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AN INVESTIGATION INTO SOME ASPECTS OF HUMAN SLOW WAVE SLEEP.

BY

BRYANIE SARA SHACKELL, BSC, SRN.

A DOCTORAL THESIS

SUBMITTED IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE AWARD OF:

DOCTOR OF PHILOSOPHY OF THE LOUGHBOROUGH UNIVERSITY OF TECHNOLOGY

MARCH 1988.

FOR MY PARENTS.
New every morning is the love
Our wakenIng and uprising prove;
Through sleep and darkness safely brought,
Restored to life and power and thought.
ACKNOWLEDGEMENTS.

I would like to express my sincere gratitude and appreciation to everybody who kindly devoted their time and energy to assisting and encouraging me in the execution of this study.

Firstly I owe my thanks to all the students and staff of the University, and the individuals from the local community for their enthusiasm, cooperation and continuing interest, without which the work could not have been completed.

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Finally, but by no means leastly, I am endebted to my parents for their eternal patience, support and encouragement in the perpetuation of this and all my work. I am also very grateful both to my sister Annabel, and to Henry, for their company and friendship throughout the preparation of this thesis.
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The thesis describes investigations into two contrasting aspects of SWS. The first is a laboratory based study of the effects of passive heating on the subsequent SWS of six healthy subjects, and the second employs home sleep recording techniques to investigate the prevalence and characteristics of the 'alpha sleep anomaly' in volunteers from the local community.

The passive heating study was designed to compare the effects of a fixed dose of body heating given at 1700hrs or 2100hrs on subsequent nighttime sleep, and to examine how pre-treatment with aspirin influences heating effects on SWS. Significant increases in SWS followed the late bath with placebo condition, but these effects were counteracted by aspirin administration. Passive heating at 1700hrs appeared to have comparatively less effect on SWS than the late bath.

Investigations into the prevalence and characteristics of the 'alpha anomaly' of SWS in fifteen subjects with musculoskeletal discomfort and nineteen symptom-free subjects, found a greater mean quantity in the groups with discomfort, although the overlap in distribution of mean percentages of alpha in SWS indicated that this 'anomaly' was not directly related to the appearance of these symptoms. Frontal EEG 'alpha-like' activity or 'kappa' activity was also more prevalent in subjects with discomfort, and may represent processes of NREM sleep mentation in individuals predisposed to psychological stress and anxiety. SWS 'alpha' activity is suggested to present as a normal and individually constant characteristic which is more prevalent in subjects showing a psychological profile that shows a greater tendency towards anxiety and depressive states, and an increased frequency of somatic complaints.
AN INTRODUCTION INTO SOME ASPECTS OF HUMAN SWS.

CHAPTER 1.

INTRODUCTION.
AN INVESTIGATION INTO SOME ASPECTS OF HUMAN SLOW WAVE SLEEP.

1. INTRODUCTION.

This thesis describes investigations into two aspects of SWS. The first is a laboratory based experiment involving a manipulation of SWS in healthy student volunteers, and is a continuation of work previously carried out in this laboratory. The effects of body heating on subsequent nighttime sleep are reported, with reference to time of day effects and the influence of daytime administration of aspirin.

The second study investigates the prevalence of an 'abnormal' aspect of SWS in patients and volunteers recorded in their homes. It is a study of the relationship of the NREM sleep 'alpha' anomaly to musculoskeletal discomfort of unknown origin. Previous authors (Smythe and Moldofsky, 1977, 1978) have suggested that 'alpha-delta' sleep represents a specific disturbance of sleep, which is related to the appearance of symptoms in the 'Fibrositis' syndrome.

The precise functions of sleep stages 3 and 4, which comprise human slow wave sleep (SWS) are not yet defined. Current knowledge of SWS indicates that some restoration of function takes place during sleep, which may be associated with body restitution (Oswald, 1970), or cerebral restitution (Horne, 1986) or both.

Sleep deprivation experiments and the effects of sleep disorders confirm the importance of SWS to performance. Clues to sleep function come from investigating factors which increase or decrease SWS, and from consideration of the functional deficits which result from the adverse effects of psychophysiological characteristics.
1.1. NOCTURNAL SWS.

SWS is particularly prominent during the first three hours of nocturnal sleep, and characterizes the first two NREM sleep cycles (Williams, Agnew and Webb, 1966). The quantity of stage 4 sleep has been found to be directly related to length of time awake, previous time asleep, and age. A possible circadian influence has also been suggested, (Webb and Agnew, 1971) although there is little evidence for this.

Despite differences in habitual sleep length, the amount of nocturnal SWS remains remarkably stable (Benoit et al, 1980; Bliwise and Bergmann, 1987), although short sleepers may have slightly more stage 4 and less stage 3 due to easier transition into stage 4 sleep from stage 2 sleep (Webb and Agnew, 1970; Webb and Friel, 1971).

Sleep limitations increase the time awake, and result in a need for sleep recovery processes to adapt to a shorter sleep period. Reducing habitual sleep periods by 2.5 to 3 hours leads to increases in stage 4 sleep, although there is no stage 4 sleep deprivation (Dement and Greenberg 1966). Stage 4 sleep returns to baseline levels within five weeks of a maintained sleep reduction, without detrimental effects (Webb and Agnew, 1974). Other studies of reduction to 4.5 to 5.5 hours (Friedmann et al, 1977) and to 4 hours (Johnson and MacLeod, 1973) confirm that subjective fatigue is the limiting factor determining tolerance to sleep reduction.

1.2 SLEEP DEPRIVATION.

During recovery from the prolonged wakefulness of total sleep deprivation, approximately one third of lost sleep is recovered, containing 50% of lost SWS (80% of stage 4 loss, and a little less of stage 3 sleep) and only 40% of lost REM sleep (Horne 1984). Significant increases in stage 4 sleep are also found during the recovery from stage 4 sleep deprivation, indicating a requirement for sleep associated
with this type of EEG activity. (Agnew, Webb and Williams, 1964).

Following total sleep deprivation, Lubin et al. (1974) reported that deprivation of stage 4 sleep during recovery does not impair recuperation, but as increases in stage 2 sleep may compensate for the stage 4 sleep loss, the quantity of sleep may be more important than the type of sleep.

According to Borbely (1981), significant increases in the power density of stage 4 sleep are seen following 40.5 hours of sleep deprivation. As the intensity of a sleep process is indicated by the prevalence of EEG slow waves, the effects of sleep deprivation on sleep are inadequately reflected by the time spent in various sleep stages. Akerstedt and Gillberg (1986) report that the lost EEG power density is almost equal to that seen during recovery, and closely determines the duration of recovery sleep.

1.3. NAPS

Naps taken soon after a normal nights sleep contain little or no stage 4 sleep, but this amount increases as naps occur later in the day, (Feinberg, 1974). Due to the limitations of visual scoring, late daytime naps appear to result in a disproportionate loss of subsequent nocturnal stage 4 sleep, but the integrated amplitude of delta activity is conserved over naps and post-nap sleep so that the total delta equates with normal nocturnal levels, (Feinberg et al. 1985).

1.4 SLEEP EXTENSION

In subjects able to extend their sleep for 15 hours, SWS reappeared 12 hours after the time of sleep onset, and corresponded to times of decreased arousal (Gagnon and De Koninck, 1984), suggesting that SWS is not entirely dependent on prior wakefulness and previous time asleep, but may also be under a weak or 'damped' circadian control, or may be associated with decreasing REM sleep tendencies. These findings complicate the concepts of SWS as an index of
a sleep need due to prior wakefulness or an index of a restoration process, (Webb, 1986).

1.5. AGE AND SWS.

SWS occupies approximately 20% to 25% of sleep from the first year until the mid-twenties then declines to 13% by the mid-thirties and to 5% by the sixties, (Williams et al, 1974). As age advances, increased stage 1 sleep and wakefulness and decreased SWS result from diminishing control of the sleep processes.

In stage 4 of a child's sleep, delta waves may reach an amplitude of 400-500μV, whereas in the elderly they rarely exceed 200-250μV, and often fall below the scoring criterion of 75μV (Rechtschaffen and Kales, 1968). These changes are responsible for the apparent fall in SWS with age, although the actual number of delta waves drops to only 75% of the young adult level.

The major age related changes in sleep cycle pattern occur within the first NREM sleep cycle, which in younger age groups is dominated by stage 4 sleep. High levels of stage 3 sleep occur as stage 4 sleep weakens in intensity, whereas in the elderly, stage 4 sleep is already weakened, and stage 3 sleep appears early. Stage 3 sleep may represent a weakened or degraded version of stage 4 sleep, but does not show the same decline during the fifties, remaining similar to and sometimes slightly greater to that of younger age groups. As the first NREM sleep period shortens with age and shows less stage 4 sleep, the first REM sleep period occurs earlier and becomes longer, (Feinberg 1974).

1.6. EXERCISE AND SWS.

Body restitution hypotheses of SWS function predict that daytime exercise should result in an increased requirement for tissue repair and a corresponding rise in SWS.

Three studies of exercise and human sleep have supported this hypothesis, of which two suffered methodological defects. Six studies have reported negative
findings, but only three of these have used an adequate design, (Walker et al, 1978).

Shapiro et al, (1975) reported graduated increases in SWS after daily increases in exercise load according to estimated VO2 max. A progressive increase in SWS over the whole night sleep record was reported with progressively increasing fatigue. In a further study they reported initial increases in stage 4 sleep followed by second night increases in both stages 3 and 4, (Shapiro et al, 1981).

SWS increases following exercise were attributed to the extreme metabolic load following the increased energy expenditure of exercise. However, Bonnet (1980) concluded that sleep variables following sleep deprivation were not related to the increased energy expenditure. Twelve subjects expended the energy equivalent of 40 hours inactive sleep deprivation during a 20 mile march, but did not show any increase in subsequent SWS.

The report of Ryback and Lewis (1971) also opposes this hypothesis. They found significant SWS increases in subjects confined to bed for five weeks. The non-exercising group showed the greatest increase compared to those allowed to exercise. However, the one baseline night used for comparison showed lower than average SWS levels.

The body restorative function of SWS derives support from the largest daily surge of Human Growth Hormone (hGH) occurring at the onset of SWS. This hormone facilitates protein synthesis and promotes growth processes in the child. As most of the child's daily hGH release is confined to sleep, it has been suggested that SWS processes actively promote tissue growth and repair during sleep, (Dunleavy et al, 1974). In addition, Mac Fadyen, Oswald and Lewis, (1973) report that the increased metabolism of acute starvation is related to coincident increases in SWS which represent restoration of losses from body tissue reserves.

If exercise increases the wear and tear on the body during wakefulness, and hGH promotes tissue repair during SWS, then a corresponding increase in SWS and hGH release would be expected during subsequent nighttime sleep.
Adamson et al (1974) reported significant increases in nocturnal hGH release following variable increases in daytime exertion, but failed to observe an increase in SWS. However, Zir et al (1971) employed a controlled rigorous or light exercise routine for two groups of five fit subjects. Four of the ten subjects showed increased SWS, but only two of these, who underwent the light exercise condition, had an increased hGH peak.

1.7. SWS AND FITNESS

Fitness levels may be significant in determining the effect of exercise on SWS. Trained subjects are more likely to show whole night increases in SWS, whereas untrained subjects may show a transient elevation during the first part of sleep following acutely increased daytime exercise, (Horne, 1981). Although there was no effect, or a negative effect of exercise on SWS in the unfit subjects studied by Adamson et al, (1974) and SWS increases were of greater magnitude in the fitter of the two highly trained subjects studied by Shapiro et al (1975), both of these experiments suffered from methodological problems.

Following exercise, Griffin and Trinder (1978), found greater quantities of stage 3 sleep in fit subjects, but disturbed sleep and increased SWS latency in their unfit subjects. The lack of SWS increase in the unfit was partly attributed to the stress of unusual exercise or changes in daily routine. Unfit subjects were suggested to have more SWS than required for their activity level, which was able to absorb any additional restitutive requirements.

SWS increases in fit subjects may be associated with the physiological and functional adaptations that occur with fitness training. Shapiro et al (1984), assessed eight army recruits throughout their initial eighteen week training programme, and reported increases in percentage SWS, SWS duration and sleep efficiency with increasing fitness.
Torsvall, Akerstedt and Lindbeck (1984), studied the effects of increasing physical effort over three days in six fit men, culminating in a race on the third day followed by a day of no exercise. There was no significant change in SWS with increasing exercise, but the power density of the EEG increased and had decreased significantly by the fourth night. This increase could be attributed to the reduced and delayed REM sleep which was replaced mainly by stage 2 sleep in the third night.

These authors draw attention to the possible influence of diminishing delta amplitude in older age groups, which may affect the way in which SWS responds to exercise. Horne and Porter (1976) suggested that the metabolic effects of exercise early in the day may dissipate during the waking period, leaving little to restore during SWS. Torsvall, Akerstedt and Lindbeck (1984) presume that such a process of restoration during wakefulness may proceed particularly rapidly in well trained subjects.

The lack of exercise effect on the SWS of unfit subjects (Griffin and Trinder, 1978) is difficult to explain in conjunction with the body restitution theory. Unfit subjects would be expected to require greater restitution than fit subjects following non-habitual exercise.

Horne and Staff (1983), suggest that SWS increases seen in fit subjects are a result of their greater endurance and ability to tolerate prolonged rises in body temperature and sweat loss, which increase the problems of heat stress.

1.8. BODY HEATING EFFECTS ON SWS.

In the study by Horne and Staff (1983), eight trained subjects underwent high or low-intensity exercise or passive heating. Significant increases in stage 3 sleep followed high intensity exercise but a greater increase in stage 4 sleep followed passive heating. Later studies by Horne and Moore (1985), Shapiro, Bortz and Mitchell (1985), and Bunnell and Horvath (1985) supported this relationship.
High intensity exercise was suggested to have affected SWS by evoking high rates of thermal loading. Temperature rises which cause a brain temperature increase of 2°C lead to a 20% rise in brain metabolism (Siesjo, 1978), and may thereby increase the brain processes of wakefulness which influence subsequent SWS (Horne, 1984). An increase in the quality of demand on the cerebral cortex during wakefulness may therefore result in an increased demand for the restorative processes associated with SWS.

A day of exceptional variety and novelty was shown by Horne and Minard (1985) to increase stage 4 sleep and SWS over two recovery nights. In addition to the possible effects of behavioural arousal on SWS in this study, Horne and Walmsley, (1976) showed that high daytime visual load initially increased stage 3 and SWS (+4.6%) during the first recovery night and stage 4 and SWS (+6.8%) during the second. Similar visual loads also provoked greater stage 4 sleep increases than low load conditions during sleep deprivation, (Horne, 1976). The failure of Potter and Heron (1980), to reduce SWS by confining subjects to a perceptual deprivation chamber for up to seven days, might suggest that as cerebral metabolic rate is already near to maximal levels, even during relaxed wakefulness, (Horne, 1984) there is potential for SWS increase but reduction is unlikely.

1.9. EFFECTS OF SWS ON SUBJECTIVE WELL-BEING.

If SWS represents processes of cerebral restitution, then the effects of sleep loss should centre on the CNS.

The effects of total sleep deprivation are confined mainly to minor psychological and neurological decrements, without signs of reduced body tissue repair (Edwards, 1941; Horne, 1985). The most severe psychological effects of sleep deprivation were reported from studies which used one to four subjects. Patrick and Gilbert, (1896) who carried out the first sleep deprivation experiment, described visual hallucinations in one subject with a nervous temperament, whereas Berger and Oswald, (1962) described abnormal behaviour and rare cases of paranoid delusions in three of their six subjects.
Symptoms more commonly reported following sleep deprivation include those of behavioural irritability, suspiciousness, speech slurring, minor visual disturbances, (Horne, 1985), and reduced binocular convergence under high visual load conditions, (Horne, 1975).

Sensations of fatigue and 'heaviness' of the body are frequently reported during sleep deprivation, (Gulevitch, Dement and Johnson, 1966), however symptoms of physical discomfort with concern over physical complaints and changes in bodily feelings, may be attributed to loss of stage 4 sleep. Agnew, Webb and Williams (1967) found that subjects undergoing stage 4 sleep deprivation became withdrawn, less aggressive and tended towards hypochondriasis and depressive reactions.

Studies of the effects of sleep deprivation on body restitution have shown negative or inconclusive results, with few apparent changes in physiological stress indices of urinary corticosteroids, blood analysis, urinary catecholamines, heart rate and blood pressure. Apparent reductions in exercise endurance during deprivation are more likely due to reduced psychological motivation than to physiological deficits (Horne, 1983).

Heightened susceptibility to infection is often considered to be an effect of sleep loss, although prolonged wakefulness may itself be associated with psychological stress, known to suppress the immune response by elevating the output of corticosteroids. Although sleep processes may promote healing (Adam and Oswald, 1984), psychological factors are also important, as they may affect the response to and recovery from illness, (Baker, 1987).

1.10. SWS AND IMMUNE FUNCTION.

There is new evidence for a relationship between sleep and immune function. Moldofaky et al (1986) believe that NREM sleep is associated with an enhancement of the immune system, whereas Krueger et al (1986) have identified a muramyl peptide, Factor S, which can promote deep NREM sleep in rabbits through the release of Interleukin-1 an endogenous pyrogen.
1.11. **THE INTERACTION OF SWS DISTURBANCE WITH PSYCHOLOGICAL AND PHYSICAL WELL-BEING.**

Symptoms similar to those reported following sleep deprivation may also result from sleep disturbances and sleep disorders, which arise as a result of psychological stress and anxiety. Depending on the age, premorbid personality and the nature of the precipitating events, sleep complaints include difficulties getting to sleep, frequent nighttime awakenings and premature morning arousal. Sleep disturbances may interrupt the restorative role of SWS and give rise to symptoms of malaise and fatigue which complicate the clinical picture of the original disorder. Depression and anxiety may then disrupt sleep, creating more cause for concern.

Patients with chronic pain syndromes frequently complain of sleep problems, and those receiving anti-depressants often highlight a beneficial improvement in their sleep rather than their pain. Amongst pain clinic patients, poor sleepers report a greater pain intensity, and show more nervousness, irritability and depression than good sleepers with pain, (Pilowsky, Crettenden and Townley, 1985).

Sleep disturbance is one of the most frequent complaints of the emotionally disturbed person, poor sleepers show more emotional and somatic symptoms than matched good sleepers (Monroe, 1967). Moldofsky, (1978) suggests that a physiological arousal during SWS, which is triggered by an emotionally disturbing event, can initiate a self-perpetuating cycle of 'non-restorative' sleep, irritability, depression and anxiety, coupled with symptoms of lethargy, malaise and musculoskeletal discomfort.

Kramer, Roehrs and Roth, (1976), suggest that sleep serves as a mood regulator, and that overnight changes in mood are dependent upon the nature of the physiologic aspects of sleep one has obtained, and the type of mental content and dreams experienced.
Sleep mentation during SWS and other NREM sleep is qualitatively different from the dreaming of REM sleep, and is more closely related to daytime concerns and environmental features. Broughton (1968) considers that certain parasomnias of psychological origin (for example enuresis) may occur coincident with emotional conflicts expressed during this activity.

As a treatment for endogenous depression, total sleep deprivation, (Van den Burgh and Van den Hoofdakker, 1975) partial sleep deprivation, (Schilgen and Tolle, 1980) and REM sleep deprivation, (Vogel et al, 1977) have all been reported as effective but short lived. Recently, Parry and Wehr (1987) have reported the successful use of one night's sleep deprivation in premenstrual syndrome patients.

1.12. SUMMARY.

The functional significance of SWS is unknown, yet age, changes in the length, pattern, and cerebral demands of wakefulness, body temperature, physical fitness and some drugs have profound effects on the quality and quantity of this type of EEG activity.

Although SWS is closely related to physiological functions and is associated with sleep related increases in hGH and immunological activity, there are few apparent effects of sleep deprivation on physiological functioning.

Cerebral functions show the greatest change following deprivation, and it has been suggested that SWS processes may be associated with restoration of cerebral function following the cerebral activity of wakefulness.

The evidence that SWS functions are concerned with body tissue repair and mood regulation is less substantial than the recent reports which suggest that sleep processes are involved in recovery from cerebral fatigue. Disturbance of SWS affects subjective experiences of somatic well-being and mood, but it is unknown whether these effects are produced by psychological changes coincident with SWS disorders or as a direct result of the disruption of SWS processes.
CHAPTER 2.

THE INFLUENCE OF BODY HEATING ON SWS WITH REFERENCE TO TIME
OF DAY AND THE EFFECTS OF ASPIRIN.

INTRODUCTION.
2. INTRODUCTION.

In this study, healthy student volunteers participated in a laboratory based experiment designed to investigate the effects of body heating on subsequent SWS. The effects of heating at different times of the day, and daytime administration of aspirin were also examined.

2.1. BODY HEATING EFFECTS ON SWS.

The interest in body heating effects on SWS stems from the report of Horne and Staff, (1983) which suggests that changes in body temperature induced by high intensity exercise may be related to subsequent increases in SWS.

Horne and Staff, (1983), studied the sleep of eight highly physically trained (VO2 max better than 55ml oxygen/kg/min) lean runners who underwent three conditions on three separate days between 1400hrs and 1800hrs, high intensity and low intensity exercise and passive heating in a warm bath.

High intensity exercise at 80% of each individual's VO2 max was performed for two forty minute periods, with an interim rest of thirty minutes. Low intensity exercise (at 40% VO2 max) consisted of an equivalent exercise load performed at half the rate over twice the length of time (two eighty minute periods with a fifteen minute rest).

In the no exercise condition, the temperature increases induced by high intensity exercise were achieved by the subject sitting in a tank of warm (42°C) water for eighty minutes, also with a thirty minute interim rest period out of the tank.

SWS increases were observed following both the high intensity exercise and the passive heating condition, but were confined to stage 3 sleep following high intensity exercise (+7.8 min) and stage 4 sleep following the passive heating condition (+17.8 min). Total sleep time was significantly increased following low intensity exercise, as
was stage 2 sleep (+15.1 min) but there were no significant increases in SWS.

Although there were no significant differences in mean weight loss between the three conditions, the rate of body weight loss was significantly higher for both the high intensity exercise and passive heating conditions. Similarly, core temperature increases resulting from the low intensity exercise (+1.0°C) were approximately half of those resulting from the high intensity exercise and passive heating conditions, (+2.0°C).

The greater increases in rates of water loss and core temperature following high intensity exercise and passive heating suggested that high rates of thermal loading may have been the key to the subsequent SWS increases.

Horne and Reid, (1984) investigated whether the bath itself may have acted as an intervening variable producing unwanted effects on the sleep EEG. Six untrained subjects were immersed to mid thorax in a thermoneutrally maintained cool bath (35.5°C) or a hot (41°C) bath for ninety minutes between 1430hrs and 1730hrs on two different occasions.

Significant increases in sleep stages 3 (+9 min) and 4 (+12 min) followed the hot condition and were apparent for both halves of the night, but not the first NREM sleep period. A reduction in REM sleep during the first half of the night was attributed to an increase in SWS 'pressure' following the hot condition. The cool bath had no significant effect on sleep.

Fit subjects undergoing high intensity exercise are capable of sustaining prolonged rises in body temperature and sweat loss, and such high rates of thermal loading may be responsible for the subsequent increases in SWS. In contrast, unfit subjects are unable to endure high intensity exercise to similar levels of thermal loading, but show increases in subsequent SWS when their body temperatures are artificially raised by passive heating in a hot bath, (Horne and Reid, 1984).
Other studies which investigated the effects of body heating upon sleep reported similar increases in SWS.

Putkonen et al., (1973) reported significant SWS increases when four healthy males underwent three ten minute periods of heat stress in a sauna, one hour prior to retiring. Oral temperatures increased to maximums of 39.5°C to 40.5°C from unknown pre-sauna levels, although subjects were allowed fifteen minute rests at room temperature after each heating period.

Although details of individual sleep stages 3 and 4 are not given, mean SWS increased significantly by 19.2 minutes during the first two hours of sleep, and non-significantly by 4.6 minutes during the first six hours.

Maloletnev and Chachanashvili, (1979) studied ten young (mean age 21y) male wrestlers after three baseline nights and then following rapid weight reduction in a steam bath.

Significant (p=0.001) increases in stage 4 sleep (+17 min) followed the steam bath. These increases were particularly evident in the first NREM sleep cycle, and were followed by increases in the length of stage 2 sleep in later NREM sleep cycles.

Body heating effects of progressively increasing block-stepping work in a hot (31.7°C ±0.2) wet environment were reported to enhance SWS and stage 4 sleep increases in four untrained males, (Shapiro, Bortz and Mitchell, 1985). In this study, one baseline night was followed by one morning of exhausting exercise in a neutral environment, two exercise free days, then three mornings during which subjects performed four hours of 'moderate' progressive block-stepping work in a hot wet environment.

