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The effects of otolithic vestibular stimulation on sleep

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THE EFFECTS OF OTOLITHIC VESTIBULAR STIMULATION ON SLEEP

by

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DEDICATION

This doctoral thesis is dedicated to my father, Dr. Edward R. Woodward, whose enthusiasm for research and continual support made the completion of my doctoral degree possible, and to my son Jeter, whose love made all the sleepless nights worthwhile.

I wish to express my gratitude to my chairman, Arthur J. Spielman, Ph.D. who inspired me to enter the field of sleep research and whose advice and encouragement have kept me there.

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Lastly, a special dedication to the memory of Edward Tauber, M.D. who was there at the beginning and saw the future.

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CHAPTER I
LITERATURE REVIEW

SLEEP DIFFERENTIATION

Sleep and arousal disorders have become so pervasive that it is quite possible to divide our society into those who can sleep and those who cannot.

In contrast to the waking state, sleep appeared to be an inaccessible phenomenon until the discovery by Hans Berger in the 1920's that electrical brain waves could be continuously recorded. In the years following the development of the electroencephalogram (EEG), other polygraphic techniques were developed which allowed researchers to observe typical changes in the EEG which occurred during sleep (Loomis, Harvey, Hobart, 1937). Subsequently, Aserinsky and Kleitman (1955) described rapid-eye-movement sleep (REM), consisting of discrete periods of sleep correlated with rapid conjugate eye movements. These discrete periods were later associated with a high incidence of dream recall (Dement and Kleitman, 1957).

A significant amount of scientific investigation has been devoted to characterizing states of sleep associated with separate and distinct EEG, electro-oculographic (EOG), and electromyographic (EMG) patterns. These consistent descriptions make up the basis of what is termed sleep architecture. This provisional working construct permits investigations into sleeping and waking rhythms, length and

depth of sleep periods, physiological concomitants of sleep and environmental factors that affect sleep to be analyzed with regard to the following sleep stages and cycles.

Sleep Stages and Cycles

Sleep is delineated into non-rapid-eye-movement (NREM) sleep and rapid-eye-movement (REM) sleep. NREM sleep consists of three stages:

Stage 1. A transitional phase between full wakefulness and sleep characterized by an EEG activity of between three and seven hertz (Rechtschaffen and Kales, 1968).

Stage 2. Marked by the appearance of EEG sleep spindles consisting of bursts of twelve to fourteen hertz activity lasting a half second to two seconds, and by K-complexes defined as well-delineated, slow, negative EEG deflections that are followed by a positive component (Rechtschaffen and Kales, 1968).

Delta Sleep. Distinguished by slow delta EEG waves of a half to two hertz and high amplitude (> 75 microvolts, peak to peak). Twenty percent of the EEG must be dominated by these waves before sleep is scored as delta. Delta sleep can be further separated into stages 3 and 4 depending on the amount of delta waves (Rechtschaffen and Kales, 1968).

REM sleep alternates with NREM sleep at about ninety-minute intervals in adults. The EEG pattern resembles that of Stage 1, except that sawtooth waves are often seen. In addition, REM sleep has both

tonic and phasic components. Phasic REM includes short episodes when the eyes move rapidly and the entire system is highly activated. Tonic REM is characterized by near paralysis in many muscles and provides the background for phasic activity (Rechtschaffen and Kales, 1968).

Sleep can also be looked at in terms of cycles. After retiring, a person first passes through a stage of relaxed wakefulness, characterized by alpha waves. Later, the sleeper passes through stage 1 into stage 2, gradually descending deeper into sleep. Most normal young adults enter delta sleep within thirty to forty-five minutes after sleep onset. Delta sleep may persist from a few minutes up to an hour; then yield again to stage 2. About seventy to ninety minutes after sleep onset, the first REM period period occurs. It usually lasts about five minutes and is mild in intensity in terms of its physiological manifestations.

The second sleep cycle begins when stage 2 sleep redevelops after the first REM period. Delta sleep may reappear, but there is generally less delta sleep in the second cycle than in the first. Following this, the second REM period occurs about three hours after sleep onset and lasts about ten minutes.

Following the second REM period and until morning awakening, stage 2 sleep and REM alternate in ninety-minute cycles. In these later sleep cycles, delta sleep is rarely seen whereas REM periods become more intense and have a longer duration. The mean length of a REM period is about fifteen minutes, but some may last for an hour (Hauri, 1982).

Daytime Sleepiness: Assessment and Measurement

Another aspect of the sleep montage is daytime sleepiness. Sleepiness is essentially a lack of daytime alertness. Dement (1982), proposes that the most obvious interaction between sleep and wakefulness is the predictable fluctuation in daytime alertness as a function of nocturnal sleep.

It is possible to assess daytime sleepiness utilizing subjective, behavioral and physiological measures. Although it is difficult to describe their subjective feelings of sleepiness, subjects can readily identify sensations that accompany the feeling: heaviness of the eyelids, heaviness in the legs and arms, loss of initiative and difficulty concentrating (Dement and Carskadon, 1982). The Stanford Sleepiness Scale (SSS) was developed to measure sleepiness quantitatively through introspective reports of sleepiness through a seven point forced choice subjective rating scale (Hoddes, 1972).

Observed behavioral changes can also be used as indicators of sleepiness; lack of animation of the facial expression, rubbing the eyes or yawning and finally a drooping head and closing eyelids (Dement and Carskadon, 1982).

Measurements of the underlying physiological state associated with subjective feelings of daytime sleepiness may be conducted through the analysis of EEG waveforms. The Multiple Sleep Latency Test (MSLT) utilizes polygraphic techniques to measure changes in the EEG during a series of five daytime nap opportunities administered every two hours. Using the Rechtschaffen and Kales (1968) standard scoring system, latency to sleep onset is seen as a

direct change from wakefulness to either three consecutive epochs of stage 1 NREM sleep or a single epoch of any other stage of sleep.

The MSLT has face validity and has been shown to distinguish between normals and patients with excessive daytime sleepiness (Mitler, 1982). This test has also been demonstrated to distinguish patients with narcolepsy from noncomplaining control individuals (Carskadon, 1982; Carskadon and Dement, 1982). In addition, the MSLT has proved reliable when conducted on normals over seven consecutive days following controlled eight-hour nocturnal sleep periods (Carskadon and Dement, 1981).

The availability of data confirming sleep stages, sleep cycles and their interactions in young, healthy normals makes it possible to examine systematic changes in these variables induced by an external stimulus. The effects of an external factor on normal sleep and daytime readiness for sleep in the form of otolithic vestibular stimulation are examined in this thesis.

THE VESTIBULAR PROPRIOCEPTIVE SYSTEM

The vestibular-proprioceptive system is an ancient system phylogenetically and ontogenetically. Although recognized as a branch of sensory physiology, its direct association with motor mechanisms is clearly a predominant functional aspect. In addition to monitoring motion, the vestibular system is concerned with control of posture and spatial orientation by detecting position and motion of the body in space. This detection, although normally not in awareness, depends on the instant integration of impulses from the proprioceptive and visual systems as well as their vestibular.

apparatus. Most of the input carried centrally via the vestibular system is purely afferent in nature (Goldberg and Fernandez, 1975). This input is used to mediate a variety of reflexes but almost never reaches consciousness. Its continuous normal operation is essential in order to keep the body balanced, to coordinate head and body movements and to enable the eyes to remain fixed on a point in space even when the head is moving (McGeer et al., 1979).

The vestibular-proprioceptive is extremely complex, including connections with the cortex, cerebellum and brainstem. The uvula and flocculus comprise the vestibulcerebellum, which acts as an extension of the vestibular nuclei (Spoendlin, 1966). Connections with the cerebral cortex and brainstem and reticular nuclei in the brainstem tegmentum are reliably known (Kelly, 1981). Afferent functions include somatic proprioception drawing upon both kinesthetic and vibratory sensation. In addition, it is now clear that visual motor perception in the peripheral field is under control of the vestibular system (Howard, 1982). These findings clarify the visual-vestibular interactions and allow a more sophisticated analysis of habituation and recalibration of the vestibular-ocular system (Collins, 1974).

The vestibular system relies primarily on two sensory receptors in the head; the semicircular canals and the otoliths. Afferent connections are made from the proprioceptive receptors in the joints and muscles. The division of the vestibular labrith of concern here is the one containing the otoliths, which detect linear acceleration and determine the position of the head under static conditions with respect to gravity. The hair cells in the otoliths have

crystals of calcium carbonate on their surface which are embedded in the otolithic membrane (Key, 1981). When the head is upright, pressure created by the weight of the crystals keeps a particular set of hair cells in a bent position. When the head is tilted or subjected to acceleration or deceleration the shift causes the otolithic membrane to slide over a different set of hair cells. This establishes the neural coding for up-down and linear movement. The fibers of the vestibular portion of the auditory nerve are relatively large in diameter and conduct impulses with greater speed. This added speed is necessary for the rapid reflex adjustments required by the rapid changes in body position (Lowenstein, et al., 1956).

Influences of the Vestibular System

The influence of the vestibular system is often exercised out of conscious awareness. We take for granted the relatively stable postures occurring during sleep; we do not ordinarily fall out of bed. Arboreal animals and birds are equally capable of maintaining sleeping postures in precarious situations. In the maintenance of motor activity, balance is often taken for granted unless coordination becomes impaired. The maintenance of body posture and orientation in space is essentially automotized and does not reach awareness unless something adverse occurs bringing out the need for corrective motor activity. Unconscious vestibular-proprioceptive processes are still registered in the central nervous system and can be brought to conscious awareness when necessary. When this is the case sensations usually out of awareness impinge all too readily on our consciousness, such as the vertigo associated with Meniere's disease.

Accompanying symptoms of nausea and sweating are referable to the autonomic nervous connections of the labyrinth (Wolfson, 1966 a).

Postural reflexes such as the tonic neck reflex and the tonic labyrinthine reflex are often manifested in pathology such as hemiplegia, brain tumor and cerebral hemorrhage (Tokizane, 1951). These reflexes are apparent in the infant prior to higher cortical maturation and subsequent influence. They were thought to disappear in the normal adult unless released due to injury of illness. Langworthy (1933), noted in his embryological studies that the sensory pathways subserving kinesthetic and tactile activities are the first to complete myelinization. Thus optic, olfactory, auditory, thermal, and pain stimuli play a secondary role in the development of body awareness. Many integral components of whole sequences of skilled movements are neither purposefully initiated nor monitored yet they occur with precision (Guedry, 1974). Furthermore, challenges to the vestibular system produced by actions requiring expert physical coordination, are now known to activate postural reflexes once thought not to survive infancy (Fududsa, 1961). This evidence emphasizes that the vestibular-proprioceptive system operates out of awareness to support specialized activity, but that is can readily be called into functional awareness and non-activity at any time cannot be assumed.

