled the degree of fragmentation seen in disorders such as nocturnal myoclonus and sleep apnea. One preliminary study reported the effects on MSLT latency of sleep fragmentation produced by delivering one second tones, ten seconds apart, in increasing intensities until electroencephalographic signs of arousal were seen (Stepanski et al, 1984). The trials were spaced an average of 5.5 minutes apart during sleep periods, resulting in approximately 8.5 awakenings per hour. Whereas total sleep time and sleep-stage percentages did not differ significantly between the baseline and second (of two) experimental nights, a significant reduction in MSLT latency, from an average of 13.8 to 8.2 minutes, was

observed.

The only comprehensive study of sleep fragmentation reported to date involved the awakening of eleven young adult subjects after every minute of sleep via the presentation of 1000 hz tones of increasing intensity (Bonnet, 1985). The Clyde Mood Scale, Stanford Sleepiness Scale and several performance tests were administered at least once after one baseline night, two experimental nights, and two recovery nights.

This disruption procedure, in contrast to that of Stepanski and colleagues, altered sleep architecture profoundly. It increased wakefulness and Stage 1 sleep, reduced Stage 2 and all but eliminated both SWS and REM sleep. Total sleep time was also significantly reduced by about one hour, despite efforts to counteract this effect of disruption by extending bedtimes thirty minutes.

Recovery sleep showed a rebound of SWS at the expense of wakefulness and Stage 1 sleep. REM sleep was nonsignificantly increased. This rebound pattern is similar to that produced after recovery from total or partial sleep deprivation, as will be discussed below.

Performance on a simple reaction time test, the Wilkinson Addition Test and a Digit Symbol Substitution test all showed decrements after two nights of sleep disruption. Only the addition test showed decrements after one night.

Subjects also reported themselves as significantly more sleepy following the second disruption night.

In comparing these results with those obtained during total sleep deprivation protocols, Bonnet noted that in general, the decrements produced by two nights of sleep disruption were at least as great as those seen after one night of total sleep deprivation, even though only one hour of sleep was lost per night. He suggested that fragmentation itself may interfere with the restorative function of sleep. Several possible mechanisms which might produce this interference were noted. The first was that changes in sleep architecture may be critical. The increased Stage 1 sleep seen during disruption may not "count" as sleep at all in terms of restorative value, if it were subtracted then total sleep time during disruption would be grossly reduced. It is also possible that a sleep-dependent process of protein synthesis is interfered with by repeated awakening. Similarly, sleep-dependent hormonal secretions may be disrupted.

b. Sleep Deprivation

The behavioral effects of total sleep deprivation have been subject to investigation since the pioneering work of Patrick and Gilbert (1896). There is a remarkable degree of agreement between the earliest writers and the more recent concerning the relative lack of catastrophic conse-

quences of prolonged sleep loss, although for a time in the early 1960's, after the differentiation of sleep into REM and NREM processes, several investigators emphasized the psychotogenic potential of total sleep deprivation (Berger and Oswald, 1962b; West et al, 1962).

Kleitman conducted a comprehensive investigation of the biochemical, physiological and behavioral consequences of 40 to 115 hours of enforced wakefulness on six young male subjects (1923). He reported a clear circadian alertness rhythm superimposed upon the progressive decrements in performance attending sleep loss. The second and third nights of deprivation, for example, were characterized by more subjective discomfort and objective impairment than the third and fourth days respectively.

Kleitman also observed that even when circadian factors reinforced the tendency towards impairment, subjects could regain normal levels of arousal and capability if sufficiently motivated, such as by a trip to an "all-night cafe".

Objectively, sleep deprived subjects could be distinguished mainly on the basis of sleep-seeking behavior, and the tendency to fall asleep immediately if not prodded by the experimenter. Biochemical variables monitored, including blood sugar levels, blood carbon dioxide saturation, erythrocyte and leucocyte counts did not show abnormal variations during sleep loss. Physiological changes were

detected, including lowered blood pressure and dampening of the circadian temperature fluctuation.

Kleitman judged his results to refute then prevailing theories which explained the need for sleep on the basis of vasomotor fatigue (producing insufficient circulation to the brain) or the accumulation of toxins during wakefulness. In formulating his own theory, he emphasized the fact that (nonpsychotic) impairment of higher brain centers (e.g. lowered concentration) appeared to be the major consequence of sleep loss. Sidestepping the question of what kind of neuronal deficiency or toxicity was responsible for this impairment, Kleitman focussed instead on the fact that sleepiness in inherently a neurological phenomenon, not simply a neurological byproduct of other physiological changes. Kleitman went so far as to propose that dreaming may occur when the higher brain centers are especially fatigued, leaving lower centers to uncritically allow irrational associations to stand uncorrected. Whereas the neuroanatomical loci of Kleitman's model must of course be refined, it remains remarkable in its foreshadowing of the division of sleep processes onto multiple neural substrates.

In the ensuing decades, before the dichotimization of sleep into REM and NREM processes, research into the effects of total sleep deprivation focused on two questions. The first was whether the overall psychophysiological reaction

to sleep loss was one of activation or deactivation. Findings in this area were equivocal; what subjects were doing while being sleep deprived was seen as a deciding factor. The question was further complicated by the fact that one of the most consistent psychophysiological effects of sleep deprivation, suppression of the waking alpha rhythm, could be interpreted either as an indicator of deactivation, since the alpha rhythm was known to attenuate prior to sleep onset (Kleitman, 1939; Bjerner, 1949) or as a sign of compensatory efforts at arousal, since the alpha rhythm was also lost when subjects were asked to concentrate on a task (Malmo, 1958).

The second area of concern was identifying the type of task which would prove sensitive to sleep loss. Early investigators posited that more complex, demanding tasks would be most sensitive (Laslett, 1924; Kleitman, 1939) whereas later workers held that less interesting and less challenging tests should be employed (Wilkinson, 1957; Ax et al, 1957).

Williams and his colleagues adopted a different approach to settle this question: Rather than delineating the type of task which would detect impairment following sleep loss, they focused on the nature of the impairment itself, and how that impairment interacts with the demands made by different types of tasks (Williams et al, 1959).

The major consequence of sleep loss with respect to

performance, according to these authors, was lapses in response. These were seen to be coincident with brief, intermittent episodes of sleep, such as would be termed "microsleeps" currently. Lapses in response would interact with the demand characteristics of a task to produce various types of decrement following sleep loss. On self-paced tasks, for example, speed would be compromised, whereas accuracy would suffer on experimenter-paced tasks.

The experimental results of several studies bore out this hypothesis, with the additional finding that subjects' performances were notably uneven following sleep deprivation. Their worst efforts (e.g. slowest reaction times within a given trial) grew markedly worse, even though they were still at times capable of baseline-level performance.

The authors next integrated these performance studies with electrophysiological data. They concluded that alpha suppression following sleep deprivation should be interpreted as a sign of deactivation, since it coincided with lapses in performance.

In general, more recent research has reinforced the notion that simple, monotonous tasks, or complex tasks which nonetheless fail to stimulate interest, are best suited to detect the effects of sleep loss (Wilkinson, 1968). In either case, tasks must be relatively long (on the order of thirty minutes or longer) and spaced throughout the day.

Wilkinson made use of signal detection theory to posit an interesting differentiation between the effects of REM vs. NREM sleep loss. Noting that d', a measure of observer sensitivity apart from motivational factors, did not fall in subjects who accumulated more than three hours of sleep over a night but did fall precipitously for sleep lengths of less than three hours, Wilkinson hypothesized that loss of slow wave sleep (which normally does not occur until sleep is curtailed to less than three hours) might be responsible for loss of performance capacity, whereas loss of REM sleep (which occurs during less drastic sleep restriction) might cause a reduction in willingness to perform.

One of the most recent investigations of the effects of total sleep deprivation monitored four subjects for 205 hours (Kollar et al, 1969). This study was similar in method and scope to the work of Kleitman 46 years earlier, albeit more sophisticated in terms of measurement. Results were also in accord with the earlier study. In general, whereas increasing levels of cognitive disorganization, temporal disorientation and visual misperception were observed as the sleepless period lengthened, psychotic-like behaviors were brief, self-contained and present against a background of intact cognitive functioning. Prolonged sleep loss was not deemed to possess significant psychotogenic potential.

Measurement of respiration rate, blood pressure, pulse rate

and oral temperature again yielded results that were equivocal in terms of supporting an "activation" or "deactivation" theory. Biochemical assays indicated "some activation of the pituitary-adrenal axis" but not a full-fledged "Selye-type stress response".

Especially noteworthy in terms of the present study was the observation of a direct correlation (+.72) between alpha suppression and subjective sleepiness on a five point scale. This finding reinforced that which was derived from performance testing, (Williams et al, 1959) demonstrating that alpha supression is associated with deactivation measured along diverse dimensions.

c. Sleep Restriction

Reduction in habitual sleep time, either acutely for short or long periods, or gradually, has been the subject of a number of studies since the mid 1960s. These studies have assessed changes in the electroencephalographic quality of the shortened sleep, as well as changes in mood, sleepiness and performance capability.

There is general agreement that sleep architecture undergoes characteristic changes when sleep is restricted. Findings concerned with mood parameters have been more equivocal--intersubject variability appears to play an important role in this area. Subjective sleepiness is

clearly increased by acute restrictions of time in bed, although apparently this effect is short-lived during extended periods of restriction. Performance during periods of sleep restriction is surprisingly resilient, especially on short or interesting tasks. Only when sleep is restricted below three hours per night do performance capabilities show a broad and consistent decline.

i. Acute Sleep Restriction

An early sleep restriction study limited subjects to three hours in bed for eight consecutive nights (Webb and Agnew, 1965). The major finding was that the truncated sleep periods were not "miniatures" of normal-length sleep periods. They more closely resembled the first three hours of a normal night's sleep, in that slow-wave Stage 4 sleep was preserved, appearing in its usual position during the first hours of sleep, and lasting a normal duration of about 90 minutes. Expressed as a percentage of Total Sleep Time (TST), Stage 4 sleep therefore increased greatly, from 20.4% at baseline to 47.5% of the shortened sleep period.

Also in keeping with this resemblance, very little REM sleep was seen. It declined in relative as well as absolute terms, constituting 7.5% of the shortened sleep as opposed to 23.1% of baseline sleep. The other sleep stages were represented in roughly typical proportions, although of course they were greatly reduced in absolute terms.

Comparing the truncated night to the first 3 hours of the baseline recordings, it could be discerned that the increase in Stage 4 sleep came mainly at the expense of Stage 3, that is, less time was spent in the transition to synchronized sleep.

Performance on a paced addition test and on auditory and visual vigilance tests showed some decrement after seven or eight nights of restricted sleep, but these decrements were seen in only about half of the eight subjects, and not consistently in these.

A second study of partial sleep deprivation was accomplished as one of two conditions of an experiment studying the effects of REM deprivation (Sampson, 1965,1966). Taking advantage of the relatively late appearance of the bulk of REM sleep during the night, REM deprivation was accomplished over three nights by limiting subjects to 2.5 hours of bedtime per night, as well as by the conventional method of multiple REM-onset awakenings.

The two conditions were not systematically compared with regards to effects on performance. It was reported that overall, subjects performed better in the multiple awakening condition, which was expected since that condition entailed less NREM sleep loss. However, subjects showed little performance impairment under either condition.

A third early study of partial sleep deprivation focussed

on changes in the amount of Stage 4 sleep (Dement and Greenberg, 1966). Performance measures were not employed in this study. The investigators found that not only was Stage 4 sleep preserved during the shortened sleep nights, its duration increased on recovery nights as compared to baseline. This "rebound" was not seen as analogous to that seen after REM deprivation since no Stage 4 deprivation was necessary to produce it--nonspecific sleep deprivation was all that was needed. The authors suggested that there may be "some quantitative interrelatedness" between the NREM sleep stages in the sense that Stage 4 sleep is "worth more" than Stage 2 (p.525), so that it takes precedence following nights of reduced sleep.

Acting on the hypothesis that subjects were able to compensate, at least for short periods of time, for actual performance decrements related to sleep loss, Wilkinson and his colleagues used a protocol requiring testing over extended periods (Wilkinson,1968). Under this protocol, impairments in vigilance (detecting randomly occurring tones which are slightly shorter than a standard half-second tone, with tones presented every two seconds) and self-paced addition (adding columns of five two-digit numbers) were detected after just two nights of restricted (5 hour) bedtimes (Wilkinson, Edwards and Haines, 1966).

An extension of this study examined the effects of 7.5 (control), 6 and 4 hour sleep periods on performance

throughout the day (Hamilton, Wilkinson and Edwards, 1972). In addition to standard vigilance and addition tasks, the investigators employed a "running digit span" test. This consisted of random digits presented at a rate of 1.5/sec. At random intervals a bell rang and the sequence stopped. Subjects were required to recall the digit which occurred four digits before the bell.

With respect to vigilance, a significant practice effect across days was seen, along with a marginal main effect across conditions. When the data were treated by signal detection analysis, it was seen that d', a measure of observer sensitivity apart from motivational factors, improved over time for subjects in the 7.5 and 6 hour sleep period conditions, but not in the 4 hour condition.

Calculation efficiency predictably showed greater impairment with greater amounts of sleep restriction, and this effect was cumulative over time. Surprisingly, subjects in the four hour condition did best on the digit span test. The authors surmised that their greater sleepiness forced reliance on passive echoic memory, which in this case was more effective than active strategies.

The first study to employ both subjective and objective measures of sleepiness following sleep restriction focussed on the course of sleepiness levels over seven nights of restriction to 5 hours time in bed (Carskadon and Dement, 1981). Subjective sleepiness was assessed by the Stanford

Sleepiness Scale (SSS) and an analog rating scale. The analog sleepiness scale requires the subject to place a vertical mark along a 100mm line which has its ends labeled "very wide awake" and "very sleepy". Objective sleepiness was measured by the Multiple Sleep Latency Test (MSLT).

The effects of the restricted sleep on sleep architecture were in agreement with those seen in earlier studies. Sleep latency, wakefulness after sleep onset, and Stages 1, 2 and REM sleep were all reduced, while Stages 3 and 4 remained at baseline levels. One of ten subjects exhibited sleep-onset REM periods (SOREMPS) on restricted and recovery nights.

All three measures of sleepiness were sensitive to sleep restriction, but their pattern of responsiveness differed. Subjective measures showed initial increases in sleepiness, but levelled off after the fifth restriction night. The MSLT did not register a significant increase in sleepiness until after the second night; thereafter it continued to show nearly linear increments in sleepiness after each restricted night. No "obvious relationship" was seen between individual sleepiness scores and changes in sleep architecture during the period of sleep restriction.

ii. Gradual sleep restriction

The first polygraphic study of sleep and waking behavior

during gradual sleep reduction confirmed the "tenacity" of slow wave sleep during sleep restriction (Johnson and MacLoed, 1973). Two subjects completed the regimen, reducing their time in bed by thirty minutes every two weeks, until a four hour sleep period was reached. Although a shortened REM latency was reported when sleep was restricted to 5.5 hours, it should be noted that the baseline REM latencies of these subjects were long at 225 minutes and 135 minutes respectively; they "shortened" to normal values.

Sleepiness was not objectively assessed, but an indirect measure was provided by the number of times subjects overslept and the number of forbidden naps they indulged in. Both subjects began oversleeping on occasion with the first half-hour reduction, but the frequency of this oversleeping did not dramatically increase as sleep was restricted. 58% of all oversleeping episodes (n=41) occurred during the second half of the experiment, when bedtimes were below six hours.

Napping behavior was relatively infrequent. All six instances of daytime napping occurred when nocturnal sleep periods were under six hours. The discouraging of napping had the effect of limiting its responsiveness as a measure of sleep loss.

Mood scales did not appear especially sensitive to sleep reduction. While subjects did report that they were less

happy, less friendly, less energetic and more fatigued at reduced sleep times, the differences were not large in relation to the intersubject variability seen. Interestingly, the two scales less clearly associated with sleepiness, reflecting degree of friendliness and happiness, showed a more uniform and consistent responsiveness to sleep reduction. Performance measures were equivocal at 5.5 hours of bedtime, but showed impairment at 4 hours.

The most significant finding of this study was that both subjects maintained a shorter bedtime at three and eight month follow-ups. The reductions were on the order of 1.5 and 2.5 hours. This voluntary reduction in sleep time was accompanied by daytime mood indicators which matched baseline levels.

A second study extended the work of Johnson and MacLoed, assessing the effects of gradual sleep reduction on four couples over the course of a year (Mullaney et al, 1977). This study confirmed the finding that Stage 4 sleep increased both in absolute and relative amounts. The increase was accomplished abruptly during the 6.5 hour sleep-period phase, and held steady through the 5.5 hour phase.

While REM sleep decreased in absolute terms, its proportion of total sleep time remained constant. A phase advance of REM sleep was seen in the sense that the first five hours of the night contained greater amounts of REM sleep as TST

was reduced. The actual latency to REM onset was not significantly shorter, and the NREM/REM cycle length and rhythmicity did not change over the course of sleep reduction. Two subjects responded to sleep reduction with SOREMPS. On twenty occasions, these subjects had REM latencies of less than twenty minutes. (The finding that a minority of normal subjects are especially prone to exhibit SOREMPS under altered sleep/wake schedules had been noted previously, e.g. Rechtshaffen and Dement, 1968.)