On the first and second days of heat, after four hours of exercise mean temperature increases were 1.3°C and 1.8°C respectively. Following these days, percentages of stage 4 sleep and SWS were greater than during either the one control night or following exhaustive exercise in a neutral environment. Although the hot wet environment could be considered responsible for the extra stage 4 sleep and SWS, different exercise regimes were employed, making a direct comparison of the three different conditions difficult.
Bunnell and Horvath (1985), reported the effects of body heating during sleep interruption at a time when SWS propensity is normally low, and REM sleep propensity is high.

Ten subjects were immersed to mid-thorax in thermoneutral (34°C) or hot (41°C) water for twenty minutes during a thirty minute period after the end of the second REM sleep period. Tympanic temperature was reported to rise by 2.5°C during heating, but did not resume pre-interruption levels until one hour after the end of heating. This indicates that temperature was still raised when they returned to bed, which may account for the observed increase in sleep latency following the hot condition, although sleep latency was also increased following the thermoneutral immersion condition.

Heating resulted in increases in SWS and NREM sleep delayed to the second cycle following post-interruption sleep onset, (the fourth NREM sleep cycle of the night). Integrated slow wave amplitude and slow wave density also showed increases of 33% and 10% respectively.

Heating resulted in small increases (+ 5.3 min) in SWS compared to non-immersion nights and compared to the tepid immersion condition (+5.7 min). Stage 2 sleep was also increased.

A significant increase in SWS (+5.6 min) was also found when the hot immersion night was compared to the mean of the tepid immersion and no immersion nights, but individual sleep stages 3 and 4 were not examined.

Unfortunately, sleep characteristics of later NREM sleep cycles in uninterrupted nights and those of the experimental conditions were not compared, but the available results do suggest that some time has to elapse before body heating affects SWS.
2.2. POSSIBLE MECHANISMS UNDERLYING THE HEATING EFFECTS ON SWS.

Horne, (1981) suggests that heat related increases in cerebral metabolism may be responsible for the observed SWS changes following passive heating or high intensity exercise.

A 2°C rise in human brain temperature, which is typical of moderate heat stress, will lead to a 20% increase in brain metabolism (Siesjo, 1978). Such changes may be initiated by the perfusion of warmer blood from the exercising muscles into the cerebrum, or by stimulation of cerebral metabolism by adenosine tri-phosphate and other nucleotides released into the blood by exercising muscles, (Forrester, 1978; cited by Horne, 1981).

Under this hypothesis, preventing increases in brain temperature should reduce the effect of high intensity exercise on SWS. Although brain temperature itself is difficult to monitor, the temperature of the tympanic artery which is close to that of the brain can be measured using a tympanic thermometer carefully positioned in the ear canal. When a continuous stream of cool air is applied to the face, cooled blood from the skin of the face drains via the angularis oculi vein into the jugular vein which runs alongside the carotid artery carrying blood to the brain. Due to the proximity of these vessels arterial blood is cooled, thereby reducing the brain temperature increase.

Horne and Moore, (1985) studied the effects of facial cooling on six very fit subjects who underwent two forty minute periods of exercise at 75% of their individual VO2 max on two occasions between 1430hrs and 1730hrs. A ten minute rest period was allowed between the two exercise periods on each occasion.

Facial cooling was given to reduce any possible rise of cerebral temperature during exercise to 0.5°C, although the hot and cool conditions produced mean rectal temperature increases of 2.3°C and 1°C respectively. Thermal comfort ratings also differed between the two conditions, but sleep disturbances indicative of stress were not evident.
Following the no-cooling (hot) condition, significant increases in stage 4 sleep (+7.5 min) and SWS (+12.1 min) were observed during the first half of the night, especially the second and third NREM sleep cycles. However, when facial cooling was applied there were no significant changes in sleep parameters.

These findings, taken with those of Horne and Staff (1981) and Horne and Reid (1984), suggest that the mechanisms by which SWS was increased following high intensity exercise or passive heating, may be associated with the effects of increases in cerebral temperature.

The precise mechanisms by which a temperature associated increase in cerebral metabolism might affect SWS processes are unknown. However, as the quantity of nocturnal SWS is related to the length of preceding wakefulness, (Webb and Agnew, 1971), a possible effect of heating might be the acceleration of waking processes associated with the production of sleep substances in the brain.

If an increased accumulation of sleep substances was still present at sleep onset, this may result in a rise in SWS, but processes of 'recovery' or removal of these substances might also be active during wakefulness.

If body heating increases SWS by the acceleration of sleep processes, then drugs which reduce SWS might affect the same physiological pathways. A widely used drug with a variety of physiological actions that has been found to affect SWS is aspirin.

2.3. THE EFFECTS OF ASPIRIN ON SLEEP.

Despite previous reports of limited and delayed hypnotic effects of aspirin in poor sleepers (Hauri and Silberfarb, 1978), a study performed by Horne, Percival and Traynor, (1980), found that aspirin reduced SWS when given in large doses to good sleepers.

Horne Percival and Traynor, (1980) gave either 600mg aspirin or placebo to six females three times daily for four days. Despite inter-subject differences in response to aspirin, significant reductions in stage 4 sleep (-11 min) and total SWS (-12 min) were found during aspirin nights.
Stage 2 sleep showed a significant increase (+17 min), which may have compensated for the reduced stage 4 sleep.

Possible mechanisms by which aspirin may affect sleep include the inhibition of prostaglandin synthesis, or the elevation of serum free tryptophan.

Prostaglandins have been implicated in the mechanism of sleep in many animal species. Prostaglandin D2 (PGD2) increases the amount of SWS dose dependently when microinjected into the preoptic area of rats, whereas PGE2 and PGF2α induce high voltage delta waves in rats, (Veno et al, 1982). The PGE series has also been shown to have sedative effects in the mouse and chick. (Chiu and Richardson, 1985). PGE1 also increases brain serotonin turnover, acetylcholine concentration and cortical activation, (Douthitt, Bugbee and Perez-Cruet, 1973).

A single dose of aspirin is sufficient to reduce the synthesis of prostaglandins by an easily detectable amount, and treatment with therapeutic doses for two to three days results in a very pronounced inhibition.

Prostaglandins are long chain fatty acids with a wide spectrum of biological activity, (Flower, 1973), and aspirin specifically inhibits 'prostaglandin synthetase' which synthesizes prostaglandins from their precursor arachidonic acid.

They possess central effects on the regulation of behaviour, body temperature, cardiovascular activity and neurotransmitter functions, and a variety subserve different physiological functions in the brain.

There is also considerable evidence that in many animal species, prostaglandins affect body temperature and are involved in producing hyperthermia and modulating the febrile response induced by pyrogens, (Chiu and Richardson, 1985).

In addition to the suppression of prostaglandin synthesis, it is possible that aspirin may affect sleep by affecting the metabolism of tryptophan.

Aspirin doses of 1800mg significantly elevate serum-free tryptophan in man, by reducing the binding capacity of serum protein for tryptophan, (Smith and Lakatos, 1971)
Tryptophan is a precursor of the neurotransmitter serotonin, which has a role in controlling mammalian sleep and waking. Although the role in sleep mechanisms is not fully established, serotonin may be involved in the initiation and maintenance of sleep, and may be required for both NREM and REM sleep, (Hartmann, 1986).

2.4. **SUMMARY**

SWS increases were observed when subjects underwent high intensity exercise or passive heating in a warm bath, which provoked high rates of thermal loading, characterised by high rates of water loss and increases in core body temperature.

The study by Horne and Staff, (1983) illustrated that the body thermal effects induced by high rates of exercise in fit subjects are more likely to underlie the mechanisms leading to an increase in SWS than other effects of exercise such as the exercise load.

Other studies of sleep and body heating have also reported increases in SWS following varying periods of increased body temperature.

The study conducted by Bunnell and Horvath, (1985) suggested that a time lapse of approximately 2 hours is required before the effects of body heating on SWS are observed.

Body heating studies previously carried out in our laboratory employed eighty to ninety minute heating periods, between 1400hrs and 1800hrs, (Horne and Staff, 1983: Horne and Moore, 1984: Horne and Reid, 1984). Putkonen et al, (1973) reported larger SWS increases (54.6 min) after three ten minute periods of raised body temperature, although the time of day of heating was not described.

It is not known whether the effect of body heating on subsequent SWS decays over interim wakefulness, and whether there is a 'dose X delay' interaction. In this case, if the interim wakefulness is contributory to 'recovery' following heating, then heating close to sleep onset might result in a greater effect on subsequent SWS.
As previous studies employed a 'dose' of one and a half hours of heating, the following study was designed to compare the effects of a smaller fixed amount of heating given either two or eight hours prior to sleep.

As aspirin administration reduces subsequent SWS in good sleepers, we were interested to know whether giving aspirin prior to nighttime passive heating would reduce any heating effects on subsequent SWS.
CHAPTER 3.

THE INFLUENCE OF BODY HEATING ON SWS WITH REFERENCE TO TIME OF DAY AND THE EFFECTS OF ASPIRIN.

EXPERIMENTAL PROCEDURE AND RESULTS.
3. EXPERIMENTAL PROCEDURE AND METHOD.

As previous studies used 1.5 hour periods of passive heating in the afternoon, a pilot study was run to examine whether this length of heating prior to retiring might be counterproductive to sleep. A shorter period of sleep, (0.5 hour) was also used to determine whether this might be more suitable.

3.1. PILOT STUDY.

3.1.1. SUBJECTS.

Two healthy female subjects (aged 21y), were recruited from the campus population. Each was a non-smoker, of medium build and moderate fitness. Subjects were interviewed to ensure that they fulfilled the criteria for participation in the study. Each subject was required to:

- Maintain regular sleeping habits.
- Report good sleep without the use of sleeping pills, or other hypnotics.
- Report good health.
- Have been free of colds, or fever for the past month.
- Be free of medication, and to report the use of the contraceptive pill.

Prior to the study subjects were requested to:

- Maintain a normal sleeping and eating routine throughout the preceding week and during 'nights out' of the laboratory, to avoid late nights, sleeping in and taking naps.
- Abstain from alcohol from the Friday preceding the beginning of the experiment (Sunday night), and throughout the experimental period.
- Avoid taking baths prior to nights in the laboratory unless requested to do so.
- Avoid exercise, or any strenuous activity likely to increase body temperature prior to nights in the laboratory.
e) Avoid using any pills or medicines for headaches etc, especially aspirin, paracetamol, cough medicines and analgesics.

f) Complete the pre and post-sleep Stanford Sleepiness Scales (SSS) for a week preceding and during the experiment.

g) To inform the experimenter if there were any problems concerning the experimental procedure.

h) Keep tea and coffee intake to a minimum.

i) Report any malaise, infection or menstruation.

Subjects were given modest payment for their participation.

3.1.2. PILOT STUDY METHOD.

Subjects were studied for one adaptation and three baseline nights. On experimental nights one subject underwent passive heating of 1.5 hours or 0.5 hour duration given at 2100hrs on separate occasions, the other subject underwent a baseline recording. The passive heating method was as described for the main study. Temperature and pulse were taken by the experimenter who remained in attendance throughout the heating session.

Subjects were asked to report to the sleep laboratory at 2200hrs each night when electrodes were applied in accordance with the standard procedure described in the main study. Following this, subjects watched television or talked with the experimenter prior to retiring at 2345hrs each night, and slept until they awoke at 0745hrs the following morning, in time for breakfast and lectures.

3.1.3. RESULTS.

The 1.5 hours heating condition was abandoned from the main study as for both subjects, this resulted in long sleep onset delays and major sleep disturbances with frequent awakenings. The 0.5 hour condition produced obvious increases in SWS with no sleep disruptions, and was adopted for the main study.
3.2. **MAIN STUDY.**

3.2.1. **SUBJECTS.**

Subjects were three male and three female good sleepers (age 21 to 33y) from the University population. All were moderately physically trained non-smokers in good health, and medication free. All subjects conformed to the same criteria set forth for the pilot study. In addition, subjects were asked if they had ever suffered an adverse reaction to aspirin, but none were reported. Each subject was informed that they would be taking either aspirin or aspirin-placebo tablets during the experiment and written consent was obtained.

3.2.2. **EXPERIMENTAL DESIGN.**

Subjects were studied in pairs and slept in the laboratory for four nights during the first week and four during the second. Subjects were requested to co-operate with the requirements of the experiment which were as for the pilot study.

Prior to the study subjects were requested to:

a) Maintain a normal sleeping and eating routine throughout the preceding week and during 'nights out' of the laboratory, to avoid late nights, sleeping in and taking naps.

b) Abstain from alcohol from the Friday preceding the beginning of the experiment, (Sunday night), and throughout the experimental period.

c) Avoid taking baths prior to nights in the laboratory unless requested to do so.

d) Avoid exercise, or any strenuous activity likely to increase body temperature, prior to nights in the laboratory.

e) Avoid using any pills or medicines for headaches etc, especially aspirin, paracetamol, cough medicines and analgesics.
f) Complete the pre and post-sleep SSS for a week preceding and during the experiment.

  g) To inform the experimenter if there were any problems concerning the experimental procedure.

  h) Keep tea and coffee intake to a minimum.

  i) Female subjects were asked to report if they were menstruating, or using the contraceptive pill.

  j) All subjects were asked to report any malaise, infection or other illness.

Each subject was invited to visit the sleep laboratory before the experimental period in order to become familiar with the surroundings and equipment. Subjects were given modest payment for their participation.

One adaptation night was allowed during which subjects underwent standard electrode fixation but without a subsequent recording. According to the different orders for each subject, the first baseline night was followed by either an experimental night or another baseline night.

Subjects arrived at the sleep laboratory at 2200hrs and prepared for bed. After the electrodes had been attached, they sat quietly relaxing and watching television until they were ready to retire at about 2330hrs. 'Lights out' followed at 2345hrs, following completion of the SSS, which has seven points 1=wide awake, to 7=struggling to remain awake. Subjects were awoken at 0800hrs, in time for breakfast and lectures, but usually awoke spontaneously before this. Half an hour after arising subjects completed the post-sleep section of the sleepiness scale, prior to leaving the laboratory.

3.2.3. PASSIVE HEATING PROCEDURE.

Each of the three experimental conditions incorporated 0.5 hour of passive heating, achieved by the subject sitting immersed to mid-thorax in water maintained at 40°C or sufficient to raise the temperature by no more than 2°C. Baths were taken privately in the subject's own residence, and the addition of bubble bath liquid (which acted as an insulation against heat loss from the water surface)
increased subject comfort. Male subjects wore swimming trunks, whilst females wore bikini costumes. Heating took place at either 1700hrs, (before supper) or at 2100hrs. Following the bath, and on some baseline nights, subjects recorded their state of alertness/drowsiness on a scale of 1-5, until they retired to bed.

The experimenter remained in attendance during the heating session to ensure subject safety. For subject convenience, temperature was recorded orally rather than rectally. Although temperature levels differ between the two methods, previous studies in our laboratory have found that rectal and oral temperatures show similar changes over time.

Oral temperature was recorded every five minutes using a calibrated digital thermometer placed under the tongue with the mouth firmly shut, and left for two minutes, or until the temperature measurement had stabilised. Pulse rate was also recorded using the digital meter.

Both recordings continued after the subject had left the bath until body temperature returned to pre-bath levels. Subjects were asked to report any discomfort and were permitted to leave the bath at any time if they were unduly stressed, although this proved unnecessary.

3.2.4. ASPIRIN ADMINISTRATION.

Three times on each experimental day, subjects took 'blindly' either 2 x 300mg aspirin or aspirin-placebo after meals. Insoluble aspirin (acetylsalicylic acid) tablets were used and had to be taken with a glass of water. The placebos tasted like aspirin and were supplied by a local pharmaceutical company.

All subjects were resident in the university, and ate within the same meal periods. Aspirin was taken on one occasion, prior to a 2100hrs heating episode and this condition always preceded nights away from the laboratory to allow for aspirin 'washout'.

3.2.5. SLEEP RECORDINGS.

For sleep EEG recordings, electrodes were attached in accordance with the standard method described by Rechtschaffen and Kales, (1968).

Skin was cleaned with methylated spirits, prior to attachment of silver-silver chloride electrodes to the scalp, using collodion glue, dried with pressurized air. For application of skin electrodes, double-sided adhesive discs were used and secured with micropore tape. Saline 'neptic' gel was inserted into the electrode cavity using a syringe with a blunted needle to increase conductivity. Instead of scarifying the skin with the blunted needle, a blunted disposable 'orange-stick' was used to reduce the risk of transmitting any blood-borne infections if the skin was accidentally grazed. Electrode resistance was reduced to less than 5kΩ prior to commencing the recording, and checked using a resistance meter.

A1-C3 and A2-C4 EEG, EOG and sub-mental EMG recordings were made for each subject on a Grass 78 Polygraph at a paper speed of 1 cm/sec with EEG and EOG gains and time constants at 50 μV/cm and 0.3 sec respectively.

In addition, Oxford Medilog ambulatory recorders were wired into the polygraph junction box, in order to obtain a taped recording of sleep. The miniature analogue tape recorder can record four channels of data, (EEG, EMG, and two EOG's) continuously for up to twenty four hours on a C-120 cassette tape.

Sleep EEG paper write outs were visually scored in one minute epochs according to the standardised criteria of Rechtschaffen and Kales, (1968). A 'blind' investigator also scored sleep records and in the 5% epochs where there was disagreement, this was resolved by discussion.
3.2.6. DATA ANALYSIS.

For manual scoring, the first 420 minutes of sleep from sleep onset were analysed for the following parameters:

Sleep onset latency: The time (minutes) from light's out to the beginning of the first period of 15 minutes of continuous sleep stage 2 or deeper.

Sleep Period Time, (SPT): The time (minutes) from sleep onset to final awakening.

Total Sleep Time: Sleep Period Time minus interim wakefulness.

REM Sleep Periodicity: The average interval (minutes) between the onset of consecutive REM sleep episodes of greater than 3 mins duration but greater than 15 mins apart.

REM Sleep Latency: The time (minutes) from sleep onset latency to the onset of the first REM sleep period of greater than 3 mins duration.

OXFORD SLEEP STAGER ANALYSIS.

The Oxford Sleep Stager automatically processes overnight sleep recordings, (recorded on C-120 tapes using Medilog Ambulatory recorders) to give clear consistent results. Signals are accepted from the replay device at twenty times the recording speed via an Input Attenuator. Sleep stage analysis and results are produced as a seven stage hypnogram and sleep statistics within thirty minutes.

This method scores sleep in real time epochs of thirty seconds at 20 X recording speed, according to standard criteria. One channel of EEG (A1-C3), two channels of EOG (left and right), and one channel of submental EMG are played through the sleep stager using an Oxford Medilog Page Mode Display playback.

Use of the sleep stager allowed a direct comparison of visual and automated scoring of sleep, analysis of inter-subject trends across conditions, and quantification of sleep stages 3 and 4 in particular.

Analysis of the taped sleep recordings was performed after visual scoring had been carried out, as a 'blind' comparison.
A direct comparison of the sleep stager output with visual scoring epochs over the whole night was not possible, as some 'drifting' is inevitable. Preliminary analysis showed the sleep stager to have a good repeatability of 92-96%.

3.2.7. EEG POWER.

The EEG was assessed for the root mean square (RMS) of the EEG. As EEG power is inversely proportional to frequency, this technique was used as an additional method of quantifying delta activity, and obtaining the total power of the EEG.

Following A-D conversion and high frequency filtering at >25 Hz real time (to remove muscle and other artifacts), the EEG voltage was sampled at 10 Hz for thirty second epochs by an Apple E2 microcomputer, with the root-mean-square (RMS) calculation done on the 300 samples. This value was stored for later use and the process repeated for 420 minutes of sleep.

3.2.8. STATISTICAL ANALYSIS.

Whole night data from visual analysis were compared between baseline and experimental conditions, and where differences were apparent, 'planned comparison' analyses of variance were performed. Baseline values were subtracted from all experimental conditions to give 'changes from baseline' for each parameter and subject. As automated analysis results were similar to those of visual analysis, it was decided not to replicate these statistical analyses, but to use only visual scorings for statistical analysis.

Use of visually scored sleep analysis which was subjected to two independent scorers, also allowed inspection of sleep stages in each of the two halves of a standardised 420 minute night. This length of time was used as it was the highest common denominator between sleep recordings of the six subjects.
Statistical analysis was performed using two-way analysis of variance for the following comparisons.

1) EARLY versus LATE BATH + PLACEBO.

2) LATE BATH + PLACEBO versus LATE BATH + ASPIRIN.

There was no a priori reason for comparing early versus late bath plus aspirin.

An overall F value was first obtained across all conditions, and the treatment effects were compared using standard errors estimated for all eighteen values (six subjects and three conditions). Estimates for the two treatment comparisons were divided by their appropriate standard error and the t-statistic applied, having ten degrees of freedom.

Two tailed significance values were used with a criterion of p<0.05.
3.3. RESULTS.

3.3.1. TEMPERATURE CHANGES.

Mean changes in oral temperature for all subjects during all baths are shown in figure (1). This shows a rapid mean temperature increase of 1.5°C within the first five minutes of heating. Temperature then rose more gradually reaching a mean increase of approximately 2.2°C after fifteen minutes. During the remaining fifteen minutes, mean temperature increased slightly, but was generally stable.

Mean temperature increases over each heating session are shown in table (1). Mean pre-bath temperature differed by 0.2°C between early and late baths, but mean increase was slightly less (0.2°C) for the early bath.

TABLE I.

<table>
<thead>
<tr>
<th>Heating Condition.</th>
<th>Early</th>
<th>Late</th>
<th>9pm+Asp</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5pm</td>
<td>9pm</td>
<td>9pm</td>
</tr>
<tr>
<td>Pre-bath temp.</td>
<td>36.2°C</td>
<td>36.0°C</td>
<td>36.0°C</td>
</tr>
<tr>
<td>s.d.</td>
<td>(0.6)</td>
<td>(0.4)</td>
<td>(0.2)</td>
</tr>
<tr>
<td>Temp °C Increase.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (n=6)</td>
<td>1.8°C</td>
<td>2.0°C</td>
<td>2.0°C</td>
</tr>
<tr>
<td>s.d.</td>
<td>(0.3)</td>
<td>(0.3)</td>
<td>(0.1)</td>
</tr>
</tbody>
</table>

Differences in mean temperature increases of the early and late baths may be attributed to difficulties in stabilising the bath water temperature, thus controlling individual temperature increases. None of the subjects reported any differences in subjective comfort between the three conditions, after which oral temperature returned to pre-bath levels within 0.5 hour.
FIGURE 1. MEAN ORAL TEMPERATURE CHANGES FROM BASELINE DURING 30 MINUTE PASSIVE HEATING SESSIONS.

NOTE:
MEAN FOR ALL SUBJECTS OVER THREE EXPERIMENTAL CONDITIONS.
STANDARD DEVIATIONS SHOWN AS BARS.
TEMPERATURES RETURNED TO NORMAL WITHIN 30 MINUTES OF TERMINATION OF HEATING.
3.3.2. **COMPARISON OF VISUAL AND AUTOMATED SLEEP ANALYSIS.**

Visually scored and analyser scored mean baseline values for sleep parameters during the first 420 minutes of sleep are shown in table (II).

| TABLE II. GROUP MEAN BASELINE VALUES FOR SLEEP STAGES OVER 420 MINUTES OF SLEEP |
|---------------------------------|---------------------------------|-----------------|----------------|----------------|----------------|----------------|
| Sleep Stages                    | Minutes                         | W+M  | 1   | 2   | 3   | 4   | SVS | REM |
| Visually scored                 |                                 | Mean  | 20.1| 29.1| 211.5| 41.1| 32.2| 73.3| 86.7 |
|                                 |                                 | s.d.  | 14.2| 8.0 | 21.7 | 13.2| 13.6| 15.5| 12.1 |
| Analyser scored                 |                                 | Mean  | 27.2| 22.2| 214.7| 54.7| 17.0| 71.7| 85.0 |
|                                 |                                 | s.d.  | 21.6| 10.2| 29.6 | 16.0| 14.3| 19.0| 7.1  |

As described in the method there was close agreement between visually scored and analyser scored sleep. Agreement was particularly good for stage 2 sleep, REM sleep and SWS as a whole, but there was less similarity with individual sleep stages 3 and 4. The agreement between wake and movement, and stage 1 sleep can be improved if these parameters are taken together, visual analysis giving 49.2 minutes and automated analysis giving 49.4 minutes.

Percentage agreement between visual and analyser scored sleep for each sleep stage and each subject is shown in table (III). For this analysis, each value for a particular sleep stage was compared between visual and automated scoring. The smallest value was calculated as a percentage of the larger value. Mean percentage agreements for each subject over all stages are shown, in addition to mean for each stage over all subjects.
TABLE III. COMPARISON OF VISUAL AND AUTOMATED SLEEP SCORING: PERCENTAGE AGREEMENT FOR EACH SUBJECT AND EACH SLEEP STAGE.

(N=6, Number of nights analysed=7).

Percentage Agreement.