Somatognosis

somatognosis, or body schema, also has a strong vestibular component. It may be regarded as a subjective model of the body

against which changes in posture, disposition of parts and integrity can be appreciated (Lishman, 1978). Underscoring the influence of vestibular inputs are studies involving patients suffering from vertigo, where abnormal perceptions of the spatial aspects of their own bodies were seen (Bonnier, 1905). There are two types of vertigo both meeting the requirement that there be an hallucination of motion. Objective vertigo refers to the sensation of objects moving while feeling that your body is stationary. The second type is termed subjective vertigo, and refers to a feeling of bodily movement in a stationary environment. Connections also exist between the vestibular apparatus and the autonomic system, which account for the perspiration, nausea and vomiting that commonly accompany an attack of vertigo. these types of abnormal perceptions also occur in conditions of experimentally produced vertigo and by experiments in elevators where the integration of impulses received from the eyes, the proprioceptive end-organs and the vestibular apparatus are examined (Hacaen and Albert, 1978).

It should also be noted that illusions of change of body schema may result from the absence of active body movements of occur while no active body movements take place. these types of changes are often seen during relaxation training sessions where sensations in body size, heaviness, lightness and body separation may be experienced in the absence of active movement. Illusions of spatial orientation are also produced in response to head movements in weightlessness. In this type of environment it is thought that vestibular afferent signals, primarily from the otolith organs, are centrally reinterpreted to represent fore-art or left-right linear

acceleration, rather than pitch or roll of the head with respect to the vertical (Young and Oman, 1984). It is evident that a person's perception of his position in space depends on valid synchronization of the vestibular system. Experimental blockade of sections of dorsal roots in the cervical region or anesthetized animals leaves them behaving as labyrinthextomized animals (Wolfson et al., 1966 b).

The phantom limb phenomenon is an excellent example of somesthetic illusion, and sedisive proof of the existence of a body schema normally existing on the fringe of awareness being brought into consciousness through focused attention. Perception of a missing body part, including all of the somesthetic and spatial characteristics, is present; the limb can assume a relaxed or cramped position, feel heavy or light and the sensation of movement is common (Hecaen and Albert, 1978). It has been suggested that peripheral afferent impulses play a role in the production of this phenomenon (Lishman, 1978), and link it directly to internal and external vestibular-proprioceptive input.

VESTIBULAR SYSTEM AND SLEEP FUNCTIONING

The role of the vestibular-proprioceptive system in sleep functioning is a dynamically important one considering that both these systems operate for the most part out of awareness, and non-activity during both types of functioning cannot be assumed.

Somnambulism, or sleepwalking as it is commonly referred to, is a state of dissociated consciousness in which phenomena of the sleeping state combine with those of the waking state (Kales, et al., 1980 a). Sleepwalking episodes frequently involve semipurposeful

automatisms and poor coordination when the higher cortical functions are inefficient. Visual inspection does operate during this behavior but despite open eyes, the person's expression is dazed. The essential features of sleepwalking consist of a sequence of complex behaviors that occur predominantly out or NREM stages 3. and 4. during the early part of the night (Mountcastle, et al., 1986). Behaviors such as eating or dressing are clearly out of context and reflect the person's general lack of awareness, and the person is usually amnesic for the episode (Kales, et al., 1966). This combination of night time awakenings associated with confusion, complex automatisms and extensive walking is rare except in sleepwalking, but it exemplifies the continuous functioning of the vestibular-proprioceptive system during what may be viewed as an abrupt liberation of motor activity under circumstances of incomplete arousal (Broughton, 1968).

In addition to somnambulism, there are a number of other clinical conditions consisting of undesirable physical phenomena exclusively appearing in sleep that possess distinctive vestibular-proprioceptive components. Night terrors are nocturnal episodes of extreme terror and panic accompanied by intense anxiety, autonomic discharge, vocalizations and motility (Kales, et al., 1980 b). Sleep-related epileptic seizures, particularly psychomotor attacks, may give rise to sleep behaviors that appear similar to those of sleepwalking. Patients with seizure-induced behavior are totally unreactive to environmental stimuli, and automatic behaviors like swallowing and rubbing of hands are common (Association of Sleep Disorders Centers, 1979). Sleep-related headbanging or total body rocking

usually occurs in the time period prior to sleep onset. If this rhythmic rocking continues into sleep, it predominates during stages 1. and 2. as REM sleep and stage 3/4 sleep may potentially inhibit rocking (Watanabe et al., 1980).

Vestibular-Proprioceptive Stimulation

A certain amount of research has been devoted to examining the effects of vestibular-proprioception on human and animal functioning. Determining physiological pathology is only one aspect of vestibular research. Studies evaluating the use of various forms of vestibular stimulation to positively influence functioning are also being conducted.

Gross motor functioning of preambulatory, normal human infants was significantly improved after exposure to mild semicircular canal stimulation (Clark et al., 1977). Stimulation was administered by holding the infant in the investigator's lap while sitting in a rotating chair in a dark room. A control group of infants were held in the chair for the same length of time but were not rotated. Although children of this age normally receive semicircular canal stimulation during passive handling and rocking and during active head movements, it was felt that the effect of such stimulation could be enhanced significantly by localizing the stimulation, maximizing the duration and increasing the magnitude. Motor development was accelerated, possible through facilitated maturation of the vestibuloocular reflex against which motor involvement with the environment developed more rapidly.

The recording of vestibular evoked potentials is relatively new compared to the extensive clinical and experimental recording or auditory and visual evoked potentials. Experiments by Hood and Kayan (1985), utilized rotational repetitive stimuli that simulated natural head movements to study vestibular evoked responses in normal subjects. Their findings indicate a clear visuo-vestibular interaction. When subjects were rotated in the light while fixating on the target there occurred a statistically significant increase in the amplitude of the evoked response recorded in darkness. All eye movements were restrained, and it appears that under experimental conditions where simultaneous visual and vestibular stimulation are administered the visual stimulation acts synergistically to enhance the vestibular.

The effects of microgravity on vestibular functioning and on visual-vestibular interactions were tested during the Spacelab mission. Pilots were a specifically designed helmet in a restraining seat during testing which contained equipment that measured EOG using amplifiers and a camera with infrared illumination in front of the right eye which allowed eye movements to be computed in real time. A television monitor in front of the left eye provided optokinetic stimuli. Values obtained during and after the flight suggest that one aspect of adaptation to microgravity is an increased dependence on visual as opposed to vestibular mechanisms in the stabilization of the retinal image during head movement. The otolithic vestibular component appears to be responsible for this finding. On the ground, the otolithics make a significant contribution to the gain of the vestibulo-ocular reflex in the pitch axis. In space,

head movements in pitch are nauseating due to the fact the the static otolithic component is absent (von Vaumgarten et al., 1984). The static labyrinthine reflex, mentioned above, was investigated by Fukuda (1958), using animals. Leghorns were blinfolded and rotated many times in both directions every day for two weeks. Normally during rotation the head is deviated against the direction of rotation, resulting in postrotary head-nystagmus which in man is accompanied by a state of disturbed body equilibrium. The well-known labyrinthine reflex is what is known as the static labyrinthine reflex. However, in this study, it was found that after repeated rotations, the animal's head turned in the direction of the rotation after a normal deviation and postrotary head-nystagmus was much less marked. Fuduka has termed this phenomenon the kinetic labyrinthine reflex.

The most interesting aspect of this study is the difference between activity and passivity with regard to rotation. Passive rotation is used almost without exception in the study of the labyrinthine physiology.

Passive rotation is an unusual stimulus for animals since their movements are generally active. Man is the first animal to impose passive stimulation on himself whether on horseback, in a car or on an airplane. The physiology of the labyrinth is intimately connected with the optic organ in order to regulate muscle tone at every active moment. It may be that the labyrinth processes passive movements as new stimuli and cannot accommodate them; as a result motion sickness may be produced. The establishment of the kinetic labyrinthine function after repeated rotations which prevents

postrotary nystagmus, is thus seen as an improvement of the labyrinthine equilibrating function and not due to fatigue, damage or paresis.

The kinetic labyrinthine reflex has been investigated further using repetitions not only of rotation, but of centrifugal, see-saw and pendulum-like motions with the same result (Fuduka, 1959).

HABITUATION

Habituation to vestibular stimulation has also been of interest to investigators. The term habituation is frequently used interchangeably with response reduction to indicate the effects of repeated stimulation. Habituation is not considered the same as fatigue, which refers to a temporary state due to the fact that brief periods of rest are usually sufficient for recovery of the response. Adaptation, a special type of response decline that occurs during a prolonged stimulus, is also different from habituation. Habituation involves a relatively long lasting change. One that even extended periods of rest are not sufficient to restore the response to its original level (Collins, 1965). The most common measurement of levels of habituation is eye nystagmus. These eye muscle movements are the clinical indication of labyrinthine response to stimulation.

McNally and Stuart (1942), have reported that repetitive vestibular stimulation in both man and animals produces a response decline in all subjective and objective aspects of the reactions, including nystagmus.

This was not always thought to be the case. Barany (1907), stated that vestibular nystagmus in a normal subject was a relatively fixed and invariable reflex.

It has subsequently been reported that habituation to stimulation can be established at least with the horizontal canals (Henriksson et al., 1961). This study used caloric stimulation on the cat and concluded that the habituation was not due to fatigue or adaptation of the vestibular receptors, but rather, the habituation phenomenon took place in the reticular formation.

McCabe and Gillingham (1964), also used cats and habituated them to rotation. Following surgical destruction of the superior and lateral vestibular nuclei, there was a release of the suppressed reaction to vestibular stimulation. They proposed that normal suppression of response may result from an inhibitory effect that the efferent nerve system exerts on the afferent discharge from the vestibular end-organs.

A study conducted with humans used a special machine to rotate subjects about a horizontal axis. Using this method, in addition to angular acceleration (canal stimulation), there was continuous alteration in relation to gravity and centrifugal force (otolithic stimulation) (Guedry, 1966). Modification of both the resulting nystagmus and the sensation of rotation resulted from the otolithic stimulation. guedry concludes that the information from the otolithic stimulation modulates the messages that the semicircular canals send to the higher center.

Fluur and Mendel (1964a, 1964b), also carried out habituation studies on human subjects. Nystagmus was recorded electro-

nystagmographically under both caloric and rotation conditions in complete darkness. The resulting habituation changes proved not to be a consequence of the particular method of stimulation, but what is interesting is their notation that the subject was spoken to in order to prevent a decline in the waking state of the subject.

Collins (1965), studied two groups of subjects each subjected to caloric stimulation daily for six days and for one day one month later. One group was tested in total darkness and the other tested in the light but fixating visually. The group tested in the darkness showed a significant rise in the number of nystagmic beats while the group tested in light showed no change in the number of nystagmic beats. What is interesting in this study as well, is that in both groups Collins kept his subjects mentally alert. It is supposed that the mental alertness tends to eliminate central inhibition and amplify the labyrinthine reactions. The effect of visual fixation would be to reduce labyrinthine function, which is not what Collins found with his alerting method.

Simple instructions, designed to relax a subject and reduce mental activity, have been found to produce a response decrement to vestibular stimulation on the very first trial (Wendt, 1951). Therefore, in the absence of alertness, central inhibition is operating.

Mental arithmetic problems were found to be most effective in maintaining alertness that resulted in an increased nystagmus level (Crampton, 1964). It is now common in testing situations where vestibular stimulation is used to elicit nystagmus in order to determine pathology, to have patients do mental arithmetic. Otherwise, it is difficult to elicit crisp nystagmus.