Subjective sleepiness during this same study of gradual sleep reduction was assessed through the Stanford Sleepiness Scale and a questionnaire assessing difficulty going to sleep, restedness, the need for more sleep, and subjective sleep latency (Friedman et al, 1977). All of these measures showed the predicted results of greater subjective sleepiness at reduced sleep times.

Behaviorally, sleepiness levels could be inferred from instances of napping and oversleeping. Oversleeping did not exceed an average of 1.33 occasions per month until sleep time was reduced to 5.0 hours, it then rose to 6.5 times on average. Subjects were able to avoid napping until time in bed reached 6.0 hours--even at lower levels naps were taken less than once per month on average.

Mood was assessed through the Profile of Mood States (POMS) and performance through the Wilkinson tests described above. Surprisingly, no significant trends were detected

by the mood or performance instruments at any level of sleep reduction.

During a twelve month follow-up period, subjects voluntarily remained at levels of Time In Bed (TIB) 1-2.5 hours below baseline. While subjectively they claimed that feelings of well-being and alertness had returned to baseline levels, it is interesting to note that these subjects, who had reported no napping during baseline adaptation and very infrequent napping during the course of sleep reduction, were now napping approximately 2-3 times per month.

iii. Chronic sleep restriction

Only one study has examined the effects of a stable, prolonged reduction in sleep length (Webb and Agnew, 1974). Fifteen subjects were restricted to 5.5 hours TIB for sixty consecutive days. EEG, performance and self-report data were collected on all subjects, however, because of experimenter error usable EEG data were available for only eight subjects.

As was seen during gradual sleep reduction, there was an increase in Stage 4 sleep during initial weeks on the regime. Stage 4 sleep returned to baseline values by the fifth week. An immediate drop in REM time was observed. Over the course of the study subjects experienced on average a 25% reduction in REM time, or thirty minutes per

night in absolute terms.

While REM sleep time decreased overall, there was significantly more REM sleep in the shortened sleep periods than had occurred during the first 5.5 hours of the baseline nights. REM latency was dramatically reduced at the end of the first week of restricted sleep. Two of eight subjects experienced SOREMPS, five of the remaining subjects showed reduced latencies. However, over subsequent weeks REM latencies remained reduced for only three subjects.

Performance on the Wilkinson Vigilance Task declined over the course of the study, but this was attributed mainly to decreased motivation: Subjects made progressively fewer responses even though they were informed that "false alarms" would not count against them. Grip strength and a word-memory test showed improvement and no change respectively.

Responses to questions such as "How easy was it to get up this morning" or "Are you drowsy during the day?" indicated significant sleepiness during the first week, with a shift toward baseline values in subsequent weeks. In the case of drowsiness, there were fewer complaints during the last week of sleep restriction than had been registered during the baseline period. Webb and Agnew interpreted these findings to mean that humans are capable of sustaining moderate amounts of sleep loss over prolonged periods with only limited behavioral consequences.

Some reservations may be raised with regard to this conclusion, both as it pertains to performance measures and to sleepiness levels. The major performance tests used in the study, while drawn from the battery of Wilkinson and colleagues, were only administered during two thirty minute periods per day. As has been emphasized by Wilkinson, detecting the effects of sleep loss requires sustained testing. An eight-hour "workday" of such testing was noted above to detect the effects of restricting TIB to 5 hours after only two restriction nights (Wilkinson, Edwards and Haines, 1966) Furthermore, performance deficits were seen to be cumulative across a four day protocol (Hamilton et al, 1972).

Webb and Agnew's chronically restricted subjects would presumably have demonstrated even greater impairment on full day's testing.

Similarly, the finding by Carskadon and Dement (1981) of a dissociation between an objective measure of sleep tendency (MSLT) and subjective assessments calls into question the conclusion of Webb and Agnew that no significant sleepiness actually resulted from chronic sleep restriction. Their subjects may have in fact begun to lose their frame of reference as to what qualifies as "normal" alertness, a phenomenon which has been observed in pathologically sleepy patients (Dement et al, 1978).

d. Selective Sleep-Stage Restriction

i. REM Deprivation in Humans

Experiments assessing the effects of REM deprivation, either on polygraphic variables or daytime mood and functioning, account for almost all the work employing selective sleep-stage deprivation. For the most part, it was taken for granted that such selective deprivation would cause daytime sleepiness, and the effect was not systematically measured. Investigators were after findings such as increased drive level, memory impairment or, in the earlier experiments, incipient psychotic processes. Against these expectations it is ironic, as Webb has noted, that perhaps the only consistently seen behavioral effect of two decades of sleep manipulations has been to make subjects sleepy (cited in Carskadon and Dement, 1982). On the other hand, a number of characteristic changes in polygraphic variables have been repeatedly demonstrated following selective sleep-stage restriction.

The first study to employ selective sleep-stage deprivation compared the effects of REM with Stage 2 deprivation (Dement, 1960). At the time, this manipulation was believed to allow for examination of the effects of dream deprivation since dreaming was thought to be confined to REM sleep. Subjects were first run in the "dream deprivation" condition; after a baseline period, they were

awakened for several consecutive nights at the onset of each occurrence of REM sleep. This was followed by a series of uninterrupted "recovery" nights. Subsequently, the effects of sleep deprivation were controlled for by waking the same subjects an identical number of times out of NREM Stage 2 sleep.

Two findings relating to polygraphic variables were noted which have been consistently replicated: First, with each consecutive night, an increasing number of awakenings was necessary to prevent subjects from returning to REM sleep. This phenomenon has since been described as a buildup of "REM pressure". Second, when subjects were finally allowed uninterrupted sleep, they spent a higher percentage of TST in REM sleep then they had on baseline. This elevation of REM sleep time was not seen after control Stage 2 awakenings. "REM rebound", as this phenomenon came to be called, provided the focus for much subsequent research.

Two other polygraphic findings are noteworthy: Dement pointed out that his procedure did not completely deprive subjects of REM sleep, because it often took a minute or two for the experimenter to make the judgment that REM sleep was in fact beginning. REM time was actually reduced 65-75% from baseline values. This methodological factor is present to a greater or lesser extent in all REM "deprivation" studies, including the present one; the procedure has thus sometimes been characterized as "partial REM

deprivation".

A second important feature of Dement's data concerns the variability seen in subjects with respect to both the rebound phenomenon and the amount of "REM pressure" generated. One of his seven subjects did not show elevated REM time on any of five consecutive recovery nights, even though this subject required the greatest increase in the number of REM-onset awakenings in order to suppress REM sleep. Differences in potential for REM rebound, both between normal subjects and between clinical groups, was to become a major research focus (Azumi et al., 1967; Zarcone et al., 1968, 1969,; Cartwright et al., 1967; Gillin et al., 1974; Nakasawa et al., 1975). Other studies examined responses to REM deprivation occurring within the deprivation night itself; these generally were concerned with changes in the nature of mentation elicited from the various stages of sleep (Pivik and Foulkes, 1966; Arkin et al, 1978).

Variability in the number of awakenings necessary to achieve (partial) REM deprivation did not prove to be of major interest: Only one early study, to be detailed below, assessed the relationship between this variable and other sleep and personality parameters (Cartwright, 1967).

In terms of daytime sequelae to "dream deprivation", increased anxiety, irritability, difficulty concentrating and increased appetite were all observed following REM

awakenings, but not following Stage 2 awakenings. These behavioral changes were described as "not catastrophic". Nonetheless, it was conjectured that "a serious disruption of the personality" might result if REM deprivation had been prolonged (p. 1707).

This possibility aroused the interest of the research community; investigations therefore concentrated on assessing mood shifts and changes in drive-related behaviors, rather than on performance testing. This line of research received impetus from the results of REM deprivation studies in animals, primarily in the cat and the rat. These studies, to be detailed below, reported hyperphagia, hypersexuality and increased aggression (Dement, 1965; Morden, 1967).

Some early studies found analogous changes in man. Sampson (1965) noted increased appetite, irritability, accident proneness and "disturbances in...subjects' relationships to reality" following REM deprivation accomplished either by REM-onset awakenings or truncation of sleep length. Dement's own work continued to find characteristic mood changes (Fisher and Dement, 1963) and changes on projective psychological testing in the direction of increased "intensity of feeling and need" and a "reduction in critical facility" (Clemes and Dement 1967) following REM but not NREM deprivation.

However, at the same time other workers were failing to replicate the findings of psychological or behavioral chan-

ges attributable to REM deprivation as opposed to generally disrupted sleep (Snyder, 1963; Kales, 1964). In a review article, Vogel (1968) demonstrated that those studies which purported to show REM-related impairments were generally poorly controlled or particularly susceptible to experimenter's and subject's expectations. As Vogel noted, this was the conclusion that Dement himself had come to by the mid 1960's.

Two REM deprivation studies from this period deserve mention within the context of the present investigation. The first was prompted by the hypothesis that psychological disturbance might follow the accumulation of many nights of moderate REM reduction, as opposed to a few nights of total or near-total deprivation, since this latter condition is rarely met outside the laboratory (Dement, Greenberg and Klein, 1966). Rather than systematically assess psychological changes, these investigators first wished to demonstrate that a rebound occurs following prolonged moderate deprivation which is comparable to that seen following relatively acute total deprivation.

Two subjects were run for an extended baseline period, after which their REM sleep was limited to 75% of their baseline mean for nineteen nights. When uninterrupted sleep was allowed, a rebound comparable in magnitude to that which would be expected after five nights of essentially no REM sleep was seen.

Two other subjects were first deprived totally of REM sleep, and then held to baseline levels for five nights, in order to determine whether the deprivation "effect" would be dissipated during the period that recovery was delayed. When finally allowed recovery sleep, these subjects showed a characteristic rebound.

Under both protocols, no significant psychological changes were seen. However, a REM "storage" property had been demonstrated. This homeostatic mechanism might be of use in other functions besides the maintenance of psychological stability. In terms of the present investigation, one might hypothesize that it serves as a physiological "reminder" of sleep loss, since, as has been detailed above, REM sleep is differentially lost (or perhaps deferred) during nights of truncated sleep.

A second study of special interest to the present investigation assessed the intercorrelations between an array of REM deprivation and personality variables (Cartwright et al, 1967). Although this study suffers from methodological weaknesses (cf. Ellman et al, 1978) it is noteworthy for considering REM "pressure", evidenced by the number of REM-onset awakenings necessary to accomplish deprivation, as a variable of interest.

Cartwright found three overall patterns of response to REM deprivation. Subjects who required a relatively large number of deprivation awakenings showed a disturbance of

typical EEG staging upon recovery sleep, in that aspects of REM and Stage 2 sleep often coincided. These subjects also tended to be highly anxious, and particularly susceptible to the effects of an hallucinogenic drug. This pattern was called "disruption".

A second group, the "substituters", tended to respond to REM-onset awakenings with vivid dream-like mentation reports, as if they were able to utilize pre-REM states as a REM substitute. This group did not show a characteristic REM rebound.

The final group, the "compensators", resembled subjects from previous studies in that they showed a rebound. This group produced few dream-like mentation reports on REM-onset awakenings.

Recently, the use of the number of REM-onset awakenings as an index of REM persistance was resumed (Adler et al, 1983). Adler ran both narcoleptic subjects and normal controls through a baseline MSLT. One week later, subjects returned to the laboratory for an adaptation night and one night of REM deprivation, followed immediately by a second MSLT.

The criteria for awakening subjects on the REM deprivation night were conservative, designed to insure that awakenings were actually made out of REM sleep, even at the expense of allowing some REM time to accumulate. All three polygra-

phic indicators of REM sleep had to be simultaneously present: Characteristic low voltage, mixed frequency EEG, reduced muscle tone and conjugate rapid eye movements. Once awakened, subjects were kept awake by voice for three minutes.

It was found that narcoleptics required more experimental REM-onset awakenings than did normal subjects, since they were much more likely to produce SOREMPS when allowed to resume sleep.

A second finding directly pertinent to the present investigation was that the number of experimental REM-onset awakenings necessary to achieve deprivation was directly correlated to levels of baseline sleepiness in the normal controls. In other words, those subjects who were sleepier before any nocturnal sleep manipulations were introduced tended to require more awakenings in order to accomplish REM deprivation.

The two findings are consistent in that narcoleptics, a group characterized by pathological sleepiness, also exhibited the greatest degree of REM recurrence. It is important to note that the two studies which investigated associations to REM pressure found seemingly contrary correlates: High anxiety and high daytime sleep tendency. The relationship between these two personality characteristics has not been systematically investigated, although there is some evidence that poor sleepers tend to be more

anxious than good sleepers (Monroe, 1967; Jovanovic, 1973).

Since it is well established that the process of REM deprivation experimentally increases the level of REM pressure, it would be profitable, in terms of resolving this issue, to examine the changes in sleepiness level produced during previous REM deprivation studies.

Although sleepiness was never explicitly and systematically assessed in the early studies, some anecdotal data are available. Wide intersubject variability in level of arousal following REM deprivation was generally seen. Referring to the handful of studies which assessed daytime effects of REM deprivation, Cartwright and Ratzel (1972) noted that some subjects "report feeling slowed down from their normal activity level, others report feeling activated."

Adler (1984) was the first to utilize the MSLT to assess changes in sleep tendency following REM deprivation in normals. He found a nonsignificant increase in sleep latency for this group overall. As this was not a primary interest in Adler's study, no NREM awakening condition was included for purposes of comparison.

Despite the relative paucity and variability of findings, some reviewers (e.g. Albert, 1975) are convinced that REM deprivation in humans tends to produce "overactivity and irritability" (p.348). This belief does not merely represent a persistence of Dement's original findings (a long

term "primacy effect") but more substantially an extension of a group of consistent findings from animal studies. These studies, to be reviewed below, indicate that REM deprivation increases central nervous system excitability.

ii. REM deprivation in animals

Acting upon the finding (Dewson, 1967) that REM deprived cats produced less attenuated evoked responses to the second click of paired stimulus clicks than equally disturbed (but not specifically REM deprived) cats, Cohen and Dement (1965) hypothesized that the REM deprivation had produced an increase in neural excitability. They confirmed this hypothesis in a study employing the threshold for electroconvulsive shock (ECS) in rats as a measure of the level of excitability.

Rats in the experimental group were deprived of REM sleep using the "flower pot" method. This technique involves placing the rats on the bottom of inverted flower pots which rise just above the surface of water. The rats can partake of NREM sleep, but the muscle atonia characteristic of REM sleep causes them to slip into the water and awaken. Control rats are placed on slightly larger pots, which allow both REM and NREM sleep.

It was found that the threshold for ECS was significantly reduced in the REM deprived group, whereas the control

group showed a nonsignificant increase. These results were confirmed in a parallel study, included to meet objections over the unequal treatment of groups by the flower pot technique, which used hand awakenings in a small group of rats to achieve differential sleep-stage deprivation with roughly equal amounts of total sleep loss.

A second study (Cohen and Dement, 1966) demonstrated that one ECS per day lowered the amount of REM sleep obtained by cats. Even when REM sleep was reduced for up to a week by this method, the expected REM rebound did not occur upon recovery sleep. It was suggested that the excitation produced by ECS substituted in part for the daily quota of REM sleep.

The investigators next evaluated the ability of ECS to substitute for REM sleep (measured by its attenuation of rebound) under the conditions of heightened need for REM sleep produced during REM deprivation (Cohen et al, 1967). It was found that REM deprived cats who were also convulsed four times prior to recovery sleep had significantly less REM rebound than equally deprived but non-convulsed cats.

This evidence of neural excitability following REM deprivation prompted Steiner and Ellman (1972) to hypothesize that thresholds for positively reinforcing electrical brain stimulation, also known as intracranial self-stimulation (ICSS), would be reduced following REM deprivation. As the measurement of ICSS involves monitoring a behavior (lever

pressing in rats) hypothesized changes in ICSS activity were deemed of special interest because they would coincidently demonstrate a discrete behavioral change following REM deprivation.

Following a period when rats were trained to self-stimulate and baseline rate-intensity functions obtained, half were REM deprived by the "flower pot" method, while half were placed on a larger platform to serve as yoked controls. This deprivation period lasted four days, and was followed by a four day recovery period.

As predicted, REM deprivation lowered the threshold intensity necessary for ICSS, and shifted the rate-intensity function such that increased rates of responding were seen at all but the highest intensity level. This feature of the data demonstrated that the results obtained were not due simply to increased activity, but rather to a specific leftward shift of the baseline rate-intensity function, which had originally exhibited a dip in responding at the highest intensity delivered.

A second experiment investigated the effect of ICSS on REM rebound. It was found that (subconvulsive) ICSS had an effect similar to that reported by Cohen and Dement for ECS: It reduced rebound by at least fifty percent. Considering the results of both experiments, Steiner and Ellman suggested that the ICSS neural network is directly activated by REM sleep, and that experimental activation of this

network (i.e. ICSS) can serve as a surrogate for REM sleep and reduce rebound patterns.

Other behavioral indices of increased excitability following REM deprivation in animals include increased shock-induced fighting responses, (Morden et al, 1968) hypersexuality (Dement et al, 1967) and nonspecific increases in activity (Albert et al, 1970; Olgilvie, 1972). Steiner and Ellman note that there is a direct link between some of the traditional "drive" behaviors elicited in animals by REM deprivation and the effects of ICSS, in that the hypothalamic areas mediating these behaviors are themselves potent ICSS sites.

iii. Stage 4 deprivation

Relatively little work has assessed the effects of Stage 4 deprivation. The first such study was modeled after Dement's REM deprivation paradigm (Agnew et al, 1964). Subjects were shifted to another stage of sleep (rather than awakened) by sounding a tone whenever delta wave activity began to account for almost 50% of the one minute epochs. On the recovery night, subjects showed a significant "Stage 4 rebound" over baseline values. Part of the motivation for performing this study may have been to demonstrate that a "psychic need" as postulated by Dement for "dreaming sleep" was not the only way to interpret rebound phenomena, since Stage 4 rebound does not readily

lend itself to this interpretation. Behavioral effects of this manipulation were not reported.