<table>
<thead>
<tr>
<th>Sleep Stages</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>SVS</th>
<th>REM</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subject 1</td>
<td>78</td>
<td>93</td>
<td>88</td>
<td>67</td>
<td>86</td>
<td>92</td>
<td>87 (90)</td>
</tr>
<tr>
<td>2</td>
<td>64</td>
<td>94</td>
<td>69</td>
<td>95</td>
<td>83</td>
<td>90</td>
<td>89 (93)</td>
</tr>
<tr>
<td>3</td>
<td>56</td>
<td>92</td>
<td>84</td>
<td>44</td>
<td>78</td>
<td>66</td>
<td>83 (85)</td>
</tr>
<tr>
<td>4</td>
<td>92</td>
<td>95</td>
<td>69</td>
<td>24</td>
<td>78</td>
<td>88</td>
<td>86 (89)</td>
</tr>
<tr>
<td>5</td>
<td>70</td>
<td>91</td>
<td>45</td>
<td>85</td>
<td>91</td>
<td>90</td>
<td>88 (90)</td>
</tr>
<tr>
<td>6</td>
<td>63</td>
<td>93</td>
<td>47</td>
<td>71</td>
<td>89</td>
<td>94</td>
<td>86 (91)</td>
</tr>
<tr>
<td>Mean</td>
<td>70</td>
<td>93</td>
<td>67</td>
<td>64</td>
<td>84</td>
<td>87</td>
<td>87 (90)</td>
</tr>
</tbody>
</table>

(Numbers in parentheses refer to percentage agreement when SWS was used instead of sleep stages 3 and 4 separately).

Figure (2) shows a comparison of mean changes from mean baseline for whole night sleep parameters as scored visually and by the sleep stager, for each condition.

Both methods show similar increases in sleep stages 3 and 4, and SWS, following the late bath and placebo condition. Changes in these stages after the early bath, and late bath with aspirin show similar trends although individual values differ. Summated waking, movement and stage 1 sleep is reduced below baseline after all bath conditions, with both scoring methods showing the largest reduction after the late bath with aspirin condition, although the magnitude of difference is greater for visual scoring.
FIGURE 2. SLEEP STAGE CHANGES FROM BASELINE UNDER EACH PASSIVE HEATING CONDITION: COMPARISON OF VISUAL AND AUTOMATED SCORING.

VISUALLY SCORED

ANALYSER SCORED

**Significant at p<0.005
***Significant at p<0.002

a Significantly different from early
b Significantly different from late plus aspirin.
Visually scored stage 2 sleep is increased above baseline levels under all conditions, showing the largest change following the late bath with aspirin. Automated scoring shows a smaller increase in stage 2 sleep after this condition with decreases following the early bath and late bath with placebo.

With both scoring methods, REM sleep shows similar trends for early and late bath plus placebo conditions, but differs for the late bath with aspirin. Automated scoring shows an increase of 12 minutes, but visual scoring shows a small decrease of 4 minutes after this condition.

3.3.3. **VISUALLY SCORED SLEEP PARAMETERS.**

Although the two scoring methods produced similar results, statistical analysis was only performed on visually scored sleep. Statistically significant differences for visually scored sleep parameters are shown on figure (2). Table (IV) shows the mean changes from baseline for visually scored sleep under each condition.

Table (IV) gives the mean whole night changes in sleep stages from baseline following the three experimental heating conditions.
Table IV. MEAN WHOLE NIGHT CHANGES FROM BASELINE FOR EACH VISUALLY SCORED SLEEP STAGE UNDER ALL CONDITIONS.

<table>
<thead>
<tr>
<th>Heating Condition</th>
<th>5pm</th>
<th>9pm</th>
<th>9pm+Aspirin</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>W+H+1</td>
<td>-7.3</td>
<td>-7.5</td>
<td>-13.6</td>
</tr>
<tr>
<td>s.d</td>
<td>21.9</td>
<td>22.4</td>
<td>(26.5)</td>
</tr>
<tr>
<td>Stage 2</td>
<td>+4</td>
<td>+4.3</td>
<td>+24.6</td>
</tr>
<tr>
<td>s.d</td>
<td>16.1</td>
<td>20</td>
<td>(28.6)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>+1.6</td>
<td>+5.8</td>
<td>-2.8</td>
</tr>
<tr>
<td>s.d</td>
<td>13.4</td>
<td>9.0</td>
<td>(12.4)</td>
</tr>
<tr>
<td>Stage 4</td>
<td>-5.3</td>
<td>+16.6</td>
<td>-2.5</td>
</tr>
<tr>
<td>s.d</td>
<td>16.3</td>
<td>19.4</td>
<td>(10.9)</td>
</tr>
<tr>
<td>SWS</td>
<td>-3.6</td>
<td>+22.5</td>
<td>-5.3</td>
</tr>
<tr>
<td>s.d</td>
<td>7.6</td>
<td>15.4</td>
<td>(13.2)</td>
</tr>
<tr>
<td>REM</td>
<td>+9.6</td>
<td>-15.5</td>
<td>-3.8</td>
</tr>
<tr>
<td>s.d</td>
<td>16.9</td>
<td>13.8</td>
<td>(18.1)</td>
</tr>
</tbody>
</table>

Table (V) shows the mean values for baseline nights and following each experimental condition, for visually scored sleep onset, sleep period, sleep time, REM sleep latency and length of the first REM sleep period. Also shown are the mean values for SSS ratings (pre and post-sleep), and the mean EEG power in RMS volts for each twenty minute sample taken over 420 minutes of the night, under each experimental condition.
TABLE V.  
MEAN VALUES FOR SLEEP PARAMETERS UNDER EACH  
EXPERIMENTAL CONDITION.  

(Visually scored parameters used where appropriate).

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>5pm</th>
<th>9pm</th>
<th>9pm+Asp</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep onset (min)</td>
<td>16.6</td>
<td>20.5</td>
<td>11.3</td>
<td>15.1</td>
</tr>
<tr>
<td>s.d</td>
<td>(8.9)</td>
<td>(14.4)</td>
<td>(8.9)</td>
<td>(12.7)</td>
</tr>
<tr>
<td>Sleep Period (min)</td>
<td>451.5</td>
<td>448.0</td>
<td>455.6</td>
<td>454.5</td>
</tr>
<tr>
<td>s.d</td>
<td>(12.0)</td>
<td>(15.6)</td>
<td>(13.8)</td>
<td>(14.0)</td>
</tr>
<tr>
<td>Total Sleep (min)</td>
<td>432.0</td>
<td>443.2</td>
<td>437.8</td>
<td>444.7</td>
</tr>
<tr>
<td>s.d</td>
<td>(11.9)</td>
<td>(17.4)</td>
<td>(16.4)</td>
<td>(13.8)</td>
</tr>
<tr>
<td>REM Latency (min)</td>
<td>76.5</td>
<td>64.7</td>
<td>113.8</td>
<td>87.0</td>
</tr>
<tr>
<td>s.d</td>
<td>(37.6)</td>
<td>(15.8)</td>
<td>(38.8)</td>
<td>(31.6)</td>
</tr>
<tr>
<td>Length 1st REMP (min)</td>
<td>6.2</td>
<td>5.0</td>
<td>3.0</td>
<td>4.5</td>
</tr>
<tr>
<td>s.d</td>
<td>(2.9)</td>
<td>(5.6)</td>
<td>(4.1)</td>
<td>(4.5)</td>
</tr>
<tr>
<td>SSS score (bedtime)</td>
<td>3.8</td>
<td>4.3</td>
<td>3.3</td>
<td>4.8</td>
</tr>
<tr>
<td>s.d</td>
<td>(1.5)</td>
<td>(1.8)</td>
<td>(1.6)</td>
<td>(2.1)</td>
</tr>
<tr>
<td>SSS score (arising)</td>
<td>2.4</td>
<td>2.8</td>
<td>3.0</td>
<td>3.0</td>
</tr>
<tr>
<td>s.d</td>
<td>(1.5)</td>
<td>(0.9)</td>
<td>(0.8)</td>
<td>(1.6)</td>
</tr>
<tr>
<td>EEG Power (RMS V)</td>
<td>1.29</td>
<td>1.32</td>
<td>1.49</td>
<td>1.32</td>
</tr>
<tr>
<td>s.d</td>
<td>(0.13)</td>
<td>(0.17)</td>
<td>(0.13)</td>
<td>(0.17)</td>
</tr>
</tbody>
</table>

(a) Significant (p<0.02).
(b) Significantly different from early.
(c) Significantly different from late plus aspirin.
(d) Includes one subject with a 'missed' first REM sleep period.

Thirty minutes of heating at 1700hrs produced no obvious change from mean baseline levels of any sleep parameter under either method of sleep scoring.

Comparison of the 1700hrs bath with the 2100hrs bath plus placebo, shows that significant increases in SWS (t=3.89, df=10, p<0.02) and stage 4 sleep (t=4.78, df=10, p<0.002) resulted from the latter condition.
Visually scored SWS increased by a mean of 22.5 minutes, and stage 4 sleep by 16.6 minutes over mean baseline values. Following the late bath plus placebo, total time spent in REM sleep was significantly reduced (t=2.43, df=10, p<0.005), as was sleep onset latency (t=2.85, df=10, p<0.02). There was also a tendency for REM sleep latency to be increased, and the first REM sleep period to be shorter following the late bath plus placebo, although these changes were statistically non-significant. One subject showed an unusually long REM sleep latency, and appeared to 'miss' his first REM sleep period.

When the two late (2100hrs) baths are compared, significant differences for SWS (t=4.32, df=10, p<0.002) and for stage 4 sleep are found (t=4.17, df=10, p<0.002), indicating that aspirin seemed to reduce most of the effects produced by the late bath.

With aspirin administration, sleep onset latency was increased, whereas total REM sleep, REM sleep latency and the length of the first REM sleep period were all reduced compared to the placebo condition. There were no significant effects of the heating conditions on these sleep variables or on SSS ratings.

Statistically significant differences in mean EEG power per twenty minutes were found between the early (1700hrs) bath and late bath (2100hrs) with placebo, (t=2.89, df=10, p<0.02), and the two late baths, (t=2.95, df=10, p<0.02).

Table (VI) shows the changes in sleep stages 2, 3, 4 and REM for each half of the night, when the first 420 minutes were analysed. These values were obtained from the means of each 210 minutes of the night, for each condition, compared to the mean baseline values.
TABLE VI MEAN CHANGES FROM BASELINE FOR EACH HALF OF THE NIGHT (420 mins).

<table>
<thead>
<tr>
<th></th>
<th>First half (0-210 mins)</th>
<th>Second half (211-420 mins)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early Bath</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 2</td>
<td>+2.8 (13.1)</td>
<td>+7.5 (12.1)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>+3.3 (14.7)</td>
<td>+0.3 (9.9)</td>
</tr>
<tr>
<td>Stage 4</td>
<td>-5.8 (17.9)</td>
<td>+1.8 (5.9)</td>
</tr>
<tr>
<td>SWS</td>
<td>-2.5 (12.9)</td>
<td>+2.1 (12.3)</td>
</tr>
<tr>
<td>REM</td>
<td>+4.3 (11.1)</td>
<td>+4.3 (16.7)</td>
</tr>
<tr>
<td>Late Bath + Placebo</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 2</td>
<td>-6.0 (6.6)</td>
<td>+9.2 (17.5)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>+1.0 (8.4)</td>
<td>+1.2 (5.4)</td>
</tr>
<tr>
<td>Stage 4</td>
<td>+14.6 (15.1)</td>
<td>+2.4 (10.5)</td>
</tr>
<tr>
<td>SWS</td>
<td>+15.6 (10.4)</td>
<td>+3.6 (14.2)</td>
</tr>
<tr>
<td>REM</td>
<td>-4.6 (8.8)</td>
<td>-7.8 (9.9)</td>
</tr>
<tr>
<td>Late Bath + Aspirin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 2</td>
<td>+4.5 (23.4)</td>
<td>+11.5 (10.2)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>+1.2 (17.2)</td>
<td>-3.8 (7.8)</td>
</tr>
<tr>
<td>Stage 4</td>
<td>+1.8 (12.3)</td>
<td>-3.0 (2.9)</td>
</tr>
<tr>
<td>SWS</td>
<td>+3.0 (18.5)</td>
<td>-6.8 (9.6)</td>
</tr>
<tr>
<td>REM</td>
<td>-2.5 (10.1)</td>
<td>+2.6 (16.5)</td>
</tr>
</tbody>
</table>

Following the early (1700hrs) bath there was a small increase in stage 3 sleep, but a decrease in stage 4 sleep during the first half of the night. A small increase in both stages seen in the later part of the night resulted in a non-significant overnight change from baseline. REM sleep showed a slight increase over baseline but this was equally divided over the two halves of the night. Stage 2 sleep showed the greatest change overnight, mainly due to an increase of 7.5 minutes in the last 210 minutes.

The late bath (2100hrs) with placebo resulted in an increase in sleep stage 4, most apparent in the first half of the night. This was associated with a slight decrease in REM sleep throughout the night, possibly as a result of an increase in SWS 'pressure'. Stage 2 sleep was also reduced during the first half of the night, but increased during the
second half. Stage 3 sleep showed little change from baseline levels.

Administration of aspirin resulted in a reduction in the effects of the late bath. Sleep stages 3 and 4 showed a small increase in the first half of the night, but this was counteracted by a larger reduction in the second half, resulting in an overall decrease from baseline levels. However, the magnitude of this difference indicates that there was very little change from baseline following the late bath with aspirin. Stage 2 sleep showed the largest increase in the second half of the night compared to either of the other two conditions, resulting in an increase of 16 minutes for the (420 min) night.

3.3.4. EEG RMS POWER

Baseline and experimental changes in mean EEG RMS power for each twenty minutes of the night are shown in figure (3). Figure (4) shows the EEG RMS power as the cumulative difference from the relevant baseline. Each point has had it's corresponding baseline value subtracted, and the result added to that of the previous point. Mean power values for the whole night are shown in table (V).
FIGURE 3. EEG POWER: MEAN VALUES OF RMS VOLTS (AFTER AMPLIFICATION) EVERY 20 MINUTES OVER 7 HOURS SLEEP.

AVERAGED ACROSS SUBJECTS FOR EACH EXPERIMENTAL CONDITION AND BASELINE.
**FIGURE 4.** EEG POWER: CUMULATIVE CHANGES FROM BASELINE LEVELS OVER 7 HOURS SLEEP, AVERAGED ACROSS SUBJECTS FOR EACH EXPERIMENTAL CONDITION.

(RMS = ROOT MEAN SQUARE.)
Figure (3) shows that EEG power surges approximate NREM sleep cycles, and show a drop during periods when REM sleep occurs.

The late bath with placebo produced an early night increase in EEG RMS power, which remained above baseline levels and those of other conditions for the majority of the night. There was a large drop in EEG RMS power after four hours of sleep followed by a surge which continued for another hour. Late night increases in sleep stages 2, 3 and 4 following the late bath plus placebo may have contributed to this, in addition to which an increase in delta activity below the criterion for SWS was apparent during visual scoring of stage 2 sleep.

Aspirin administration reduced the increase in EEG RMS power seen following the late bath plus placebo condition, such that EEG RMS power after aspirin was similar to that of baseline nights. Following the early bath, there was an early surge in EEG RMS power, (although not as large as that of the late bath with placebo) but this gradually diminished and became close to baseline levels within four hours.

Figure (4) shows that for the late bath plus placebo condition there is an increase in EEG RMS power which is sustained throughout most of the night, particularly during SWS episodes, (indicated by the peaks). The graph shows changes from baseline levels, which are low in the later part of the night, but did not show such a great decline under the late bath with placebo condition.

EEG RMS power is greater than baseline for the early (1700hrs) and late bath (2100hrs) with aspirin conditions. Table (VI) shows that compared to baseline there is an increase in sleep stages 2 and 3 during the first half of the night, and a continuing increase in stage 2 sleep during the second half, which may account for this increase over baseline.
Individual and group mean values for total EEG RMS power over the first 420 minutes of the night, for each condition are shown in table (VII). EEG RMS power was analysed using taped recordings, but as difficulties were encountered analysing one subject's recordings, only five subjects' data are shown. Means of EEG RMS power per twenty minutes of each record under each condition are shown in table (V).

### TABLE VII. INDIVIDUAL MEAN TOTAL EEG POWER FOR THE FIRST 420 MINUTES UNDER EACH CONDITION.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Early 5pm</th>
<th>Early 9pm</th>
<th>Late 9pm</th>
<th>Late 9pm + Asp</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subject 1</td>
<td>30.5</td>
<td>30.0</td>
<td>33.4</td>
<td>32.2</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>25.7</td>
<td>26.8</td>
<td>27.5</td>
<td>24.3</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>25.7</td>
<td>25.3</td>
<td>31.4</td>
<td>26.6</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>29.9</td>
<td>33.6</td>
<td>35.1</td>
<td>31.6</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>23.7</td>
<td>23.5</td>
<td>28.8</td>
<td>24.2</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Group Mean</th>
<th>27.1</th>
<th>27.8</th>
<th>31.2</th>
<th>27.8</th>
</tr>
</thead>
<tbody>
<tr>
<td>s.d</td>
<td></td>
<td>(2.6)</td>
<td>(3.5)</td>
<td>(2.8)</td>
<td>(3.5)</td>
</tr>
</tbody>
</table>

The late bath with placebo produced the greatest values seen for EEG power for each of the five subjects. This effect was diminished following the aspirin condition which resulted in mean EEG power values closer to those of the early bath and baseline nights. However, four of the five subjects did show a slight increase in EEG RMS power following the late bath with aspirin, although it was less than that seen following the late bath with placebo.

There was no significant effect of the early bath on the group as a whole; although two subjects showed a slight increase in EEG power.
3.3.5. REM SLEEP LATENCY.

All subjects showed a non-significant increase in REM sleep latency following the late bath with placebo but REM sleep latency was shortened non-significantly following the late bath with aspirin, although two subjects showed increase of 85 and 15 minutes respectively. The early bath also resulted in a shortened REM sleep latency for five of the six subjects.

3.3.6. DAYTIME SLEEPINESS RATINGS.

Subjects rated their subjective alertness/drowsiness on a scale of 1 (alert) to 5 (drowsy) from 1700 hrs half hourly until retiring. Figure 5 shows the mean group ratings for each condition from 1700 hrs until 2330 hrs.

During baseline days, there was a steady fall in alertness from 1700 hrs onwards, reaching the lowest rating at 2300 hrs, then increasing slightly whilst subjects prepared for bed. Alertness during baseline days was close to that of late bath plus aspirin days, and did not show any drop at 1700 hrs, although it fell by 0.3 points at 2100 hrs.

Each heating condition resulted in a small increase in drowsiness compared to pre-bath levels. For the early bath, alertness dropped by 0.2 points during the heating, then improved within an hour of terminating the bath and remained above pre-bath levels until 2230 hrs, when it began to fall. Pre-bath alertness was lowest prior to the early bath compared to the other experimental days.

For both late bath conditions, drowsiness ratings began to increase prior to the bath, and continued to increase during the heating. Alertness dropped by 0.5 points after the late bath plus aspirin then quickly returned to pre-bath levels until falling again at 2230 hrs and increasing while subjects were preparing for bed.

After the late bath with placebo, alertness dropped by 0.75 points and continued to fall for 0.5 hour then gradually improved towards bedtime, but remained below pre-bath levels throughout.
FIGURE 5. MEAN SUBJECTIVE ALERTNESS AFTER EACH BATH CONDITION.
3.3.7. PRE-SLEEP AND POST-SLEEP STANFORD SLEEPINESS SCALE RATINGS.

No significant differences were found for pre-sleep ratings or post-sleep ratings between conditions. However, analysis of the overnight change in ratings given by each individual following each condition (obtained by subtracting the post-sleep score from the pre-sleep score) showed an improvement in 'alertness' towards the morning.

Table (VIII) shows the individual overnight changes in sleepiness scale ratings for each subject. Positive signs indicate an improvement in ratings (ie increased alertness), whereas negative signs indicate a drop in the rating (ie decreased alertness). SSS ratings are given on a scale of 1 (Feeling active and vital, alert, wide awake) to 7 (Sleep onset soon, struggling to remain awake).

TABLE VIII.

INDIVIDUAL DIFFERENCES IN PRE AND POST-SLEEP STANFORD SLEEPINESS SCALE RATINGS FOLLOWING EACH CONDITION.

(standard deviation in parentheses).

<table>
<thead>
<tr>
<th>HEATING CONDITION</th>
<th>BASELINE</th>
<th>EARLY</th>
<th>LATE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ave.</td>
<td>5pm</td>
<td>9pm</td>
</tr>
<tr>
<td>Subject</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>0 (0.7)</td>
<td>+2</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>+3 (1.8)</td>
<td>+3</td>
<td>+4</td>
</tr>
<tr>
<td>3</td>
<td>+0.8 (1.1)</td>
<td>+3</td>
<td>-1</td>
</tr>
<tr>
<td>4</td>
<td>+0.6 (1.0)</td>
<td>0</td>
<td>-2</td>
</tr>
<tr>
<td>5</td>
<td>0 (1.4)</td>
<td>+2</td>
<td>-1</td>
</tr>
<tr>
<td>6</td>
<td>+2 (0)</td>
<td>-1</td>
<td>0</td>
</tr>
<tr>
<td>Mean.</td>
<td>+1.0</td>
<td>+1.5</td>
<td>0</td>
</tr>
<tr>
<td>s.d</td>
<td>(1.0)</td>
<td>(1.5)</td>
<td>(2.0)</td>
</tr>
</tbody>
</table>

(+) Indicates increased alertness rating, decreased sleepiness.

(-) Indicates decreased alertness rating, increased sleepiness.
Following baseline nights, the early bath and the late bath with aspirin, the majority of subjects recorded an improvement in subjective alertness, by rating their subjective alertness as higher on the SSS (i.e., nearer to 1 than to 7).

Examination of individual scores suggests a trend towards least improvement in sleepiness ratings the mornings after the late bath with placebo compared to other conditions.

Five subjects rated themselves to be as sleepy or sleepier than they had been on retiring after the late bath and placebo, although one subject reported a large improvement in alertness.

### 3.3.8. DIFFICULTY GETTING UP AND QUALITY OF SLEEP

The number of individual ratings of difficulty getting up and quality of sleep reported following each experimental condition are given in table (IX). The number of ratings given for the total of 22 baseline nights are also shown.
TABLE IX. NUMBER OF RATINGS GIVEN FOR QUALITY OF SLEEP AND DIFFICULTY GETTING UP ON 22 BASELINE NIGHTS, AND FOLLOWING EACH EXPERIMENTAL CONDITION.

<table>
<thead>
<tr>
<th>CONDITION</th>
<th>22 Baseline Nights</th>
<th>Early Bath</th>
<th>Late Bath + Placebo</th>
<th>Late Bath + Aspirin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Difficulty Getting Up</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very Difficult</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Difficult</td>
<td>7</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Moderate</td>
<td>13</td>
<td>2</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Easy</td>
<td>2</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Very Easy</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Quality of Sleep</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Much better</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Better</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Normal</td>
<td>12</td>
<td>4</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Worse</td>
<td>8</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Much worse</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

(***Compared to normal.

Although there were seven reports of difficulty getting up following baseline nights, the majority of reports were of moderate difficulty.

All subjects reported some difficulty getting up after both late bath conditions, whereas there were more reports of no difficulty the morning following the early bath.

Following baseline nights, sleep was usually rated as of normal quality, although eight nights were considered to be worse than normal. These were generally amongst the first few nights each individual spent in the laboratory.

The distribution of ratings of sleep quality is the same for both the early bath and late bath with aspirin, with four of the six subjects reporting normal sleep quality.
The morning after the late bath with placebo, one extra subject reported his sleep as 'worse than normal', but the distribution of ratings was otherwise similar to the two other heating conditions.

3.3.9. **SEX DIFFERENCES IN RESPONSE TO ASPIRIN.**

Table (X) shows the mean changes in sleep stages from mean baseline for males and females on aspirin nights compared to placebo nights. Differences were tested for significance using a two tailed t-test, but as there were only three subjects in each group, the results should be considered with caution.