SWINGING STIMULATION

In the above experiments which examined general vestibular stimulation and vestibular stimulation and its connection with nystagmus, no attempt was made to distinguish between specific semicircular canal and specific otolithic stimulation. Although differences in findings were attributed in some cases to either one or the other of these labyrinthine organs, the initial method of stimulation was not selective.

Breuer (1975), was one of the first investigators to distinguish between the effect of semicircular canal stimulation and otolithic stimulation. In an important paper Jongkees and Groen (1946), went on to hypothesize that the labyrinthine reflexes could be divided according to acceleration. This division specified reflexes and sensation caused by angular accelerations, and reflexes and sensations caused by linear accelerations. These findings made it highly probable that the proper stimuli for the semicircular canals was angular acceleration, and that linear acceleration was the proper form of stimulation for the otolith organs.

Based on the results of the acceleration studies of Jongkees and Groen, Groen (van Egmond, et al., 1954), conducted studies looking at the role of the vestibular apparatus in motion sickness, a disease experienced by predisposed persons when they were exposed to changes of acceleration. Groen proposed that the vestibular system and its connections with autonomic functioning were of primary importance in the pathogenesis of motion sickness. In addition, the symptoms associated with motion sickness were due to changes of

linear acceleration and that research should be concentrated on the sense organs that mainly react to these types of accelerations, namely the otoliths.

The particular technique of measurement being used at that time was measurement of blood pressure in the central retinal artery. correlations between linear acceleration and motion sickness depended on discovering a reliable quantitative method of assessing a subject's responses to linear acceleration, which was thought to be the use of a parallel swing. Stimulation of the vestibular organ with linear stimuli was thought to cause an increase of the pressure of the central retinal artery, especially in subjects susceptible to motion sickness. Pressure was measured before and after a subject had been swung on a parallel swing for one minute. The swing used consisted of a stretcher suspended on four, three meter parallel ropes. Initial investigations conducted with a parallel swing at Groen's clinic were favorable, indicating it to be the method of choice, but it proved not to be suitable as a routine method when in conjunction with the current mode of blood pressure measurement. A reliable method of examining the semicircular canals, cupulometry, was available however, and the subsequent studies were completed based on subjects' reactions to rotary stimuli perceived by the semi-circular canals.

The development of electronystagmography, the method of electrically recording eye nystagmus, has made it possible to dependably measure and record vestibular reactions. It is readily adaptable to routine studies of vestibular physiology and has made it possible to study vestibular eye reflexes accurately. Electro-

nystagmography is considered to be an important advancement in vestibular research.

Jongkees and Philipszoon (1962), developed a method to record electronystagmographically eye movements by the linear accelerations of a parallel swing. This method now made it possible to study Jongkees and Groen's hypothesis that the proper stimulation for otoliths and semicircular canals are linear and angular accelerations respectively. A parallel swing was used to provoke rhythmically changing linear accelerations causing compensatory eye movements. To provoke angular accelerations a torsion swing was used and compensatory eye movements and nystagmus elicited by this stimulation were also recorded (Philipszoon, 1962). In all the following experiments rabbits were used and the head of the rabbit was covered to prevent optokinetic influences. Total and partial labyrinthectomies were performed. The results of these studies showed that after total labyrinthectomy on both sides rabbits presented no compensatory eye movements on the parallel swing, while clear reactions on the torsion swing could still be observed. This same finding held true for human subjects with two inexcitable labyrinths.

After partial labyrinthectomy, compensatory eye movements could still be provoked on the parallel swing in rabbits as well as in humans. Under this situation, where the semicircular canals were left intact, nystagmus was also found following angular acceleration. In addition, findings also indicate that an intact set of canals in the plane of rotation is necessary, while reaction to linear acceleration is not affected by damage to the canals. (See Figure 1.)

Figure 1

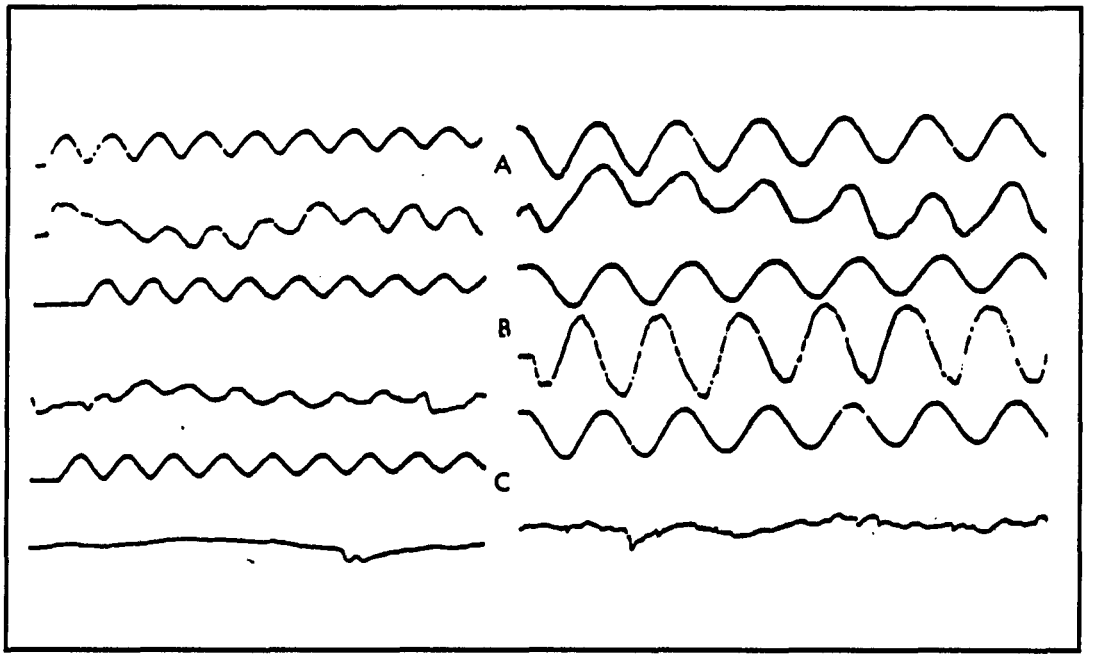


Figure 1 -Eye Movements on the parallel swing

Left part rabbit
A. Normal rabbit.
B. After unilateral labyrinthectomy.
C. After labyrinthectomy on both sides.

upper lines - movements of the swing.
lower lines - movements of the eyes.

Right part human
A. Normal Human
B. Patient after one dead labyrinth.
C. Patient with two inexcitable labyrinths.

Another significant study was looked at the influence of position on eye movements provoked by linear accelerations on a parallel swing (Bos et al., 1962). Rabbits placed in different positions were swung sideways and it was found that eye movements in the normal and lateral positions were definitely smaller than eye movements which resulted from swinging the rabbit in the supine position.

Jongkees states (1966), that it has been sufficiently proven that the otoliths are responsible for the compensatory rolling of the eyes. In the case of vertical rolling of the eyes, the use of the four pole suspension swing should be used to produce linear acceleration in head to foot direction. On the parallel swing the vertical counter rolling of the eyes is normal as long as one labyrinth is normal.

VESTIBULAR STIMULATION AND SLEEP

Very few studies have been performed looking at the effects of vestibular stimulation during sleep and most of these focused on the elicitation of nystagmus, only incidentally noting the role of sleep itself. The mechanisms governing sleep and wakefulness have also been investigated, for the most part analyzing the effects of varying types of vestibular stimulation on arousal levels. These studies, conducted with infants children and young adults as well as with animals do suggest however, that vestibular stimulation during sleep may alter sleep architecture as well as some aspects of sleep efficiency. In none of the studies conducted so far was an attempt made to selectively stimulate the otolithic system and exclude semi-circular canal stimulation.

Vestibular Stimulation and Levels of Arousal

An experiment involving normal unrestrained cats demonstrated that EEG synchronization could be regularly induced with low frequency, low intensity electrical stimulation of cutaneous nerves (Pompeianp and Swett, 1961). The opposite effect of arousal could be obtained if the intensity of the stimulation was increased. A rocking platform was used to stimulate various proprioceptive systems thought to be mediated by the vestibular, subcutaneous and kinesthetic organs. The resulting increases and decreases were thought to be the result of repetitive stimulation of the rocking affecting centers controlling sleep and wakefulness directly.

Oswald (1959), conducted a number of studies based on the concept he adopted from Pavlov that internal inhibition and sleep are one and the same process. Using the modern technique of electroencephalography in addition to measurement of other physiological variables, such as respiration, heart rate and electro-dermal activity, parameters of sleep and wakefulness were continuously monitored. Human subjects were exposed to different modes of vestibular stimulation with eyes closed. It was found that signs of sleep appeared in persons subjected to repeated strong electric shocks, that signs of sleep could come and go rhythmically in time with regular stimuli at intervals of only a few seconds, and that signs of sleep appeared while subjects continued to move in time to prolonged, rhythmic music. Two tendencies appeared in the EEG data, one, a tendency for sleep and alertness to alternate at the rate of the signals, the other, a slower general downward drift of variable

steepness and duration towards sleep. One thing of note here is the "harness" effect. Although not subjected to a physical restraint often used in animal studies, Oswald instructed his subjects either not to move or to make only certain movements. This physical positional readiness for sleep may have played a major role in the elicitation of sleep signs in these studies and could be a significant contributor to lowered cerebral vigilance.

In a follow up study, subjects were exposed to simultaneous, synchronized, rhythmic electric shocks, loud rhythmic music and strong flashing lights while their eyes were glued widely open (Oswald, 1960). It was believed that all of the subjects did go to sleep and evidence of reality adjusted thinking was found during these periods of light sleep. This type of adjusted thinking was pointed out to the probable occurrence of similar brief episodes of light sleep in persons, such as some drivers, who are exposed to prolonged, monotonous sensory stimulation.

A study by Webb and Agnew (1979), reviewed the studies above by Oswald, but in their experimental design stimulation was given in the form of three basic sound stimuli: silence, monotonous sound and an intermittent tone. In addition, conditions of intermittent tone plus counting and intermittent tones with synchronized eye opening-eye closing were used. Measures of sleep latencies were compared with a sleep deprived group run under silent conditions. Subjects were exposed to the variable stimuli after a full night's sleep in what Webb termed as an "artificial insomnia". The intermittent tone and the tone plus counting proved to be the most effective in reducing sleep latencies under these conditions.

The experimental situation involved a strong suggestion to sleep. Although not restrained, subjects were retired in sound attenuated chambers and given taped instruction which informed them that their primary task was to go to sleep. The shorter sleep latencies associated with the intermittent tone were thought to be facilitated not evoked; the mediating variable being the reduction of extraneous stimuli incompatible with sleep.

The effects of vertical rocking frequencies on the arousal level of two-month-old infants resulted in a positive monotonic relationship between frequency of vertical rocking and effectiveness of rocking (Ter Vrugt and Pederson, 1973). More of the infants slept and fewer cried when they were rocked at higher frequencies. The authors suggest that the soothing effect of the rocking resulted from stimulation of the proprioceptive organs, and its effectiveness depended on the intensity of the rocking.