An extension of this study directly compared Stage 4 and REM sleep deprivation across seven nights (Agnew et al, 1967). Electric shocks were used to keep subjects out of the targeted stage. This study assessed polygraphic, performance and personality changes.

During Stage 4 deprivation, subjects spent more time in Stage 2 sleep then they had on baseline, with no significant decrease in REM sleep. Subjects in the REM deprivation condition experienced significantly less Stage 2 sleep along with sharply increased Stage 1 (without rapid eye movements or loss of muscle tone). Slow wave sleep in this condition was not significantly changed.
It was necessary to deliver four times as many shocks to achieve Stage 4 deprivation than to produce REM deprivation.

Subjects in both conditions experienced rebound of the deprived stage on recovery nights. REM time remained elevated over three recovery nights, whereas the Stage 4 rebound was complete after one night. The authors were surprised by the finding that elevated REM times were seen after recovery from Stage 4 deprivation. However, as noted by Ellman (1978) this finding can be explained by the fact that a small amount of REM sleep was lost, on average, during each deprivation night.

No significant performance decrements were noted in subjects under either deprivation condition. This could have resulted from the use of relatively short performance tests; but it may also reflect the fact that the deprivation technique resulted in very little extra wakefulness.

The personality tests employed showed changes which distinguished between the two manipulations. Overall, Stage 4 deprivation resulted in "a depressive and hypochondriacal reaction" whereas REM deprivation made subjects "less well integrated and less interpersonally effective" (p.856). It was noted that the "chief complaint" heard from subjects under both conditions was of sleepiness.

The most comprehensive study of the effects of Stage 4 vs. REM deprivation on performance utilized a novel procedure: Rather than assessing these effects directly, the investigators compared the recuperative value of the two stages on performance following two nights of total sleep loss (Lubin et al, 1974). On the two nights immediately following this total sleep deprivation, subjects were either deprived of Stage 4 sleep, deprived of REM sleep or allowed uninterrupted sleep. Deprivation was accomplished by 30-60 second awakenings accompanied by questions concerning personal information (in the case of the Stage 4 condition) or dream content (in the REM deprivation condition). A second control group slept uninterrupted through the entire experiment, in order to assess the effects of two nights of

total sleep loss.

Performance was measured on a variety of tasks generally lasting about one hour apiece, including the Williams Word Memory Test, the Wilkinson Addition test, a "Plus Seven" self-paced mental addition test, an "X-Crossout" visual vigilance test and an auditory vigilance test. Drowsiness was indirectly assessed through performance on a "Counting" task. Subjects were required to press panel keys in the order 9,8,7,6,5,4,3,2,1,9,8,etc, for fifty minutes. The score was the time spent counting; gaps longer than 2.5 seconds were excluded. Mood was assessed by a checklist composed of selected items from the Profile of Mood States (POMS).

About 50% more awakenings were required to accomplish Stage 4 deprivation than REM deprivation. The REM group averaged 70 and 68 awakenings over the two partial recovery nights; the corresponding numbers of Stage 4 awakenings were 121 and 98.

Every test employed showed significant decrements following two nights of total sleep loss, and all groups recovered functioning during the two partial and two full recovery days. However, no significant differences were found in recovery rates between the three groups. In addition, most subjects recovered to baseline levels of performance following the two partial recovery nights.

The authors concluded from these results that, insofar as

restoration of performance is concerned, sleep architecture is of relatively little consequence: Sleep quantity rather than sleep quality appears to be paramount.

In a second experiment, the same group of investigators reversed the order of deprivation: After a four night baseline period, three nights of REM or Stage 4 deprivation were administered, followed by one night of total sleep deprivation and, finally, two nights of recovery sleep (Johnson et al, 1974). The purpose of this experiment was to assess any interactive effects between selective REM or Stage 4 deprivation and total sleep deprivation. The authors hypothesized that both stages of sleep would potentiate the effects of total sleep loss.

In addition to the performance tests employed in the first experiment, a reading comprehension test and a test measuring immediate, short-term and long-term memory was administered. Additional psychometric scales employed included the Primary Affect Scale, the Spielberger State-Trait Anxiety Inventory and the McReynolds Rorschach Concept Evaluation Test (CET).

As a result of the prolonged awakenings necessary to keep REM-deprived subjects from immediately re-entering REM sleep, total sleep time for this group was significantly lower than the Stage 4 deprived group on the third selective deprivation night, but not for the three nights overall.

Both groups experienced a Stage 4 rebound on the first recovery night. This result was expected, since the REM deprived group was also deprived of Stage 4 sleep on the total deprivation night, and the recovery of Stage 4 sleep is known to take precedence following total sleep loss. The limiting of recovery sleep to two nights under these conditions prevented the occurrence of a REM rebound even in the REM deprived group, which had been held to an average of 3% REM sleep over the three selective deprivation nights.

Performance on the Counting task declined following selective and total deprivation for both groups. Stage 4 deprived subjects showed a significant decrement in long-term memory following the third night of selective deprivation, this decrement persisted even following recovery sleep. The Stage 4 deprived subjects improved on the Williams Word Memory Test and the Wilkinson Addition Test following total sleep loss. However, no significant between group differences were seen on any performance measures. Likewise, no between group differences were seen on the Rorschach CET measure or the mood scales. The Spielberger inventory did show the Stage 4 deprived subjects decreasing from baseline anxiety while the REM-deprived group showed a slight increase. However, the actual scores of the two groups following selective deprivation were nearly the same--they had differed initially.

When the investigators compared the results of their two studies, they found that one night of total sleep loss was less detrimental to performance if it followed three nights of selective deprivation (experiment 2) than if it followed baseline sleep (experiment 1). Performance was either less impaired, or as noted above, improved in absolute terms.

The authors concluded that three nights of selective REM or Stage 4 deprivation, with attendent frequent interruptions, did not potentiate the detrimental effects of one night of total sleep loss. In addition, the results of this second experiment provided further evidence that selective sleep stage deprivation produced little if any differential effect on waking behaviors.

These rigorous and sophisticated studies demonstrated that performance tasks which had been proven sensitive to sleep loss in general failed to detect differential effects of selective sleep-stage deprivation. Given the "lapse" hypothesis of Williams et al (1959) concerning the means by which these tests are sensitive (i.e. they measure momentary lack of response due to what are now called "microsleeps") it would appear reasonable to infer that the subjects in the REM deprivation, Stage 4 deprivation and non-deprived conditions within each experiment were equally sleepy. However, at least when comparing the two awakening conditions with that of uninterrupted sleep. this interpretation conflicts with more recent evidence from the

sleep fragmentation studies cited above. These produced significant impairment and, in the study of Stepanski et al, significant increases in sleepiness as measured by the MSLT with only half the number of awakenings.

While it is conceivable that skilled maneuvers, complex problem solving or nuanced judgments (in short, the very tasks which have been discredited as indicators of general sleep loss) may have been necessary to ferret out differential effects, on a more basic level, sleep tendency as measured by the MSLT may have been differentially affected.

Just as sleep latency on standardized tests has been shown to vary with the kind of instructions subjects receive (Hartse et al, 1982) transitions from a relaxed waking state in a prone position to sleep as measured by the MSLT may be quite a different gauge of sleepiness than intrusions of microsleep episodes into enforced, monotonous tasks. As an example of the factors which may complicate inferences of sleepiness based on performance measures, it may be recalled that drowsiness in the Lubin et al study was inferred from gaps in performance on a fifty minute key-press counting task. Time was subtracted from a subject's score both when the subject inadvertently stopped counting (presumably because of sleep onset) and when the subject signalled to the experimenter that a short break was needed to relieve wrist strain or general fatigue. These two conditions are clearly not equivalent in terms of

arousal level. Also, the presence of motivational influences was suggested by the (nonsignificant) trend towards lower scores following four baseline nights, when subjects presumably received adequate sleep.

The choice of comparing REM with Stage 4 deprivation could be viewed as a consequence of earlier work, which established a number of dichotomies regarding the two stages: Stage 4 sleep is defined by the most synchronized, high voltage slow wave EEG activity whereas REM sleep is characterized by a desynchronized, low voltage mixed frequency pattern. Physiological functions such as respiration and heart rate are generally regular during Stage 4 sleep and irregular during REM. Reports of vivid, perceptual mentation are expected out of REM sleep and unexpected following Stage 4 awakenings. Stage 4 sleep is accumulated early in the night, REM sleep later in the night. These and other differences would seem to indicate that a REM-Stage 4 comparison is the "natural" choice.

Nevertheless, it is difficult to extrapolate from the findings of this pair of studies to the question of what normally determines levels of sleepiness. In the drug-free, healthy human from childhood through middle age, slow wave sleep is rarely lost. The two occurrences which are most likely to intrude upon sleep are nocturnal awakenings and restricted bedtimes. Both of these are relatively powerless to disrupt slow wave sleep.

On the other hand, reduction of sleep length has been demonstrated to primarily reduce REM and Stage 2 sleep (Webb and Agnew, 1965; Carskadon and Dement 1981). Since reduction of sleep length has also been shown to produce increased sleepiness on the MSLT, the question may be asked whether this increase in sleepiness is due to the reduction in REM sleep alone, Stage 2 sleep alone or a non-specific reduction in sleep. The present study addresses this question.

CHAPTER 2. METHODS

A. Subject recruitment

Subjects were recruited through advertisements posted on a college campus. The poster offered $75 to adults who completed a study requiring "log-keeping at home plus several sessions in a sleep laboratory."

Those interested were contacted by the principal investigator (P.G.) by telephone, and asked a series of standardized questions concerning biographical information, sleep habits, alcohol and drug use, and the presence of any conditions requiring ongoing medical treatment (appendix A).

Criteria for further consideration were as follows:

1. Age between 18 and 38 years.
2. No major medical illness or current pharmacologic treatment.
3. No subjective sleep complaint.
4. Report of naps occurring less than twice a week.
5. Willingness to refrain from coffee, alcohol and other mood altering substances during the laboratory portions

> of the study, and limit intake of these substances to moderate levels during the log-keeping period.

Subjects were told that the purpose of the study was to investigate the relationship between the quality of nighttime sleep, sleepiness rhythms and dreams. They were also told that the study required several weeks of log-keeping at home, followed by three consecutive nights and two non-consecutive days spent in the sleep lab, where sleep would be recorded by standard procedures.

Subjects were told that they would experience an unspecified number of interruptions on some of the nighttime recordings, and would have the opportunity to take naps during the days spent in the lab. It was specified that at the end of the log-keeping period, a small percentage of subjects would be asked to withdraw from further participation, in which case they would be paid $10 for keeping the log. Finally, subjects were told that they could expect to receive no benefit from the study other than the monetary compensation and the opportunity to focus on their sleep, sleepiness rhythms and dreams. Subjects were told that no deleterious effects other than mild discomfort were anticipated from the procedure. No mention was made of possible changes in sleepiness level.

Those who agreed to participate were sent a two week sleep log which included the Stanford Sleepiness Scale, (appendix

c) a Casio Model F-5 Alarm Chronograph wristwatch set to "chime" on the hour, and an index card for recording sleepiness scores. They were instructed to carry on their normal routine, recording an SSS rating whenever they heard the chime during waking hours. They were also instructed to keep a careful log of bedtimes and sleep, including nocturnal awakenings and daytime naps.

B. Subject attrition

The criteria for disqualification were as follows:

1. Average of more than two naps per week. A nap was defined as a sleep episode occurring more than one hour before or after the main sleep period.
2a. Median bedtime length equal to or greater than 9.5 hours.
2b. Median bedtime length equal to or less than 5.5 hours.
3. Baseline MSLT latency of less than five minutes.

Twenty nine prospective subjects completed at least some of the sleep log. Eight dropped out before the scheduled laboratory dates, generally citing unavoidable schedule conflicts. Three subjects were disqualified by the experimenter, on the basis of averaging more than two naps per week (2) or median time in bed of at least 9.5 hours (1).

One subject discontinued participation in the study after the first night, citing aggravation of an existing leg injury while outside the laboratory, and one subject completed the study, but was replaced when a "blind" scorer judged his average baseline MSLT latency to be less than five minutes.

C. Pre-laboratory procedure

Subjects were asked to stabilize their sleep by being in bed at assigned bedtimes for one week prior to their first laboratory sessions. The assigned bedtimes were the median bedtimes recorded over the two week log-keeping period (henceforth referred to as "habitual bedtime".) Female subjects were scheduled so as to avoid their being in the laboratory during the last week of their menstrual cycles.

On the date of the first laboratory session, subjects were asked to appear at the sleep laboratory within two hours of their habitual rising times.

Groups of four subjects were assigned to either the REM awakening or Stage 2 (S2) awakening condition in the following manner: The first group was assigned to the REM awakening condition. The next was assigned to the Stage 2 awakening condition. The third group was again assigned to the REM condition and the final group of four subjects was assigned to the Stage 2 condition.

The REM awakening group contained 7 females and 1 male.

The mean age was 26.9 years (6.1) and the mean bedtime, as calculated by sleep log, was 460.0 minutes (50.4). The Stage 2 awakening group contained 5 females and 3 males. The mean age was 24.5 years (4.0) and the mean bedtime 461.1 minutes (59.0). Individual subject variables are given in Table 1. Differences between the groups with respect to age and habitual bedtime were not statistically significant. Individual habitual bedtimes are given in Table 2.

Initially, no attempt to match the two conditions with respect to the variables of age and sex was made. However, after twelve subjects had been run and the last four scheduled, it was discovered that chance would have placed females almost exclusively in one group and males exclusively in the other. Therefore, the experimenter intervened and disqualified the last four subjects, replacing them with subjects of the opposite sex in an effort to roughly equate the two conditions with respect to this variable.

D. Laboratory procedure

Subjects were generally scheduled to be run in pairs. However, because of last minute cancellations and schedule changes, 10 of the 16 subjects were run alone. The following schedule applied to all subjects, regardless to which experimental condition they were assigned:

Day 1: Baseline MSLT and psychometric

testing.

Night 1: Experimental awakening night.

Day 2: No laboratory study; subjects forbidden to nap.

Night 2: Experimental awakening night.

Day 3: Post-treatment MSLT.

Night 3: Partial recovery night with two awakenings for mentation reports. Data from this night will be presented elsewhere.

All subjects gave their informed written consent to participate in the laboratory study (appendix d). Electrodes were then applied in preparation for the first MSLT nap.

The following EEG montage, based on the International 10-20 System (IFSECN, 1958) was employed for all MSLT recordings:

Channel 1: 02 referenced to A1+A2. The left occipital placement O1 was applied as a backup.

Channel 2: C3 referenced to A1+A2. The right central placement C4 was applied as a backup.

Channel 3: LOC (Left outer canthus) referenced to A1+A2.

Channel 4: ROC (Right outer canthus) referenced to A1+A2. Placements at the canthi were made at a slight angle to the

horizontal to pick up vertical eye movements.

Channel 5: Left EMG referenced to right EMG. Mentalis muscle placements were used.

All face electrode resistances were under 20 Kohms. Scalp electrodes were under 10 Kohms, as were A1 and A2. A ground electrode was applied to the forehead.

The following polygraph settings were used:

Paper speed: 10mm/sec.

60 cycle notch filters ON for all amplifiers.

EEG channels:

- Low Frequency Filter: .3Hz
- Sensitivity: 7.5
- Rise Time Constant: 3ms
- High Frequency Filter: 90Hz

EOG channels:

- Low Frequency Filter: .1Hz
- Sensitivity: 5.0
- Rise Time Constant: 3 ms
- High Frequency Filter: 30Hz

EMG channel:

- Low Frequency Filter: 3.0 Hz
- Sensitivity: 1.5
- Rise Time Constant: 3.0 ms
- High Frequency Filter: 90 Hz

All channels were calibrated such that a 50 microvolt pulse

registered as a one centimeter pen deflection.

1. MSLT Procedure

The first MSLT nap was scheduled to occur three hours after each subject's habitual bedtime. Subsequent opportunities were spaced every two hours, until a total of five nap opportunities had accumulated.

Two minutes before each scheduled nap opportunity, subjects entered a soundproofed chamber and got into bed. Recording quality was checked, and any adjustments to electrodes were made. At the start of the nap, the lights were turned off, the subject was told "time for your nap" and the door was closed. Subjects were allowed to remain in bed until two consecutive minutes of any stage of sleep were accumulated, or until twenty five minutes had elapsed. If a subject fell asleep on the twenty fifth minute, one extra minute of recording time was allowed.

At the end of the nap opportunity, the subject's name was called over an intercom, and a series of questions relating to any mentation occurring just before the subject was called were answered and recorded on tape. (All results pertaining to mentation will be presented elsewhere.) Lights were then turned on, and the subject either completed study questionnaires or was allowed quiet activity (e.g. reading, walks, eating, but no time in bed) until the next nap opportunity.