**TABLE X. MEAN CHANGES FROM BASELINE IN SLEEP STAGES ON ASPIRIN AND PLACEBO NIGHTS. (420 MINS)**

<table>
<thead>
<tr>
<th>SLEEP STAGE</th>
<th>Mean</th>
<th>W+M</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>SWS</th>
<th>REM</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Placebo</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>4.6</td>
<td>-2.6</td>
<td>+0.3</td>
<td>+1</td>
<td>+16.3</td>
<td>+17.3</td>
<td>-17.0</td>
<td></td>
</tr>
<tr>
<td>s.d</td>
<td>(7.1)</td>
<td>(6.3)</td>
<td>(9.1)</td>
<td>(14.7)</td>
<td>(16.2)</td>
<td>(17.9)</td>
<td>(6.5)</td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>-11.5</td>
<td>-7.5</td>
<td>+7.5</td>
<td>+4</td>
<td>+18</td>
<td>+22</td>
<td>-5.5</td>
<td></td>
</tr>
<tr>
<td>s.d</td>
<td>(36)</td>
<td>(6.3)</td>
<td>(41.7)</td>
<td>(2.8)</td>
<td>(32.5)</td>
<td>(29.6)</td>
<td>(10.6)</td>
<td></td>
</tr>
<tr>
<td><strong>Aspirin.</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>+2</td>
<td>-6.3</td>
<td>+5.6</td>
<td>-6</td>
<td>+3</td>
<td>-3</td>
<td>+6.6</td>
<td></td>
</tr>
<tr>
<td>s.d</td>
<td>(7)</td>
<td>(5.6)</td>
<td>(31.2)</td>
<td>(5.5)</td>
<td>(13.5)</td>
<td>(12.2)</td>
<td>(20)</td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>-20.6</td>
<td>-8.6</td>
<td>+26.3</td>
<td>+0.6</td>
<td>-5.3</td>
<td>-4.6</td>
<td>+4.3</td>
<td></td>
</tr>
<tr>
<td>s.d</td>
<td>(17)</td>
<td>(9.6)</td>
<td>(29)</td>
<td>(18.5)</td>
<td>(10.7)</td>
<td>(16.5)</td>
<td>(2.1)</td>
<td></td>
</tr>
</tbody>
</table>

Comparison of each sleep stage for males and females under the aspirin condition shows a large but non-significant difference in sleep stage 2. Males showed an increase of 28.3 minutes following aspirin whereas the females showed an increase of only 5.6 minutes. Males also exhibited a decrease in wake and movement following aspirin. There were no significant differences in sleep stages between males and females for either aspirin or placebo conditions.
3.3.10. **SUMMARY OF RESULTS.**

Artificially raising body temperature by passive heating in a warm bath for half an hour, two hours prior to retiring, caused significant increases in subsequent SWS, mainly due to stage 4 sleep, whereas the same quantity of heating ending six hours before retiring had little effect. Aspirin given in three daytime doses of 600mg counteracted the potential rises in SWS observed following the late heating condition.

Each heating session resulted in mean oral temperature increases of 1.8 to 2.0°C and caused immediate reductions in alertness for the duration of the bath. These improved gradually following termination of heating, and except for the late bath with placebo condition, resumed pre-bath levels before bedtime.

There were no significant effects of the three heating conditions on total sleep, sleep period, REM latency, length of the first REM sleep period, or SSS ratings. However, there was a tendency for subjects to report less improvement in sleepiness ratings the morning after the late bath with placebo.

The late bath plus placebo resulted in significant and consistent overnight increases in EEG RMS power coincident with periods of SWS, but such increases were not found following aspirin administration.
CHAPTER 4.

THE INFLUENCE OF BODY HEATING ON SWS WITH REFERENCE TO TIME OF DAY AND THE EFFECTS OF ASPIRIN.

DISCUSSION.
4. DISCUSSION.

SWS increases observed by Horne and Staff (1983), following late afternoon high intensity exercise, were attributed to high rates of thermal loading induced by prolonged increases in body temperature and sweat loss. Artificially raising body temperature by passive heating in a warm bath also produced these effects, but in the present study, using one third of the heating dose, SWS increases were seen following late evening heating, but not after an identical afternoon session. This suggests that potential SWS increases may be subject to a 'time of day' effect, with body heating having more 'potent' effects on subsequent SWS when given later in the day, in addition to a dependence on the 'dose' of heating given.

4.1. DOSE AND TIME OF DAY EFFECT.

Late evening heating which is neither too stressful nor too close to retiring appears to have consistently positive effects on SWS and stage 4 sleep in particular. This suggests that some effect induced by body heating provokes an intense form of 'recovery' during the first few hours of sleep, although the processes underlying this 'recovery' are as yet unknown.

Similar periods of heating earlier in the day have no effect on SWS although table (VI) shows a small increase in stage 3 sleep (+3.3 min) during the first half of the night but this was non-significant for the night as a whole.

The present study increased body temperature by 1.8°C to 2.0°C for an uninterrupted thirty minute period for each experimental condition, and observed mean whole night stage 4 sleep increases of 16.6 minutes and significant increases in SWS of 22.5 minutes following the 2100hrs heating session, whereas the 1700hrs bath had little effect on these sleep stages. Non-significant decreases in the length of the first REM sleep period and an increase in REM sleep latency were also found following the late bath and placebo condition. Other studies have reported similar effects of body heating on SWS.
In a similar later study, Bunnell and Horvath (in press), passively heated three women and three men (age 20-32y) in a tank of water at 41.¢C (±0.5) for one hour at four different times on separate days. Each heating session allowed a thirty minute rest period out of the tank half an hour after commencing and resulted in mean tympanic temperature increases of 2.58°C.

Late evening heating resulted in significant increases during the first NREM sleep cycle in stage 4 sleep, (7 min), SWS (7.7 min) and integrated slow wave activity, (ISWA) and a significant reduction in the first REM sleep period, whereas early evening heating (6 hrs before 'lights out') tended to result in non-significant increases in ISWA in later sleep cycles. Whole night changes in stage 4 sleep (7.1 min) and SWS (2.5 min) following early evening heating were non significant.

Horne and Staff, (1983) gave eight physically trained subjects two forty minute periods (80 minutes total) of heating in a 42°C bath between 1400hrs and 1800hrs. Rectal temperature increased by 2°C during the heating, which resulted in significant increases in stage 4 sleep (17.2 min), and SWS (18 min), confined mainly to the first 225 minutes of sleep.

Horne and Reid, (1985) raised body temperature by 1.8°C in six unfit subjects during three half hour heating periods (90 minutes total) between 1430hrs and 1730hrs. Significant increases in SWS of 21 minutes were composed of non-significant increases in stage 3 sleep (9 min) and stage 4 sleep (12 min). These were apparent during both halves of the night but not during the first NREM sleep cycle.

The mean whole night stage 4 sleep increases, (5.1 min) and SWS increases (2.5 min) reported by Bunnell and Horvath are smaller than those reported in the present study. Although the subjects are similar in age and sex, the heating periods were longer, and the temperature increases higher (2.68°C for the late evening bath). However as late evening heating ended just prior to subjects retiring to bed, with electrodes attached before the heating session, the slight increase in stage 2 sleep (+9.8 min) and movement
during the first part of the night could indicate that the full effect of heating was counteracted by discomfort.

These authors employed laboratory water tanks, whereas in the present study subjects underwent heating in private bathrooms within a hall of residence. This allowed easier control of the surrounding temperature and ensured that the subject was not stressed through lack of privacy or embarrassment from observers. Only the experimenter was present, and all subjects found the bath sessions relaxing and soporific.

In our pilot study, a longer period of heating, (1.5 hours) which ended an hour before bedtime, also disrupted sleep and resulted in a mean SWS decrease of 17 minutes.

Comparison of the effects of the 2100hrs bath and 1700hrs bath suggests that the proximity of heating to sleep, and therefore the length of the intervening period of wakefulness, may be important in determining the magnitude of effect of body heating on SWS.

There may also be a 'time of day' effect of body heating on SWS in which case larger 'doses' of body heating may be required earlier in the day to produce SWS increases of the same magnitude produced by late evening heating.

Three times the length of heating used in the present study when given between 1430hrs and 1730hrs resulted in mean increases in stage 4 sleep (14.6 min) and SWS (18.5 min) (Horne and Staff, 1983; Horne and Reid, 1985), which are comparable with those produced by 30 minutes of heating at 2100 hrs in the present study, (stage 4 sleep +16.6 min; SWS +22.5 min).

The dose of thermal loading required to produce SWS increases may be dependent on the relative increase in temperature and the length of time for which this is maintained.
SWS increases following body heating have been suggested to be related to increases in cerebral metabolism resulting from increased body temperature, (Horne, 1984). Although in most studies, temperature was raised by approximately 2°C, (Horne and Staff, 1983; Horne and Reid, 1985) few others give the pre and post heating temperatures. It is unclear whether the absolute change in recorded temperature is the determining factor, or the increase above normal temperature, and therefore the degree of heat stress.

In the present study mean pre-bath temperatures for the two conditions were similar, 36.2°C (5pm) and 36.0°C (9pm) rising by a maximum of 2.5°C during the sessions and resulting in larger SWS increases than those reported by Bunnell and Horvath. These authors reported that temperature increases during heating tended to be greater when the pre-bath temperature was lower, as it was prior to their late evening and early morning sessions, (35.21°C and 34.49°C respectively), which suggests that the timing of heating relative to the circadian rhythm of body temperature may be important. However it is worth noting that in the Bunnell and Horvath study, pre-bath temperature was quite low before each of the conditions, and remained within the normal temperature range, (oral, 35.8 to 37.7°C; rectal 35.9 to 37.8°C; Passmore and Robson, 1976) during heating. Consequently the actual thermal load experienced may have been minimal.

Following passive heating and low intensity exercise, Horne and Staff, (1983) reported large drops in rectal temperature following sleep onset which were greater than those seen on baseline nights and persisted for the first two to four hours of sleep. These were accompanied by significant stage 4 sleep increases, suggesting that more intense SWS accompanied a greater temperature fall. Sewitch, (1987) suggested that a regulated rapid drop in core body temperature following sleep onset is a necessary prerequisite to subsequent sustained stage 4 sleep. If body temperature is still raised at bedtime due to passive heating immediately beforehand then this may contribute to sleep disturbance and a lack of SWS effect.
4.2. CIRCADIAN EFFECTS.

Heating given earlier in the day may have less potent effects on SWS, either due to circadian effects, or as a result of unknown processes during intervening wakefulness which cause a 'decay' of the factors influencing the quantity of subsequent SWS.

Under normal circumstances, body temperature is low on awakening and rises rapidly during the morning, to reach a maximum in the late afternoon or early evening, after which it falls gradually until bedtime. Temperature generally falls rapidly after this and reaches its minimum during sleep, usually in the middle or latter half of the hours in bed, (Minors, Mills and Waterhouse, 1976). Hence the 1700hrs bath is on the incline of the circadian metabolic rate and behavioural arousal rhythm, whereas the 2100hrs bath is on the decline.

The 1700hrs bath may act as a boost to the already rising temperature, whereas the late evening bath temporarily reverses the trend of the temperature rhythm. As a result of increasing body temperature, both baths may temporarily increase the rate at which the body's metabolic processes proceed.

If the quantity of SWS is determined by the amount of build up of a substrate which accumulates during wakefulness, then passive heating might cause an increase in the rate of production of such a substrate. However, it could also be suggested that the extra substrate might be removed by metabolic activity in the intermittent time preceding sleep.

Such a removal of accumulated substrate might be accentuated following the 1700hrs bath, when the temperature and metabolic rhythms are close to their daily peak. However, after the late bath, these rhythms are on their decline, and substrate removal may not proceed at the same rate as following the 1700hrs bath. If there were a direct relationship between length of heating and accumulation of substrate, this theory could support a dose X time of day effect on SWS.
When heating immediately precedes sleep, the sudden increase in temperature has an opposing effect on sleep processes. Both fever, (Karacan et al, 1968) and elevated ambient temperatures during sleep have been found to be associated with a greater number of awakenings, increased total waking time and reduced amounts of REM sleep and SWS, (Muzet, Libert and Candas, 1984). Also, the phase of the body temperature rhythm at sleep onset influences sleep duration, such that the higher the temperature is at sleep onset, the longer the duration of sleep, (Gillberg and Akerstedt, 1982).

4.3. POSSIBLE HEATING EFFECTS ON SWS MECHANISMS,

It has been suggested that a mechanism by which heating affects SWS involves the increase in cerebral temperature and hence cerebral metabolism, which is equivalent to an accelerated form of wakefulness (Horne, 1984).

An increase in the metabolic processes associated with wakefulness would lead to an increase in the production of cerebral by-products, and possibly the rate at which accumulating products are removed, unless this is specific to processes within sleep. However, as the amount of preceding wakefulness is one of the major determinants of subsequent stage 4 sleep and therefore SWS, (Webb and Agnew, 1971) if body heating results in an increase in the cerebral processes associated with wakefulness then this could contribute to the observed rise in SWS and the predominance of stage 4 sleep after the late bath.

If it is assumed that the two heating sessions at 1700hrs and at 2100hrs have the same effect on the processes underlying SWS, then body heating must induce some temporary change which only affects delta sleep if still present at sleep onset. The intervening wakefulness preceding sleep after the 1700hrs bath must accommodate a process, or processes responsible for reversing or removing this effect.
If as suggested by Horne and Porter, (1976) some recovery takes place during wakefulness which is sufficient to offset the need for increased SWS, then under controlled experimental conditions, the intervening wakefulness should be roughly proportional to the magnitude of the subsequent effect on SWS.

The nature of these processes is as yet unknown. If SWS mechanisms are affected by circadian rhythms of metabolic processes, then other variables affected by the time of day might also be affected.

Ratings of alertness/ drowsiness from 1700hrs suggest that there may be 'time of day' differences in the immediate effects of each bath. Mean baseline ratings of alertness show a gradual fall from 1700hrs to retiring, with a small improvement at 1930hrs. Figure (4) shows that alertness recovers fairly rapidly to above pre-bath levels following the 1700hrs bath whereas following the 2100hrs plus placebo condition, alertness continues to fall then gradually improves but fails to reach pre-bath levels before retiring. The differences between the early and late bath plus placebo condition are also masked by activity and behavioural differences between subjects, who were more likely to participate in arousing activities after the early bath, whereas as the late baths were closer to bedtime, they were more psychologically prepared to retire following these heating sessions.

Self-rated alertness assessments are correlated with the body temperature rhythm. In a free-running experiment, subjects elected to retire to bed just after the circadian temperature minimum, which corresponded to the nadir of the subjective alertness assessment rhythm, (Czeisler et al, 1980).

An improvement in alertness following the early bath, and the poor recovery following the late bath plus placebo might be expected in line with the relationship between arousal and the body temperature rhythm. However, the differences between the late bath with aspirin and the late bath plus placebo suggest that aspirin may have counteracted any increases in sleepiness induced by the heating by blocking the underlying mechanisms.
4.4. ASPRIN.

The effect of aspirin in counteracting the potential increases in SWS seen after the late bath with placebo, suggests that aspirin might block the sleep promoting processes which may be accelerated by heating. This could be executed in a number of unknown ways, for example it may block the heating effect on mechanisms responsible for SWS, or accelerate waking processes which remove accumulated sleep 'factors'.

In the present study the late bath resulted in a 14% increase in visually scored stage 3 sleep, a 51% increase in stage 4 sleep and an overall increase of 30% in SWS from mean baseline values. The need for the most intense form of delta sleep was reflected in the significant increases in EEG power particularly evident coincident with the first two NREM sleep cycles, (figure 3), which remained above baseline values for most of the night.

Pre-treatment with three 600mg doses of aspirin resulted in an attenuation of potential EEG power increases associated with a reduction in SWS stages which were 18% (stage 3 sleep), 39% (stage 4 sleep) and 29% (SWS) less than those achieved following the late bath plus placebo condition. Stage 2 sleep was increased by 11% compared to baseline nights.

Under non-heating conditions, Horne, Percival and Traynor (1980) found no significant changes in mood or tiredness as a result of the aspirin condition, although the questionnaire used was of limited sensitivity. In the present study, which empl oyed the SSS, there were no significant differences in pre-and post-sleepiness scores between the three heating conditions, but table (VIII) shows that following the late bath plus aspirin, the mean difference between ratings of sleepiness before and after sleep suggests greater overnight improvement in alertness following this condition. The results also showed that following the late bath with placebo condition, SSS scores showed less overnight improvement than they did following the other heating conditions. Also, following the late bath
with placebo condition, EEG power was still greater than baseline after seven hours of sleep. (Figures 3 and 4).

This suggests that the underlying processes responsible for increased SWS and EEG power were still in evidence prior to awakening, and may have accounted for the residual fatigue in the morning. However, there was no carry over of SWS into the next night.

Although aspirin counteracted the effects of late evening body heating, aspirin or it's metabolites may affect sleep by mechanisms other than those affecting SWS. These might also account for the differences in recovery of alertness following the two late heating conditions, and the fatigue on awakening following placebo which was not seen after the aspirin condition.

Horne, Percival and Traynor, (1980) found that the same daytime dosage of aspirin as used in the present study resulted in significant reductions from mean baseline values of stage 4 sleep (15%) and SWS (12%), opposed by a significant increase in stage 2 sleep (9%) in normal subjects studied under non-heating conditions.

Menguy et al, (1972) reported a possible sex difference in the metabolism of aspirin. They compared rates at which the drug was metabolised in the two sexes and reported that females had less serum aspirin esterase in liver homogenate and therefore eliminated the aspirin at a slower rate than the males. In order to eliminate sex differences in aspirin metabolism, Horne, Percival and Traynor (1980) used six female subjects, but in the present study we used equal numbers of males and females. Differences between the sexes in sleep stages on placebo and aspirin nights (table X) show that on placebo nights both sexes had large increases in stage 4 sleep and SWS, and decreases in stage 1 sleep and REM sleep.

Aspirin nights resulted in a decrease in SWS for both sexes, although this was greater for males, who showed a large but non-significant increase in stage 2 sleep. Males had more stage 2 sleep than females on placebo nights, although this difference was also non-significant.
Wake and movement time was decreased from baseline for males after aspirin but showed very little difference from mean baseline values in the female subjects. The differences suggest that the males in this study experienced less sleep disruption following aspirin than females did, although they showed less SWS and a greater increase in stage 2 sleep.

Due to the small number of male and female subjects these differences could be attributed to individual variation rather than sex. Horne, Percival and Traynor, (1980) reported inter-subject differences in response to aspirin, which were particularly evident in sleep stages 2, 3, 4, and SWS.

4.4.1. **ASPIRIN AND L-TRYPTOPHAN.**

Aspirin may influence sleep by competing for the L-tryptophan binding site on serum albumin, thereby elevating serum free-tryptophan, which is a precursor of serotonin, a neurotransmitter involved in processes of sleep induction and sleep maintenance.

L-tryptophan is the only amino acid bound to protein in the human serum, and salicylates cause a release of tryptophan from its binding sites. Less than 20% of L-tryptophan is in a freely diffusible form, but in the presence of sodium salicylate at a concentration of 20mg per 100mls of serum, this proportion rises to 85%, (Mc Arthur and Dawkins, 1969).

Administration of L-tryptophan to rats results in an increase in serotonin levels at sites at which it normally occurs in the brain, accompanied by drowsiness and a decrease in sleep latency. Serotonin and serotoninergic neurons of the raphe system play a major part in the biochemical mechanisms of sleep, (Hartmann, 1977).

L-tryptophan also takes part in a variety of other metabolic pathways leading to proteins and polypeptides, kynurenine and related products, and tryptamine, (Hartmann, Cravens and List, 1974). Any interference with binding could cause a greater availability of a variety of diffusible molecules to enter cells, so that one or more of the metabolic or pharmacological actions of salicylates might be
mediated by the release of such substances, (Mc Arthur and Dawkins, 1969).

Smith and Lakatos, (1970) gave 5g of L-tryptophan orally at 1000hrs to six subjects, and on the same day, 1800mg of acetylsalicylic acid were ingested between 2100hrs and 2300hrs, and then at 0830hrs and 0930hrs the following day. Blood and urine samples were taken at regular intervals. Total, free and bound serum tryptophan concentrations rose to a maximum between 1 and 2 hours after ingestion of a load of L-tryptophan, but following salicylates, at 6 hours, the tryptophan concentrations were much lower and approached normal.

In the present study the total daily aspirin consumed was the same as that used by Smith and Lakatos, but the last dose of aspirin was given at 1800hrs, and subjects retired at 2345hrs, but any effects on tryptophan levels would have been unlikely to have affected sleep after 2359hrs, unless the passive heating delayed the rate of aspirin metabolism.

4.4.2: ASPIRIN AND PROSTAGLANDINS.

One of the most potent properties of aspirin is it's suppression of prostaglandin synthesis. Prostaglandins have a wide spectrum of biological activity and can be generated by all tissues of the body, but enzymes responsible for their synthesis may be differentially sensitive to inhibition by aspirin and aspirin like drugs.

It is possible that specific prostaglandins modulate neurotransmission in discrete areas of the brain, although their precise regional effects are difficult to determine. There is compelling evidence that prostaglandins are formed at multiple sites in the CNS, both neural and non-neural, and interact in a varied manner in physiological and pathological situations. Prostaglandin levels in the cerebrospinal fluid may affect brain function directly or through local changes in the circulation.

Prostaglandins participate in the pathogenesis of fever and inflammation, and aspirin has anti-pyretic, anti-inflammatory and analgesic actions, (Flower, 1973). Different prostaglandins can have opposing actions on the
same cells, but one of the most widely studied central actions of prostaglandins is their effect upon body temperature. Various prostaglandins (A1, E1, E2, Flα and I2) produce hyperthermia in many animal species, and microinjection of PGD2 into the preoptic area of the rat brain results in a fall of colonic temperature, (Ueno et al, 1982) but prostaglandins probably do not contribute to normal temperature regulation. Furthermore, prostaglandin levels in the CSF remain unchanged during thermoregulatory adjustments to hot or cold environments, (Wolfe and Coceani, 1979).

4.4.3. PROSTAGLANDINS AND SLEEP.

Recent studies of the EEG and behavioural effects of prostaglandins in rats have demonstrated that PGE1 produces sedation and cortical activation, a 30 to 60% increase in serotonin turnover and a 30% increase in acetylcholine concentration in the rat brain.

Douthitt, Bugbee, and Perez Cruet, (1973) observed the EEG effects of four prostaglandins injected into Sprague-Dawley rats. All prostaglandins resulted in the rats becoming unresponsive and extremely relaxed, although the EEG was indistinguishable from waking. The duration of response was PGE1, (146.5 min), PGE2 (93.8 min), PGF2α, (77.3 min) and PGFlα, (38.4 min). The evidence was insufficient to conclude that prostaglandins are capable of inducing a state of normal sleep, but it was suggested that endogenous prostaglandins may be involved in the mechanisms underlying normal sleep.

Prostaglandin D2 increased the amount of rat SWS (all NREM sleep in the rat) dose dependently when injected into the pre-optic area of conscious male Wistar rats, suggesting that in rats this area is a site of action of PGD2 on inducing sleep. The injection also resulted in bradycardia and hypothermia. Prostaglandins E2 and F2α induced high voltage EEG waves, but their effect was less than half that of PGD2. Synchronized EEG recordings were observed when the colonic temperature was maintained 1 to 2°C above the initial level, but as hyperthermia synchronizes the cortical
EEG in rats, (Ten Cate et al, 1949) it was not certain whether the effect of PGE2 and PGF2α on inducing delta waves was a direct effect or a secondary effect caused by the high body temperature, (Ueno et al, 1982).

More recently, Inoue et al, (1984) studied the differential sleep promoting effects of five sleep substances nocturnally infused into male Sprague-Dawley rats. Although all the substances, (muramyl dipeptide; delta-sleep inducing peptide, δSIP; SPS-B; uridine and PGD2) exhibited compound specific sleep-promoting characteristics, a ten hour infusion of 0.36nmol of PGD2 resulted in a rapid increase in SWS and a gradual increase in paradoxical sleep. The sleep-enhancing effect was apparent at the beginning of the infusion period but more prominent during the later (dark) period. In the latter half, the cumulative increment of rat SWS was 64.4 minutes (31.7%) and of paradoxical sleep 13 minutes, (36.5%) which differed significantly from baseline from seven hours after the onset of the dark period until the end of the dark period. It was suggested that a property of PGD2 might be to primarily induce sleep and to secondarily activate the sleep-maintaining mechanism.

The role of prostaglandins in sleep processes has been demonstrated in experiments with a variety of assay methods and in various animals, but ethics prevent these experiments on man, consequently the role of specific prostaglandins in sleep of other species must be treated speculatively when considered in relation to human sleep.

The mechanisms underlying SWS production may not necessarily be those affected by aspirin in the present study, but may share a common pathway of biochemical reactions.

Recent evidence suggests that sleep is regulated by a number of humoral factors that have a specific role in sleep processes. As PGD2 and other prostaglandins have been implicated in sleep mechanisms, in the present study it might be suggested that as tissue warming increases prostaglandin turnover, a rise in brain prostaglandin levels as a result of body heating may have contributed to the observed increase in SWS, which was inhibited by aspirin.
Body heating induced temperature rises result in a perfusion of warm blood to the brain via the carotid and cerebral arteries. A 2°C rise in brain temperature which is typical of moderate heat stress, will lead to a 20% increase in brain metabolism, (Siesjo, 1978).

In the brain, the local [H+] is the main determinant of cerebral vascular resistance, and a rise in regional cerebral metabolism increases the local [H+] which causes vasodilatation in that region, (Keele and Neil, 1975). However it is now well accepted that prostaglandins synthesized in the cerebral blood vessel, and thromboxanes, contribute to vascular homeostasis through a direct action on the smooth vessel wall, and are involved in the normal control of cerebral blood flow, and possibly a modulation of muscle responses to neural and hormonal stimuli. PGI2 and thromboxane A2 are relaxant and constrictor agents respectively, at all sites.