Sleep and Rapid Eye Movements

Normal young adults were studied for a total of fourteen nights to determine whether vestibular stimulation could elicit nystagmus during different sleep stages, particularly during rapid eye movement sleep (Tauber, et al., 1972). The experimental design required that the subjects sleep in a sitting position throughout the night. A constant angle torsion swing technique permitting per-rotary stimulation, was used to produce the vestibular stimulation. The duration of rotation trials consisted of six to ten sequential rotations, each period of oscillation lasting five seconds. Results indicated that although slow compensatory conjugate deviation of the

yes occurred with each oscillation, nystagmus was never elicited by this type of vestibular stimulation during unequivocal REM sleep. During NREM sleep stages the effects of vestibular stimulation confirmed the absence of vestibular nystagmus, therefore suggesting that vestibular stimulation constitutes a powerful means of selective suppression of eye movements during REM sleep.

Tauber (Hirsch and Tauber et al., 1973), followed up the previous investigation by noting the effects of vestibular stimulation in the form of cradle rocking on neonates. The cradle was rocked in the longitudinal axis in order to determine the incidence of nystagmus resulting from this type of vestibular stimulation. Conditions were designed to control for possible sequence and adaptation effect. Only two out of twenty-one babies showed no nystagmus and of the 90 observed incidents of nystagmus, 85 occurred in REM. Sixty percent occurred during rocking and forty percent when the rocker was off. More importantly, two of the records were randomly selected and surveyed for sleep cycle shifts, giving some indication of possible changes in sleep efficiency. The analysis of records of sleep efficiency suggested that rocking extended the periodicity of REM parameters (EOG, EEG, EMG,), regardless of the sequence of stimulus presentations. This controlled cradle rocking elicited fewer arousals and a significant increase in total sleep time in the newborn infants studied.

Bilateral lesions limited to the medial and descending vestibular nuclei have been shown to abolish not only REM bursts, but all the phasic events related in time to them (Pompeinao, 1965). These findings in animals clearly implicate the vestibular nuclei in

the control of rapid eye movements in REM sleep. Pompeiano (1970), has also examined the mechanisms of sensory motor integration during sleep, proposing that the clustering of rapid eye movement reflects the phasic activity of central vestibular mechanisms in the modulation of sensory input and motor output.

An interesting study by Ornitz (Ornitz, et al., 1972), looked at the effect of vestibular and auditory stimulation on the rapid eye movement of REM sleep in normal children. Subjects were studied throughout the night under three different conditions; vestibular stimulation, no stimulation and auditory stimulation. The vestibular stimulation consisted of a horizontal bed with angular velocity providing a mild sinusoidal acceleration and deceleration. Auditory stimulation consisted of exposure to alternating single and paired clicks. With regard to changes in sleep architecture, there was a slight tendency for longer REM sleep periods, longer total sleep time on the vestibular stimulation nights and a greater proportion of wake time on the auditory stimulation nights. None of the differences was significant.

The main findings of this study concerned rapid eye movement activity. Compared to nights with no stimulation, both vestibular and auditory stimulation increased the total amount of rapid eye movement activity, the measures of rapid eye movement clustering and the variability of these measures, although auditory stimulation to a lesser extent. A cumulative influence on the clustering of the rapid eye movements during the nights of conscious vestibular stimulation was also found. These results confirm the findings of Pompeiano's animal studies, reviewed above.

Vestibular stimulation in the form of a "hammock-like" rocking bed was tested for effects on respiratory effort, and sleep states in kittens (McGinty, 1985, personal communication). Vestibular stimulation augmented respiratory effort tonically during both NREM and REM sleep. Rocking at frequencies near fifty percent of the NREM baseline respiratory rate produced significantly greater facilitation of respiratory effort than higher or lower frequencies in REM, and greater facilitation than higher frequencies in NREM. A dramatic increase in REM percentage of sleep was unexpectedly found under the rocking condition. The experimental protocol was repeated after vestibular-auditory nerve destruction, and no increase in either respiratory effort or REM percentage sleep was observed. The author suggests that vestibular stimulation activates reticular formation neurons which play a role in the facilitation of respiratory effort. Facilitation of reticular formatin unit activity may also have mediated the vestibular influence on REM sleep, confirming Pomeiano and Morrison's (1965), view that the vestibular nuclei participate in the complex ponto-geniculo-occipital waves of REM in the cat. Clearly, further studies investigating the mechanisms of REM modulation by vestibular stimulation are needed.

Nocturnal sleep parameters were assessed in chronic insomniacs after all night rocking on a "swing bed" (Barthlen, 1985). Subjects alternated between rocking and non-rocking nights. The number of arousals and awakenings were significantly decreased. The amount of stage three sleep was increased, although not significantly, and there were no changes in REM sleep parameters.

The research conducted to date has shown varied and interesting effects of vestibular stimulation on arousal levels, eye movements and nocturnal sleep stages. Associations between the mechanisms governing sleep and wakefulness and the vestibular proprioceptive system clearly do exist. What is not clear is precisely what form of vestibular stimulation is the most appropriate for tapping these mechanisms of sleep and wakefulness.

There is evidence that the otolithic system is consistently accessible for stimulation. A constant maintained input from the otoliths is required for all positions of the body, including normal erect posture and the maintenance of postural tone. This system, which responds to linear acceleration has no natural zero value since the utricles are normally inclined several degrees to a constant field force of one g acceleration of gravity (Howard, 1982). The semi-circular canals, on the other hand, do possess a natural zero value when rotary stimulation is not being inputed. A parallel swinging bed would selectively stimulate the otolithic system by means of linear acceleration, thereby avoiding stimulation of the semicircular canals. As the whole body would be in motion, the potentially confounding influence of the neck reflex would also be avoided.

No studies have been conducted which evaluate the effects of vestibular stimulation, either angular or linear, on physiological and subjective measures of daytime sleepiness following a normal night's sleep. Placing a subject in an environment situation where stimuli counterproductive to sleep are reduced, would allow the effects of the stimulation to be more clearly seen. It may be that specific stimulation received while in this passive state may confirm

quiescence and inform the individual that high levels of vigilance are no longer required.

The question is then, whether specific otolithic stimulation in the form of linear acceleration will affect sleep architecture or sleep efficiency either during nighttime sleep or daytime sleep opportunities. The present study will attempt to answer this question.

CHAPTER II
METHODS

PARALLEL SWING

A parallel swing bed was constructed in order to examine the effects of a combination of accelerated linear movement and the always active gravitational force, on the otolithic system. The swing consisted of scaffolding that used eight tubes of eight-foot length and four tubes of four-foot length in order to produce a rectangular box frame. Four ropes with height adjusters were suspended from the horizontal crosspieces and attached to a platform on which the mattress was placed. The swinging bed was of standard twin bed size, fitted with high quality mattress and installed in a 14 by 12 foot bedroom of a research laboratory specifically designed for conducting all-night sleep investigations. The room was temperature controlled, sound attenuated and equipped with an infra-red video camera and a two-way auditory monitor. The swing was driven by a variable frequency electric motor, which was covered by an insulated box in order to reduce motor noise to a barely detectable level. Connecting cables were fed through a wall porthole which was blocked to sound and light.

The swing was mechanically activated to move between the scaffolding frame in a longitudinal direction with an excursion set at one-and-a-quarter inches at the level of the bed. The frequency of movement was 0.42 cycles per second. This amplitude of the swing

was expressed as one half the total excursion; . It has been shown that the soothing effect of the swinging, or rocking, motion may depend on the intensity of the rocking, which is a function of both frequency and amplitude (Ter Vrugt and Pederson, 1973). Intensity was expressed in terms of "peak g load," which can be defined as the maximum level of linear acceleration attained during movement. Peak g levels were computed by means of the following formula suggested by Ter Vrugt and Pederson:

$$\text{peak g load} = a_{max} = 4 f A/32.$$

where a max represents peak g load, f the frequency in hertz and A the amplitude of swing. The lower threshold in which linear acceleration can be perceived by beings is 0.01 g (DeWitt, 1953). The length of the suspending cables was long enough to produce sinusoidally changing horizontal accelerations. The vertical acceleration, although present, was negligible as it remained below the level of perception. A person lying on the swing with eyes closed may have the slight perception when the bed moves in the direction of his head, that his head is lowered and his feet are rising. The opposite is perceived when the bed moves in the direction of the feet (fig. 2).

SUBJECTS

Subjects were recruited by word-of-mouth from the university population after a verbal announcement was made by the investigator offering $400.00 to young healthy adults for charting their sleep habits for two weeks, and spending six nights and two days in a the

Figure 2

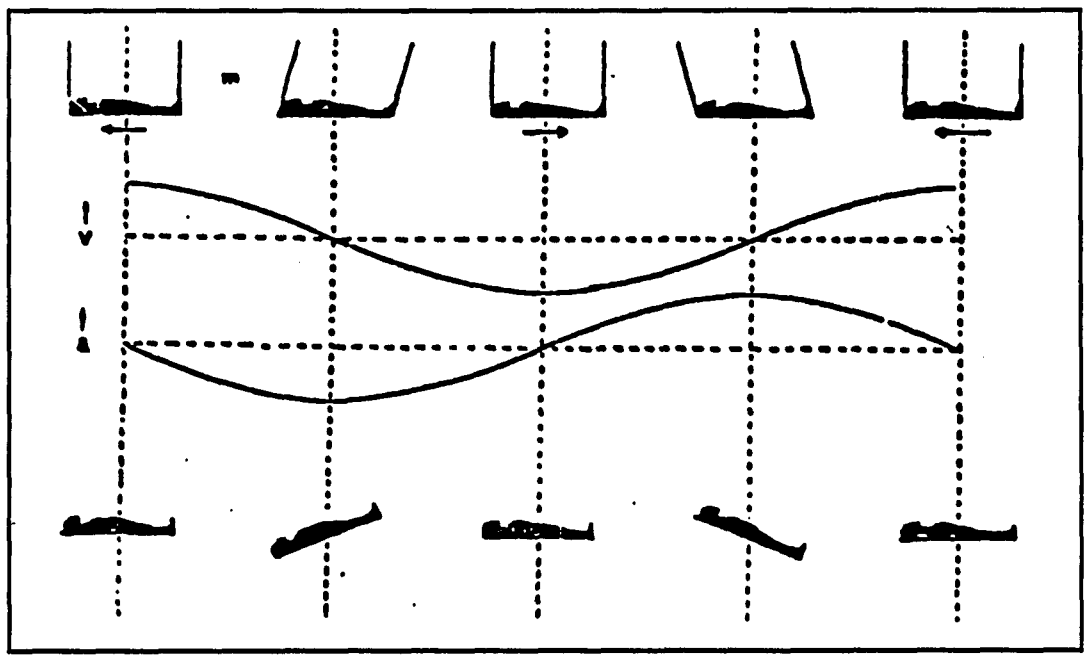


Figure 2 - Scheme of the place, movement and acceleration of the parallel swing, with the subjective position of the test person.
(m) movement (v) velocity (a) acceleration (s) sensation

sleep laboratory. Those interested contacted the investigator by telephone and were told that the purpose of the study was to investigate the effects of motion on sleep. They were then asked a series of standardized questions pertaining to general health, in particular any current or past vestibular pathology, demographics, sleep habits and medication needs known to affect sleep functioning. If age and subjective health criteria were met, subjects were mailed a two-week log of their sleep habits to be completed at home, A medical index, and a comprehensive sleep questionnaire. Upon completion of the sleep log, subjects were asked to mail the log, along with the other completed forms back to the investigator and a determination would be made with regard to acceptance into the study. Criteria for acceptance were as follows:

1. Age between 18 and 36 years.
2. No major medical history.
3. No current condition requiring pharmacological treatment.
4. No subjective sleep complaint.
5. Sleep log reports of fairly regular sleep/wake times and naps occurring less than twice a week.
6. Willingness to stabilize bed-times for three nights prior to laboratory visits.
7. Willingness to refrain from caffeine or alcohol intake during the time spent in the laboratory.