2. Psychometric testing procedure

During the first laboratory session, between nap opportunities but before any nocturnal sleep, subjects completed the following psychometric tests:

1. Minnesota Multiphasic Personality Inventory (Hathaway and McKinley, 1951)
2. Embedded Figures Test (Witkin, 1969)
3. State/Trait Anxiety Inventory (Spielberger et al, 1968)

In addition, before each nap opportunity on both the baseline and post-treatment MSLT runs, subjects rated their sleepiness according to the Stanford Sleepiness Scale.

3. Auditory Evoked Response procedure

Seven times over the course of the entire study, each subject underwent auditory evoked response (AER) recording. Methodological details and results pertaining to the AER recording will be presented elsewhere. Insofar as the recording technique may have influenced subjects sleepiness levels, the following information will be presented here:

On each AER trial, subjects sat in a comfortable chair with headphones on for approximately twelve minutes. The overhead lights were off during this time. Subjects were required to tap their fingers when a particular stimulus

was presented over the headphones; they were visually monitored to assure that they were awake and tapping.

The schedule for AER recording was as follows:

1. After baseline MSLT nap 1.
2. After baseline MSLT nap 5.
3. After awakening night 1.
4. After awakening night 2.
5. After post-treatment MSLT nap 1.
6. After post-treatment MSLT nap 5.
7. After recovery night 1.

4. Experimental Awakening Procedure

All nighttime recordings began at each subjects habitual bedtime as determined from the sleep log and ended at the habitual time of arising. Subjects were required to lie in bed with the lights out between these bedtimes, apart from use of the lavatory. The montage used for nocturnal recordings included all of the placements used for the MSLT recording plus:

Channel 6: Right superior orbital (RSO) referenced to A1+A2.

Subjects in the REM deprivation condition were awakened by calling their names over an intercom until they responded with the words "I'm awake" each time there were polygraphic

indicators of REM sleep, defined as concurrent

1. Low voltage mixed frequency EEG.
2. Conjugate rapid eye movements.
3. Reduced muscle tone.

This stipulation of concurrence differs from the criteria of Rechtshaffen and Kales (1968) who score REM onset "backwards" from the first rapid eye movement to the beginning of tonic REM manifestations, i.e., reduced muscle tone and characteristic EEG. This conservative methodology was meant to insure that REM awakenings were indeed made from REM sleep.

Once awakened, subjects were kept awake for three minutes by playing an interactive "geography" game, which required each player to recall the name of a geographic feature such as a city or river which began with a letter corresponding to the last letter in the previous name.

On two occasions per night, instead of playing the geography game subjects were kept awake by answering the series of mentation questions used after each MSLT nap opportunity.

Subjects in the Stage 2 awakening condition were awakened out of Stage 2 sleep, as defined by standard criteria (Rechtshaffen and Kales, 1968) according to the following schedule:

1. The number of awakenings per night was set equal

to the mean number of experimental awakenings undergone by subjects in the corresponding REM awakening group.

2. The number of awakenings per quarter of the night was also matched to the mean number of experimental awakenings per quarter night undergone by corresponding REM awakening subjects, in an attempt to control for "time of night" effects.

3. No Stage 2 awakenings were performed until after the first REM period had transpired, in an attempt to avoid interference with Stages 3 and 4 sleep.

Once awakened, subjects in the Stage 2 awakening condition were treated in the same manner as were the REM awakening subjects.

5. Recovery night procedure

On the night following the second awakening night, all subjects underwent a recovery night. Habitual bedtimes were also used for this night. Subjects were woken out of an early and late REM period and asked the series of questions pertaining to mentation noted above. Data from this night will be presented elsewhere in conjunction with mentation findings.

E. Primary Statistics

All nocturnal sleep recordings were scored by the principal investigator. Ten records (31.25%) were also scored by a

polysomnographic technician employed at the Montefiore Sleep-Wake Disorders Center. The concordance between scorers for Total Sleep Time was .98 and for Total REM Time .97.

All MSLT recordings were scored by a certified clinical polysomnographer who was blind to each subject's condition.

Sleep stage data from nocturnal recordings were analyzed using standard SWDC programs on a Digital Equipment Corporation PDP 1123 computer. Statistics concerned with the effects of awakenings on the REM/NREM cycle were generated using programs developed during a previous SWDC investigation (Adler, 1984).

MSLT records were scored for the following variables:

Total Recording Time (TRgT): Time between lights out and lights on.
Sleep Latency (SL): The time from lights out to the first of two consecutive 30 second epochs scored as any stage of sleep (Carskadon and Dement, 1979).
Total Sleep Time: The sum of all sleep epochs.

Nocturnal recordings were scored for the following variables in addition to those scored on the MSLT:

Total Stage 1 Time (TST1)
Total Stage 2 Time (TST2)
Total Stage 3 Time (TST3)

Total Stage 4 Time (TST4)

Total REM Time (TREMT)

Rem Latency (RL): Time from sleep onset to first 30 second epoch of REM sleep or first experimental awakening, whichever occurred first.

Total Number of Experimental Awakenings (EA1 and EA2)

Number of Experimental Awakenings Per Hour of Sleep (EA/TST)

Number of Experimental Awakenings Per Hour of Recording Time (EA/TRgT)

Following Adler, REM period cycles during the awakening procedure were defined in two ways: The first was according to the "fifteen minute rule" as used by Hartmann (1968) and Vogel (1980) whereby REM offset is defined as an epoch of REM sleep followed by fifteem minutes of any state but REM (including wakefulness).

The second method of defining REM cycle length is in response to claims that the REM cycle is sleep dependent, and that therefore intervening wakefulness should be omitted when delineating REM cycles (Moses, 1977; Johnson, 1980). The "ten minute rule" defines REM offset as an epoch of REM sleep followed by ten minutes of either consecutive or non-consecutive NREM sleep.

REM Period Onset (REMP Onset): The first epoch of REM sleep which followed Sleep Onset or a REM Offset.

REM Period Interval (REMP Interval): The time between suc-

cessive REMP Onsets

REM Period Length (REMP Length): The time between REMP Onset and REMP Offset

REM Period Number (REMP Number): Total number of REMPs per night

SOREMP Frequency following EA (SOREMP Frequency): The percentage of instances when an EA was followed by a SOREMP (REM sleep occurring within fifteen minutes after sleep resumes)

F. Secondary Statistics

Mean sleep latency on Pre and Post-Treatment MSLT was calculated for each subject. If one minute of consecutive sleep did not occur on a particular nap opportunity, a 25 minute sleep latency was entered into the formula for computing the mean.

Nocturnal sleep stage parameters were averaged over the two awakening nights for each subject. These averages were employed in comparing nocturnal PSG parameters in the two conditions.

Within-subject differences, such as Pre- vs. Post-Treatment mean sleep latency on the MSLT, were compared using the correlated T test. Comparisons were made for all subjects pooled together, as well as within each awakening condition. Between-group differences were compared using the T test for independent means. These included comparisons of

averaged nocturnal parameters, and comparison of changes in mean MSLT latency following two nights of awakenings.

The degree of association between selected variables both within treatment conditions and across pooled subjects was calculated using Pearson correlations.

A .05 rejection region was adopted for all statistical tests. T tests are two-tailed unless otherwise specified. Tests were carried out on an IBM 370 computer using programs from SPSS Version M Release 9.1, as well as on a PDP 1123 using Saturn-Calc 2.2 programs.

CHAPTER 3. RESULTS

A. Baseline Measures

Baseline MSLT

The average basal sleep latency across five nap opportunities of subjects in the REM awakening (REM) group did not differ significantly from that of the Stage 2 awakening (S2) group. (REM mean = 15.5 min [7.6]; S2 mean = 12.8 min [4.8], NS.)

Baseline SSS

Hourly subjective sleepiness scores during the nine hours corresponding to the hours spanned by each subject's MSLT testing (i.e. beginning three hours after habitual time of arising, ending twelve hours after habitual time of arising) were averaged across the two week log keeping period. The two groups did not differ significantly on this baseline measure (REM mean = 2.17 (.53); S2 mean = 1.99 (.52) NS).

Baseline Psychometric Measures

The two groups did not differ significantly with respect to

level of anxiety as measured by either the Spielberger State-Trait Anxiety Inventory (STAI) (REM State mean = 33.00 (7.76); S2 State mean = 29.75 (4.43), NS; REM Trait mean = 36.00 (9.67); S2 Trait mean = 35.13 (6.51), NS) or the Taylor Manifest Anxiety Scale (MAS) of the MMPI (REM mean = 53.29 (15.77); S2 mean = 45.37 (7.87), NS).

There was no significant difference between the groups with respect to level of depression as measured by the Depression Scale (DS) of the MMPI (REM mean = 58.25 (18.58); S2 mean = 52.87 (7.85), NS).

There was a trend towards greater field independence, as measured by Witkin's Embedded Figures Test, in the Stage 2 awakening group (REM mean = 815.6 seconds (374.6); S2 mean = 520.1 seconds (226.3), $p<.10$).

Correlations Between Baseline Measures

The objective and subjective baseline sleepiness measures were not significantly correlated with each other ($r= -.208$, NS). Neither measure was significantly associated with subjects' habitual bedtime length ([Bedtime/MSLT] $r= -.226$, NS; [Bedtime/SSS] $r= -.366$, NS).

B. Awakening Nights

Data presented represent the average of the two awakening nights, except where indicated.

The two groups did not differ significantly with respect to Total Recording Time (REM mean = 463.6 min [48.5]; S2 mean = 458.5 min [59.7], NS) or Total Sleep Time (REM mean = 331.1 min [39.7]; S2 mean = 348.8 min [75.6], NS).

The number of experimental awakenings could be only imperfectly matched between the two groups. The means of the two groups of subjects run in the REM awakening condition were used as "targets" in awakening S2 subjects. S2 subjects who spent more time in REM sleep towards the end of the night than anticipated escaped the last few awakenings. Statistically, the two groups did not differ significantly either on night one (REM mean = 12.4 [4.6]; S2 mean = 11.8 [2.2], NS) or on night two (REM mean = 18.0 [6.2]; S2 mean = 15.8 [1.1], NS.)

The percentage of time spent in Stage 1 sleep was virtually the same in the two conditions. (REM mean = 10.8 [3.4]; S2 mean = 10.0 [3.6], NS.) Stage 2 percent was reduced in the subjects who were awakened from that stage (REM mean = 59.0 [5.9]; S2 mean = 46.5 [7.2], $p<.01$); there was a reciprocal decrease in REM percent in the REM awakening group (REM mean = 9.3 [4.5]; S2 mean = 23.2 [7.3], $p<.001$).

There was an unexpected difference in the distribution of Stages 3 and 4 sleep. Considering Stage 3 percent alone, the REM group had a higher percentage than did the S2 group (REM mean = 12.5 [5.2]; S2 mean = 7.2 [1.7], $p<.05$). The S2 group had a nonsignificantly greater percentage of Stage

4 sleep (REM mean = 8.5 [6.9]; S2 mean = 13.4 [6.2], NS). When the two slow wave sleep stages were combined, there was virtually no difference between the two groups (REM mean = 21.0 [8.7]; S2 mean = 20.6 [5.0], NS

C. Pre/Post Sleepiness Comparisons

MSLT

Pooling both groups, subjects exhibited shorter mean sleep latencies on the MSLT following the two awakening nights (Pre-MSLT mean = 14.2 min [6.3]; Post-MSLT mean = 10.2 [4.6], $p<.01$). However, when each group was analyzed separately, only the S2 group's increase in sleepiness remained significant (S2 Pre-MSLT mean = 12.8 [4.8]; S2 Post-MSLT mean = 8.4 [3.6], $p<.025$). The lack of a significant result in the REM awakening group was a function of both a nonsignificantly smaller increase in sleepiness and increased variability, as compared to the S2 group (REM Pre-MSLT mean = 15.5 [7.6]; REM Post-MSLT mean = 12.1 [5.0], NS).

There was no significant difference between the changes in average sleep latency seen in the two groups (REM mean = -3.4 min [5.9]; S2 mean = -4.4 min [4.0], NS).

SSS

Due to experimenter error sufficient SSS data are available for only twelve of the sixteen subjects (REM N = 5; S2 N =

7). Given this inequality in sample size, where there were substantial differences in sample variance, differences between group means were evaluated using a non-parametric test (Mann-Whitney U).

Pooling across twelve subjects, there was no significant difference between Pre- and Post-Treatment SSS scores (Pre mean = 2.73 [.68]; Post mean = 2.82 [.88], NS).
No significant differences in this measure were seen within either the REM condition (Pre mean = 2.92 [.64]; Post mean = 2.92 [1.38] NS) or the S2 condition (Pre mean = 2.59 [.71]; Post mean = 2.74 [.36] NS).

Finally, the two groups did not differ with respect to subjective sleepiness on baseline testing, (REM Pre mean = 2.92 [.64]; S2 Pre mean = 2.59 [.71] NS) post-treatment (REM Post mean = 2.92 [1.38]; S2 Post mean = 2.74 [.36] NS) or in terms of pre/post differences (REM Pre/Post mean = 0.00 [.78]; S2 Pre/Post mean= -0.15 [.91] NS).

D. Pooled Group Correlations

Several significant correlations were obtained when subjects were pooled across both conditions. These associations may be interpreted as relating to the effects of nonspecific (with regard to REM or NREM sleep) nocturnal awakenings, rather than to selective sleep stage interruptions.

There was a trend towards inverse association between the number of forced awakenings on the second awakening night and mean sleep latency during the day immediately following ($r = -.496$, $p<.10$). (Scatterplots of all correlations having p values less than .10, with linear regression lines depicted, are presented in Appendix D).

Age was inversely correlated with change in sleep latency following two nights of awakenings, with older subjects tending towards greater decrements in mean sleep latency ($r = -.609$, $p<.05$).

A second significant correlation involving changes in sleep latency, that with initial sleepiness, ($r =-.687$, $p<.01$) is most likely a statistical artifact related to the phenomenon of regression to the mean.

Two other significant correlations involving baseline sleepiness were obtained from the pooled groups. Baseline sleep latency was inversely associated with total sleep time across the two awakening nights ($r= -.538$, $p<.05$) Subjects who were sleepier to begin with tended to accumulate relatively large amounts of sleep despite the awakenings. Perhaps initial sleepiness "innoculated" subjects against the disruptive effects of forced awakenings.

In addition, basal sleep latency was directly correlated with post-treatment sleep latency ($r = +.636$, $p<.01$). Subjects who were relatively sleep to begin with also tended

to be sleepier following two nights of disturbed sleep.

It is interesting to note that total recording time, which was matched to subject's habitual bedtimes and thus may have been expected to correlate with basal sleepiness levels, in fact did not do so ($r = -.226$, NS).

E. REM Group Correlations

Table 11 presents the correlation matrix for selected subject, sleep architecture and sleepiness variables in the REM awakening group. Within group correlations are based on a small sample size (N=8), and therefore are to be interpreted with caution, pending replication. Two-tailed t tests were employed in all cases except those involving the relationship of REM recurrence to basal sleepiness, as these were planned attempts to replicate earlier findings.

A main objective of the present study was to confirm the finding of a strong inverse correlation between the number of REM awakenings necessary to achieve deprivation across a night and basal sleep latency (Adler, 1984). In the present study, there was a trend towards confirmation of this relationship on the first awakening night, ($r = -.579$, $p < .10$. On the second awakening night, this association was strongly in evidence ($r = -.848$, $p < .01$).

The observed association between basal sleepiness and REM recurrence during deprivation was not an artifact of bed-

time length. When an index of experimental awakenings per hour of sleep or hour of recording time was substituted into the correlation equation in place of the raw number of awakenings, the relationships were weakened somewhat but still achieved statisical significance on the second night (Night one: [EA(TST)/MSLT] r= -.379, NS; [EA(TRgT)/MSLT] r= -.512, NS; Night two: [EA(TST)/MSLT] r= -.736, $p<.05$; [EA(TRgT)/MSLT] r= -.753, $p<.05$).

SOREMP probability, defined as the percentage of instances when a REM-onset awakening was followed by REM sleep within fifteen minutes, also displayed a significant inverse correlation with basal MSLT sleepiness, both on night one (r= -.667, $p<.05$) and night two (r= -.808, $p<.01$).

A trend towards inverse association was observed between the age of subjects in the REM group and the amount of total sleep time they accumulated over the two awakening nights (r= -.668, $p<.10$). This association was not due to any correlation between age and total recording time (r= +.033, NS) or to a greater number of awakenings in older subjects (in fact these subjects tended to have [nonsignificantly] fewer awakenings). The decreased total sleep time may reflect a greater disruptive potential of REM awakenings in older subjects. An alternate interpretation is that the greater age variance found in the REM awakening group allowed a nonspecific correlation between age and the sleep-disruptive effects of awakenings to be expressed. No

analogous association was seen in the S2 awakening group (r= +.147, NS).

A significant correlation was observed between REM awakenings necessary on the first and second nights (r=+.638, p<.05, one-tailed test).

Correlations with psychometric variables

Neither the State-Trait Anxiety Inventory (STAI) or the Manifest Anxiety Scale (MAS) yielded scores showing a signficant correlation with the number of REM-onset awakenings necessary to accomplish deprivation in the REM group over the two awakening nights ([STAI-State/EA] r = -.240, NS; [STAI-Trait/EA] r = -.332, NS; [MAS/EA] r = -.636, p<.15). Although the [MAS/EA] correlation was close to reaching significance, it should be noted that the present study is reporting an inverse correlation whereas Cartwright et al (1967) observed a direct association. The MAS/EA correlation is based on a sample size of seven, since one subject's MMPI was returned without this scale scored because of doubtful validity.