Indomethacin, an aspirin like drug which has more potent inhibitory effects, (Flower et al, 1972) reduces cerebral blood flow, suggesting that vessels are normally maintained in a relaxed state by a prostaglandin, (Wolfe and Coceani, 1979).

In the present study, aspirin counteracted the potential SWS increases resulting from the late heating session. It is possible that the mechanism underlying this inhibition may involve the blocking of prostaglandin action on the cerebral vessels in response to the increased cerebral metabolism induced by heating. A reduction in cerebral blood flow would then prevent a substantial increase in cerebral activity.

Proposed mechanisms of increased SWS production following heating have been suggested as a result of an heating related increase in cerebral activity equivalent to increased wakefulness, (Horne, 1984), or an accelerated form of wakefulness, which increases the rate of production of sleep inducing substances, (Bunnell and Horvath, in press).
4.5. BODY HEATING EFFECTS OF EXERCISE AND SWS: DISCUSSION OF EXERCISE STUDIES.

Following the study by Horne and Staff, (1983) Horne proposed that SWS increases observed following high intensity exercise may be related to the exercise induced thermal load and sweat loss. Low intensity exercise failed to produce an increase in SWS although there were significant increases in stage 2 sleep.

Although the body restorative theory of sleep function predicts a positive relationship between SWS and the increased wear and tear of wakefulness associated with exercise, other studies examining the effects of exercise on SWS have shown both positive and negative results.

Some of the problems with these studies outlined by Horne (1981) may account for the lack of SWS increase following exercise and the inconsistency of results.

Many major methodological problems have affected the outcome of exercise and sleep studies. These are; small subject numbers, insufficient control groups or baseline nights, failure to quantify fitness differences, to use individually designed exercise loads based on VO2 max and heart rate, and to look at small segments of the sleep EEG. In addition, no exercise and sleep study has actually measured core body temperature, water loss or other physiological measures directly related to the degree of heat stress, (Horne, 1981).

Zloty et al, (1973), had no baseline data of their own, but studied sixteen distance runners for one night only, and reported greater SWS than normative groups. Shapiro et al, (1981) following a single baseline night, reported initial increases in stage 4 sleep, followed by second night increases in sleep stages 3 and 4, in six subjects following a marathon race.

Walker et al, (1978) gave different non-standardized exercise regimes to matched runners and non-runners and failed to observe any SWS increases in either group, although habitual runners showed significantly more NREM sleep than non-runners during the one baseline night.
Hauri, (1968) saw no change in sleep stages during the first 3.5 hours of sleep in fifteen subjects undergoing strenuous exercise prior to retiring, but did not analyse later NREM sleep cycles for a delayed effect.

Shapiro et al, (1975) studied two highly trained males and reported progressive increases in SWS when cycling was increased from two, to four and then six hours at 50% \( \dot{V}O_2 \) max in the early afternoon over three days and then to 75% \( \dot{V}O_2 \) max for eighty minutes on the fifth day. This study suffers from a small subject number and insufficient baseline recordings which enhance the effects of inter and intra-subject variation.

There appear to be similar findings in the effects of different times of day of heating and exercise and the subsequent changes in sleep.

Body heating immediately prior to retiring has been shown to disrupt sleep, and similarly, exercise taken in the evening may disrupt subsequent sleep if taken just before bedtime. Forty five minutes of light dynamic exercise, immediately before retiring had no effect on SWS, but increased sleep latency, in nine subjects studied by Browman and Tepas, (1976).

In a later study, following a single baseline recording, seven subjects were given eighty minutes of sustained static exercise with a hand dynamometer two hours prior to retiring. This exercise load of 40% of maximal levels, resulted in decreased sleep latency and increased SWS during the first NREM sleep cycle, (Browman, 1980) but this was followed by a subsequent decrease in SWS, resulting in no net increase, (Horne, 1981).

Baekeland and Lasky, (1966) found SWS increases in trained (fit) subjects after afternoon exercise but not evening exercise, which may have disturbed sleep.

Desjardins et al, (1974) also suggested evening exercise may have a disruptive effect. Untrained subjects given high and low intensity exercise loads in the evening showed only a significant decrease in REM sleep in the first third of the night, although there were also non-significant increases in sleep stages 3 and 4.
Similarly Hauri (1968) found only an arousing effect of six hours of evening exercise with no change in sleep stages.

Horne and Porter (1976) conducted a controlled study, of eight subjects of average fitness. A standardized workload of 45% of assessed VO2 max was given in the morning or afternoon on different occasions. Although whole night sleep stages remained within baseline levels, significant increases in stage 3 sleep within the first NREM sleep cycle followed the afternoon exercise. Despite a non-significant increase in stage 4 sleep there was a significant decrease of total NREM sleep stages 2 (>$10\%$ delta activity), 3 and 4 during the first half of the night following the morning exercise.

As the exercise was considered tolerable and within the subjects' capability, it was suggested that the wakefulness following the morning exercise may have been sufficient for recovery, without incurring a need for additional SWS, whereas insufficient wakefulness followed the afternoon exercise so that the recovery processes may have intruded into sleep.

If the level of fitness of a subject determines how exercise affects subsequent SWS, then fitness training would be expected to result in SWS increases following exercise.

Griffin and Trinder (1978) conducted a comparison of eight subjects described as fit or non-fit based on physical work capacity, and found more SWS in fit subjects, mainly due to greater amounts of stage 3 sleep.

Although both groups underwent the same late afternoon exercise of running over a 7.3 km hilly course, level of exertion was not controlled as unfit subjects were allowed to walk when necessary and the time in which the exercise was completed was not specified. The exercise, resulted in a non-significant increase in stage 3 sleep in the four fit subjects, although there was no effect of exercise on SWS as a whole.
Torsvall, Akerstedt and Lindbeck, (1984) found that compared to one night following a no-exercise condition, six fit men showed an increase in the power density of the EEG delta band over two days of increasing exertion, although percentage SWS was not significantly affected.

Eighteen weeks of fitness training resulted in increases in lean body mass of eight army recruits studied by Shapiro et al, (1984), coincident with significant improvements in SWS, maximal oxygen consumption and lactic acid turn point. However, Paxton et al, (1984) saw no difference in SWS between matched athletes and non-athletes, although SWS was found to be significantly correlated with lean body mass.

Comparison of trained and untrained subjects should also take into consideration other factors such as dietary habits and regular sleeping habits, which may affect sleep, (Horne, 1981).

Producing a thermal load by exercise might be expected to result in increased SWS within subsequent NREM sleep cycles, normally low in SWS.

In an exercise study similar in design to their study of body heating during sleep interruption, Bunnell, Bevier and Horvath, (1984) asked ten subjects of mixed fitness to walk on a treadmill at 60% \( \dot{V}O_2 \text{ max} \) for fifty minutes, in order to produce a state of elevated temperature and metabolism during a forced sleep interruption.

Comparison of the third and fourth NREM sleep cycles of sleep interrupted with exercise or no-exercise found no change in any NREM sleep variable. There was however an increase in slow wave activity during the interrupted night without exercise compared to baseline which may have compensated for the reduced NREM sleep during the third cycle.

As Horne and Moore (1983) found SWS increases following exercise were delayed to the second and third NREM sleep cycles, any delayed effect on SWS following exercise during sleep interruption may have been missed in this analysis as only the third and fourth cycles were examined.
The controlled study conducted by Horne and Staff (1983), suggests that the rate of thermal loading during exercise in fit subjects is the key to subsequent increases in SWS.

The lack of SWS effect in the study by Bunnell et al (1984) could be attributed to the exercise loads, which may not have been sufficient to induce a thermal load. Unfortunately, temperature was recorded from one subject only, who showed only a 1°C rise during the exercise condition.

The present study utilised a passive heating procedure similar to that used by Horne and Staff, (1983) and Horne and Reid, (1984), and found that raising body temperature in untrained subjects lead to comparable increases in subsequent SWS. Time of day effects and the 'dose' of heating were found to be important influences in this relationship.

4.6. CONCLUSIONS.

Late evening passive heating in a warm bath causes SWS increases which are not found following identical heating conditions in the late afternoon. Administration of 1800mg of aspirin in three doses prior to late evening heating counteracts the heating effect on subsequent SWS.

The SWS effect is reflected by an increase in whole night EEG power, and is mainly confined to stage 4 sleep, which has been suggested to represent a more intense form of 'recovery' during sleep. It is possible that increased cerebral activity associated with an increase in brain temperature, may act as an accelerated form of wakefulness, (Horne, 1984), and as the length of preceding wakefulness is one of the main determinants of stage 4 sleep, (Webb and Agnew, 1971) this may account for the predominance of stage 4 sleep in the extra SWS.

It is possible that heating effects on SWS are subject to a 'dose X time of day' interaction which accounts for the lack of effect following the 1700hrs condition and the 1.5 hours period of heating used in the pilot study.
Although the precise mechanisms are unknown, the 'time of day' effect may be subject to circadian control of underlying metabolic processes, and/or related to processes active during intervening wakefulness and therefore the proximity to sleep onset. The duration of heating and/or the temperature increases resulting from passive heating may influence the effect on SWS.

The mechanisms underlying SWS may not be the same as those affected by aspirin, yet aspirin counteracted the effects of the late evening bath. Aspirin or it's metabolites may affect SWS by the inhibition of prostaglandin synthesis or by elevating serum free-tryptophan which is a precursor of the neurotransmitter serotonin.

4.7. SUGGESTIONS FOR FUTURE STUDIES.

It would be of interest to perform similar late evening body heating sessions raising body temperature by approximately 2°C, achieving a range of pre-heating temperatures in an environmental chamber. Circadian effects could be investigated by performing the experiment at different times of the day.

It would be useful to have regular daily temperature recordings from subjects in a controlled environment in order to observe the phase of the temperature rhythm prior to each heating condition, without the rhythm being masked by behavioural activities and environmental variables.

In order to determine whether raising the brain temperature alone is the key to SWS increases, or whether other physiological reactions to exercise or passive heating are responsible, a controlled experiment to increase brain temperature and observe subsequent sleep would be worthwhile.
CHAPTER 5.

A STUDY OF THE RELATIONSHIP BETWEEN THE NREM SLEEP ALPHA ANOMALY AND MUSCULOSKELETAL DISCOMFORT IN A LOCAL POPULATION.

INTRODUCTION.
5. INTRODUCTION.

In contrast to the last experiment, this study investigated an 'abnormal' aspect of sleep in subjects with a specific disorder. The prevalence of the 'alpha-delta' sleep anomaly was investigated in patients with musculoskeletal discomfort, and in a normal population. Alpha frequency waves have previously been observed in the SWS of patients with 'Fibrositis' in association with morning muscular pain and stiffness. These symptoms may be mediated by a specific SWS disturbance 'alpha-delta' sleep, which has been suggested to disrupt the restorative role of SWS.

5.1. 'ALPHA-DELTA SLEEP.'

'Alpha-delta' sleep was first observed in the sleep of nine psychiatric patients who complained of somatic malaise and fatigue. These patients were frequently classified as neurotic depressives, as diagnosis of a specific disorder had proved difficult on several occasions. The 'alpha-delta' phenomena was observed in chronic schizophrenics, depressed patients, morphine addicts and a uremic syndrome patient, and commonly in normal two year old children, (Hauri and Hawkins 1973).

'Alpha-delta' sleep occurred "whenever one would expect delta sleep in normal subjects: early in the night and opposite from REM sleep in the ninety minute cycle". Patients occasionally alternated between 'alpha-delta' sleep and stage 3 sleep, but stage 4 sleep was generally absent. During NREM alpha sleep, the auditory awakening threshold was greater than during stage 2, and sleep mentation appeared more dreamlike than normal. REM sleep seemed unaffected by 'alpha-delta' sleep events in the same sleep cycle, although the time between subsequent REM sleep periods was increased.
'Alpha-delta' sleep was described as:-

"...a mixture of 5-20% delta waves (>75μV, 0.5-2c/sec) combined with relatively large amplitude, alpha-like rhythms (7-10 c/sec). These alpha rhythms are usually 1-2 c/sec slower than waking alpha. No sleep spindles are found during 'alpha-delta' sleep, and this stage is never scored if there are enough delta waves to score stage 3 sleep". (Hauri and Hawkins, 1973).

Five patients with complaints of poor quality sleep, daytime fatigue and malaise, who showed 'alpha-delta' sleep underwent psychiatric interviews and the Minnesota Multiphasic Personality Inventory (MMPI). Although none met the criteria for psychiatric disorder, all were mildly anxious, sensitive, conscientious and concerned with maintaining control. The alpha component of 'alpha-delta' sleep was clearly different from the subject's waking alpha and was predominantly central and frontal compared to posterior waking alpha, (Watson et al, 1982).

5.2. ALPHA ACTIVITY DURING WAKEFULNESS.

EEG alpha activity (7-11Hz) normally occurs during relaxed wakefulness when the eyes are closed, but disappears when the eyes open or attention becomes fully engaged, (Adrian and Yamagiwa, 1935). There is a normal slowing of the alpha pattern with advancing age, and the alpha frequency shows a relationship to changes in basal metabolic rate. Conditions such as mild anoxia, hypoglycaemia and hypothyroidism may cause reductions in alpha frequency, whereas hyperthermia and hyperthyroidism cause an increase, (Kleitman, 1967). Berger, (1930; cited by Kleitman, 1967) considered the alpha pattern to be "an expression of the psycho-physical processes taking place in the brain", whilst Lemere, (1936; cited by Kleitman, 1967) related the ability to produce a good alpha pattern to the affective capacity of the individual.
Waking alpha activity is normally maximal within the parieto-occipital regions and may spread to the central and temporal regions, but the alpha frequency component of 'alpha-delta' sleep is concentrated frontally and parasagittally on the scalp surface, (Weber et al, 1983).

The 'kappa' rhythm, which is an alpha variant, occurs fronto-temporally and has different functional properties to occipital alpha. 'Kappa' activity is related to thinking, and increases during intellectual tasks such as reading, discriminating, learning and mental arithmetic, (Chapman et al, 1962), and problem solving behaviour, (Kennedy et al, 1949). Kappa has also been identified fronto-centrally during sleep stages 3 and 4 in a healthy adult man, (Sewitch et al, 1978).

5.3. THE 'FIBROSITIS SYNDROME'.

Although fibrositis has been variously described since 1904, it is not generally recognized among physicians, but is a common cause of disability, resulting in a high proportion of work absenteeism, (Yunus et al, 1981).

Fibrositis has a 5:1 female to male ratio, and is most commonly seen between the ages of 40 and 60y, (Wolfe, 1986) although the syndrome is common and readily identifiable in children, (Calabro, 1986). For most fibrositics, symptoms begin gradually, and a history of 'growing pains' and leg aches in childhood is a common feature.

Fibrositic disorders can be divided into a primary form in which there is no underlying disease, and a form which may be secondary to trauma, rheumatic diseases, hypothyroidism and malignancy.
5.4. ALPHA-DELTA SLEEP IN THE 'FIBROSITIS SYNDROME'.

'Alpha-delta' sleep has not previously been observed in normal sleep, and appears to be particularly prevalent in psychiatric illness, (Hauri and Hawkins, 1973), and in disorders in which psychological factors affect the course and severity of the disorder, for example the 'Fibrositis syndrome', and rheumatoid arthritis, (Moldofsky et al, 1983).

Weber et al, (1983) studied thirteen patients who had been diagnosed as fibrositics, and two who complained of a 'non-restorative' sleep syndrome, and who had shown 'alpha-delta' on previous recordings. Only one fibrositic demonstrated 'alpha-delta' sleep whereas both of the non-restorative patients demonstrated this anomaly.

Moldofsky et al (1975), observed a NREM sleep disturbance similar to 'alpha-delta' sleep in ten patients who conformed to the clinical criteria for the 'Fibrositis syndrome'.

In a later study by Catesby Ware et al, (1986) five depressed patients and nine fibrositics showed alpha intrusions in more than 50% of their NREM sleep. As depression is a common feature in fibrositis, it was suggested that alpha intrusions may be one of a number of markers in sleep that accompany depression, (Catesby Ware et al, 1986).

5.5. PSYCHOLOGICAL FACTORS IN 'FIBROSITIS'.

Fibrositis is a poorly defined and controversial term commonly used to describe widespread and variable musculoskeletal aching and stiffness, in the absence of an organic disease, (Beetham, 1979). In several of the studies published in the last thirty years, 70% of patients diagnosed as having fibrositis actually suffered from psychological disorders. Compared to matched rheumatoid arthritis sufferers, fibrositics showed higher and more variable scores on neurotic scales of hypochondriasis and hysteria and on psychotic scales of paranoia and schizophrenia, suggesting that they were more
psychologically disturbed, (Payne et al, 1982). Eighteen female fibrositics studied by Marks et al, (1983) reported anxiety and depressive symptoms, showed a psychological profile similar to that of somatizing and pain prone patients, and claimed that their symptoms influenced their interpersonal relationships with males and with their mothers. A variety of non-rheumatic complaints, such as 'tension' headaches and irritable bowel syndrome, (Wolfe, 1986), sleep disturbance, anxiety disorders and 'dry eyes' are common to both fibrositics and depressives, (Goldenberg, 1986).

Payne, (1982) suggests that fibrositic patients probably do not resemble each other to an unusual degree, except in their physical complaints and the fact that they are psychologically disturbed.

Fibrositic patients typically report an emotionally distressing event at the time of onset of their symptoms. Most prominent among these are 'stress', 'emotions', family change, and fatigue, but physical factors such as trauma, surgery, and overactivity are also frequently reported, (Yunus et al, 1981). Smythe and Moldofsky, (1977, 1978) suggest that such incidents may induce a psychophysiological arousal mechanism or internal alpha inducer, which competes with the NREM sleep system impairing the restorative role of SWS and producing the symptoms of musculoskeletal pain and fatigue. Similarly, in acute rheumatoid flares, nocturnal intrusions of articular pain stimuli during sleep may also induce an alpha NREM sleep disturbance and subsequent morning symptoms, (Moldofsky, Lue and Smythe, 1983).

Fibrositic patients are demanding of themselves and others, and although they give the impression of exaggerating their symptoms on examination, they deeply resent any suggestion that they are using their complaint as a means to escape responsibility, and they dislike using drugs, alcohol or other crutches, (Smythe, 1979).
5.6. MUSCULOSKELETAL DISCOMFORT.

Musculoskeletal aching and stiffness are aggravated by fatigue, weather change or chilling. Although many patients report "I hurt all over", low back and neck pain are among the commonest symptoms and are often the most predominant initial and persistent discomforts. Neck pain often radiates to the shoulders and arms, whilst in low back pain, there is pain in the buttocks and hip regions often extending down the legs. Patients are often suspected of having intervertebral disc disease and may undergo hospitalization and even surgery for this, (Wolfe, 1986).

Fibrositis sufferers feel stiff on arising and often improve with moderate activity, although they may also suffer with 'gelling', which is a form of aching and stiffness that commonly occurs after inactivity or rest, (Beetham, 1979).

5.7. NEUROVASCULAR COMPLAINTS.

Neurovascular complaints which may also be present, include coldness, numbness, paraesthesia, mottled skin, a reticular skin pattern and swelling, and in some fibrositics they are the most prominent and disturbing feature. The hands are 'always cold' and painful in the cold, sometimes having a mottled appearance. Thirty per cent of fibrositics at a university clinic were diagnosed as having Raynauds syndrome, (Wolfe, 1986).

5.8. 'TENDER POINTS'.

One of the essential features of fibrositis is exaggerated tenderness at anatomical locations of high density muscle and other tissue types, generally unrelated to the areas of pain. These points are found at precisely predictable points which are slightly tender in normal individuals, and are consistent across patients of widely differing age, shape and origin. Tenderness at any of the fourteen bilateral sites may be amplified in the presence of
referred pain, SWS deprivation or disturbance, and in fibrositis, (Smythe, 1979).

5.9. PSYCHOGENIC RHEUMATISM.

Fibrositis is often misdiagnosed as 'psychogenic rheumatism', which describes the aching of anxious and depressed patients. Whereas fibrositic patients are influenced by heat, cold, weather, exercise, rest and medications and are described as being at the mercy of their external environment, patients with psychogenic rheumatism are at the mercy of their internal environment and are moody, hostile, anxious or depressed. These patients often have an inappropriate attitude and may exaggerate their symptoms, acting as though they are in great distress, and appearing tense, nervous and defensive.

Patients with psychogenic rheumatism wake up tired rather than stiff, and nothing seems to give them relief. The pattern of their symptoms is inconsistent, and although they report 'severe pain' they actually describe fatigue, weakness, numbness, pressure, fullness or burning sensations. They are often worried about constipation, rectal itching, mild rashes, tension headaches, eye irritation or indigestion, (Beetham, 1979).

5.10. SUBJECTIVE SLEEP QUALITY IN FIBROSITIS.

In a study conducted by Campbell et al, (1983), fibrositics and control subjects complained with equal frequency of difficulty falling asleep, waking frequently and early. Almost all fibrositics awoke tired, with stiffness, aching and daytime fatigue.

Most patients describe a disabling exhaustion, which is unrelieved by a lessened activity load and exacerbated by exercise. Patients emphasize their physical complaints which are intensified on awakening, but minimize their sleep disturbance, although they arise feeling unrefreshed and more exhausted than the night before, (Smythe, 1979).
The fibrositic constellation of non-restorative sleep disturbance, with overnight increase in musculoskeletal discomfort, daytime fatigue and emotional distress describe a specific syndrome, the Rheumatic Pain Modulation Syndrome, (Moldofsky, 1982) in which sleep physiology may serve as a modulating influence on the noxious subjective experiences in patients with non-articular and articular rheumatic disease.

In an earlier study by Moldofsky et al, (1975), seven female and three male fibrositics showed an overnight increase in musculoskeletal tenderness at thirteen bilaterally symmetrical sites assessed by a single observer. This was coincident with the appearance of a NREM sleep alpha rhythm contaminating sleep stage 2, 3 or 4 in seven patients. Three patients not showing 'alpha-delta' had little or no sleep stage 3 or 4. Alpha appeared throughout all the NREM sleep stages in four of the patients, whilst in two it was present in sleep stages 2 and 3 only, and in another patient, in sleep stages 3 and 4 only. As a control group was not used, other sleep parameters were compared with those of middle aged (41 to 46y) males from Williams et al, (1972), but different criteria for delta sleep scoring made comparison of stages 2, 3 and 4 impossible. Stage 1 sleep was found to be twice as long in the fibrositics, which may have been attributable to their greater ages (37 to 64y).

Patients reported reductions in energy and anxiety on awakening which were statistically non-significant. Mood ratings according to an adjective check list derived from Nowlis and Nowlis, (1956) by Moldofsky and Chester, (1970) characterized the subjects moods as 'anger-irritability' and 'anxiety-sluggishness' throughout the study. Investigator bias may be influential in this study, and there is a lack of data for the reported mood changes, (Moldofsky et al, 1975).

As various psychologic, environmental, and physiologic disturbances may adversely affect sleep, Moldofsky proposed that any of these could be shown to be associated with the EEG sleep arousal disorder, and pain and mood symptoms.
5.11. STAGE 4 SLEEP DEPRIVATION EXPERIMENT.

Moldofsky et al, (1975), hypothesized that stage 4 sleep deprivation in healthy subjects would be associated with the appearance of musculoskeletal and 'mood' symptoms. Six young (19 to 24y) sedentary males were screened for medical and emotional abnormality using the Cornell Medical Index and Eysenck Personality Inventory. Mood assessment was omitted as a 'blind' investigator was not available. Musculoskeletal tenderness and subjective symptom ratings were collected as in the previous study. Three nights of stage 4 sleep deprivation were achieved by delivering auditory stimuli up to 90db against a background of 55db whenever four delta waves, (>75μV, <2Hz) appeared in one 40 second epoch of record. This was given in anticipation of the emergence of stage 4 sleep and continued until a shift into stage 1 sleep or a movement arousal was indicated on the EEG. Awakening the subject was avoided where possible.

Stage 4 sleep deprivation resulted in a shift into stage 3 sleep, which was increased compared to baseline nights. Dolorimeter scores of musculoskeletal tenderness were highest during the deprivation period, largely due to an increase over the first deprivation night, and a decrease over the first recovery night. Details of the scores over the second and third deprivation nights are not given.

All subjects described a 'heaviness' of the entire body, and an unusual somatic fatigue during the deprivation period, some subjects also reported gastro-intestinal symptoms, loss of appetite, depression and irritability. Regions commonly affected with aching and stiffness included shoulders, lower limbs, head, neck and upper back.

Stage 4 sleep deprivation became increasingly difficult over the three experimental nights, requiring increases in frequency, duration and intensity of the arousing stimulus. As delta became more resistant, the stimulus frequently gave rise to a period of mixed alpha, (7-11Hz) and delta (<2Hz) waves lasting up to twenty seconds, terminated by a movement arousal, or a shift to sleep stage 1 or 2 or transient wakefulness.
Three physically fit subjects who underwent stage 4 sleep deprivation on a different occasion, failed to show a change in either dolorimeter scores or subjective well-being. As Moldofsky, (1986) comments, physical fitness may have been an important modulating influence in the emergence of symptoms following artificial alpha induction into NREM sleep. It is also possible that psychological changes and increases in subjective feelings of well-being which accompany physical training, (Mc Cain, 1986) influence the likelihood of subjects reporting somatic discomfort.