The medical index and sleep questionnaire were reviewed by the attending neurologist who specialized in sleep disorders.

Interviews were conducted with any subject whose responses on the above forms were suspect. In addition, all subjects were given the Vertical Writing With Eyes Covered Test (Fuduka, 1958). This test is designed to diagnose vestibular pathology by having the subject writer letters vertically with eyes covered. Marked deviation indicating vestibular impairment. All subjects were asked to sign an informed consent form specifying the research protocol an any mild discomfort anticipated from the procedure (Appendix A).

Subjects were excluded from the study, either in progress or prior to beginning, for the following reasons:

1. Vestibular dysfunction was reported on either the medical index or sleep questionnaire, or discovered after completing the vertical writing test.
2. The two-week sleep log was sent back filled out haphazardly.
3. Stable bed times were not possible due to work schedules.
4. Baseline screening polysomnography revealed evidence of myoclonus, excessive awakenings, sleep respiration disturbance or inability to sleep with electrode array.
5. Sleep latency was found on the MSLT of less than five minutes.

Nine subjects met the initial requirements for participation in the nighttime studies. One was excluded due to mean sleep latencies on the MSLT of less than five minutes. Eight subjects met the requirements for the MSLT studies. One subject dropped out citing unavoidable schedule conflicts. The nighttime experiments ultimately involved eight normal subjects, (5M, 3F, mean age 26).

The daytime studies involved seven normal subjects, (3M, 4F, mean age 27).

EXPERIMENTAL PROTOCOL

The experimental design required the subjects to spend two three-night periods of two one-day periods in the sleep laboratory. Each of the periods was separated by four or more days. The two three-night periods consisted of one adaptation night followed by either two nights when the bed was in motion or two stationary nights. The first adaptation night involved the monitoring of polysomnographic (PSG) sleep parameters for sleep pathologies such as sleep apnea, nocturnal myoclonus, excessive awakenings, or insomnia. The first three-night period was always conducted prior to either of the daytime periods. The two one-day periods consisted of multiple sleep latency tests, one given when the bed was in motion and the other given when the bed was stationary. The condition order, motion or stationary, was counterbalanced between subjects, such that half of the subjects were studied while the bed was in motion on the first three nights and the other half were studied while the bed was stationary on the first three nights. The MSLT condition was also counterbalanced between subjects. The condition order was randomly determined by drawing numbers from a hat.

LABORATORY PROCEDURES

Subjects were requested to come to the laboratory at least two hours prior to any session. All subjects, during both night and day

studies, were alone in the laboratory except for the continuous presence of the investigator.

Subjects were asked to stabilize their sleep schedule for three days prior to any laboratory session by adhering to an assigned bedtime. The assigned bedtime was the median lights-out time recorded from the two-week sleep log.

Continuous polygraphic monitoring of the standard sleep parameters of EEG, EOG, EKG and chin EMG were conducted on all six nights and both days. One channel was used to record oscillations of the swing, using a measured current offset reostat. Electrode impedances were only accepted under 20 Kohms except for scalp electrodes, which were accepted at only 10 Kohms or less. A ground electrode was applied to the forehead. Recordings were made on a Grass model 78D polygraph.

Night Studies

Two hours prior to bedtime, subjects were asked to prepare for bed, change clothes, brush teeth, etc. Total night recording time was set for eight hours for all subjects. A bedtime questionnaire was filled out by the subject before retiring and a morning questionnaire was filled out immediately upon awakening in the morning. Electrodes were then applied in preparation for the night recording.

The following polygraph montage was employed for all six recording nights, with the exception of channels 8, 9, and 11, which were used only during the adaptation recording. The EEG montage is based on the international 10-20 system (IFSECN, 1958).

CHANNEL	DERIVATION	LOW	HIGH	SENSITIVITY
1.	BED			
2.	C4/A1, A2 central EEG	0.3	60	7.5uV/mm
3.	02/A1, A2 occipital EEG	0.3	60	7.5uV/mm
4.	LOC/A1, A2	0.3	60	7.5uV/mm
5.	ROC/A1, A2	0.3	60	7.5uV/mm
6.	LOC/ROC	0.3	60	7.5uV/mm
7.	Chin EMG	10	60	1.0uV/mm
8.	LAT/RAT leg EMG	10	90	1.0uV/mm
9.	Airflow combined	0.1	30	5.0mV/cm
10.	EKG	0.1	90	2.0mV/cm
11.	EMG intercostal	30	60	1.0uV/mm

The paper was run at a speed of 10mm/sec., and 60 cycle notch filters were used on all amplifiers. Channels were calibrated such that a 50 microvolt pulse registered as a one centimeter pen deflection.

Day Recordings

Multiple sleep latency tests were conducted during the two one-day periods. Subjects were asked to come to the laboratory as soon as possible after awakening from a stabilized eight-hour night's sleep. Electrodes were applied and the polygraph calibrated

according to the montage normally used during the nighttime recordings specified above. The series of five-nap opportunities comprising the MSLT were begun two hours after the subject's arising time. At the start of each nap the lights were turned off, and the subject was told to "go to sleep". Subjects were allowed to remain in bed until ten minutes of sleep had accumulated or twenty minutes had elapsed, whichever came first. Immediately after arising from the nap, regardless of the PSG results, the subject was asked to answer verbally four questions:

1. Did you sleep?
2. If so, how long did it take you to fall asleep (in minutes)?
3. How many minutes did you sleep?
4. Did you dream?

The subject was allowed to move around freely between naps, but was not allowed to sleep or spend time in bed.

Starting thirty minutes prior to each nap opportunity, subjects were asked to complete the following scales and psychometric tests:

1. The Standord Sleepiness Scale - A seven point scale used to assess subjective sleepiness (Hoddes, 1973). (Appendix B).
2. The Wilkinson Addition Test - A performance test used to assess alertness applicable to a task (Wilkinson, 1968).
3. The Profile of Mood States - An assessment of mood (Pillard, 1967).

4. The Symbol Substitution Test - A subset of the Wechsler adult intelligence scale, used to assess ability to orient to a task (Wechsler, 1955).

Upon completion of each nap, subjects rated their mood and subjective sleepiness again.

ASSESSMENTS

All nocturnal and MSLT recordings were scored by a certified polysomnographic technician. All identifying data was removed from the recordings and replaced with codes, to ensure that the scorer was blind to the condition of the run; motion or stationary, and night or day number.

MSLT data were also scored by the investigator for particular measures. Records were scored using conventional criteria (Rechtschaffen and Kales, 1968), and by REM period techniques developed at Montefiore Sleep/Wake Disorders Center.

Nocturnal recordings were scored for the following variables:

a) Total sleep time: the sum of all sleep epochs.
b) Sleep latency: the time from lights out to the first four consecutive thirty-second epochs of any sleep stage.
c) Total recording time: the time between lights out and lights on.
d) Percent stage 1.
e) Percent stage 2.
f) Percent stage 3.
g) Percent stage 4.

h) Percent stage 5.

i) Latency to REM: the time from sleep onset to the first thirty-second epoch of REM sleep.

j) REM density: REM density was defined as the number of eye movements per REM period divided by the number of minutes of the same REM period.

k) Number of REM periods per night.

MSLT data were scored for the following variables:

a) Total recording time: the time between lights out and lights on.

b) Sleep latency: the time from lights out to sleep onset, or twenty minutes, whichever came first. Sleep onset defined as the first four consecutive epochs of stage 1, or any single epoch of any other stage of sleep.

c) Total sleep time.

d) REM periods: used for exclusion criteria.

CHAPTER III

RESULTS

PRIMARY AND SECONDARY STATISTICS

All nocturnal and MSLT sleep recordings were scored by a certified polysomnographic technician blind to subject and condition. REM density measures on all nocturnal recordings were scored by the principal investigator.

Sleep stage data from nocturnal recordings were analyzed using standard SWDC programs on a Digital Equipment Corporation PDQ 1123 computer.

Nocturnal sleep stage parameters were averaged over the second two motion and the second two stationary nights. These averages were employed in comparing nocturnal PSG parameters in the two conditions.

Mean sleep latency on both the motion and stationary MSLT were calculated for each subject. If three epochs of consecutive sleep did not occur on a particular nap opportunity, a twenty-minute sleep latency was entered into the formula for computing the mean.

A paired T-test was used to compare within-subject differences between motion and stationary conditions on the mean sleep latencies of the MSLT.

2X2 repeated measures ANOVAs (condition by night) were conducted on the nocturnal sleep parameters, where condition was defined as motion versus stationary and night was defined as nights

two and three of the condition. The following variables were considered in the analysis sleep latency, percent stage 1, percent stage 2, percent stage 3, percent stage 4, percent stage REM, REM latency, sleep efficiency, REM density index, number of awakenings, and number of REM periods.

A 2X2 repeated measures ANOVA (condition by time), was also conducted on sleep latency, where time was defined as day versus night and condition was defined as motion versus stationary. Newman-Keuls post-hoc tests were run on all significant interactions.

A .05 rejection region was adopted for all statistical tests. T-tests were two-tailed unless otherwise specified. Analyses were carried out on a Amdahl 470V/8 computer using the BMDP4V program.

NOCTURNAL SLEEP PARAMETERS

The two conditions, motion versus stationary, did not differ significantly with respect to Sleep Latency (rocking mean = 14.25 [standard deviation = 11.86], non-rocking mean = 18.44 [18.44]) or REM Latency (rocking mean = 74.88 [31.21], non-rocking mean = 73.56 [21.01].

The number of awakenings per night also did not differ significantly between conditions (rocking# mean = 19.81 [6.15], non-rocking# mean = 17.88 [5.60], and neither did the total number of REM periods per night (rocking# mean = 9.25 [2.74], non-rocking# mean = 8.56 [1.67]).

The sleep efficiency, expressed in percent, of the two pooled rocking nights was almost identical to that of the two pooled non-rocking nights (rocking mean = 92.37 [3.21], non-rocking mean = 92.49 [3.79]). This high sleep efficiency was expected from the population of young healthy normals used in this study.