No significant correlation was observed between the awakening index and the Depression Scale of the MMPI (r= -.469, NS).

Field Dependence was assessed using Witkin's Embedded Figures Test (EFT). No significant association was observed between field dependence and the number of awakenings

necessary on the two experimental nights (r= -.191, NS). Gillin et al (1974) had reported a (nonsignificant) trend towards field independent subjects exhibiting a greater change in the number of REM awakenings necessary between REM deprivation nights one and two. In the present study, this association was not observed (r= .238, NS).

F. Stage 2 Group Correlations

Table 12 presents the correlation matrix for selected subject, sleep architecture and sleepiness variables in the S2 awakening group.

Total recording time, which was matched to subjects' habitual bedtimes as determined by a two week sleep log, was correlated with sleep time accumulated over the two awakening nights only for the S2 awakening group (REM r= +.223, NS; S2 r= +.780, $p<.05$).

A very strong association was observed within the S2 awakening group between the number of experimental awakenings on the second night and average REM percent over the two nights (r= +.897, $p<.005$). This result is probably an artifact of the awakening procedure: On the second night, more control awakenings were performed than on the first night (to match the larger number of REM awakenings made on the second night). It was generally difficult to fit all these S2 awakenings within each subject's prescribed recording time--hence S2 subjects experienced, on average,

approximately two less awakenings on the second night than did their REM counterparts. Those S2 subjects who had longer sleep periods were likely to undergo more scheduled awakenings ($r = +.804$, $p < .05$). These longer sleepers exhibited a trend towards greater REM percentages ($r = +.678$, $p < .10$; Verdone, 1968). This combination of factors may help account for the very high association observed.

G. REM Period Statistics

As REM period (REMP) comparisons between experimental groups were essentially similar regardless of which termination rule was adopted, data will be presented using the "15 minute rule", i.e., the end of a REMP was defined as the epoch of REM sleep followed by fifteen minutes of any non-REM state, including wakefulness.

There was no difference between groups in terms of REM latency on the first awakening night (REM mean = 63.9 min (29.8); S2 mean = 73.9 min (18.7), NS). On the second night, there was a trend towards shorter latencies in the REM deprived group (REM mean = 55.1 min (20.2); S2 mean = 71.4 (14.9), $p < .10$).

Subjects undergoing REM deprivation had significantly more REMPs than Stage 2 awakening subjects both on the first awakening night (REM mean = 7.4 (1.5); S2 mean = 3.8 (1.2), $p < .005$) and on the second night (REM mean = 7.4 (1.3); S2

mean = 3.9 (1.0), $p<.005$). REM group subjects had significantly shorter REMPs on the first but not the second deprivation night (Night one: REM mean = 11.9 min (6.3); S2 mean = 24.7 min (10.9), $p<.05$; Night two: REM mean = 20.6 min (11.6); S2 mean = 25.9 min (10.1), NS). REMP intervals were significantly shorter in the REM-deprived group on both nights (Night one: REM mean = 59.1 min (17.1); S2 mean = 113.8 min (18.8); $p<.005$; Night two: REM mean = 53.4 min (8.3); S2 mean = 110.7 min (19.6), $p<.005$).

No significant correlations were observed between first night REMP variables and basal MSLT sleep latency within the REM awakening group. The correlation between the mean baseline MSLT latency and the mean REMP duration on night one was -.460 (NS). However, on the second awakening night, the average REMP duration was highly associated with basal MSLT sleep latency ($r = -.912$, $p<.005$). Relatively sleepy subjects tended to have longer REMPs.

H. Multiple Regression: Prediction to Changes in Sleep Latency

The finding that several variables were strongly correlated with changes in sleepiness following two nights of awakenings, in conjunction with the acceptance of the null hypothesis regarding the differential effects of selective sleep-stage awakenings on changes in sleepiness, led to the decision to employ multiple regression analysis to deter-

mine whether some combination of variables would account for significantly more of the variance in sleepiness changes than the highest simple correlation.

This analysis is clearly to be considered exploratory in nature. The small sample size of 16, together with the relatively large number of variables considered, yield a sample size/variable ratio which is insufficient to warrant use of the technique on other than an exploratory basis.

A stepwise multiple regression was performed predicting to changes in sleep latency. The following variables were entered into the analysis: Age, Sex, Total Recording Time, Total Sleep Time, and Percentages of Stage 1, Stage 2, Stage 3, Stage 4, Stages 3+4 and REM sleep. The number of experimental awakenings was not included because of the difficulties inherent in interpreting the contribution of a variable which was experimentally manipulated in some subjects and free to vary in others. Variables were entered into the regression equation only if their partial correlations remained significant at the .05 level.

This procedure yielded a two step regression equation with Age and Total Recording Time as the two variables entered. A summary of the analysis is given in Table C.

The multiple correlation of 0.7333 can be seen to account for approximately half the variance observed in the change in sleep latency. Older subjects who had habitually short

bedtimes (and thus short total recording times in this study) were likely to exhibit greater drops in sleep latency following two nights of awakenings.

Inspection of variables remaining outside of the regression equation after the first variable, age, had been entered revealed that Stage 4 percent had very narrowly missed entering the equation ahead of total recording time (TRT partial r = .51527; S4 percent partial r = -.51489). A second analysis performed while excluding TRT yielded a nearly identical multiple correlation of 0.7331. Thus, older subjects who accumulated relatively high percentages of Stage 4 sleep on the two awakening nights tended to have more precipitous drops in sleep latency.

Non-parametric comparisons

Inspection of the data suggested that males tended towards larger decreases in sleep latency following two nights of awakenings than did females. Since only one quarter of all subjects were male, this possibility was evaluated using a non-parametric comparison, the Mann-Whitney U Test. Pooling subjects across both groups, there was no significant difference in the the ranks assigned to sleepiness changes for males and females (U=17, N1=4, N2=12, NS). There was a trend towards confirmation of this hypothesis in the S2 group (U=1, N1=3, N2=5, $p<.10$).

Table 1

Subject Variables

Subject	Sex	Age
REM Awakening Group		
JN	F	31 years
RS	F	24
KS	M	24
CK	F	27
JD	F	21
MC	F	19
NM	F	37
MA	F	32
MEAN (S.D.)	7F, 1M	26.9 (6.1)
Stage 2 Awakening Group		
MN	F	21
SH	M	31
FN	M	28
JK	M	21
RT	F	21
CM	F	24
GW	F	22
VS	F	28
MEAN (S.D.)	5F, 3M	24.2 (4.0)

Table 2

Baseline Bedtime Length and Subjective Sleepiness

Subject	Bedtime	SSS
REM Awakening Group		
JN	480 min	1.80
RS	435	2.10
KS	420	2.82
CK	435	1.94
JD	420	2.26
MC	530	2.69
NM	540	1.21
MA	420	2.51
MEAN	460.0	2.17
(S.D.)	(50.4)	(.53)
Stage 2 Awakening Group		
MN	410	1.79
SH	405	2.97
FN	470	1.42
JK	405	2.05
RT	485	2.44
CM	540	1.48
GW	427	1.71
VS	547	2.09
MEAN	461.1	1.99
(S.D.)	(59.0)	(.52)
REM vs. Stage 2	NS	NS

Table 3

Psychometric Data

Subject	EFT	STAI-1	STAI-2	MMPI-DS	MAS
REM Awakening Group					
JN	542 sec	27	25	42	44
RS	788	39	43	67	80
KS	695	43	49	94	71
CK	321	37	34	49	42
JD	1078	31	30	40	45
MC	496	23	27	46	40
NM	1337	24	31	55	51
MA	1268	40	49	73	--
MEAN	815.6	33.0	36.0	58.2	53.3
(S.D.)	(374.6)	(7.8)	(9.7)	(18.6)	(15.8)
Stage 2 Awakening Group					
MN	537	34	38	53	54
SH	329	33	43	68	53
FN	406	22	34	51	37
JK	899	30	34	58	40
RT	263	32	36	42	45
CM	637	24	24	49	37
GW	750	33	43	47	56
VS	340	30	29	55	41
MEAN	520.1	29.8	35.1	52.9	45.4
(S.D.)	(226.3)	(4.4)	(6.5)	(7.8)	(7.9)
REM vs. Stage 2					
	$p<.10$	NS	NS	NS	NS

Table 4

Pre/Post Sleepiness Scores

Subject	Pre SSS	Pre MSLT	Post SSS	Post MSLT
REM Awakening Group				
JN	--	7.1 minutes	--	6.4 min
RS	--	23.9	--	13.4
KS	3.2	13.7	4.8	13.4
CK	--	21.4	--	17.5
JD	3.4	5.2	3.8	8.7
MC	3.0	10.3	2.0	13.2
NM	1.8	25.0	1.4	19.0
MA	3.2	17.4	2.6	5.1
MEAN	2.92	15.5	2.92	12.1
(S.D.)	(.64)	(7.6)	(1.38)	(5.0)
STAGE 2 Awakening Group				
MN	--	11.9	--	9.2
SH	2.6	14.2	2.2	3.6
FN	1.6	21.7	3.0	11.7
JK	2.5	15.9	2.8	12.8
RT	3.4	10.8	2.6	11.6
CM	2.4	5.2	3.2	3.6
GW	3.6	10.5	2.4	7.8
VS	2.0	12.5	3.0	7.0
MEAN	2.59	12.8	2.74	8.4
(S.D.)	(.71)	(4.8)	(.36)	(3.6)
Pooled Groups				
MEAN	2.73	14.2	2.82	10.2
(S.D.)	(.68)	(6.3)	(.88)	(4.6)
REM vs. Stage 2	NS	NS	NS	NS

Table 5

Sleepiness Change Scores

Subject	SSS	MSLT
REM Awakening Group		
JN	--	-0.7
RS	--	-10.5
KS	+1.6	-0.3
CK	--	-3.9
JD	+0.4	+3.5
MC	-1.0	+2.9
NM	-0.4	-6.0
MA	-0.6	-12.3
MEAN	0.0	-3.4
(S.D.)	(.78)	(5.8)
Stage 2 Awakening Group		
MN	--	-2.7
SH	-0.4	-10.6
FN	+1.4	-10.0
JK	+0.3	-3.1
RT	-0.8	+0.8
CM	+0.8	-1.6
GW	-1.2	-2.7
VS	+1.0	-5.5
MEAN	+0.16	-4.4
(S.D.)	(.92)	(4.0)
REM vs. Stage 2	NS	NS

Table 6

Awakening Night Parameters
(Average of Two Nights)

Subj.	TRgT	TST	1%	2%	3%	4%	REM%
REM Awakening Group							
JN	476 min	338 min	13.6	64.8	13.1	1.6	6.8
RS	433	339	11.4	64.9	6.9	13.4	3.5
KS	416	343	7.8	58.0	13.4	12.4	8.4
CK	435	266	7.7	62.8	15.1	5.5	8.9
JD	480	352	16.2	58.6	5.5	0.6	19.1
MC	529	396	11.7	53.3	8.2	17.4	9.4
NM	532	288	12.0	61.4	17.2	1.6	7.9
MA	408	327	5.9	48.1	20.4	15.3	10.3
MEAN	463.6	331.1	10.8	59.0	12.5	8.5	9.3
(S.D.)	(48.5)	(39.7)	(3.4)	(5.9)	(5.2)	(6.9)	(4.5)
Stage 2 Awakening Group							
MN	412	225	17.1	41.8	5.5	18.8	16.5
SH	404	333	7.8	41.8	8.6	13.6	28.2
FN	464	288	11.8	60.7	7.3	11.3	12.1
JK	398	330	8.2	50.2	9.9	5.8	25.9
RT	481	386	9.4	48.1	6.7	17.2	18.6
CM	538	461	5.9	41.9	8.5	8.3	35.3
GW	423	339	7.9	38.2	5.0	23.8	25.0
VS	548	428	12.3	49.2	6.0	8.4	24.1
MEAN	458.5	348.8	10.0	46.5	7.2	13.4	23.2
(S.D.)	(59.7)	(75.6)	(3.6)	(7.2)	(1.7)	(6.2)	(7.3)
REM vs. Stage 2							
	NS	NS	NS	$p<.005$	$p<.05$	NS	$p<.001$

Table 7

Night One Awakening Data

Subj.	EA	EA/TRGT	EA/TST	SOREMP%
REM Awakening Group				
JN	13	1.66	2.38	.38
RS	10	1.42	1.84	.30
KS	9	1.29	1.57	.22
CK	12	1.65	3.08	.25
JD	15	1.85	2.52	.53
MC	20	2.28	3.09	.65
NM	5	0.57	1.22	.20
MA	16	2.36	2.83	.44
MEAN	12.4	1.64	2.32	.37
(S.D.)	(4.6)	(.57)	(.70)	(.16)
Stage 2 Awakening Group				
MN	10	1.47	2.68	
SH	10	1.49	1.78	
FN	9	1.13	2.17	
JK	10	1.53	1.83	
RT	14	1.51	1.83	
CM	13	1.57	1.82	
GW	14	2.12	2.70	
VS	14	1.53	2.02	
MEAN	11.8	1.54	2.10	
(S.D.)	(2.2)	(.27)	(.38)	
REM vs. Stage 2				
	NS	NS	NS	

Table 8

Night Two Awakening Data

Subj.	EA	EA/TRgT	EA/TST	SOREMP%
REM Awakening Group				
JN	25	3.09	4.31	.76
RS	11	1.51	1.88	.36
KS	13	1.85	2.29	.38
CK	16	2.20	3.23	.50
JD	25	3.16	4.33	.80
MC	22	2.44	3.26	.68
NM	10	1.11	1.83	.20
MA	22	3.23	4.18	.73
MEAN	18.0	2.32	3.16	.55
(S.D.)	(6.2)	(.80)	(1.06)	(.22)
Stage 2 Awakening Group				
MN	14	2.09	3.07	
SH	16	2.37	2.91	
FN	15	2.00	2.74	
JK	16	2.37	2.89	
RT	15	1.89	2.43	
CM	18	2.11	2.48	
GW	16	2.41	2.99	
VS	16	1.74	2.18	
MEAN	15.8	2.12	2.82	
(S.D.)	(1.2)	(.25)	(.54)	
REM vs. Stage 2				
	NS	NS	NS	

Table 9

Night One REM Period Data

Subj.	REM Lat	REMP	REMP Dur	REMP Int
REM Awakening Group				
JN	57.0 min	9	8.8 min	50.6 min
RS	72.0	7	6.7	54.8
KS	42	7	9.2	55.0
CK	64.0	9	7.9	43.1
JD	56.0	6	15.2	65.5
MC	131.5	7	26.1	64.1
NM	55.0	5	11.2	96.1
MA	33.5	9	10.2	43.8
MEAN	63.9	7.4	11.9	59.1
(S.D.)	(29.8)	(1.5)	(6.3)	(17.1)
Stage 2 Awakening Group				
MN	79.0	2	22.5	153.0
SH	78.0	3	38.5	110.0
FN	65.5	4	7.1	113.3
JK	96.5	3	30.3	98.5
RT	102.5	5	17.0	92.2
CM	61.0	5	36.1	103.6
GW	60.0	3	30.3	124.5
VS	48.5	5	15.8	115.6
MEAN	73.9	3.8	24.7	113.9
(S.D.)	(18.7)	(1.1)	(10.9)	(18.8)
REM vs. Stage 2				
	NS	$p<.005$	$p<.05$	$p<.005$

Table 10

Night Two REM Period Data

Subj.	REM Lat	REMP	REMP Dur	REMP Int
REM Awakening Group				
JN	45.5 min	6	35.7 min	52.9 min
RS	60.5	7	9.6	61.8
KS	53.0	7	13.6	49.2
CK	60.0	8	14.1	50.5
JD	34.0	6	39.1	69.5
MC	55.5	8	23.1	66.1
NM	98.0	10	8.2	47.3
MA	34.5	7	21.4	53.8
MEAN	55.1	7.4	20.6	53.4
(S.D)	(20.2)	(1.3)	(11.6)	(8.3)
Stage 2 Awakening Group				
MN	60.0	3	16.3	131.0
SH	89.0	3	28.8	141.5
FN	58.5	4	14.6	104.0
JK	88.0	3	34.2	100.5
RT	86.5	5	12.8	79.2
CM	73.5	5	35.5	110.4
GW	63.5	3	38.0	119.0
VS	52.0	5	27.2	100.1
MEAN	71.4	3.9	25.9	110.7
(S.D.)	(14.9)	(1.0)	(10.0)	(19.6)
REM vs. Stage 2				
	$p<.10$	$p<.005$	NS	$p<.005$

Table 11

REM Group Correlation Matrix

	TRgT	TST	EA1	EA2	1%	2%	3+4%	REM%	Lat1	Lat2	dLat
Age	+.03	-.67 °	-.59	-.26	-.23	+.10	+.18	-.31	+.48	+.07	-.56
TRgT	xx	+.22	+.05	+.10	+.64 °	+.11	-.40	+.14	-.16	+.32	+.48
TST		xx	+.63 °	+.46	+.35	-.37	+.02	+.18	-.65 °	-.46	+.45
EA1			xx	+.76 *	+.03	-.61	+.19	+.40	-.58	-.52	+.31
EA2				xx	+.33	-.36	-.19	+.58	-.85 **	-.78 *	+.44
1%					xx	+.43	-.87 **	+.37	-.49	-.09	+.56
2%						xx	-.65 °	-.37	+.19	+.40	+.09
3+4%							xx	-.41	+.37	-.08	-.55
REM%								xx	-.59	-.30	+.52
Lat1									xx	+.64 °	-.76 *
Lat2										xx	+.02

° = p<.10
* = p<.05
** = p<.01

Table 12

Stage 2 Group Correlation Matrix

	TRqT	TST	EA1	EA2	1%	2%	3+4%	REM%	Lat1	Lat2	dLat
Age	+.19	+.14	-.25	+.19	-.09	+.25	-.32	+.08	+.36	-.50	-.88 **
TRqT	xx	+.78 *	+.59	+.46	-.10	+.18	-.50	+.23	-.39	-.29	+.20
TST		xx	+.71 *	+.80 *	-.64 o	-.12	-.43	+.68 o	-.57	-.44	+.29
EA1			xx	+.36	-.30	-.40	+.16	+.36	-.69 o	-.24	+.61
EA2				xx	-.81 *	-.27	-.40	+.90 **	-.52	-.61	+.08
1%					xx	+.21	+.08	-.73 *	+.32	+.34	-.07
2%						xx	-.53	-.59	+.78 *	+.62	-.38
3+4%							xx	-.23	-.17	-.00	+.20
REM%								xx	-.68 o	-.71 *	+.17
Lat1									xx	+.57	-.68 o
Lat2										xx	+.22

o = p<.10
* = p<.05
** = p<.01

Table 13

Multiple Regression Summary Tables

Dependent Variable = dLat
(Change in MSLT Latency)

Var.	Mult.R	R Sq.	RSQ Chng.	Simple R	B	Beta
Age	0.609	0.371	0.371	-0.609	-0.625	-0.654
TRgT	0.734	0.538	0.167	0.340	0.382	0.411
Constant						-5.467

Disallowing TRgT From the Regression Equation

Age	0.609	0.371	0.371	-0.609	-0.757	-0.791
4%	0.733	00.538	0.167	-0.125	-0.321	-0.447
Constant						19.037

CHAPTER 4. DISCUSSION

This study assessed the effects of selective sleep-stage awakenings on changes in sleep tendency, and found those changes to be comparable whether the awakenings were made from REM or Stage 2 sleep. It is therefore in accord with earlier studies which had compared the effects of REM and Stage 4 awakenings on performance measures and found no appreciable differences (Agnew et al, 1967; Lubin et al, 1974).