Moldofsky and Scarisbrick, (1976) studied the effects of REM sleep deprivation in seven subjects, achieved using the same technique for depriving stage 4 sleep. However, there were difficulties in REM sleep identification, so that REM sleep was reduced to 6-6.8%, compared to 0.8-3.1% for stage 4 sleep deprivation. This resulted in shifts into stage 1 sleep or wakefulness with a significant increase in the number of arousals, and a decrease in stage 2 sleep. Three subjects showed an increase in dolorimeter scores whereas four showed a decrease, but there were no differences observed for musculoskeletal symptoms. One subject reported widespread and painful musculoskeletal symptoms with headache, generalized aching, tiredness and sluggishness, which continued through the recovery period. This was attributed to an alpha NREM sleep disturbance in his sleep, the effects of which were suggested to have been potentiated by the REM sleep deprivation. Other complaints included tiredness (5 subjects), irritability, (4 subjects), headache and leg stiffness, (1 subject) and loss of appetite, (1 subject).

5.12. SLEEP DISTURBANCE AND FIBROSITIC SYMPTOMS.

If impaired sleep is of pathogenic significance in fibrositis, other disorders associated with frequently interrupted sleep might also be strongly associated with this syndrome, for example sleep apnea. Molony et al, (1986) observed brief bursts of NREM sleep alpha activity concomitant with episodes of sleep apnea in a 54 year old male who complained of musculoskeletal discomfort, daytime
lethargy and somnolence. A review of eleven past apneac patients showing at least two apneas per hour showed three to have generalized aches and pains of more than three months duration, with at least five typical and consistent tender points. However three of fifty general medical clinic patients also fulfilled the criteria for primary fibrositis. A significantly increased time in transitional sleep, an increased frequency of miniarousals and longer sleep latencies suggested a poorer quality of sleep in the patients with fibrositic symptoms, (Molony et al, 1986).

Moldofsky and Lue, (1980) reported that NREM sleep alpha time and power correlated with overnight increases in subjective pain and muscle tenderness, and alpha power also correlated with increased hostility. NREM delta time correlated with overnight decrease in subjective pain, decreased hostility and anxiety and increased energy. Whilst these results appear to indicate that NREM sleep alpha may be associated with morning pain and mood symptoms, this may be limited to mood, as alpha during REM sleep also correlated with overnight increases in subjective pain, and decreased energy.

5.13. TREATMENT AND MANAGEMENT OF FIBROSITIS.

The fibrositic patient presents a clinical picture which is difficult to classify under a distinct diagnosis. The disturbed sleep physiology may account for the apparent relationship between depression, anxiety and somatic symptoms.

Current therapeutic strategies are only partly successful and are aimed at modifying those factors which seemingly influence the severity and course of the condition, for example, sleep disturbance, mechanical and psychic stresses and other causes of chronic pain, (Bennett, 1986). Most patients benefit with a clear explanation of the nature of their symptoms and an attempt at diagnosis.
Drug treatment of the sleep disturbance has focussed on removing the alpha anomaly, but has failed in most cases. Barbiturates do not restore the sleep pattern, benzodiazepines are often ineffective and unable to remove the alpha anomaly, and chlorpromazine produces poorly tolerated side effects such as morning lethargy, (Smythe, 1979).

Watson et al, (1985) reported successful reduction of 'alpha-delta' sleep and coincident malaise in seven of nine patients treated with amytryptiline (a tri-cyclic anti-depressant) 10 to 30 mg daily, although REM sleep was significantly depressed. Cyclobenzaprine, a tri-cyclic non-anti-depressant not available in the UK also showed favourable results when tested in 120 fibrositics. Cyclobenzaprine has muscle relaxant properties which may offer additional advantages, (Bennett et al, 1986)

Surgical supports, exercise programmes analgesics, massage, heat applications and short term use of tranquillisers may alleviate some of the symptoms, but Smythe, (1979) suggests that changes in lifestyle and attitude may be most beneficial although difficult to achieve.

5.14. SUMMARY.

A sleep phenomenon not previously reported in normal sleep has been identified in depressed patients and fibrositics, in whom psychological factors may affect the course and severity of the disorder. The clinical picture of sleep disturbance, musculoskeletal aching and stiffness, daytime fatigue and mood changes which have been described as a 'non-restorative' sleep syndrome, have been associated with psychophysiological arousal during SWS giving rise to 'alpha-delta' sleep.

Moldofsky and his colleagues identified the 'alpha-delta' phenomenon in fibrositic patients, and subsequent studies focussed on the presence of this anomaly in patients with similar symptoms of 'non-restorative' sleep and musculoskeletal discomfort. It was hypothesized that the intrusion of alpha into NREM sleep may mediate the
appearance of these symptoms. However, 'alpha-delta' sleep was absent in the sleep of some fibrositics studied by Weber et al, (1983), indicating that 'alpha-delta' sleep alone does not result in the appearance of symptoms. Furthermore, 'alpha-delta' and subsequent musculoskeletal symptoms were induced in healthy volunteers, although the sleep of normal subjects was not examined for evidence of this anomaly.

More recent reports (Sewitch et al, 1978; Scheuler et al, 1983, Weber et al, 1983) have identified the alpha activity of 'alpha-delta' sleep to be predominantly frontal and central rather than occipital in origin, and 1Hz slower than waking alpha. This suggests that the NREM alpha anomaly may differ from waking alpha in its functional characteristics.
As previous studies have been laboratory based, involving patients selected for their conformity to clinical criteria for fibrositis, they have been 'sterile' though controlled in their approach, and may have inadvertently focussed on only one characteristic of these individuals. As 'alpha-delta' sleep has been identified in a variety of clinical disorders, the following study aimed to investigate more fully the incidence and characteristics of the NREM 'alpha' sleep phenomenon.

A group of subjects with musculoskeletal discomfort, and a group of symptom-free control subjects were studied at home under 'normal' living conditions, rather than in the confines of the sleep laboratory. Questionnaires and interviews were used to obtain a 'complete' picture of each subject, in contrast to a 'clinical' impression, in order to identify any common underlying characteristics of their personalities or lifestyles.

An attempt was made to identify the origin of the alpha anomaly, and to investigate whether it was predominantly frontal in origin as previously reported, and whether there are any variations in it's presentation on the sleep EEG.

The following flow diagram shows the order in which the different components are described in this part of the thesis.
SCHEMA OF COMPONENTS OF THESIS.

A STUDY OF THE RELATIONSHIP OF THE NREM SLEEP ALPHA ANOMALY TO MUSCULOSKELETAL DISCOMFORT IN A LOCAL POPULATION.

PRELIMINARY QUESTIONNAIRE SURVEY.

MAIN SLEEP EEG STUDY.

MAIN SLEEP QUESTIONNS
STANDARD SLEEP RECORDINGS
FRONTAL-OCCIPITAL RECORDINGS
+ NEUROMAPPER QUESTIONS
PRE/POST SLEEP RECORDINGS

SUB-STUDIES.

CONSISTENCY SLEEP MENTATION DAILY LOGS TEA/COFFEE INTAKE STUDY
STUDY STUDY STUDY

A STUDY OF THE INCIDENCE AND CONSISTENCY OF NREM SLEEP ALPHA IN YOUNG HEALTHY STUDENTS.

PRELIMINARY QUESTIONNAIRE ANALYSIS.

SLEEP EEG STUDY QUESTIONNAIRE

SLEEP EEG STUDY.

STANDARD SLEEP RECORDINGS
FRONTAL-OCCIPITAL RECORDINGS
PRE/POST SLEEP QUESTIONNAIRES.

CONSISTENCY STUDY.
The study began with a preliminary questionnaire survey. Suitable subjects were then chosen for the sleep EEG study, which involved home sleep recordings using two different EEG montages. In addition to the standard sleep EEG montage, an alternative montage was employed to study the distribution of alpha activity between frontal and occipital electrode placements.

Four sub-studies were performed to investigate areas of interest which arose during the course of the main sleep EEG study, and these will be discussed later.
CHAPTER 6.

A STUDY OF THE RELATIONSHIP BETWEEN THE NREM SLEEP ALPHA ANOMALY AND MUSCULOSKELETAL DISCOMFORT IN A LOCAL POPULATION.

PRELIMINARY QUESTIONNAIRE SURVEY.
6. PRELIMINARY QUESTIONNAIRE SURVEY.

6.1. METHOD.

Two questionnaires were compiled for distribution to subjects with a) Back pain and muscular problems, and b) Sleep problems. These can be found in appendix (I).

As the questionnaires were intended for distribution to the general public, questions were designed to be easily understood, avoiding medical terminology and ambiguity.

The questionnaires were designed to obtain information about the subjects' sleep and subjective symptoms in order for the experimenter to:

a) Assess whether the individual's symptoms were relevant to the study.

b) Assess whether there were any confounding characteristics that would:-

   (i) Interfere with sleep, eg, dependence on hypnotics, excessive consumption of alcohol.

   (ii) Contribute to the symptoms, eg, severe chronic arthritis, organic diseases.

   c) Obtain an overall picture of each individual's health and sleep in order to determine the presence of any common characteristics in the group as a whole.

   d) Determine the prevalence of sleep problems in patients with musculoskeletal discomfort, and the prevalence of musculoskeletal discomfort and somatic malaise in patients with sleep problems.

Each covered personal details of age, sex, height, weight, occupation, recreational activities and marital status, in addition to number of cigarettes smoked, alcohol consumed per night, and use of medications and sleeping tablets. Details on recent or current stressful experiences were also requested.

The questionnaire entitled 'Survey on Muscular Discomfort' obtained details of the characteristics of musculoskeletal discomfort in addition to sleep. Subjects were requested to shade in areas of the body affected on a diagram taken from the McGill Pain Questionnaire, (1975).
The questionnaire entitled 'Survey on Sleep Problems' aimed to identify whether any somatic symptoms might be associated with the initial complaint of poor sleep.

6.2. SUBJECTS.

Potential subjects were introduced to the study by:

a) Questionnaires distributed to patients via:

(i) General Practitioners from a local Health Centre
(ii) A Consultant Rheumatologist.

Both (i) and (ii) practised locally, and were approached by the experimenter, who explained the nature of the study and requested their assistance. The general practitioners agreed to distribute relevant questionnaires to patients who presented with sleep problems or unresolvable and chronic (more than three months duration) muscular discomfort, and the consultant rheumatologist gave out 'Survey on Muscular Discomfort' questionnaires.

All subjects were informed that participation in the study did not affect their treatment in any way, and that they were obliged to remain anonymous if they so wished, but if they were willing to participate further, their name and address would be required. They were ensured that all information would be kept in confidence, and were asked to return completed questionnaires in a sealed envelope (supplied with the questionnaires) to the Health Centre reception. From there they were collected by the experimenter.
b) **Advertisements.**

(a) These were placed in the personal column of a local newspaper, describing the study and requesting suitable volunteers to write in confidence.

(b) Posters were distributed around the University departments, student buildings and halls of residence.

(c) An advertisement was also placed in the University gazette (primarily read by University staff), volunteers were asked to write or telephone for a private interview.

Payment was not offered for participation, in order to encourage only individuals with a genuine interest in the study.

**Control subjects.**

Posters requesting healthy good sleepers were distributed around the University in order to obtain a control group. Control subjects were also obtained from personal communication between friends and spouses of subjects. Control subjects completed the 'Survey on Sleep' questionnaires and underwent the same procedure as those with muscular discomfort or poor sleep.

6.3. **ANALYSIS OF QUESTIONNAIRE DATA.**

Data collected from the sleep and muscular discomfort questionnaires was analysed using computer programs written in BBC Basic. For each questionnaire, an initial program was written to collate the answers and another to perform simple statistical analyses, (appendix II).
6.4. RESULTS OF PRELIMINARY QUESTIONNAIRE SURVEY.

Returned questionnaires which were adequately completed, legible, and suitable for the study were analysed using computer programs written for the 'Survey on Sleep' and 'Survey on Muscular Discomfort' questionnaires, (appendix III). If the details on the 'Survey on Sleep' questionnaire indicated that the subject's details were more relevant to the 'Survey on Muscular Discomfort' questionnaire, then this was supplied for completion, and analysed accordingly. However, in most cases the relevant questionnaires were completed.

The results of analysis of the two questionnaires are shown in the following tables. Subjects who completed the 'Survey on Muscular Discomfort' questionnaire are referred to as the 'Discomfort' group, subjects who completed the 'Survey on Sleep' questionnaire are the 'Poor Sleep' group, and controls subjects are the 'Control' group.
TABLE XI.

RESULTS OF QUESTIONNAIRE SURVEY:
CHARACTERISTICS OF EACH GROUP OF SUBJECTS.

<table>
<thead>
<tr>
<th></th>
<th>Discomfort</th>
<th>Poor Sleep</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>13</td>
<td>10</td>
<td>11</td>
</tr>
<tr>
<td>Females</td>
<td>16</td>
<td>6</td>
<td>13</td>
</tr>
<tr>
<td>Total</td>
<td>29</td>
<td>16</td>
<td>24</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (y)</td>
<td>32.8</td>
<td>31.2</td>
<td>28.0</td>
</tr>
<tr>
<td>Range (y)</td>
<td>19-51</td>
<td>19-57</td>
<td>19-44</td>
</tr>
<tr>
<td>s.d. (y)</td>
<td>10.6</td>
<td>11.7</td>
<td>7.6</td>
</tr>
<tr>
<td>Marital Status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married%</td>
<td>48</td>
<td>38</td>
<td>17</td>
</tr>
<tr>
<td>Single%</td>
<td>41</td>
<td>50</td>
<td>62</td>
</tr>
<tr>
<td>Divorced%</td>
<td>10</td>
<td>12</td>
<td>21</td>
</tr>
<tr>
<td>Smokers%</td>
<td>27</td>
<td>31</td>
<td>25</td>
</tr>
<tr>
<td>Ave no smoked</td>
<td>13</td>
<td>17</td>
<td>25</td>
</tr>
<tr>
<td>Occupation%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-manual</td>
<td>96</td>
<td>100</td>
<td>95</td>
</tr>
<tr>
<td>Manual</td>
<td>4</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>PNS%</td>
<td>13</td>
<td>25</td>
<td>8</td>
</tr>
<tr>
<td>Activity Level%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frequent vigorous</td>
<td>7</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Regular exercise</td>
<td>27</td>
<td>38</td>
<td>50</td>
</tr>
<tr>
<td>Light exercise</td>
<td>38</td>
<td>38</td>
<td>42</td>
</tr>
<tr>
<td>No undue exertion</td>
<td>27</td>
<td>18</td>
<td>8</td>
</tr>
</tbody>
</table>

(PNS% refers to percentage of subjects who had worked night shifts in the past).
Table (XII) summarises the sleep characteristics reported in the preliminary questionnaires for each of the three groups of subjects.

**TABLE XII**

RESULT OF QUESTIONNAIRE SURVEY: SLEEP CHARACTERISTICS.

<table>
<thead>
<tr>
<th>%Discomfort</th>
<th>%Poor Sleep</th>
<th>%Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group (n=29)</td>
<td>Group (n=16)</td>
<td>Group (n=24)</td>
</tr>
<tr>
<td>USUAL SLEEP LATENCY.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 15 mins</td>
<td>41</td>
<td>25</td>
</tr>
<tr>
<td>15 mins-1hr</td>
<td>52</td>
<td>56</td>
</tr>
<tr>
<td>&gt; 1hr</td>
<td>7</td>
<td>19</td>
</tr>
<tr>
<td>&gt; 2hrs</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>AVE HOURS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SLEPT/NIGHT.</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Ave hours felt needed</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>TIME OF WAKING UP.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before 0600hrs</td>
<td>7</td>
<td>25</td>
</tr>
<tr>
<td>0600-0800hrs</td>
<td>79</td>
<td>56</td>
</tr>
<tr>
<td>0800-1000hrs</td>
<td>10</td>
<td>19</td>
</tr>
<tr>
<td>After 1000hrs</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>TIME OF GETTING UP.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before 0600hrs</td>
<td>4</td>
<td>13</td>
</tr>
<tr>
<td>0600-0800hrs</td>
<td>66</td>
<td>50</td>
</tr>
<tr>
<td>0800-1000hrs</td>
<td>27</td>
<td>37</td>
</tr>
<tr>
<td>After 1000hrs</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>FEELINGS ON AWAKING.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alert</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Relaxed/awake</td>
<td>32</td>
<td>13</td>
</tr>
<tr>
<td>Tired/Sleepy</td>
<td>68</td>
<td>87</td>
</tr>
</tbody>
</table>
Table (XIII) shows the percentage of sleep problems reported by each group, and their usual ratings of sleep quality and sleep latency.

<table>
<thead>
<tr>
<th>%Discomfort</th>
<th>%Poor Sleep Group</th>
<th>%Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group (n=29)</td>
<td>Group (n=16)</td>
<td>Group (n=24)</td>
</tr>
<tr>
<td>USUAL SLEEP RATING.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very well</td>
<td>31</td>
<td>6</td>
</tr>
<tr>
<td>Well</td>
<td>44</td>
<td>25</td>
</tr>
<tr>
<td>Poor</td>
<td>24</td>
<td>69</td>
</tr>
<tr>
<td>WORRY AT NIGHT.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>3</td>
<td>38</td>
</tr>
<tr>
<td>Sometimes</td>
<td>34</td>
<td>44</td>
</tr>
<tr>
<td>No</td>
<td>63</td>
<td>18</td>
</tr>
<tr>
<td>WAKE DURING NIGHT.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>62</td>
<td>62</td>
</tr>
<tr>
<td>USE OF SLEEPING PILLS.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Past/present</td>
<td>7</td>
<td>19</td>
</tr>
<tr>
<td>DIFFICULTY AWAKING.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>41</td>
<td>37</td>
</tr>
<tr>
<td>Sometimes</td>
<td>3</td>
<td>19</td>
</tr>
<tr>
<td>No</td>
<td>55</td>
<td>44</td>
</tr>
<tr>
<td>DAYTIME</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TIREDNESS</td>
<td>62</td>
<td>100</td>
</tr>
<tr>
<td>AVE. NO SLEEP ANOMALIES.</td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
</table>
Table (XIV) gives the number of subjects within each group who reported symptoms on awaking in the morning, and indicates which symptoms were most common in each group.

### TABLE XIV. RESULTS OF QUESTIONNAIRE SURVEY.
NUMBER OF SUBJECTS WITH SYMPTOMS ON AWAKING.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Poor Sleep (n=16)</th>
<th>Control (n=24)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aching</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Pain</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Stiffness</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Tenderness</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Cramps</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Headache</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>&gt;1 type of discomfort</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>&gt;2 types of discomfort</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>&gt;3 types of discomfort</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Bad Temper</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Lethargy</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Discomfort and Bad Temper</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Discomfort and Lethargy</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Discomfort and Headache</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Headache and Bad Temper</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Lethargy and Bad Temper</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Symptom free</td>
<td>2</td>
<td>12</td>
</tr>
</tbody>
</table>
Table (XV) shows the number of subjects who worry about their health, who report stress and those who use medications, and the type of disorders for which these were prescribed.

**TABLE XV.**

<table>
<thead>
<tr>
<th></th>
<th>Discomfort Group (n=29)</th>
<th>Poor Sleep Group (n=16)</th>
<th>Control Group (n=24)</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVE. NO HEALTH PROBLEMS</td>
<td>2</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>% WHO WORRY ABOUT HEALTH</td>
<td>44</td>
<td>56</td>
<td>8</td>
</tr>
<tr>
<td>% WITH STRESS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At Home</td>
<td>17</td>
<td>31</td>
<td>21</td>
</tr>
<tr>
<td>At Work</td>
<td>7</td>
<td>25</td>
<td>0</td>
</tr>
<tr>
<td>NO ON MEDICATIONS</td>
<td>14</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>REASON FOR MEDICATION</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>4</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Gastro-int.</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Circulation</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Psychiatric</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Respiratory</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Nerve Disorders*</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Genito-Urinary</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Endocrine</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Allergies</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Cardiac</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Skin</td>
<td>2</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Others</td>
<td>4</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Two or more of above</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

*'Nerve disorders' refers to diseases affecting the nervous system.*
Table (XVI) shows the results of the 'Survey on Muscular Discomfort' questionnaire analysis, and gives the number of subjects who reported discomfort in particular areas of the body.

**TABLE XVI.**

**RESULTS OF 'SURVEY ON MUSCULAR DISCOMFORT' QUESTIONNAIRE:**

**AREAS OF BODY AFFECTED BY DISCOMFORT.**

<table>
<thead>
<tr>
<th>Area</th>
<th>No of Subjects (n=29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Generalised</td>
<td>10</td>
</tr>
<tr>
<td>Lower trunk &amp; Parts of Legs</td>
<td>5</td>
</tr>
<tr>
<td>Shoulders only</td>
<td>2</td>
</tr>
<tr>
<td>Lumbar region</td>
<td>2</td>
</tr>
<tr>
<td>Parts of arms and legs</td>
<td>2</td>
</tr>
<tr>
<td>Head and/ or Neck</td>
<td>1</td>
</tr>
<tr>
<td>Shoulders and Neck</td>
<td>1</td>
</tr>
<tr>
<td>Areas of Spine</td>
<td>1</td>
</tr>
<tr>
<td>Hips and/ or Buttocks</td>
<td>1</td>
</tr>
<tr>
<td>Thighs (Back and Front)</td>
<td>1</td>
</tr>
<tr>
<td>Lower Leg (Back and Front)</td>
<td>1</td>
</tr>
<tr>
<td>Upper trunk and Arms</td>
<td>1</td>
</tr>
<tr>
<td>None described (ommitted)</td>
<td>1</td>
</tr>
</tbody>
</table>
Table (XVII) shows the suggested causes of discomfort given by the subjects completing the 'Survey on Muscular Discomfort' questionnaire, the number of subjects reporting them, and the number who thought that another family member was affected with the same problem.

### TABLE XVII. RESULTS OF 'SURVEY ON MUSCULAR DISCOMFORT' QUESTIONNAIRE: SUGGESTED CAUSES OF DISCOMFORT.

<table>
<thead>
<tr>
<th>Cause</th>
<th>No of Subjects (n=29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unknown</td>
<td>12</td>
</tr>
<tr>
<td>Injury</td>
<td>0</td>
</tr>
<tr>
<td>Deformity</td>
<td>1</td>
</tr>
<tr>
<td>Rheumatoid Arthritis</td>
<td>1</td>
</tr>
<tr>
<td>Arthritis</td>
<td>3</td>
</tr>
<tr>
<td>Ankylosing Spondylitis</td>
<td>3</td>
</tr>
<tr>
<td>Hereditary</td>
<td>0</td>
</tr>
<tr>
<td>Other</td>
<td>9</td>
</tr>
<tr>
<td>Other family member affected</td>
<td>9</td>
</tr>
</tbody>
</table>
Table (XVIII) summarises the various words and phrases used by subjects who completed the 'Survey on Muscular Discomfort' to describe their discomfort. Table (XIX) shows when subjects first noticed their discomfort.

### Table XVIII  
**RESULTS OF 'SURVEY ON MUSCULAR DISCOMFORT'**  
**QUESTIONNAIRE: TYPE OF DISCOMFORT DESCRIBED.**

<table>
<thead>
<tr>
<th>Description</th>
<th>No of Subjects (n=29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aching</td>
<td>7</td>
</tr>
<tr>
<td>Stiffness</td>
<td>8</td>
</tr>
<tr>
<td>Tenderness</td>
<td>2</td>
</tr>
<tr>
<td>Heaviness</td>
<td>0</td>
</tr>
<tr>
<td>Soreness</td>
<td>0</td>
</tr>
<tr>
<td>Need to stretch</td>
<td>0</td>
</tr>
<tr>
<td>Difficult to get going</td>
<td>0</td>
</tr>
<tr>
<td>Two or more of above</td>
<td>12</td>
</tr>
</tbody>
</table>

### Table XIX  
**RESULTS OF 'SURVEY ON MUSCULAR DISCOMFORT'**  
**QUESTIONNAIRE: TIME FIRST NOTICED DISCOMFORT.**

<table>
<thead>
<tr>
<th>Time</th>
<th>No. of Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 3 months ago</td>
<td>1</td>
</tr>
<tr>
<td>More than 3 months ago</td>
<td>0</td>
</tr>
<tr>
<td>More than 1 year ago</td>
<td>6</td>
</tr>
<tr>
<td>More than 3 years ago</td>
<td>22</td>
</tr>
</tbody>
</table>
Table (XX) summarises the descriptions of 'what kind of things increase your discomfort', and 'what kind of things relieve your discomfort', and gives the number of subjects giving these descriptions.

Table (XXI) shows how subjects who completed this questionnaire describe the pattern of their pain, and whether their discomfort was worse in the morning.