The percentage of time spent in stage 3 and stage 4 sleep was virtually identical in the two conditions (stage 3 rocking mean = 9.78 [2.93], stage 3 non-rocking mean = 9.04 [2.52]; stage 4 rocking mean = 6.32 [5.03], stage 4 non-rocking mean = 6.47 [5.03]. There was a non-significant increase of Stage 1 sleep on the rocking nights (rocking mean = 4.66 [1.60], non-rocking mean = 3.56 [1.57]), but a non-significant decrease during the second night of both conditions when compared to the first night of either rocking or non-rocking (first night condition mean = 4.37 [1.68], second night condition mean = 3.85 [1.64]).

There was a significant difference in the distribution of stage 2 sleep ($F [1,7] = 17.63$, $p < .01$). The non-rocking nights yielded a higher percentage of stage 2 sleep than the rocking nights (rocking mean = 51.82 [4.81], non-rocking mean = 54.19 [4.30]).

Subjects exhibited a significantly higher REM density index on the rocking nights than on the non-rocking nights ($F [1,7] = 12.21$, $p < .05$). The REM density index mean for the rocking nights was 4.79 [1.53], and 3.75 [0.97] for the non-rocking nights. The index remained approximately the same for both nights under each condition. The percent of stage REM sleep did not difference between conditions or between nights.

Unexpectedly, a significant night by condition interaction was found with respect to sleep latency ($F [1,7] = 7.77$, $p < .05$). The mean sleep latency, in minutes, for the first night of rocking was 18.00 [13.78], and 10.50 [8.91] for the second night of rocking. For the first night of non-rocking the mean was 15.39 [10.79], and 21.50 [20.81] for the second night of non-rocking. However, the Newman-Keuls post-hoc test found no significant difference between these means.

DAYTIME SLEEP PARAMETERS

A paired student's t-test was used to compare within subject differences between rocking and non-rocking conditions on the mean sleep latency of the MSLT. Mean sleep latency on the MSLT in the rocking condition was 10.5 minutes and 12.3 minutes when the bed was not rocking. This difference failed to reach statistical significance, given the small sample size of six subjects.

MSLT performance tests yielded no significant differences. The SSS and Profile of Mood States administered during the MSLT were also not different under condition.

SUBJECTIVE REPORT

Subjects reported no adverse side effects following a night on the rocking bed. Subjects did however, verbally report an improvement in their quality of sleep and an increase in their dream recall. Those subjects who "rocked" first reported disappointment in their night's sleep following the first night of non-rocking.

Table 1

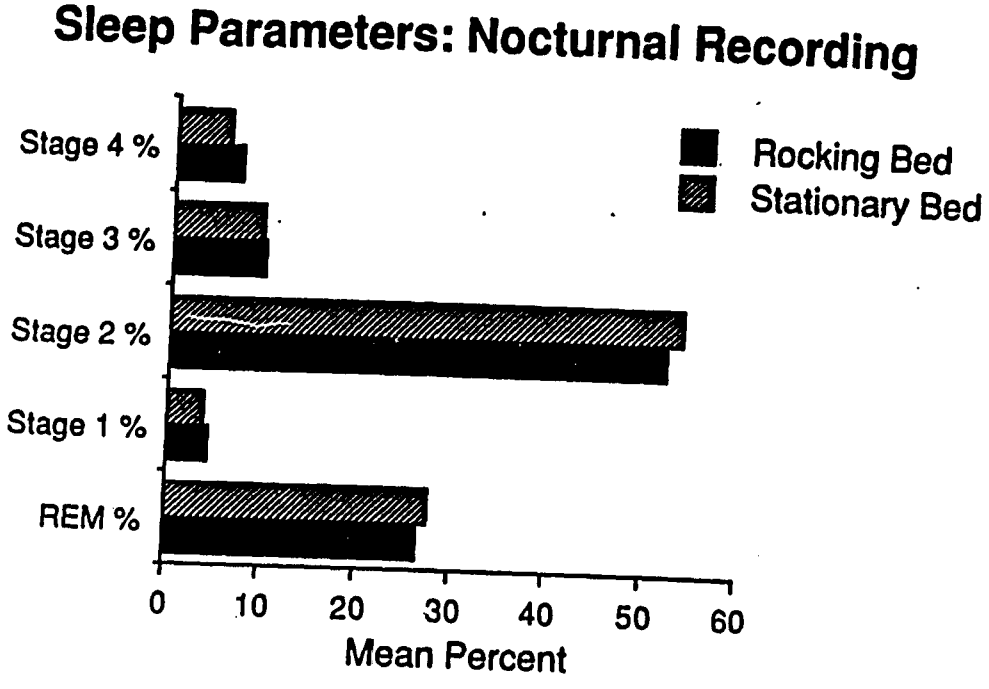
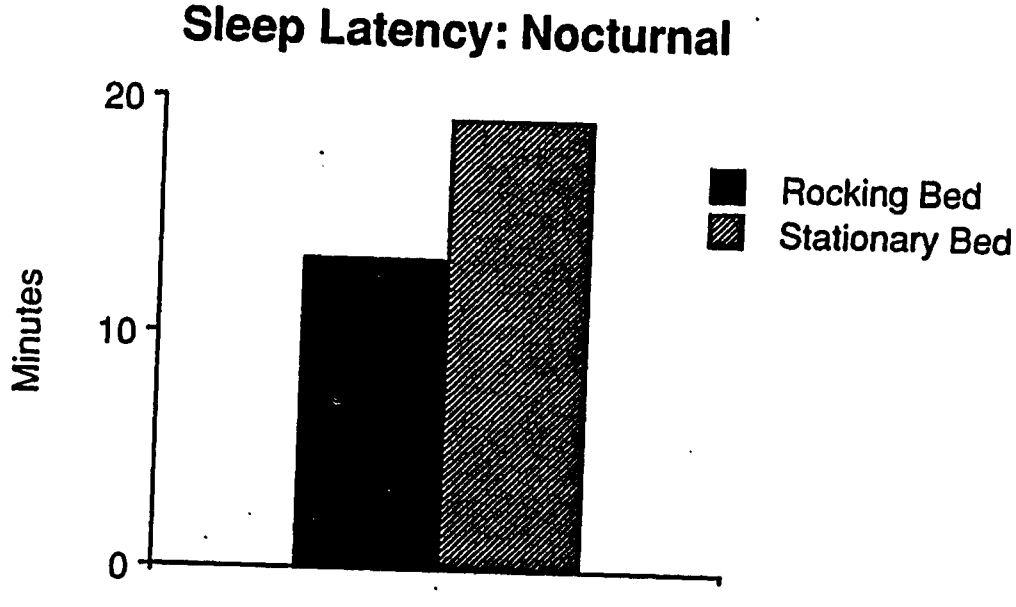


Table 2



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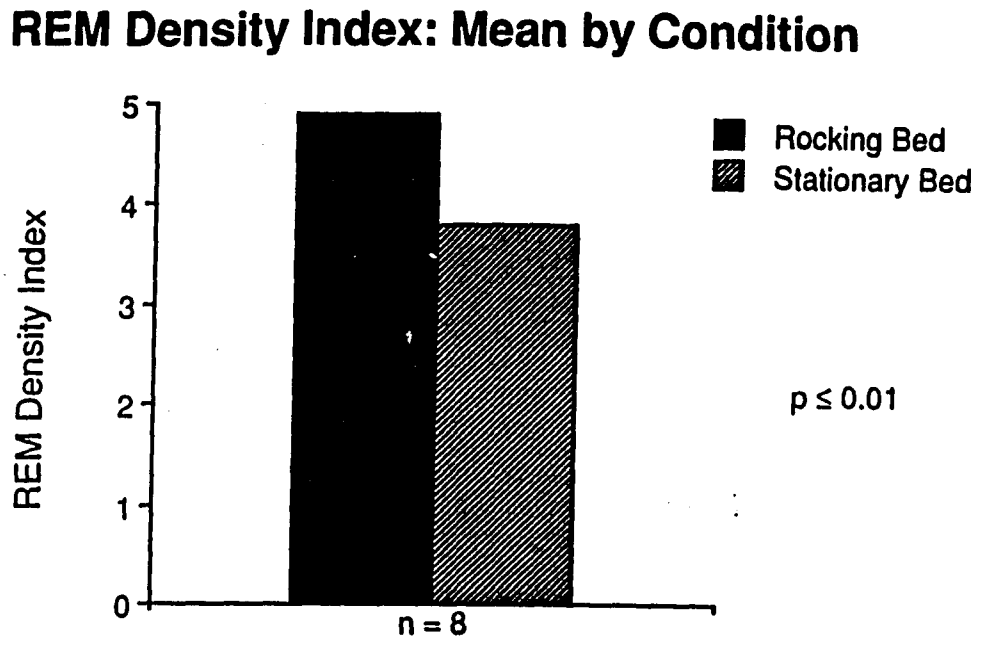

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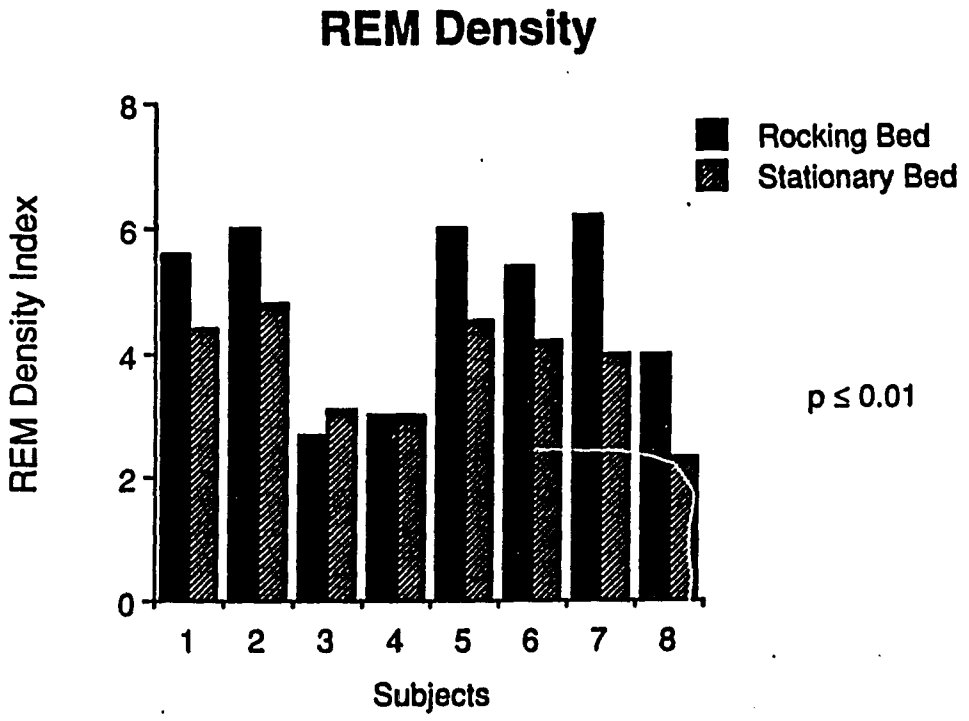