That sleepiness changes were comparable between groups reinforces and broadens the suggestion of Lubin et al that sleep composition may not be as important as sleep quantity in terms of subsequent functioning. It suggests that nocturnal awakenings produce subsequent sleepiness regardless of whether REM or NREM processes are being interrupted.

The number of experimental awakenings were not controlled in the earlier studies, since maximal deprivation of each target stage was desired. Circadian factors prevented controlling for time of night of the awakenings. Both of these factors would seem to be especially pertinent to changes in sleep tendency, and both were controlled in the present study. Finally, whereas sleepiness changes were inferred from performance measures in the earlier studies,

they were directly assessed in the present study using a widely accepted and validated measure.

It is surprising that subjective sleepiness as measured by the SSS did not register any effect of repeated nocturnal awakenings (regardless of experimental condition) and loss of total sleep time. Possibly, the fact that subjects had previously recorded SSS scores hourly during waking periods over a span of two weeks had caused a loss of sensitivity in this measure: These "veteran" subjects did not appear to consider the entire scale carefully before making their choices in the laboratory. Instead, they were often heard to impulsively exclaim "I'm a two" or some other score just as the experimenter approached for a rating.

The nocturnal sleep achieved by subjects in the two conditions was comparable in terms of total sleep time, sleep efficiency, number of forced awakenings and percentage of time spent in Stage 1 sleep. In addition to the differences introduced by the manipulation (i.e. significant differences in amount of REM and Stage 2 sleep between the two groups) subjects in the Stage 2 awakening condition experienced more intense delta wave sleep than did their REM-awakening counterparts. They had significantly less Stage 3 sleep, and (nonsignificantly) more stage 4. As Dement and Greenberg noted (1966) there may be a quantitative relationship between the NREM stages so that a relatively small amount of Stage 4 sleep can "compensate"

for larger amounts of lost Stage 2.

Considering all subjects together, an average of just under thirty three-minute forced awakenings unevenly distributed over two nights caused a reduction in MSLT sleep latency of about four minutes. It would be most appropriate to compare this change with the results of a series of studies which used the MSLT to measure changes in sleepiness produced by a range of manipulations, from sleep extension through partial sleep restriction to total sleep deprivation (Carskadon and Dement, 1982). Unfortunately, this comparison is hampered because of varying initial conditions between studies.

To cite the most extreme example, the four minute reduction in average latency produced in the present study appears comparable to the 4.9 minute reduction reported by Carskadon and Dement following two nights of total sleep deprivation (1979). However, the average baseline latency obtained in that study was 5.4 minutes, which would today be considered near the borderline of the "pathologically" sleepy range (Reynolds et al, 1982). Thus after total sleep deprivation sleep latencies were essentially nil. Carskadon and Dement noted that several of their subjects reported sleeping less than usual on the ten days prior to their experiment.

The baseline value obtained in the present study is by contrast slightly less sleepy than the figures reported for

normal subjects in standardization studies (Richardson et al, 1978 [11.2 minutes]; Mitler, 1982 [13.4 minutes]. This is probably an artifact due to the allowance in the present study of 25 rather than 20 minutes in which subjects could fall asleep. Furthermore, on those occasions when subjects did not sleep, a greater than usual upwards bias of the mean was introduced.

Similarly, departures from normal routine in Carskadon and Dement's study of the effects of 5 hour bedtimes (1981) make comparison with the present study problematic. In order to assure minimal sleep debt going into the restriction period, Carskadon and Dement had their subjects spend ten hours in bed for the three baseline nights. This intervention was at least partially responsible for the average basal sleep latency of 16.7 minutes obtained in that study. A second factor which probably contributed to the long latencies obtained was the administration of six rather than five naps every day. The sixth nap, given at 1930, extended the testing into the evening alertness "peak" of the circadian sleepiness cycle.

The four minute decrement observed after two nights in the present study (each averaging fifteen interruptions and 5 2/3 hours Total Sleep Time) amounted to a reduction in average latency of about thirty percent, comparable to that reported in the Carskadon and Dement study after two nights of 5 hour bedtimes. In absolute terms, the ten minute

average latency obtained in the present study was not reached until after the fourth 5 hour restriction night.

These comparisons suggest that the initial sleepiness level of a subject may be critical in determining what effect a given manipulation will have. As Ellman has suggested (personal communication) it would be of interest to determine the means necessary to alter sleepiness from various MSLT baseline values. It is possible, for example, that the MSLT does not provide an interval scale of sleepiness. In the case of the present study, this would imply that a decrease of approximately four minutes from an initial sleep latency of 15.5 minutes (the REM group average) may have quite a different meaning than a similar decrease, in either absolute or percentage terms, from a 12.8 value (the initial S2 group value).

It is not yet established as to whether MSLT intervals correspond to equal increments of sleep loss. Reviewing their own data, Carskadon and Dement (1982) were impressed with the abrupt drop in MSLT latency exhibited by subjects who underwent two nights of 4 hour (as opposed to 5 hour) sleep restriction. They suggested that subjects may in fact become much more sleepy at this cutoff, just as performance begins to suffer in earnest between four and three hours of total sleep time. The MSLT, then, would simply be reflecting this abrupt change. In other words, these authors endeavored to retain the interval properties of the MSLT

scale.

Such complicated reasoning may not be necessary. As noted above, the MSLT latencies in the 5 hour sleep restriction study may have been inflated. If the point corresponding to the results of this study is adjusted downwards, the graph depicting the results, in terms of post-treatment sleep latencies, of all the manipulations shows a clearly linear relationship between bedtime length and subsequent MSLT latency.

Further evidence suggesting a linear relationship between sleep loss and MSLT latencies is provided by the 5 hour sleep restriction study alone, which involved MSLT testing after each of the seven restriction nights. Each treatment night resulted in approximately the same amount of decrement in MSLT latency.

Apart from sleep loss, subjects in the present study experienced two related yet distinct effects of the manipulation: Decreased sleep continuity and lowered sleep efficiency. The awakenings yielded an average sleep efficiency of about 75%, that is, subjects were awake for one quarter of the total time they spent in bed. This is in contrast to the nearly 100% sleep efficiency achieved by subjects undergoing sleep restriction to 5 hours.

Given that subjects in the present study accumulated, on average, nearly one more hour of sleep each night (340 vs.

approximately 290 minutes) the question arises as to whether this advantage was offset solely by the higher initial alertness of the subjects in Carskadon and Dement's study, or whether the decreased sleep continuity and efficiency endured by subjects in the present study also played a role.

This question is of primary importance to understanding the daytime consequences of insomnia, and could be addressed by comparing results of these two studies with those of a third, in which subjects would be allowed five hours of relatively consolidated sleep while remaining in bed for nearly seven hours, so that a 75% sleep efficiency is achieved.

In the sleep fragmentation pilot study of Stepanski et al (1984) two nights of disruption produced by presenting tones every 5.5 minutes on average resulted in a decrease in average MSLT latency from 13.8 to 8.2 minutes (N=5). It is important to note that not only was baseline sleep stage architecture preserved on the second experimental night (at least in terms of sleep stage percentages) total sleep time was (nonsignificantly) increased. This preliminary finding suggested that sleep continuity may be a more potent factor than either sleep quality or sleep quantity, insofar as maintenance of alertness is concerned.

This was in direct contrast to the finding of Lubin et al (1974) that 50-100 arousals per night did not produce a

significantly different rate of recovery of performance capabililty, as compared to undisturbed sleep, following total sleep deprivation.

The increase in sleep tendency observed in the Stepanski et al study was only slightly more pronounced than that produced in the present study by awakenings occurring, on average, every half an hour. A second major difference between the two protocols may account for the similar results: Whereas transient arousals were produced in the earlier study, three minute forced awakenings, replete with verbal interaction, were the rule in the present investigation. This underscores the importance of a second quantitative factor (apart from frequency of arousals) in sleep fragmentation studies, namely, the degree of arousal produced by the manipulation. A relatively high degree of sleep continuity is present in "fragmentation" studies, when compared to the disruption of sleep entailed by more complete and prolonged awakenings.

The hypothesis that sleep continuity plays a critical role in maintaining daytime functioning received support from the sleep fragmentation study of Bonnet (1985), when arousals after every minute of sleep resulted in performance decrements approximating those seen following a night of total sleep deprivation, even though only about one hour of total sleep time was lost. Sleep stage architecture as well as sleep continuity was profoundly altered by this

manipulation .

Bonnet cited the negative results produced by earlier selective sleep stage deprivation studies to argue that the dramatic effects seen in his investigation should be attributed to fragmentation, not to shifts in sleep architecture. The present study supports Bonnet's interpretation.

Whereas earlier investigations had noted anecdotally that selective sleep deprivation, whether of REM or NREM sleep, leads to increased sleepiness, there was also evidence, primarily from animal studies, which suggested that REM deprivation might lead to increased activation and thus longer, not shorter MSLT latencies. The present study cannot lend support to either position, since the decrease in MSLT latency within the REM awakening condition failed to reach statistical significance.

Considering this result in conjunction with the findings of Adler (1984), who reported a (nonsignificant) increase in MSLT latency following one night of REM deprivation in normals, suggests that REM deprivation is not a particularly potent producer of sleepiness, if it is one indeed.

How then, may this be reconciled with the finding of Carskadon and Dement (1977) which reported that subjective sleepiness tended to be greater prior to naps containing relatively large amounts of REM sleep? In general, the

present study does not directly contradict this claim, because the state produced by REM deprivation is not equivalent to that produced by the "ninety minute day", where REM sleep was allowed expression, albeit in allotments of up to thirty minutes.

The "ninety minute day" study detected an association between subjective sleepiness and a pre-REM waking state, a state which may have more to do with NREM "satiation" or the influence of an underlying circadian oscillator than with REM deprivation. Carskadon and Dement avoid attributing the increased sleepiness to a REM process altogether, likening it instead to "a prodromal state for REM sleep in much the same manner that SWS or NREM sleep normally precedes REM sleep in nocturnal sleep periods" (p.132).

To ascertain whether objective sleep tendency does in fact increase prior to REM onset in normal subjects undergoing a less disruptive sleep wake manipulation than a "ninety minute day", one might analyze sleep latency following a series of awakenings systematically spaced near the beginning, middle and end of NREM periods, such as was performed by Spielman et al (1983) in order to ascertain SOREMP probability following these awakenings.

The lack of a clear link between reduced levels of REM sleep and changes in sleepiness is in contrast to the confirmation of a direct association between REM recurrence on deprivation nights and pre-existing sleepiness levels.

Increased REM recurrence has been recognized as a consequence of REM deprivation since Dement's pioneering study. The most parsimonious explanation of the linkage between REM recurrence and basal sleepiness is that those subjects who are chronically sleep deprived because of schedule demands show a tendency both towards increased sleepiness and more insistent REM. In this view, the sleepiness is the result of non-specific sleep loss whereas the insistent REM is due to partial REM deprivation--both phenomena would be eliminated if adequate sleep were obtained.

While this REM deprivation "state" model can account for the observed association, there is some evidence which speaks for the presence of an additional factor, that of individual differences in REM-related characteristics, or REM traits.

A REM "trait" model posits the existence of a cluster of relatively stable REM-related characteristics, including the ease with which REM can be triggered and its inertia once present, (Spielman et al, ms. submitted) the ability to "gate" the expression of REM processes so that they occur at appropriate times in the circadian cycle (Vogel, 1968; Schulz and Tetzlaff, 1982; Adler, 1984) and perhaps, basal sleepiness levels. As Adler emphasized, narcoleptics provide an example combining sleepiness and highly recurrent REM during deprivation procedures in the absence of demonstrated chronic REM deprivation.

In this regard, it is interesting that a sizable minority of normal individuals share the DR-2 HLA antigen which has been demonstrated to occur in virtually all narcoleptics (Honda, 1985). Perhaps these normals are also characterized by relatively high sleep tendency and REM insistence.

Sleepy normals resembled narcoleptics not only with respect to the higher number of REM-onset awakenings needed to accomplish REM deprivation, but also in the greater average duration of their REM periods.

As Pivik and Foulkes (1966) demonstrated, normals undergoing REM deprivation generally respond to this manipulation with shortened intervening NREM periods between more frequent REM onsets. Fiss and Ellman (1973) showed that when the REM sleep of normals is interrupted (as opposed to deprived) recovery night REM periods tend to be of shorter duration than those present on baseline nights. Adler (1984) noted this same response occurring on the deprivation night itself--that is, normal subjects will return to NREM sleep after a relatively short span of (thwarted) attempts to enter REM sleep.

Adler observed that narcoleptics lack this response. Rather, they tend to have the same number of REM periods during deprivation procedures as on baseline, with an increased average period duration.

Some normal subjects tended to show this narcoleptic-like

response to REM deprivation in the present study. Initially (i.e. on the first deprivation night) they exhibited shorter REM periods than those subjects who were awakened from NREM stage 2 sleep. Under a strong enough challenge to their REM systems, they began to lose the ability to disengage from REM sleep. The observation of a high inverse correlation (-.91) between average REM period duration on the second deprivation night and basal sleep tendency raises the possibility that impairment of the ability to defer REM sleep until later in the night may in fact lead to increased sleepiness. This view was advanced by Adler with respect to the pathological sleepiness seen in narcoleptics.

Gillin et al (1974) posited a mechanism whereby inability to delay the expression of REM might lead to increased sleepiness. These investigators noted that REM rebound following REM deprivation of their control subjects was not merely a function of increased total sleep time, as would be consistent with the views of Verdone (1968) and Agnew and Webb (1970) but was in fact an independent phenomenon which could serve as the basis for sleep extension -- "REM sleep may function to preserve sleep" (p. 660).

The hypothesis of individual differences in the ability of normal subjects to regulate sleep length through REM deferral should be distinguished from the view of Hartmann et al (1971, 1973) that extended habitual bedtimes and greater

amounts of REM sleep are responses to differing requirements based on lifestyle.

This hypothesis provides a bridge between the "state" and "trait" formulations regarding REM sleep and sleepiness. Thus, not only will some individuals become partially sleep deprived because of greater external (social and environmental) pressures, but also because they have less ability to extend their sleep when schedules permit.

It may seem paradoxical to suggest that some sleepy people are unable to sleep. After all, their sleepiness is operationally defined through the MSLT as the tendency to fall asleep when given the opportunity. However, experience with insomniac populations has demonstrated that the ability to initiate sleep is quite distinct from the ability to maintain it, especially "at the margin", that is, after a minimally adequate quota of sleep has been accumulated.

It is here that Gillin's "sleep preservation" function for REM sleep may come into play. If REM has been prematurely expressed, the result may be intrusions of wakefulness rather than REM sleep in the early morning hours. This sequence is often cited in chronobiologically oriented discussions of depression. It is clear from the results of sleep fragmentation studies that this would produce sleepiness, even if normals, unlike depressives, are able to repeatedly resume sleeping.

The presence of a "REM trait" contribution towards sleepiness might be tested in the following manner. Normal subjects who exhibit high basal sleepiness scores and who require many REM-onset awakenings during deprivation could be entered into a sleep extension protocol. In general, MSLT sleep latency tends to be increased by such a procedure (Carskadon and Dement, 1979). The REM "trait" model would predict that high-awakening subjects would 1) have relative difficulty extending their sleep, and 2) still require relatively large numbers of REM onset awakenings during subsequent deprivation, just as narcoleptics do not deter their abnormal REM manifestations through ad libitum sleep.