### TABLE XX

**RESULTS OF 'SURVEY ON MUSCULAR DISCOMFORT' QUESTIONNAIRE: CHARACTERISTICS OF DISCOMFORT.**

<table>
<thead>
<tr>
<th>Increases Discomfort</th>
<th>No of Subjects (n=29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weather</td>
<td>0</td>
</tr>
<tr>
<td>Exercise*</td>
<td>15</td>
</tr>
<tr>
<td>Tiredness</td>
<td>4</td>
</tr>
<tr>
<td>Cold</td>
<td>0</td>
</tr>
<tr>
<td>Others</td>
<td>7</td>
</tr>
<tr>
<td>All of above</td>
<td>0</td>
</tr>
<tr>
<td>Two or more of above</td>
<td>3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Relieves Discomfort</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Massage</td>
<td>3</td>
</tr>
<tr>
<td>Exercise*</td>
<td>7</td>
</tr>
<tr>
<td>Pills</td>
<td>2</td>
</tr>
<tr>
<td>Sleep</td>
<td>0</td>
</tr>
<tr>
<td>Rest</td>
<td>6</td>
</tr>
<tr>
<td>Holiday</td>
<td>0</td>
</tr>
<tr>
<td>Time</td>
<td>2</td>
</tr>
<tr>
<td>Own methods</td>
<td>4</td>
</tr>
<tr>
<td>Nothing</td>
<td>2</td>
</tr>
<tr>
<td>Heat</td>
<td>3</td>
</tr>
</tbody>
</table>

* Note: Some subjects found that exercise increased their discomfort, but was also useful in relieving their discomfort.
TABLE XXI RESULTS OF 'SURVEY ON MUSCULAR DISCOMFORT' QUESTIONNAIRE: CHARACTERISTICS OF DISCOMFORT: PART 2.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>No of Subjects (n=29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PATTERN OF PAIN</td>
<td></td>
</tr>
<tr>
<td>Continuous</td>
<td>7</td>
</tr>
<tr>
<td>Periodic</td>
<td>20</td>
</tr>
<tr>
<td>Brief</td>
<td>2</td>
</tr>
<tr>
<td>WORSE IN MORNING</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>17</td>
</tr>
<tr>
<td>Sometimes</td>
<td>5</td>
</tr>
<tr>
<td>No</td>
<td>7</td>
</tr>
</tbody>
</table>

6.5. SUMMARY OF RESULTS.

Of the returned questionnaires, twenty nine muscular discomfort and sixteen sleep questionnaires were accepted for analysis. Twenty four control subjects also returned suitable sleep questionnaires.

The total numbers of males and females were thirty four and thirty five. The control group contained eleven males and thirteen females, whereas the discomfort group had slightly more females than males, and the sleep group had more males than females, (table XI).

Sleep survey subjects and discomfort survey subjects were similar in mean age and age range, but the control subjects were slightly younger. This may have accounted for some of the differences observed in sleep characteristics.

Whereas the majority of the discomfort group were married, more than half of the sleep and control groups were single.

Most subjects were engaged in some sort of non-manual occupation, and undertook regular or light exercise. However a larger percentage of the discomfort group did not take any exercise at all.
More than half of the sleep survey subjects claimed to take up to or more than one hour to fall asleep, and felt tired and sleepy on awaking. In comparison, discomfort and control subjects generally reported taking less than fifteen minutes, or no more than one hour to fall asleep. Many subjects with discomfort felt 'tired and sleepy' on awaking, although compared to the subjects with poor sleep, a larger percentage felt 'relaxed and awake'.

Control subjects varied in their reported feelings on awaking, most felt 'relaxed and awake', but over a third also felt 'tired and sleepy'. There were no major group differences in times of waking, getting up or mean number of hours slept, although more of the subjects with poor sleep got up before 0600hrs.

Most of the sleep survey subjects reported poor sleep, although a quarter slept well, and a small percentage slept very well.

All control subjects slept well or very well, and most of the discomfort survey subjects reported sleeping well, although a quarter slept poorly.

A higher percentage of the control group admitted to sometimes worrying at night, compared to either the sleep or discomfort subjects, although half claimed that they did not. More than eighty percent of the sleep survey subjects admitted to worrying at night, but the majority of the discomfort subjects did not. Equal percentages (62%) of the sleep and discomfort groups awoke at least once during the night, but a smaller percentage (29%) of the control subjects had nighttime awakenings.

All sleep survey subjects and sixty two percent of the discomfort survey subjects reported daytime tiredness, whereas only four percent of the controls reported this. Nineteen percent of the sleep subjects had used sleeping pills in the past or recently, and seven percent of the discomfort subjects had at some time.

Both sleep and discomfort groups had a greater mean number of sleep anomalies than the control subjects. Difficulty waking in the morning was most common in the sleep survey subjects, with 41% of the discomfort subjects having experienced this difficulty.
Comparison of the sleep survey questionnaires returned by sleep survey subjects and control subjects showed that discomfort symptoms were most frequently reported by the sleep subjects although they were not uncommon in the control subjects.

The most common symptoms reported by the sleep survey group were 'discomfort and lethargy'. All those with discomfort but without lethargy had a combination of more than one type of symptom for example aching and tenderness, or aching, stiffness and headache. Two subjects reported lethargy as the only symptom on waking. Only two of the sleep subjects were totally symptom free.

In the control group individual subjects reported a variety of discomfort symptoms, and two reported lethargy only, but half the group claimed to be symptom free.

The majority (34.5%) of the discomfort survey subjects described generalised discomfort, with discomfort in the lower trunk and legs being the second most common area. Discomfort in the shoulders, lumbar region and parts of the arms and legs were also common. Individual subjects described other areas shown in table (XVI).

The most frequently used descriptions for the type of discomfort in this group were stiffness (28%), aching (24%) and tenderness (7%). Two or more terms were used by forty one percent of the group, and included 'difficult to get going', and a 'need to stretch'.

Over seventy five percent of the group had first noticed the discomfort more than three years previously. Twenty percent had noticed it more than a year ago, and a few had started experiencing the problem less than three months before.

Most subjects described the discomfort as 'periodic' (68.9%) or continuous (24.1%), and over half found it was usually worse in the morning.

Single factors which made the discomfort worse were exercise (52% of subjects), tiredness (14%), and a variety of other factors (24%). For ten percent of subjects, more than two things could increase their discomfort.
Although more than half of the subjects found exercise made their discomfort worse, some of these subjects also found exercise beneficial, a total of 24% of subjects found exercise relieved their discomfort. Other methods described were rest (21%) and heat and massage (20%). Time, pills and own methods were used by twenty eight percent of subjects, but seven percent found no relief at all.

The majority of this subject group did not know the cause of their discomfort, but rheumatic diseases were most commonly suggested.

Almost half of the discomfort survey subjects were on medications prescribed for problems which included musculoskeletal, respiratory, circulatory and skin disorders. Just less than a third of the sleep survey subjects were receiving treatment for problems classified as musculoskeletal, gastro-intestinal, psychiatric, and respiratory disorders.

Three of the control subjects took medications for skin problems, allergies, or respiratory disorders, but the total number on medications comprised only a quarter of the whole group.
6.6. DISCUSSION AND CONCLUSIONS.

These questionnaires were distributed in several different ways; by four General Practitioners (GP) from a local health centre; a Consultant Rheumatologist, and by the experimenter to unknown volunteers from the local community and to students and staff of the University.

Some problems which could arise as a result of these different methods of distribution might include:

6.6.1. PROBLEMS WITH QUESTIONNAIRE DISTRIBUTION.

Problems with Health Centre Distribution.

(1) General Practitioners often differ in their opinions of a patient's diagnosis, therefore a patient considered to be suitable for the study by one doctor may not have been by another. For example, a back pain patient may be considered a malingerer by one doctor, but a genuine victim of discomfort by another.

(2) Depending on how much importance the GP placed on the study, his selectivity in approaching subjects for their cooperation may have been biased by how well he got on with the patient, rather than the interest of the study.

(3) The questionnaires were distributed over a period of eighteen months. Subjects who paid frequent visits to their GPs during this time would therefore have been more likely to have received a questionnaire. This raises the possibility of whether the sample is biased towards:
(a) Patients who have a higher incidence of other problems requiring medical treatment.
(b) Patients who seek medical advice more often than necessary, that is they are more concerned than most over minor ailments.
(c) Patients who frequently report with minor physical problems as a method of seeking attention.
(4) Bearing in mind the possibilities outlined in (3), some subjects completing and returning questionnaires may have given information which was exaggerated or false, especially if they thought their doctor might have access to the questionnaire.

Problems with Specialist Distribution.

Two types of subjects would be more likely to seek specialist treatment:
(a) Those with severe or chronic rheumatic diseases, and deformity.
(b) Those who persistently present with symptoms which pose a diagnostic problem to their GPs.

Subjects obtained from this source might therefore have an underlying disorder which confuses the clinical picture and exacerbates the severity of symptoms. Such factors would not always be apparent from the questionnaire data, and medical diagnoses were unavailable.

In addition, the type of patient described in (3c) might also apply here.

Subjects from the Local Community, and University Personnel.

These subjects replied to newspaper and poster advertisements, which meant that the subjects' own doctors were unaware of their participation in the study. The suitability of these subjects was therefore left to the discretion of the experimenter, as medical opinion was unavailable.

However, as stated elsewhere (Beetham, 1979; Koldofsky et al, 1975; Payne et al, 1982: Yunus et al, 1981), subjects with the symptoms under study often present as a diagnostic puzzle, so that recognition of the presence of a 'disorder' depends on the background and attitude of the individual's doctor.

For the purposes of this study, the subjective experience of the symptoms described earlier was considered to be the important criteria, rather than the presence of a specific diagnostic 'label'.


Some of these subjects had sought medical advice for their discomfort or sleep problems in the past, but had been 'treated' without success, and had therefore had to 'put up with' their problem since.

Some discomfort subjects had been previously diagnosed as 'fibrositics', whilst others had considered their aching and stiffness as 'normal', and had been surprised when their symptoms were described in the advertisements.

Although it is possible that these respondents may have included people who liked to participate in studies, the following points should be considered.
(a) Advertisements invited correspondence only, and did not describe any experimental procedures.
(b) No payment or benefits were offered for participation in the study.
(c) Preliminary questionnaires were 'vetted' and subjects interviewed prior to mentioning any need for sleep recordings. Unsuitable subjects were not pursued, but politely thanked for their help. Only suitable questionnaires were included in the analysis.

6.6.2. GENERAL PROBLEMS WITH PRELIMINARY QUESTIONNAIRE ANALYSIS.

(1) Assumptions of Questionnaire Analysis

Unless there were obvious inconsistencies in the information supplied on the questionnaires, the experimenter had to assume that the answers given reflected the subjects own experiences and were genuine in content.

(2) Summarising Information for Computer Analysis.

The computer programs written to quantify the answers and information given on the preliminary questionnaires were designed in consideration of the variety of responses observed. However, the following problems were encountered with some of the questions asked:
(a) Answers were omitted or incomplete: In this case the answers sometimes had to be considered as negative unless other information suggested otherwise.

(b) Where there was a choice of answers, two were given; or if a descriptive answer was required, there was some ambiguity. The most likely answer had to be deduced from other information supplied.

(c) With the 'Survey on Muscular Discomfort' questionnaire, subjects were requested to shade in the areas affected on a diagram of the body. As a variety of different combinations of areas were given, this was sometimes difficult to quantify specifically, and generalisations had to be used, (Table XVI). Other areas were included in the program analysis, although none of the subjects shaded these areas. (Appendix III).

In general the questionnaires used for analysis were those supplying clear comprehensible information.
6.6.3. DISCUSSION.

Questionnaires were distributed to subjects who described symptoms of musculoskeletal discomfort or sleep problems. Subjects were volunteers from the local community and University population, who were informed of the study via their local health centre, or by advertisements placed around the University and in the local newspapers.

Twenty nine subjects returned preliminary 'Survey on Sleep' or 'Survey on Muscular Discomfort' questionnaires, which obtained details of individual sleep habits, somatic, and muscular discomfort symptoms. Twenty four control subjects also completed the 'Survey on Sleep' questionnaires.

Reported sleep characteristics indicated that the subjects describing poor sleep experienced problems with sleep onset and sleep maintenance in addition to daytime discomfort and lethargy.

Subjects who completed the 'Survey on Muscular Discomfort' questionnaire generally described periodic or continuous aching and stiffness in areas of the upper and lower trunk, arms and legs. Most of this group claimed that they slept well, although many experienced daytime tiredness and lethargy.

Although the control subjects initially described sleeping well and were symptom free, some of the questionnaires returned indicated that symptoms of lethargy or discomfort were occasionally experienced by these subjects.

To recap on the objectives of the questionnaire survey; Once individuals had been assessed as suitable for the study, that is their symptoms were relevant to those under investigation and they were free of confounding characteristics eg. alcohol or drug habituation and severe organic diseases, their questionnaire details were used for the following purposes:-
a) To obtain an overall picture of each individual's health and sleep, in order to determine the presence of any common characteristics in the group as a whole.

b) To determine the prevalence of sleep problems in subjects with musculoskeletal discomfort, and the prevalence of musculoskeletal discomfort and somatic malaise in subjects with sleep problems.

Some of the subjects studied were approached by their general practitioners, who had a good knowledge of the patient's medical history and personality, and were able to assess the suitability of the individual for the study. Despite this, precise medical diagnoses of these patients' symptoms were unavailable due to medical confidentiality. Consequently, it is important to note that the information obtained from these and other questionnaires is the subject's own interpretation of his or her symptoms and subjective experiences.

'Fibrositis' is described by Beetham, (1979) as 'a poorly defined and controversial term used to describe widespread and variable musculoskeletal aching and stiffness, in the absence of an organic disease'.

The subjects who completed the 'Survey on Muscular Discomfort' questionnaire reported similar musculoskeletal symptoms to those reported by fibrositics, although it was impossible to confirm the absence of any underlying disorders. However, it should be remembered that the purpose of the questionnaire survey was to investigate the incidence of discomfort symptoms in people with sleep complaints and vice versa, and not to seek out 'Fibrositis' sufferers.

Additionally, although many of the subjects in this questionnaire survey reported previous diagnoses of 'Fibrositis' and many of the symptoms described were common to those of the 'Fibrositis Syndrome', it would be wrong to give this label to the subjects who complained of musculoskeletal discomfort, as the presence of definite criteria required for this diagnosis was not investigated. For example, the existence of exaggerated tenderness at specific and normally tender anatomical locations is central to the acceptance and recognition of this syndrome, and skeletal X-rays and blood tests which show normal results in
fibrositics, (Moldofsky and Smythe, 1977), were not available for this study.

In the present study, sixteen females and thirteen males returned acceptable (ie. legible and adequately completed) 'Survey on Muscular Discomfort' questionnaires. It is interesting to note the female majority in this group, although the difference in numbers of the two sexes was small, considering that Wolfe (1986), reported a 5:1 female to male ratio in Fibrositis sufferers. Conversely, there was a notable male majority in the subjects who completed the 'Survey on Sleep' questionnaire, (ten males and six females).

There was very little difference in mean age of the discomfort subjects and the poor sleep subjects, although the latter group contained the older subjects, (max=57y). Unlike fibrositics, who are more frequently aged between forty and sixty years, (Wolfe, 1986), in this study, age ranged between nineteen and the fifties in each group.

Although our subjects with muscular discomfort were without a specific diagnosis, their reported musculoskeletal discomfort characteristics bore a striking similarity to those reported by sufferers of 'Fibrositis'.

Generalised discomfort and discomfort in the lower legs and trunk were most frequently described amongst the subjects with muscular symptoms, although discomfort in the shoulders, lumbar region and parts of the arms and legs was also common. Wolfe (1986) also reported that many fibrositic patients describe their symptoms as 'I hurt all over' although low back pain and neck pain are among the commonest symptoms. Other more specific areas of the body were also described by our subjects but much less frequently, (one subject each).

In most cases, muscular discomfort symptoms were first noticed more than three years ago, were periodic in nature, and worse in the morning, although just less than half of the subjects were not affected on awakening.
Most subjects did not know the cause of their symptoms, although various suggestions were given, such as driving long distances or a soft bed. Eight of the twenty nine subjects (28%) reported that they had an underlying disorder, of which arthritis and ankylosing spondylitis were most commonly suggested. The majority of the muscular discomfort group were engaged in non-manual occupations, but a large number participated in some kind of physical exercise.

Most subjects used more than one term to describe their discomfort, but aching, stiffness and tenderness were most commonly used. Exercise was the most common aggravator, but tiredness and other factors also potentiated the problem. Interestingly, none of the subjects found that weather changes or coldness made their symptoms worse, but these factors in addition to fatigue, are commonly described as aggravating discomfort in Fibrositis patients, (Wolfe, 1986).

Fibrositis patients often improve with moderate activity, (Beetham, 1979), and similarly in this study, the most frequently used method of relieving discomfort was also exercise, which was sometimes, but not always used by the same subjects who reported that exercise aggravated their discomfort. Rest was also favoured as a method of relief. Only two subjects took pills for their symptoms, and two subjects could not find relief with any method.

Only a small percentage of the discomfort group (24%) reported sleeping poorly, although the majority described feeling tired and sleepy on awakening, and during the day. An equal percentage of the discomfort group and poor sleep group awoke during the night, which was twice the percentage of the control group who awoke.

Sleep characteristics of the subjects who completed the 'Survey on Sleep' questionnaire, (the 'poor sleep group') indicated that there was a greater percentage of prolonged sleep latencies, use of sleep medications (past or present), difficulties awaking and tiredness on awakening compared to the other two groups. The majority of subjects in this group reported poor sleep, but almost a third described sleeping well or very well, despite their daytime tiredness. A higher
percentage of this group had also worked on night shifts in the past, and a third of the group smoked a mean of seventeen cigarettes a day.

Seven of the sixteen poor sleepers (44%) reported that they awoke with more than one type of discomfort, but symptoms of bad temper and lethargy, or discomfort and lethargy were equally common. Although a larger percentage of the control group (50%) claimed to be symptom free on awakening, symptoms found in the poor sleepers were also reported by half of the control subjects who were good sleepers, suggesting that these symptoms are not specific to the discomfort and poor sleep groups, although they might be more common. Two control subjects reported lethargy on awakening, but other than this only one subject reported each of the ten types of symptoms reported by the control group.

Both the discomfort group and the poor sleep group had a greater mean number of self-reported health problems and sleep anomalies than the control group, suggesting either that these subjects had a higher incidence of health and sleep problems, or were more likely to exaggerate the frequency and severity of their symptoms. Although the questions asked if sleep anomalies or health problems were experienced 'regularly', this may have been overlooked and interpreted as 'at all' in some cases. A larger percentage of the poor sleep and discomfort groups admitted to worrying about their health, which could suggest they would be more attentive to minor ailments and possibly more likely to 'over-report' their symptoms.

However, as more discomfort subjects actually took medications for musculoskeletal, dermatological, respiratory and circulatory problems than the other two groups, this supports the higher incidence of reported health problems in this group, but not in the poor sleepers. One poor sleeper took medication for musculoskeletal disorders, but two others were taking medication for psychiatric and gastrointestinal disorders respectively. Three control subjects took medications for respiratory, allergic and skin disorders respectively.
Although a small percentage of each group reported some stress at home, this was greatest for the poor sleepers, who also had the highest percentage of reports of stress at work. Over half of the poor sleep group reported some stress, which was twice the percentage of subjects who reported stress from the other two groups. This suggests that the symptoms reported by the poor sleepers might be stress related, or that these subjects exhibit a personality type which is more likely to report stressful experiences.

In addition to reports of stress, most of the poor sleepers admitted to worrying at night either occasionally or frequently, whereas the majority of the discomfort subjects and half of the control subjects said they did not.

6.6.4. CONCLUSIONS.

Subjects with muscular discomfort and those with complaints of poor sleep differ from control subjects in both the severity and regularity of their symptoms. Although a minority of the control subjects reported similar symptoms of bad temper and lethargy, in volunteering to participate in the study they described themselves as good sleepers, who had never experienced muscular discomfort of unknown origin, and were not affected by daytime tiredness and fatigue: The reports of morning lethargy, pain or stiffness and bad temper in half of the group are therefore surprising, but indicate that these symptoms are not uncommon in a population of healthy good sleepers.

The discomfort subjects and poor sleepers also differed from the control subjects by describing more self-reported health problems and sleep anomalies, their concern about their health, and their reports of stress at home and at work.

As it is impossible to judge the impact of specific life events on individual subjects, and the severity and regularity of reported health problems and sleep anomalies, it would be wrong to suggest that these subjects exaggerated their symptoms and their assessment of subjective stress.
It is possible that the two symptom groups (subjects with discomfort and poor sleepers) genuinely show a greater incidence of these problems when compared to control subjects, and these other health and sleep problems could be contributory to their experiences of poor sleep and muscular discomfort. (It is also important to remember the possible biases in subject selection outlined in section 6.6.1.).

Alternatively, the greater number of reported health, stress and sleep problems found in the symptom groups may arise in accordance with a psychological profile characterised by an oversensitivity to and an heightened perception of health and interpersonal or social problems.
CHAPTER 7.

A STUDY OF THE RELATIONSHIP BETWEEN THE NREM SLEEP ALPHA ANOMALY AND MUSCULOSKELETAL DISCOMFORT IN A LOCAL POPULATION.

MAIN SLEEP EEG STUDY.
7. MAIN SLEEP E.E.G STUDY.

7.1. CRITERIA FOR INCLUSION IN THE MAIN STUDY.

Subjects who were invited to continue with the study, and undergo home sleep recordings, satisfied the following criteria.
(a) They were under sixty years of age.
(b) They were generally in 'good health' and fully mobile.
(c) Their preliminary questionnaires and interviews described musculoskeletal discomfort, which:
   (i) Was considered to be of unknown origin, that is, present in the absence of a chronic underlying disorder, and not related to exercise.
   (ii) Was not clinically diagnosed as being related to any other medical problem.
   (iii) Had been present either periodically or continuously for three months or more.
(d) They showed no dependence on alcohol, sleeping pills, psychoactive or other drugs.

7.2. PROCEDURE.

Completed questionnaires were studied and subjects who fulfilled the criteria outlined above were contacted by post to ask if they were willing to participate further in the study. They were then contacted to arrange a personal interview in their homes to discuss the questionnaire details.

The interview took the form of a relaxed discussion and discrete appraisal of the individual's suitability for the study. Circumstances under which subjects did not continue in the study included, lack of sufficient forthcoming information, frequent use of CNS stimulant drugs or dependence on sleeping pills, subject too old or incapacitated to cope with recording, subject unwilling or unable to participate further.
If the subject agreed to undergo a home sleep recording, the Oxford Ambulatory recorder was demonstrated, with an explanation of the procedure involved. The main sleep study questionnaire was then given to the subject.

(ii) Volunteers replying to advertisements were sent the relevant questionnaires by post. On return they were examined and suitable volunteers contacted to request a personal interview, which took place in the volunteers' homes or in a private room. The procedure was then the same as above:

Twenty subjects agreed to undergo home sleep recordings, with fifteen subjects completing the study. Subjects elected to leave the study before or during its course due to the following reasons.

Some subjects were unwilling to participate further due to obligations and commitments to their partners, families, spouses, occupations or social lives, and felt that they could not cope with the inconvenience of a home sleep recording.

One subject reported exacerbation of conjunctivitis as a result of the electrode fixation procedure, in addition to a complaint of a poor night's sleep, and therefore elected to leave the study.

Other subjects also encountered difficulties during the first recording night, which were generally described as problems falling asleep and getting comfortable, concern over getting tangled up in the recorder leads, and damaging the equipment. Some felt these conditions were intolerable and refused to participate any further.

Under some circumstances subjects were not asked to participate further as it was considered that the following circumstances were apparent.

(a) The subjects' living conditions were largely contributory to the sleep disturbances, for example as a result of noise from other occupants.

(b) On meeting the subject, his or her circumstances were not conducive to any inconvenience imposed by the recording equipment, for example one elderly gentleman who volunteered was caring for his wife who was crippled with arthritis.
(c) Habitual consumption of alcohol was high and might therefore have affected sleep.
(d) The subject was using psychoactive drugs or sleeping pills.
(e) The subject was unwilling to cooperate with the conditions requested for recording nights.
(f) The subject lived more than fifteen miles away, which made evening and early morning transport impracticable for the experimenter.

Brief profiles of all subjects who participated in the main sleep EEG study can be found in appendix (V), but a summary of group data and subject details is given below.

7.3. SUMMARY OF GROUP DATA AND SUBJECT PROFILES.

Seven males and eight females complained of symptoms of daytime lethargy and musculoskeletal discomfort affecting areas of upper and lower legs, shoulders, neck and lower back. Seven subjects reported discomfort mainly in the lower trunk and legs, and seven described generalised discomfort, but only one subject described discomfort confined to the upper trunk.

Eleven of these subjects described previous or current stressful experiences, such as divorce or separation, illness, and difficulties at work, which had occurred before or since the onset of their muscular problems. All subjects described a tendency towards periodic anxiety and depression.