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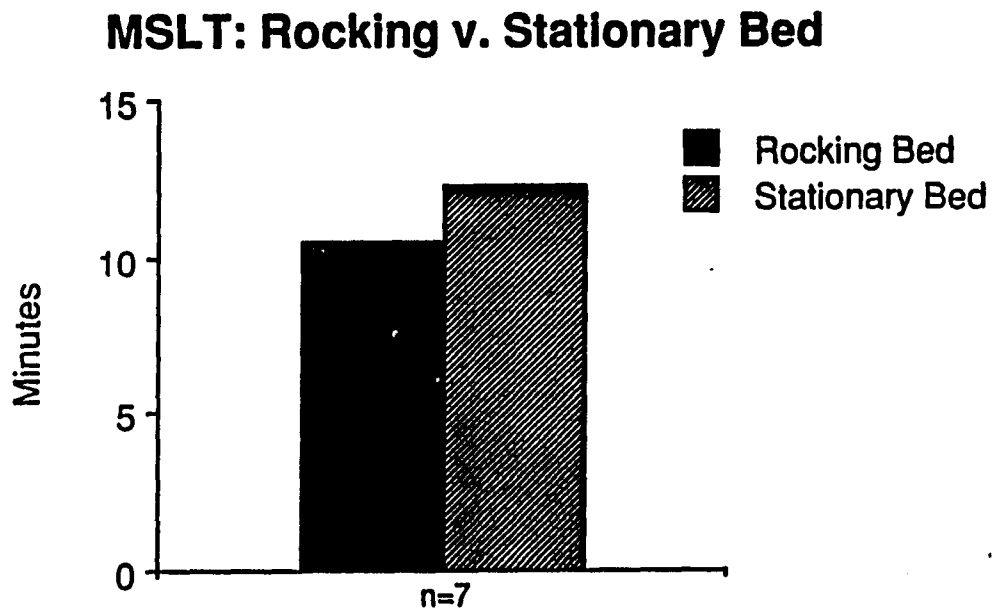

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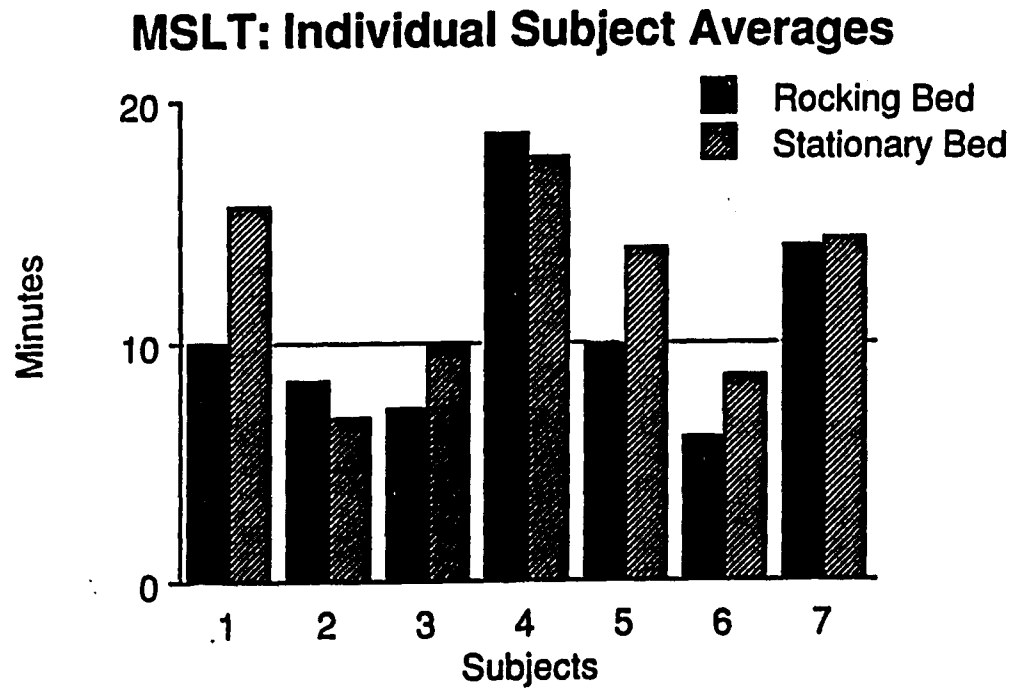

Table 7

Sleep Latency

BMDP4V Condition x Night ANOVA on LATENCY

CELL STATISTICS

FACTOR LEVEL
Cond Rock
==>

FACTOR	LEVEL	COUNT	MEAN	STDERROR	STD_DEV	WTD_MEAN	MAXIMUM	MINIMUM
Night	Second	8	18.00	4.873	13.78	18.00	35.00	1.000
	Third	8	10.50	3.151	8.912	10.50	28.00	0.0

FACTOR LEVEL
Cond NoRock
==>

FACTOR	LEVEL	COUNT	MEAN	STDERROR	STD_DEV	WTD_MEAN	MAXIMUM	MINIMUM
Night	Second	8	15.38	5.937	16.79	15.38	55.00	3.000
	Third	8	21.50	7.356	20.81	21.50	65.00	2.000

Table 8

Percent Stage 1

BMDP4V Condition x Night ANOVA on STAGE 1

LEVEL 1 MARGINALS

FACTOR	LEVEL	COUNT	MEAN	STDERROR	STD_DEV	WTD_MEAN	MAXIMUM	MINIMUM
Cond	Rock	16	4.662	0.4009	1.604	4.662	7.400	2.600
	NoRock	16	3.569	0.3931	1.572	3.569	6.600	0.7900
Night	Second	16	4.377	0.4203	1.681	4.377	7.400	1.970
	Third	16	3.854	0.4116	1.646	3.854	6.600	0.7900

Table 9

Percent Stage 2

BMDP4V Condition x Night ANOVA on STAGE 2

LEVEL 1 MARGINALS

FACTOR	LEVEL	COUNT	MEAN	STDERROR	STD_DEV	WTD_MEAN	MAXIMUM	MINIMUM
Cond	Rock	16	51.82	1.205	4.818	51.82	60.05	44.82
	NoRock	16	54.19	1.076	4.304	54.19	60.58	45.70
Night	Second	16	53.72	1.253	5.014	53.72	60.05	44.82
	Third	16	52.29	1.076	4.302	52.29	60.58	45.70

Table 10

Percent Stage 3

BMDP4V Condition x Night ANOVA on STAGE 3

LEVEL 1 MARGINALS

FACTOR	LEVEL	COUNT	MEAN	STDERROR	STD_DEV	WTD_MEAN	MAXIMUM	MINIMUM
Cond	Rock	16	9.785	0.7334	2.933	9.785	15.62	4.400
	NoRock	16	9.047	0.6313	2.525	9.047	12.70	4.080
Night	Second	16	9.561	0.7561	3.024	9.561	15.05	4.080
	Third	16	9.271	0.6165	2.466	9.271	15.62	5.650

Table 11

Percent Stage 4

BMDP4V Condition x Night ANOVA on STAGE 4

LEVEL 1 MARGINALS

FACTOR	LEVEL	COUNT	MEAN	STDERROR	STD_DEV	WTD_MEAN	MAXIMUM	MINIMUM
Cond	Rock	16	6.329	1.260	5.039	6.329	15.05	0.0
	NoRock	16	6.474	1.258	5.032	6.474	16.25	0.0
Night	Second	16	6.002	1.215	4.860	6.002	14.32	0.0
	Third	16	6.801	1.293	5.173	6.801	16.25	0.0

Table 12

Percent REM

BMDP4V Condition x Night ANOVA on REM

LEVEL 1 MARGINALS

FACTOR	LEVEL	COUNT	MEAN	STDERROR	STD_DEV	WTD_MEAN	MAXIMUM	MINIMUM
Cond	Rock	16	27.41	1.136	4.545	27.41	36.00	19.52
	NoRock	16	26.72	1.051	4.206	26.72	33.45	18.51
Night	Second	16	26.34	1.064	4.255	26.34	36.00	19.52
	Third	16	27.79	1.100	4.401	27.79	33.45	18.51

Table 13

REM Latency

BMDP4V Condition x Night ANOVA on REMLAT

LEVEL 1 MARGINALS

FACTOR	LEVEL	COUNT	MEAN	STDERROR	STD_DEV	WTD_MEAN	MAXIMUM	MINIMUM
Cond	Rock	16	74.88	7.803	31.21	74.88	166.0	46.00
	NoRock	16	73.56	5.254	21.01	73.56	120.0	46.00
Night	Second	16	76.63	7.908	31.63	76.63	166.0	46.00
	Third	16	71.81	5.023	20.09	71.81	120.0	46.00

Table 14

REM Density

BMDP4V Condition x Night ANOVA on REMDENS

LEVEL 1 MARGINALS

FACTOR	LEVEL	COUNT	MEAN	STDERROR	STD_DEV	WTD_MEAN	MAXIMUM	MINIMUM
Cond	Rock	16	4.797	0.3826	1.530	4.797	7.300	2.470
	NoRock	16	3.757	0.2449	0.9795	3.757	5.700	2.310
Night	Second	16	4.249	0.3505	1.402	4.249	7.300	2.310
	Third	16	4.305	0.3457	1.383	4.305	6.890	2.310

Table 15

REM Periods

BMDP4V Condition x Night ANOVA on REMPERS

LEVEL 1 MARGINALS

FACTOR	LEVEL	COUNT	MEAN	STDERROR	STD_DEV	WTD_MEAN	MAXIMUM	MINIMUM
Cond	Rock	16	9.250	0.6862	2.745	9.250	13.00	6.000
	NoRock	16	8.563	0.4180	1.672	8.563	12.00	6.000
Night	Second	16	8.688	0.5753	2.301	8.688	13.00	6.000
	Third	16	9.125	0.5692	2.277	9.125	13.00	6.000

Table 16

Sleep Efficiency Percentage

BMDP4V Condition x Night ANOVA on EFF

LEVEL 1 MARGINALS

FACTOR	LEVEL	COUNT	MEAN	STDERROR	STD_DEV	WTD_MEAN	MAXIMUM	MINIMUM
Cond	Rock	16	92.37	0.8044	3.217	92.37	97.41	85.30
	NoRock	16	92.49	0.9497	3.799	92.49	95.67	83.49
Night	Second	16	92.35	0.9440	3.776	92.35	96.49	83.49
	Third	16	92.51	0.8107	3.243	92.51	97.41	85.41

Table 17

Percentage of Awakenings

BMDP4V Condition x Night ANOVA on WAKES

LEVEL 1 MARGINALS

FACTOR	LEVEL	COUNT	MEAN	STDERROR	STD_DEV	WTD_MEAN	MAXIMUM	MINIMUM
Cond	Rock	16	19.81	1.539	6.156	19.81	32.00	9.000
	NoRock	16	17.88	1.402	5.608	17.88	30.00	9.000
Night	Second	16	18.75	1.233	4.933	18.75	30.00	11.00
	Third	16	18.94	1.714	6.855	18.94	32.00	9.000

CHAPTER IV
DISCUSSION

This study assessed the effects of otolithic vestibular stimulation on selective nocturnal sleep parameters and on daytime nap sleep latencies in healthy adult normals. This population was specifically chosen in order to provide baseline information regarding the effects of linear acceleration on the sleep architecture and sleep efficiency of normal sleepers. This information is currently unavailable on human adult subjects. Normative sleep data on healthy young human subjects under non-stimulation conditions is readily available and provided the normal data against which changes in the variables in question were measured. A normal population was also necessary in order to determine whether the type and amount of vestibular stimulation selected for this study would produce any adverse effects on the sleep parameters measured. The intention is to design protocols assessing the effects of otolithic vestibular stimulation using subject populations suffering from pathological sleep disturbances such as insomnia or narcolepsy.