This model would not predict that high-awakening subjects have shorter bedtimes. In fact, no association was noted in the present study between these two variables, whether considering the number of awakenings on the first night ($r = .048$, NS) or the second ($r = .096$, NS). The determinants of habitual bedtime remain largely unexplored. Rather, the concern here is with compensatory responses to alterations in bedtime and sleep length.

Cartwright et al, (1967) added personality characteristics to the REM trait "cluster", notably, an association between increased levels of anxiety and the requirement of high numbers of REM-onset awakenings to accomplish REM deprivation. The present study failed to replicate this finding.

Furthermore, no significant associations were observed between REM recurrence and measures of depression and field dependence. Evidently, measures which are more specific to the phenomenology of the REM state, such as the scale used by Weinstein (1981) to assess attitudes towards daydreaming, are necessary to ferret out personality differences with regard to REM deprivation response.

Given that REM recurrence is associated with sleepiness, either through the "state" mechanism of chronic partial sleep deprivation alone or additionally as part of a trait cluster, the finding that REM-onset awakenings were no more soporific than an equivalent number of Stage 2 awakenings suggests that there are several mechanisms which can produce sleepiness. As the work of Broughton (1982) and Pressman (1982,1983) has suggested, sleepiness may be a complex state, its composition at any one time the result of interaction between REM, NREM and wakefulness processes.

It is posssible that each of these mechanisms, when under challenge, becomes more easily triggered and less easily disengaged. Agnew et al (1967) noted that it took four times as many disruptions to deprive subjects of Stage 4 sleep as it did to accomplish REM deprivation. Similarly, an individual attempting to sleep more hours than usual or necessary can be conceptualized as challenging the wakefulness system, which responds in an analogous fashion: With more frequent nocturnal arousals and more persistent awak-

enings.

Viewed in this way, maintenance of an optimal sleepiness/alertness rhythm would require the temporal restraint of all three state-processes, each of which has the potential to fragment across the entrained twenty-four hour cycle, producing not only the behavioral manifestation of polyphasic sleep, but various fluctuating admixtures of sleepiness as well.

The ability to consolidate state processes has been noted to peak in prepubescent children (Carskadon et al, 1980) and thereafter decline with age (Feinberg et al, 1969, Webb and Swinburne, 1971; Miles and Dement, 1980). A surprising result of the present study is that the ability to tolerate nocturnal interruptions (in terms of maintaining subsequent daytime alertness) is substantially impaired between the onset of adulthood and the fourth decade of life. Age was in fact the single most important determinant of sleepiness following forced nocturnal awakenings, regardless of experimental condition.

Sleep fragmentation has been demonstrated to correlate with daytime sleep tendency both in middle aged (Roth et al, 1980) and elderly (Carskadon et al, 1982) individuals. In addition, the susceptibility of sleep to disruption by external stimuli has been shown to increase with age (Zepelin et al, 1982). The present study is the first to report that forced awakenings, as opposed to spontaneous arousals,

tend to produce greater daytime sleep tendency as the first decades of adulthood progress.

Most of the subjects in the present study were women of childbearing age (although only one had a child). All subjects, if they were to care for an infant, could expect to experience nights not too dissimilar from the interrupted sleep produced in this study, as opposed to fragmentation studies which model the disruption seen in sleep apnea. The implication is that older parents are at a clear disadvantage in terms of maintaining daytime alertness.

A second factor which apparently predisposes older subjects to increased sleepiness following nocturnal awakenings is habitually short bedtimes. It is important to note here that habitual bedtime was not related to basal sleepiness levels, only to changes in sleepiness. The "cost" of voluntary chronic sleep restriction (Webb and Agnew, 1975) if not apparent in daily functioning, may be uncovered by a challenge to the sleep systems as is afforded by repeated forced awakenings. Conversely, controlled extension of bedtime may be a rational approach to relieving the sleepiness encountered by those who face inevitable nocturnal awakenings.

The finding that older subjects who achieved relatively high amounts of Stage 4 sleep during the awakening procedure were also likely to exhibit greater increases in sleepiness subsequently is more difficult to interpret.

Presumably, accumulation of intense SWS would have served to compensate these subjects for lost NREM Stage 2 sleep and thus attenuated sleepiness. One possible explanation for this puzzling finding is that the increased Stage 4 represented a "same night effect" registering response to sleep interruption, just as Stage 4 is known to rebound upon recovery sleep. In this light, relatively high amounts of "same night" Stage 4 sleep may be analogous to "same night" REM effects (e.g. increased attempts to re-enter REM sleep during deprivation procedures)--that is, an indication of greater disruption as well as an attempted means of compensation.

Appendix A

Telephone Questionnaire

Identifying Data

Name: Date of Birth:

Sex: Phone:

Address:

Sleep Pattern:

Bedtime Length:

Disruptions/Night:

Naps/Week:

General Health and Drug Use

Currently under treatment by physician?

Taking medications which affect arousal level?

Regular user of caffeinated beverages?

If yes to either of obove two questions,

What type? Frequency?

Scheduling

If female, date of onset of last menstrual period:

Date log begins:

Date bedtime is stabilized:

Date laboratory run is scheduled:

Appendix B

Stanford Sleepiness Scale

Choose the number corresponding to your present level of alertness or sleepiness:

1 Alert. Wide Awake. Energetic.

2 Functioning at a high level, but not at a peak. Able to concentrate.

3 Awake, but not fully alert.

4 A little foggy, let down.

5 Foggy. Beginning to lose interest in remaining awake. Slowed down.

6 Sleepy. Prefer to be lying down. Woozy.

7 Cannot stay awake. Sleep onset soon.

x Asleep.

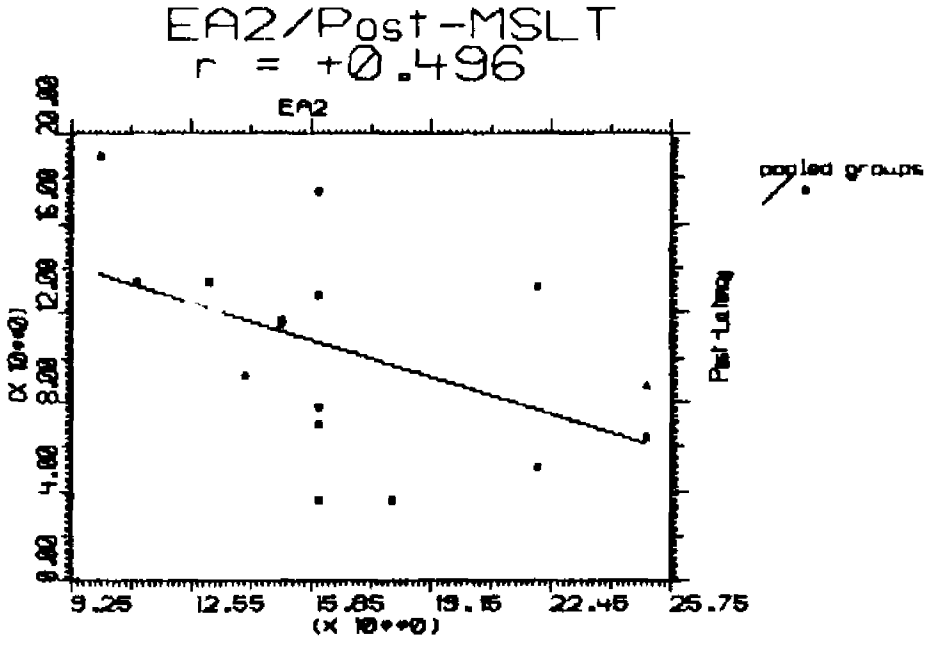
Appendix C

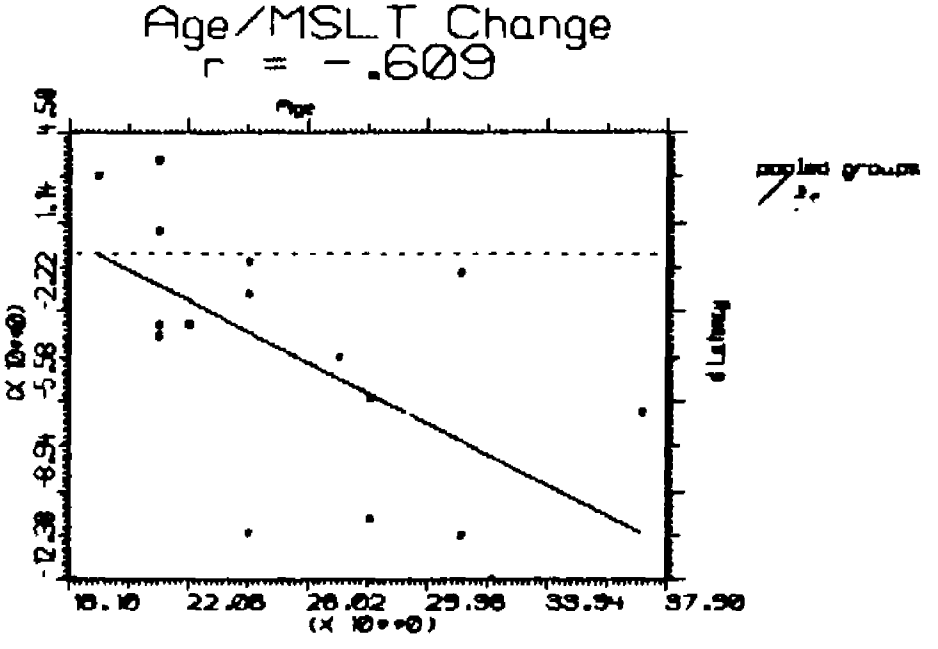
List of Abbreviations

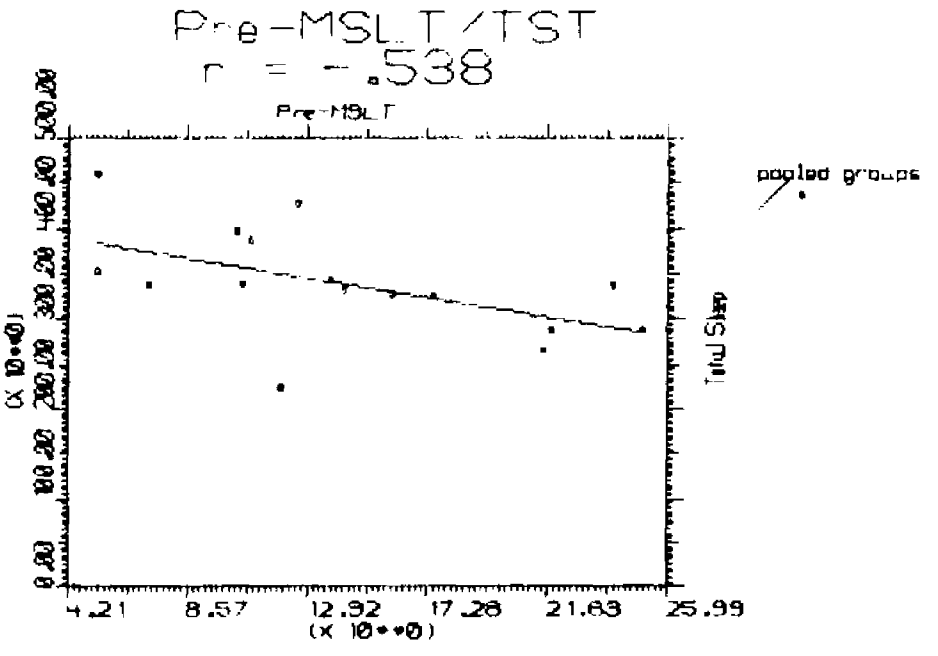
AER	Auditory Evoked Response
EA	Experimental Awakening
EEG	Electroencephalograph
EFT	Embedded Figures Test
EMG	Electromyogram
EOG	Electro-oculogram
MSLT	Multiple Sleep Latency Test
MMPI	Minnesota Multiphasic Personality Inventory
NREM	Non-Rapid Eye Movement Sleep
REM	Rapid Eye Movement Sleep
REMP	REM Period
RL	REM Latency
SL	Sleep Latency
SOREMP	Sleep-Onset REM Period
SSS	Stanford Sleepiness Scale
STAI	State-Trait Anxiety Inventory
SWS	Slow Wave Sleep
TRgT	Total Recording Time
TST	Total Sleep Time

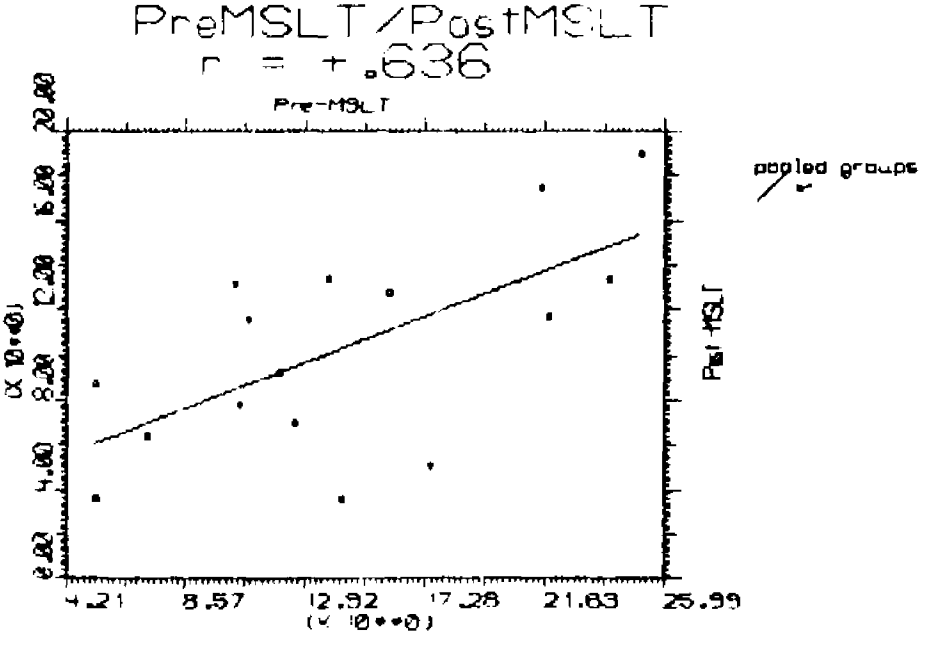
Appendix D

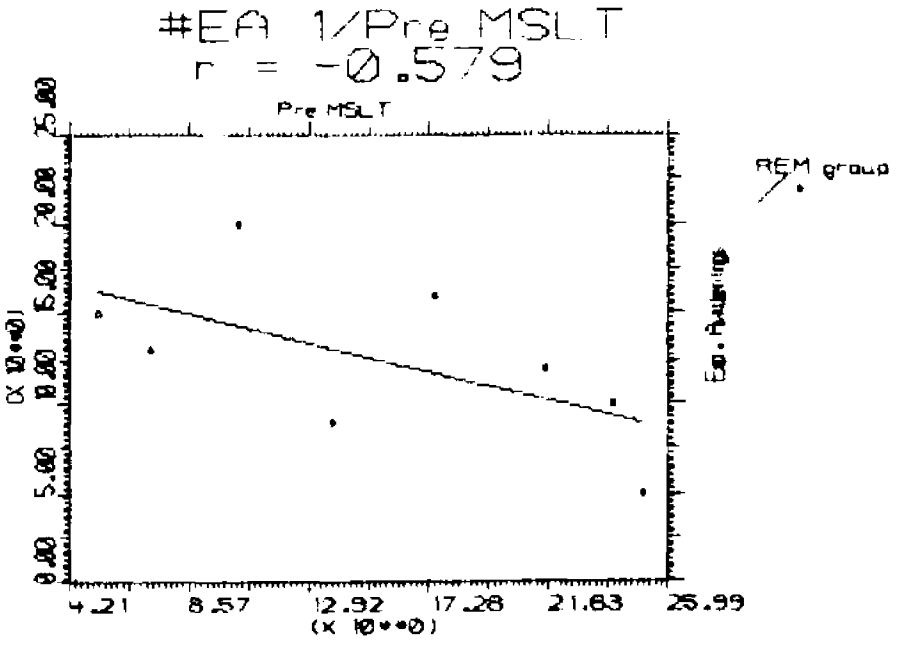
Scatterplots of Significant Correlations

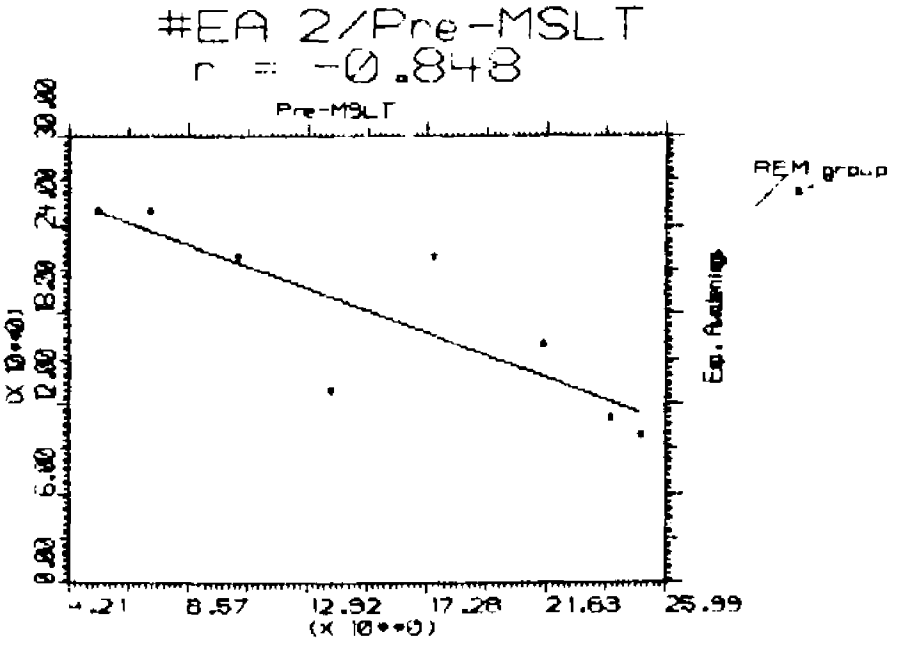

EA2/Post-MSLT
r = +0.496
EA2
pooled groups
9.25
12.55
15.85
19.15
22.45
25.75


Age/MSLT Change
r = -.609
Age
pooled groups
-2.22
-5.58
-8.94
-12.30
16.10
22.06
28.02
33.98
39.94
(x 10**0)

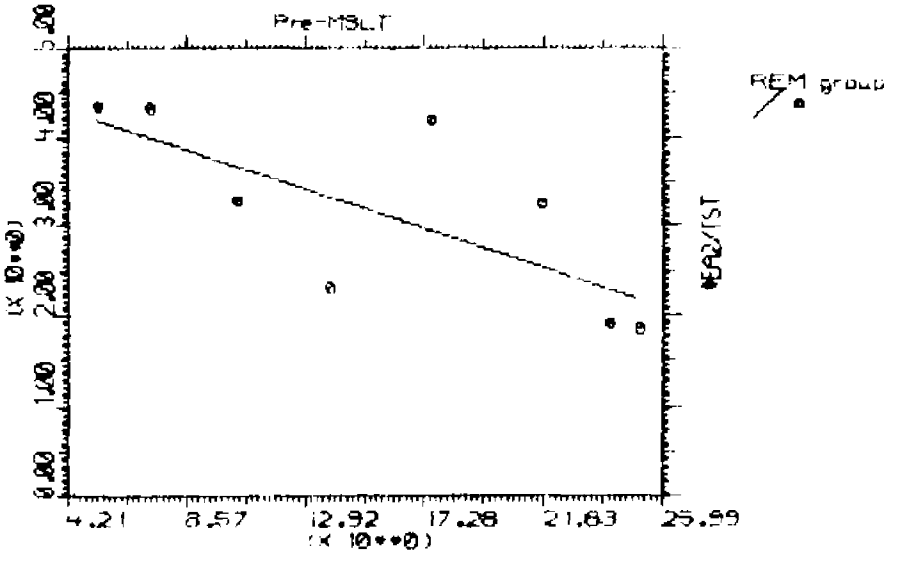

Pre-MSLT/TST
r = -.538
Pre-MSLT
pooled groups
Total Sleep
(X 10**0)
4.21
8.57
12.92
17.28
21.63
25.99
(X 10**0)


PreMSLT/PostMSLT
r = +.636
Pre-MSLT
pooled groups
Post MSLT
(X 10**0)
4.21
8.57
12.92
17.28
21.63
25.99
(X 10**0)

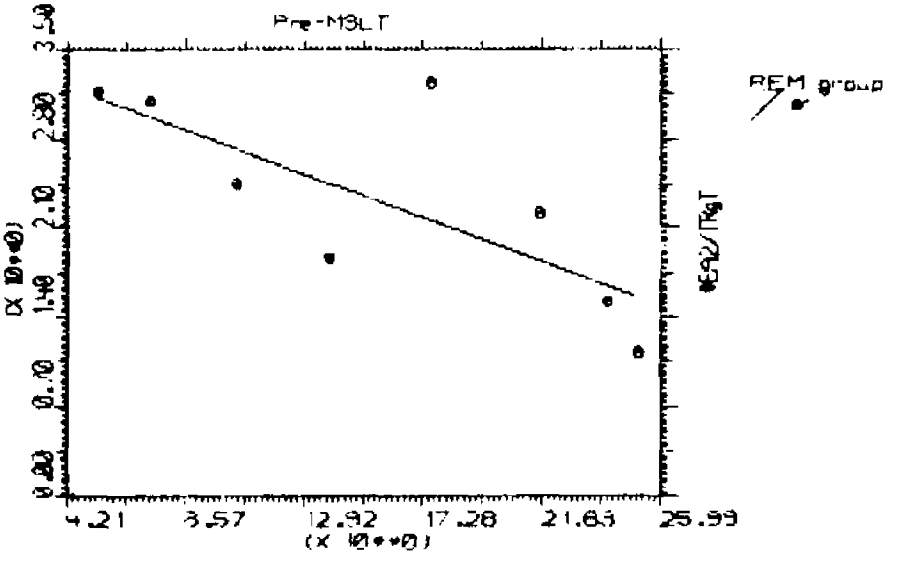

#EA 1/Pre MSLT
r = -0.579
Pre MSLT
REM group
Exp. Autonomy
(X 10**0)
4.21
8.57
12.92
17.28
21.63
25.99

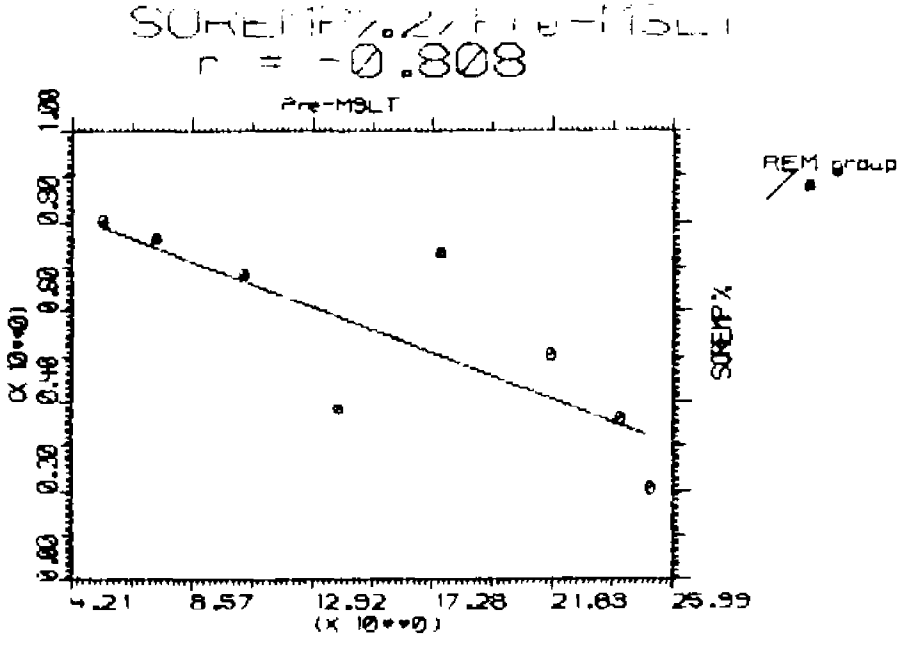

#EA 2/Pre-MSLT
r = -0.848
Pre-MSLT
REM group
Exp. Autonomy
(X 10**0)
4.21
8.57
12.92
17.28
21.63
25.99

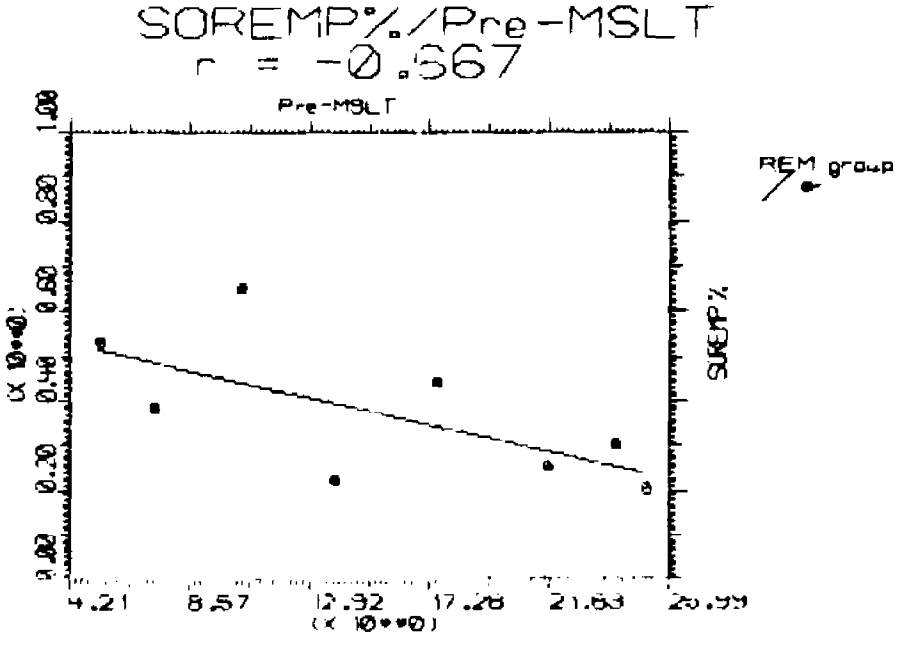
#EA2-TST/Pre-MSLT
r = -0.736



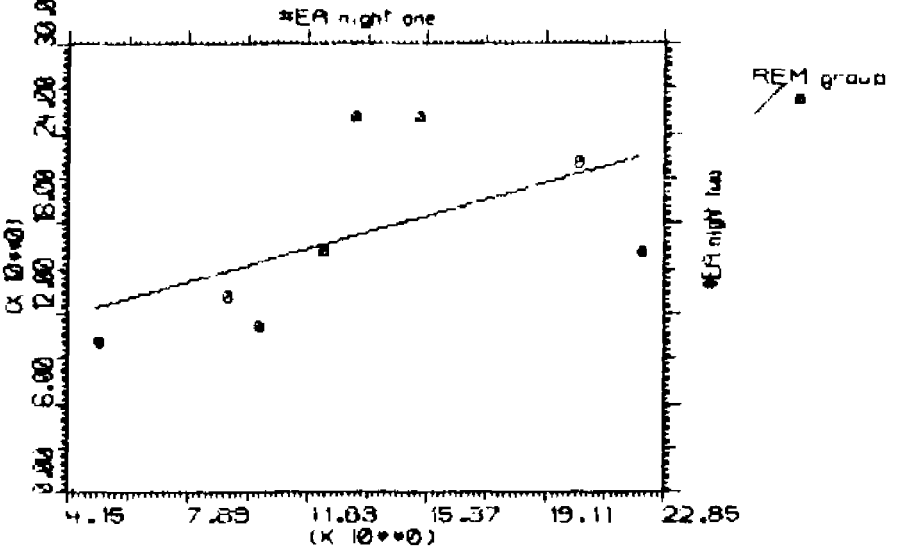
EA2-TRgT/Pre-MSLT
r = -0.753



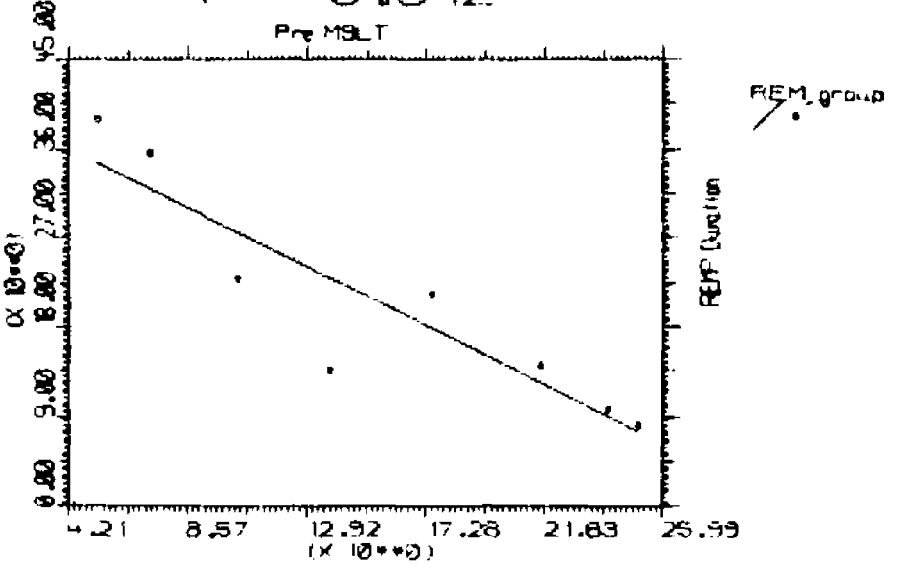

r = -0.808
Pre-MSLT
REM group
SOREMP%
4.21
8.57
12.92
17.28
21.63
25.99
(x 10**0)

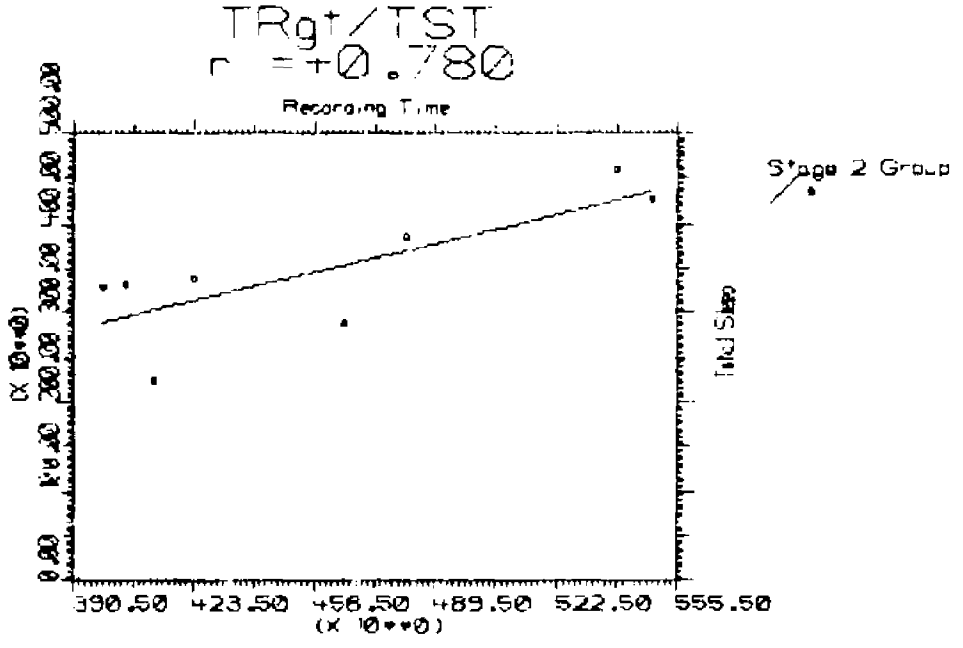

SOREMP%/Pre-MSLT
r = -0.567
Pre-MSLT
REM group
SOREMP%
4.21
8.57
12.92
17.28
21.63
(x 10**0)

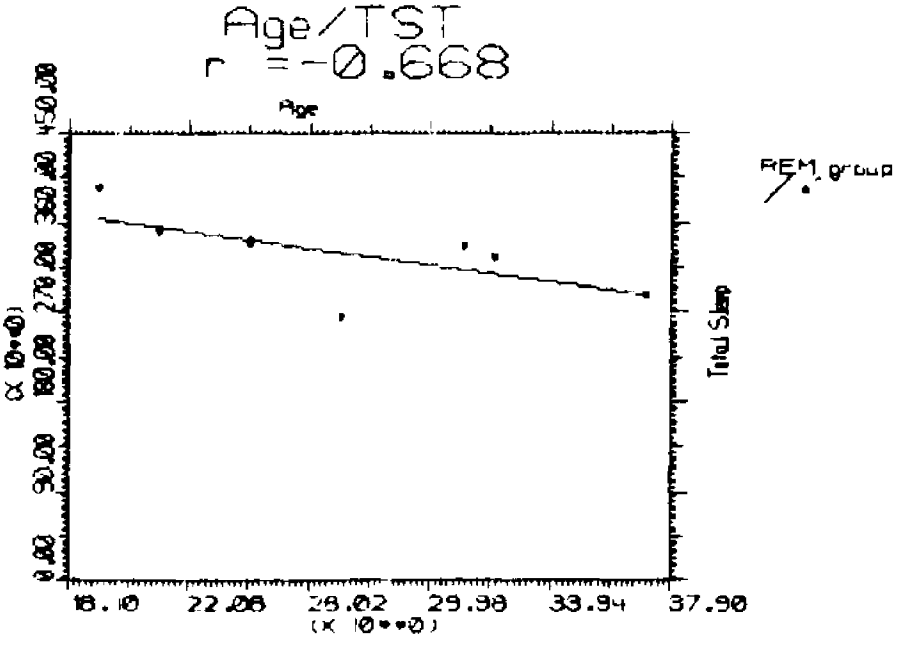
#EA 1/#EA2
r = +0.638



REMPdur2/Pre-MSLT
r = -0.912




TRgt/TST
r =+0.780
Recording Time
Stage 2 Group
Total Sleep
390.50
423.50
456.50
489.50
522.50
555.50
(x 10**0)


Age/TST
r =-0.668
Age
REM group
Total Sleep
18.10
22.06
26.02
29.98
33.94
37.90
(x 10**0)

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