The expected findings of this study were based on vestibular stimulation studies conducted with both humans and animals, where the mode of stimulation was not selectively otolithic. It was therefore difficult to predict what aspects of sleep efficiency and sleep quality would be altered as a result of specific otolithic stimulation. It was also difficult to assess whether the changes found

were specific to the manner of stimulation used or simply variations that did not correspond to findings found in nonselective vestibular stimulation studies. In order to clarify the results of the present study, the results presented here are assumed to be directly attributable to the otolithic stimulation.

Very few significant differences were found between the rocking and non-rocking nights, although non significant changes were often in the expected direction. Improvements in sleep quality were not expected from the population of healthy normal adults studied, and basically no improvements were found. Sleep efficiency did not vary significantly between conditions, but a ceiling effect may have been in operation which prevented significant improvement in this population since mean sleep efficiency did not fall below ninety-two percent at any time during the study.

A reduction in the number of nighttime awakenings was expected, based on the findings of Barthlen (1985), and Hirsh and Tauber (1973). Hirsh and Tauber were able to elicit fewer arousals by cradle rocking newborn infants, and Bathlen found a significant decrease in the number of arousals of chronic insomniacs after all night rocking on a "swing bed". The results found in the present study showed no significant difference in the number of awakenings between rocking and non-rocking nights, although the mean number of awakenings less than five minutes long was higher on the rocking nights, particularly during REM sleep. The number of awakenings did not differ significantly from the first to the second night of non-rocking, but did continue to rise again non-significantly, on the second night of rocking. The number of REM periods per night

followed this exact sequence of change indicating that the increased number of REM periods found on the rocking nights may have contributed to the higher number of awake-nings found on the same rocking nights. It would be hoped that these number counts would average out between conditions, given more study nights and a larger subject population. In addition, it is important to note that sleep efficiency was not affected by number of awakenings or the number of REM periods on either night, and in fact, showed a minimal although non-significant increase on the second rocking night and a comparable amount of non-significant decrease on the second non-rocking night. A question to consider is whether this minimal change would be amplified with a population such as the elderly where nighttime awakenings tend to disrupt sleep more than in normal controls.

Eight-hour sleep periods were preset for all subjects for all nights so total sleep length was not different between condition. It was noted, after surveying the data, that more subjects had to be awakened from REM sleep following an eight-hour rocking night than following a night of non-rocking. In addition, it was also noted that fewer subjects were awake at the predetermined lights-on time following a night of rocking, and more subjects recalled vivid dreams after a rocking night.

Considering the population studied, significantly more changes were expected with regard to sleep architecture than to sleep quality. given the high sleep quality of normal adults, changes brought about specifically by the otolithic stimulation would have to appear within the context of sleep architecture.

Based on the findings of previous studies, differences in REM sleep were most likely to be altered. Ornitz (1972), found that non-specific vestibular stimulation during sleep in young children resulted in a tendency to longer REM sleep periods. McGinty, (1985), found a dramatic in REM percentage under a rocking condition. The present study however, found no significant changes in either increases or decreases of REM percentage between night or condition. Findings from the present study did show a non-significant increase in REM percentage on the rocking nights as well as a non-significant increase in REM percentage on night two of rocking as compared to night one. It is possible to speculate that with an extended series of rocking nights REM percentages would continue to increase by occupying sleep time left by a minimal decrease in stage 4 sleep which was found on the rocking nights. REM periods were not measured for length in this study, but it was assumed that they did not increase in length on the rocking nights based on the following findings: there was virtually no difference in total percentages, and there were slightly more although non-significant REM periods on the rocking nights. It would appear that REM sleep percentages remained the same but were divided into more periods, not longer ones. REM latency did not differ between the two conditions, so it is assumed that the division into more REM periods occurred later in the night's sleep and therefore may account for the non-significant increase in the number of awakenings considering that an awakening is often seen at either the beginning or the end of a REM period.

Only one significant difference in sleep staging between condition was found in this otolithic vestibular stimulation study.

Barthlen (1985), had found an increase in stage 3 sleep under the "swing bed" condition, but this increase was not found in the present study. Stage 3 and stage 4 sleep were not significantly different under the rocking and non-rocking conditions. Stage 1 sleep was found to slightly increase, although non-significant, under the rocking condition, but this may have been due to the number of awakenings. It should be noted that subjects in the "swing bed" study were chronic insomniacs and that the experimental design used allowed subjects to alternate swinging and non-swinging nights. Considering these major differences in design and subject population, any comparison between results of the present study and the "swing bed" study are speculative.

Stage 2 sleep was significantly decreased in the present study on the rocking nights. In light of the fact that the other sleep stages remained approximately equal, it is possible again to speculate that REM sleep may be occupying the sleep time left by the decrease in stage 2 on the rocking nights. The minor, non-significant stage 1 increase added to the time spent in the awake state on the rocking nights may have been sufficient to prevent REM percentages from showing more than a slight increase. Interestingly, both the minor, non-significant increases in REM percentages and stage 1 sleep continued into the second night of rocking stimulation, while the stage 2, non-significant decrease was not sustained.

An index of REM density was calculated for the two rocking and non-rocking nights and a significant difference was found between conditions. No other parameters of rapid eye movements were assessed except for the number appearing in each REM epoch.

This finding is somewhat confirmed by the study of Popeiano (1970) where it was proposed that the clustering of rapid eye movements during sleep reflect the phasic activity of central vestibular mechanisms in the modulation of sensory input and motor output. However, EOGs were recorded and paired so that horizontal, vertical and oblique eye movements could be obtained and differentiated from several types of artifact simulating rapid eye movements. Electrode (see Fig. 1) placements allowing for these types of measurements were not implemented in the present study. Jongkees (1962) has pointed out that a parallel swing is a suitable apparatus with which to stimulate the otolithic system but that it should be combined with an electronystagmograph in order to determine otolithic function. All subjects in the present study were tested for vestibular pathology and found normal so no attempt attempt was made to delineate types of eye movements in order to assess otolithic function. A selective number of subjects had compensatory eye movements recorded on the rocking bed to ensure that the frequency chosen was indeed resulting in otolithic stimulation. AS a result of the decision to record only horizontal eye movements on the study and control nights it is impossible to make anything other than speculations as to the significance of the REM density effect. No other studies have been conducted measuring the REM density index during vestibular stimulation. Even taking into account major differences in EOG recording, it is interesting to note findings by Ornitz et al. (1973), that were similar to those found in the present study. Normal young children under horizontal stimulation exhibited fewer REM periods per night, an increased number of awakenings,

higher REM percentages and longer REM periods than those found in the present study. Normal young children under horizontal stimulation exhibited a significant increase in the total number of individual eye movements per minute of REM sleep time and an increased number of awakenings. Ornitz also reported that the phasic activity of REM sleep may be related to serotonergic mechanisms and that 5-hydroxy-tryptophan is the only pharmacologic agent found to increase rapid eye movement activity in man. IT is then, at least feasible to suggest that in the present study otolithic vestibular stimulation taps the sleep/wake center by stimulating output of serotonin via its precursor.

Only one significant interaction was found in the present study and that was of sleep latency (condition X night). mean sleep latency was significantly decreased on the second night of rocking from the first and increased on the second night of non-rocking from the first. Although this finding was not significant on the post-hoc test, the changes were in the expected directions. If subjects were habituating to the rocking stimulation sleep latency would be expected to increase on the second night or remain the same. This second night increase was clearly seen on the non-rocking nights. Subjects actually had a mean sleep latency of seventeen-and-one-half minutes less on the second night of rocking as compared to the first.

Sleep latency was the only variable of interest on the daytime recordings and it did not differ significantly between conditions. Mean sleep latency was reduced however, on the MSLT. This is an important finding when viewed in the context of the population

studied. Mean MSLT sleep latency for a normal young adult after an eight-hour night is approximately fifteen minutes depending on the time of the nap. The fact that sleep latencies were reduced at all in the same subjects when naps were conducted under the otolithic stimulation condition as compared to latencies elicited under no stimulation conditions is indicative of an effect. All experimental conditions were kept constant between the two days of testing, so it may be assumed that the reduced sleep latencies found on the MSLTs were due solely to the effects of the stimulation.

This study has addressed the question of whether or not it is feasible to physically induce or enhance sleep by means of a parallel swinging bed that selectively stimulates the otolithic vestibular system. The results clearly indicate that this manipulation does not adversely affect the sleep of normal subjects.

COMMENTS

Due to the fact that this was a preliminary as well as innovative study, an attempt was made to discuss the results in a speculative as well as a statistical manner. It is suspected that a power analysis would yield significance concerning a number of different variables, given an increase in sample size. However, the main thrust of the present study was to establish sufficient baseline data under the stimulation condition, and this was achieved with the number of subjects studied. It would be prudent at this time to select subject populations with definitive sleep disorders for further investigation.

APPENDIX A

INFORMED CONSENT

I,_____, have been asked by Suzanne Woodward to volunteer to be a subject in a research study designed to look at the effects of movement during sleep, in the form of a parallel swing, on sleep.

I have been told that my normal health condition makes me a suitable subject to take part in this research. I have been told that my participation in this study is expected to last for six nights and two days. I have been told that the procedure to be followed in this study involves a standard sleep recording which involves placing surface sensors on the head which monitor signals from the brain, eye movements and chin movements. In addition, heart beat will also be monitored with surface sensors. This recording will be conducted while lying on a parallel swing. The procedure listed above may not be considered to be experimental.

I have been told that there are no known risks associated with this study.

I have been told that I may reasonable expect to receive no direct benefit from my participation in this study. It it hope though, that the results of this study may be of benefit to others in the future.

I have been told that no published or unpublished report or visual or oral presentation of any aspect of this study will include any material that will permit identification of me as a subject to any person other than to the named collaborators of this study, to the sponsors of the research, to the appropriate federal agencies, and to the Institutional Review Board for the Protection of Human Subjects (IRB).

I have been told that should I have any questions or concerns that I may call Dr. Michael J. Thorpy, at 920-4842 or the Office of Research Administration at 920-4151.

I have been given the opportunity to ask any questions I wish regarding the purposes and procedures of the study in which I will participate. I have been told that I may refuse to participate or discontinue my participation in the study at any time without in any way prejudicing my future treatment or my future relations with the hospital or its doctors.

Signed:_____ Date:_____

APPENDIX B

Stanford Scale (SSS)

Name _____

Day and Date_____

Time right now _____:_____ AM PM (circle one)

Please answer this question by circling one answer:

This is the 1st 2nd 3rd 4th 5th 6th time today that I have answered this questionnaire/

Instructions: Please choose the number of the statement which best describes how you feel right now.

1. Alert. Wide awake. Energetic

2. Functioning at a high level, but not at 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.

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