Subjects were subsequently divided into groups of good sleepers (eleven subjects) and poor sleepers (four subjects). Good sleepers reported sleeping well (7) or very well (4), whereas poor sleepers reported problems with sleep onset, sleep maintenance and daytime fatigue.

One subject (B) reported disturbing dreams, whereas subjects (E) and (I) reported occasional nightmares. Subject (C) described rumination of daytime events and anxieties during his sleep, being "unable to get things out of his mind".
Some subjects also described other sleep anomalies, namely sleepwalking (subject C), sleeptalking (subject E), and an overwhelming need to move the legs in bed (subject D). Subjects (C), (E), (F) and (N) described frequent lethargy and bad temper or irritability on awakening. Two subjects reported headaches on awakening, (E) and (N), and two others described occasional migraines.

The most current health problems described were peptic ulcer (subject J), Raynauds disease (subject M) and hormonal imbalance (subject P).

Seven female and eight male symptom-free good sleepers were selected as the control subjects.

Although none of this group described any stressful experiences, five control subjects had been divorced or separated from their spouses within the last five years and three control subjects were studying for final examinations at the time of study. Control subjects (5) and (6) described occasional lethargy and control subjects (12) and (13) suffered occasional headaches on awakening.

Although health problems were rare in this group, two control subjects occasionally suffered with migraine (control subjects (6) and (13)), one control subject suffered with hay fever and asthma, (control subject (7)) and control subject (8) reported occasional bouts of anxiety and depression.

Reported sleep anomalies included a need to move the legs in bed (control subject (6)), bruxism (control subject (9)), snoring (control subject (15)), and sleeptalking (control subject (14)).

Exercise and fitness levels of subjects with discomfort suggest that these two factors may affect the way in which an individual perceives his symptoms.

Eleven subjects with discomfort participated in some type of physical activity. Of these, two subjects who reported high levels of physical activity found that exercise exacerbated their symptoms, but one also found exercise helpful in relieving his discomfort.
Of four other subjects who were physically active, one reported an increase of discomfort with exercise, but relief with strengthening exercises, whereas two primarily described an increase in discomfort with inactivity and two found relief from gentle exercises.

Twelve control subjects reported some physical activity, and as in the discomfort group, two of these subjects were highly physically active.

7.4. **MAIN SLEEP STUDY QUESTIONNAIRE**

This questionnaire gave multiple choice answers in an attempt to derive concise answers to the questions posed. In addition to sleep habits, questions focussed on identifying reasons for, and the effects of poor sleep, qualifying subjective mood, anxiety and stress, and obtaining an overall picture of the individuals attitude towards work home and friends. Information was requested regarding past, current and regular health disorders, (appendix II).

In addition to the questions formulated by the experimenter, other multiple choice questions used in this questionnaire were taken from sleep questionnaires composed by Dr M. Herbert of the Psychophysiology section of the Medical Research Council's Applied Psychology Unit at Cambridge.

Unfortunately, only eighteen subjects returned these questionnaires, of which there were three from the four poor sleepers with discomfort, nine from the eleven good sleepers with discomfort and six from the fifteen controls. Reasons for the poor return of questionnaires included:

(a) Loss of questionnaire by subject.

(b) Subjects left university, or moved house before returning the questionnaires.

(c) Loss of interest in the study after recordings were completed.

(d) Pressures of other commitments prevented subjects from completing the questionnaires despite requests from the experimenter.
The results obtained from the 18 returned questionnaires are given in appendix (VI). Results supplementary to those given in the subject profiles are summarised below.

7.4.1. SUMMARY OF RESULTS.

All subject groups reported that their sleep quality varied to some extent. Most subjects in the control group and good sleep with discomfort group said this was only a slight variation, but some of the latter also felt their sleep varied 'moderately' or 'very much'.

Most of the good sleepers with discomfort felt fairly tired, or very tired on awakening, whereas two of the three poor sleepers with discomfort felt very tired, although one felt fairly refreshed.

The majority of the good sleepers with discomfort felt tired and drowsy on awakening in the morning. Three subjects claimed to be 'contented' or 'calm', but most of this group described themselves as 'uneasy', 'tense' or 'distressed' after awakening.

Four of the nine good sleepers with discomfort described their level of wakefulness after getting up as 'drowsy/ tired'. Only two subjects felt 'active' or 'aroused', the remainder describing themselves as 'idle/ sluggish' or 'sleepy/ passive'.

Results for the three poor sleepers with discomfort suggested a similar trend in feelings on awakening and getting up.

In contrast to the two discomfort groups, all of the control subjects felt fairly or very refreshed, clear headed and alert in the morning. Most were either 'contented' or 'calm', and once they had got up, their general level of wakefulness was described as 'lively/ activated', 'active/ energetic', or 'vigorous/ alert'.

Prolonged sleep latency and feeling tired on awakening were most frequently chosen by the good sleepers with discomfort to describe a poor night's sleep. Second to this was 'parts of me ached when I awoke'. Also common were 'I was aware of thinking all night' and 'I awoke a great deal'.

Two subjects in this group chose 'my dreams made me anxious' but none of the subjects in the other groups chose this.

Four subjects in the good sleep with discomfort group felt that feeling very tired on awakening was the most important factor regarding a poor night's sleep, two subjects felt that taking a long time to fall asleep was important. Other reasons given were 'I moved a lot', 'I awoke a great deal', and 'I was aware of thinking all night'. Interestingly, none of this group considered 'parts of me ached when I awoke' as the most important factor describing a poor night's sleep.

The most important statements used to describe a poor night's sleep by the three poor sleepers with discomfort, were 'I had a headache on awakening', 'I awoke a great deal', and 'I felt very tired when I awoke'. Other statements used included 'I was aware of thinking all night', and 'parts of me ached when I woke up'. Dizziness on awakening and prolonged sleep latency were also important.

The statements 'I awoke a great deal', 'I was aware of thinking all night' and 'I took a long time to fall asleep' were most commonly chosen by the six control subjects to describe a poor night's sleep. Headache, dizziness and feeling tired on awakening were selected by two subjects each. Other control subjects chose 'I had many dreams', 'I moved a lot' and 'parts of me ached when I awoke'.

Half of the control subjects felt that 'I was aware of thinking all night' was the most important statement describing a poor night's sleep, but 'I woke a great deal', and 'I moved a lot', were also important.

Both discomfort groups found that spontaneous awakening, and nervous tension or worries caused them to awake during the night. One good sleeper with discomfort awoke because of pain in the legs. Need to pass urine, and noise caused the control subjects to awake, although one control subject awoke because of nervous tension.

Most subjects in all groups felt that a poor night's sleep affected both their efficiency, and how they felt. Most of the good sleepers with discomfort were affected the following day although the remaining four subjects were affected either the day after, or for two days following the
poor night. Two of the three poor sleepers with discomfort were affected on both days, but most of the control group were affected the day after the poor night's sleep.

More than half of the good sleepers with discomfort and all of the poor sleepers with discomfort felt the effects of a poor night's sleep throughout the day, whereas most of the control subjects and a third of the good sleepers with discomfort were affected mainly in the morning.

Difficulty staying awake in the day was reported by only one control subject, who experienced this problem once a month or less. In the two discomfort groups the problem was more frequent. Three of the good sleepers with discomfort experienced difficulty staying awake most days per week, and three several times a month. The remaining three subjects either never had any difficulty, or had difficulty once a month or less.

One poor sleeper with discomfort had never experienced difficulty staying awake during the day, whereas one had difficulty several times a month, and one had difficulty once a month or less.

The majority of both the good sleepers with discomfort and the poor sleepers with discomfort considered themselves to be evening types, whereas only one of the control subjects considered himself to be 'more evening type than morning type'. Three control subjects thought they were more likely to be morning types.

It is important to remember that this data describes only a portion of the three subject groups in the main sleep EEG study.
7.5. **STANDARD SLEEP RECORDINGS.**

All subjects (11 good sleepers with discomfort, 4 poor sleepers with discomfort and 15 control subjects) underwent sleep recordings using the standard sleep EEG montage.

Electrodes were attached to the subjects scalp in accordance with the advised Medilog montage. EEG was recorded from C4-A2 placements, EOG from bilateral eye electrodes and EMG from sub-mental electrodes. Two indifferent or 'ground' vertex electrodes were also attached. EEG, EMG and EOG were then recorded on the four channels of the pre-calibrated tape.

7.5.1. **METHOD.**

(1) A full written explanation of the recording procedure and necessary preparations were sent to the subject one week prior to the pre-arranged date on each occasion. Each subject was asked for one recording at a time, then further requests were made within a short period. This was necessary as many subjects were apprehensive about committing themselves to more than one recording, and following some initial recordings it became apparent that some subjects were unsuitable for the study.

Subjects were requested that prior to the recording they should:
(a) Get a good night's sleep the night preceding the recording night.
(b) Abstain from alcohol from the previous day.
(c) Avoid taking naps.
(d) Maintain a normal routine, including diet and exercise.
(e) Avoid taking medication other than that prescribed by their doctor, and to inform the experimenter of any they took.
(f) Inform the experimenter of any known allergies.
On the night of the recording, they were requested:
(a) To ensure they had washed or bathed before having electrodes fixed.
(b) To have shaved (if male) to help adhesion of EMG electrodes.
(c) To wear loose fitting clothing, or nightwear.
(d) Not to allow anyone to play with the recorders or touch the electrodes in case they became dislodged from the skin.
(e) Not to sit too close to loudspeakers or television sets, due to possible magnetic effects on the equipment.
(f) Not to allow any part of the equipment to get wet.
(g) Not to try to remove the electrodes without the assistance of the experimenter.
(h) To complete the pre and post-sleep questionnaires supplied.
(i) Not to comb or brush the hair whilst the electrodes were in place.

7.5.2. PREPARATION OF EQUIPMENT FOR SLEEP RECORDING.

(i) Each C-120 cassette was demagnetised before use, since partial magnetisation is possible during manufacture or transit.
(ii) Each tape was checked for free-running, using the playback device.
(iii) Batteries were checked using an Oxford Medilog XM-2 monitor unit.
(iv) A demagnetised tape was inserted into the recorder and calibrated for fifteen to thirty minutes to allow adjustment for gains on the playback unit.

(a) For a standard sleep EEG recording, calibration signals of 100µV were used for EEG and EOG channels, and 25µV for the EMG channel.
7.5.3. ELECTRODE ATTACHMENT.

A special abrasive paste (OMNI), was used to gently clean the skin before EEG scalp electrodes were attached with collodion glue (dried with a hair drier) and skin electrodes attached using double-sided adhesive discs. Saline gel was injected into the electrode cavity, and the scalp gently scratched with a new blunted orange stick. A small spot of glue was then applied to the electrode hole to prevent evaporisation of the glue during the night. Facial electrodes were secured with flesh-coloured micropore tape, positioned discretely and comfortably to allow facial and jaw movements. Inter-electrode impedance was reduced to less than 5kΩ, checked using an impedance meter.

The electrode leads were then attached to the respective pre-amplifiers. These record from a bipolar pair of electrodes, and are secured with collodion glue so that all leads run to the back of the head, where they are held together with tape. Hair was arranged to give the best appearance, and small strands were glued across the pre-amplifiers to conceal and secure them.

The collected leads were passed down the inside of the subjects clothes, and the recorder belted around the waist. A pre-calibrated tape and batteries were inserted into the recorder.

7.5.4. PRE AND POST-SLEEP QUESTIONNAIRES.

Pre and post-sleep questionnaires were completed prior to sleep and half an hour after getting up. These questionnaires comprised:-
Pre-sleep.

(a) Stanford Sleepiness scale as follows;
   (1) Active, alert and wide awake.
   (2) Functioning at high level, not at peak, let down.
   (3) Relaxed, awake, not at full alertness, responsive.
   (4) A little foggy, not at peak, let down.
   (5) Fogginess, starting to lose interest in staying awake all day, slowed down.
   (6) Sleepiness, preferred to be resting, fighting sleep, woosy.
   (7) Almost unable to stay awake, struggling to remain awake.

(b) Level of general activity during previous day, (from greatly below normal to greatly above normal).

(c) Subjective feelings of well-being over the day.
   (Active and cheerful, Alert and contented, Tired and relaxed, Lethargic and uneasy).

Post-sleep.

(a) Stanford Sleepiness scale.

(b) At what time did you fall asleep last night?
   - Wake up this morning?
   - Get up this morning?

(c) How difficult was it getting up this morning?

(d) Which of the following do you consider to best describe the quality (not quantity) of your sleep?
   (Much better than normal: Better than normal: Normal: Worse than normal: Much worse than normal).
   (If you did not sleep very well, please state reason, eg noise, too hot, worried).

(e) How would you describe your physical condition this morning?
   (Better than normal: No discomfort: Slight discomfort: Aching and stiff: Worse than normal).
Payment was not given for recordings, but subjects were informed of the results of their sleep analysis in order to maintain their interest in the study.

7.5.5. **ANALYSIS OF RESULTS.**

(i) Sleep recordings were analysed in 20 second samples using the Oxford Sleep Stager. The sleep stager results were previously tested to obtain a measure of repeatability, (see appendix X).

Taped calibration signals of 100μV (EEG and EOG channels) and 25μV (EMG channel) were used to calibrate the analyser for standard EEG montage recordings, whereas all channels were calibrated with 100μV for the frontal-occipital montage recordings.

(ii) Analysis of percentage alpha and delta activity per epoch, plus number of epochs containing spindles in sleep stages 2, 3, 4 and REM was performed using the analyser output of percentage alpha and delta per 20 second epoch of sleep, and the percentage of mini (2 second) epochs containing spindles, a mean value was obtained for each NREM sleep stage (stages 2, 3, 4) of mean percentage alpha per epoch, mean percentage delta per epoch and mean percentage of epochs containing spindles. Mean alpha was also calculated for REM sleep epochs.

This was done by selecting five periods of uninterrupted sleep stages 2, 3, and 4, (or REM sleep for alpha only) from the epoch by epoch sleep stage analysis. Epoch numbers for each sample were recorded, and these epochs were then analysed for alpha, delta and spindles. Mean values over all three stages were also computed.

This sampling method was compared with a similar method using fifteen samples for each stage instead of five, (see appendix IV). The two methods gave an agreement of 84% for mean NREM alpha, 96% for mean NREM delta and 77% for percentage of epochs containing spindles.
7.5.6. **STATISTICAL ANALYSIS.**

All data collected was written to a Minitab file on Multics and statistically analysed.

Group means and standard deviations were obtained for each sleep variable analysed. Histograms and graphs were prepared using the Tellagraf graphics package.

The Alpha-Delta and Spindle-Delta Indices of sleep stages 2, 3, and 4 were formulated for each individual using the following equations.

\[
\text{ALPHA-DELTa INDEX} = \frac{\text{Mean } \% \text{ Alpha Per Epoch}}{\text{Mean } \% \text{ Delta Per Epoch}} \times 100
\]

\[
\text{SPINDLE-DELTA INDEX} = \frac{\text{Mean } \% \text{ Epoch with Spindles}}{\text{Mean } \% \text{ Delta Per Epoch}} \times 100
\]

These indices reflect the ratio of alpha to delta and of spindles to delta in each epoch, but as they represent percentages, they have certain limitations which should be considered. As the percentage of one EEG activity increases, (for example percentage delta activity per epoch increases in the transition from stage 2 to stage 3 and stage 4 sleep) the relative percentage of other activities will decrease, or may become 'masked' by the dominant activity.

Group differences in sleep stages and other sleep variables including mean alpha, delta and spindles for each sleep stage were tested for significance using Mann-Whitney tests. This test is used with the assumptions that:

(a) The two samples were drawn from two populations with the same distribution characteristics.

(b) The two samples were drawn independently of one another.

(c) The scores in each sample were drawn at random.

(d) The scores are at least rankable. (Meddis, 1975).

Pearson product moment correlation coefficients were obtained for correlations of sleep variables within and between sleep stages, and were then tested for statistical significance.
7.6. **RESULTS OF SLEEP EEG ANALYSIS.**

The following tables give the sleep staging results and analysis of alpha, delta and spindles per epoch of sleep stages 2, 3, 4 and REM. The means and standard deviations for each group of subjects are shown.

Table (XXII) shows the mean percentages of sleep stages 2, 3, 4 and SWS, for each group of subjects. Obvious differences between two groups were tested for significance using a Mann-Whitney test, with a significance level of \( p < 0.05 \).

<table>
<thead>
<tr>
<th>GROUP</th>
<th>SLEEP STAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2</td>
</tr>
<tr>
<td>CONTROL (n=15)</td>
<td>48.9</td>
</tr>
<tr>
<td>s.d.</td>
<td>(7.1)</td>
</tr>
<tr>
<td>GOOD Sleepers (n=11)</td>
<td>48.2</td>
</tr>
<tr>
<td>s.d.</td>
<td>(7.4)</td>
</tr>
<tr>
<td>POOR Sleepers (n=4)</td>
<td>51.4</td>
</tr>
<tr>
<td>s.d.</td>
<td>(4.8)</td>
</tr>
</tbody>
</table>

No significant differences were observed for mean percentage sleep stage 2, 3, 4 or SWS between any pair of groups.
Table (XXIII) shows the mean percentage of REM sleep and the mean REM sleep latency for each group. There were no significant differences.

**TABLE XXIII**

**MEAN PERCENTAGE REM SLEEP, AND REM SLEEP LATENCY FOR ALL GROUPS.**

<table>
<thead>
<tr>
<th>Group</th>
<th>% REM Sleep</th>
<th>REM Latency</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONTROL Group (n=15)</td>
<td>22.4 (3.9)</td>
<td>105.9 (75.8)</td>
</tr>
<tr>
<td>GOOD Sleepers (n=11)</td>
<td>20.7 (5.1)</td>
<td>73.3 (32.5)</td>
</tr>
<tr>
<td>+ Discomfort</td>
<td></td>
<td></td>
</tr>
<tr>
<td>POOR Sleepers (n=4)</td>
<td>18.0 (7.0)</td>
<td>133.9 (102.4)</td>
</tr>
<tr>
<td>+ Discomfort</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table (XXIV) shows the means and standard deviations of percentage alpha per epoch of sleep stages 2, 3, 4, and REM for each group. The mean NREM percentages for alpha (mean over stages 2, 3, and 4), are also shown. As one poor sleeper with discomfort (subject G) did not show any stage 4 sleep, the stage 4 mean for this group is based on three subjects only.

**TABLE XXIV MEAN PERCENTAGE ALPHA PER EPOCH OF SLEEP STAGES 2, 3, 4 AND REM AND MEAN NREM VALUES FOR ALL GROUPS.**

<table>
<thead>
<tr>
<th></th>
<th>Mean Alpha%</th>
<th>s.d</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control Group (n=15)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 2</td>
<td>2.2 (a)</td>
<td>(1.2)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>2.1 (b)</td>
<td>(1.4)</td>
</tr>
<tr>
<td>Stage 4</td>
<td>1.6</td>
<td>(1.1)</td>
</tr>
<tr>
<td>Mean NREM</td>
<td>2.0 (c)</td>
<td>(1.1)</td>
</tr>
<tr>
<td>REM</td>
<td>1.8</td>
<td>(2.2)</td>
</tr>
<tr>
<td><strong>Good Sleep + Discomfort (n=11)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 2</td>
<td>3.3</td>
<td>(3.0)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>5.5</td>
<td>(4.8)</td>
</tr>
<tr>
<td>Stage 4</td>
<td>5.0</td>
<td>(5.9)</td>
</tr>
<tr>
<td>Mean NREM</td>
<td>3.8</td>
<td>(2.8)</td>
</tr>
<tr>
<td>REM</td>
<td>2.0</td>
<td>(2.3)</td>
</tr>
<tr>
<td><strong>Poor Sleep + Discomfort (n=4)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 2</td>
<td>5.4 (a)</td>
<td>(3.4)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>6.4 (b)</td>
<td>(4.5)</td>
</tr>
<tr>
<td>Stage 4</td>
<td>1.2*</td>
<td>(0.4)</td>
</tr>
<tr>
<td>Mean NREM</td>
<td>5.1 (c)</td>
<td>(3.0)</td>
</tr>
<tr>
<td>REM</td>
<td>2.9</td>
<td>(2.4)</td>
</tr>
</tbody>
</table>

* This mean is based on three subjects only.
(a) and (a) are significantly different (p<0.02)
(b) and (b) are significantly different (p=0.02)
(c) and (c) are significantly different (p<0.05)
Group means and standard deviations of percentage delta per epoch of sleep stages 2, 3, and 4, and mean NREM delta are shown in Table (XXV). Good sleepers with discomfort showed the smallest mean percentage of delta in stage 4 sleep, but figures for the poor sleepers with discomfort are based on three subjects only. One subject did not show any stage 4 sleep on recording nights, whilst the youngest poor sleeper with discomfort showed a particularly high percentage of delta in stage 4 sleep, (74%; subject E).

<table>
<thead>
<tr>
<th>TABLE XXV. MEAN PERCENTAGE DELTA PER EPOCH OF SLEEP STAGES 2, 3, AND 4 FOR ALL GROUPS.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean Delta%</strong></td>
</tr>
<tr>
<td>Control Group (n=15)</td>
</tr>
<tr>
<td>Stage 2</td>
</tr>
<tr>
<td>Stage 3</td>
</tr>
<tr>
<td>Stage 4</td>
</tr>
<tr>
<td>Mean NREM</td>
</tr>
<tr>
<td>Good Sleep + Discomfort (n=11)</td>
</tr>
<tr>
<td>Stage 2</td>
</tr>
<tr>
<td>Stage 3</td>
</tr>
<tr>
<td>Stage 4</td>
</tr>
<tr>
<td>Mean NREM</td>
</tr>
<tr>
<td>Poor Sleep + Discomfort (n=4)</td>
</tr>
<tr>
<td>Stage 2</td>
</tr>
<tr>
<td>Stage 3</td>
</tr>
<tr>
<td>Stage 4</td>
</tr>
<tr>
<td>Mean NREM</td>
</tr>
</tbody>
</table>

* This mean is based on three subjects only.
(a) and (a) are significantly different (p<0.05).
Table (XXVI) gives the mean and standard deviations of percentage of epochs containing spindles in sleep stages 2, 3, and 4, and the mean NREM value for all groups. The stage 4 mean for poor sleepers with discomfort is again based on only three subjects.

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean % of epochs</th>
<th>s.d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (n=15)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 2</td>
<td>8.9</td>
<td>(6.2)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>1.6</td>
<td>(1.9)</td>
</tr>
<tr>
<td>Stage 4</td>
<td>0.7 (a)</td>
<td>(0.8)</td>
</tr>
<tr>
<td>Mean NREM</td>
<td>6.6</td>
<td>(4.6)</td>
</tr>
<tr>
<td>Good Sleep + Discomfort (n=11)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 2</td>
<td>7.2</td>
<td>(5.4)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>1.1</td>
<td>(1.0)</td>
</tr>
<tr>
<td>Stage 4</td>
<td>0.3</td>
<td>(0.6)</td>
</tr>
<tr>
<td>Mean NREM</td>
<td>5.5</td>
<td>(4.6)</td>
</tr>
<tr>
<td>Poor Sleep + Discomfort (n=4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 2</td>
<td>7.6</td>
<td>(6.0)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>1.2</td>
<td>(1.5)</td>
</tr>
<tr>
<td>Stage 4</td>
<td>0.1 flooding (a)</td>
<td>(0.2)</td>
</tr>
<tr>
<td>Mean NREM</td>
<td>5.9</td>
<td>(3.9)</td>
</tr>
</tbody>
</table>

* This mean is based on three subjects only.
(a) and (a) are significantly different (p=0.02)
Table (XXVII) shows the mean alpha-delta indices for sleep stages 2, 3, and 4, for each group. Alpha-delta indices represent the ratio of percentage alpha per epoch to the percentage delta per epoch, (see section 7.5.6.).

**TABLE XXVII  MEAN VALUES OF ALPHA-DELTA INDEX FOR SLEEP STAGES 2, 3 AND 4 FOR ALL GROUPS.**

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean Alpha-Delta Index</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CONTROL GROUP (n=15)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STAGE 2</td>
<td>58.5 (a)</td>
<td>36.8</td>
</tr>
<tr>
<td>STAGE 3</td>
<td>7.2 (b)</td>
<td>5.2</td>
</tr>
<tr>
<td>STAGE 4</td>
<td>2.9</td>
<td>2.0</td>
</tr>
<tr>
<td><strong>GOOD SLEEP + DISCOMFORT (n=11)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STAGE 2</td>
<td>74.6</td>
<td>72.0</td>
</tr>
<tr>
<td>STAGE 3</td>
<td>17.5</td>
<td>15.5</td>
</tr>
<tr>
<td>STAGE 4</td>
<td>9.5 (c)</td>
<td>10.8</td>
</tr>
<tr>
<td><strong>POOR SLEEP + DISCOMFORT (n=4)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STAGE 2</td>
<td>129.0 (a)</td>
<td>112.9</td>
</tr>
<tr>
<td>STAGE 3</td>
<td>18.2 (b)</td>
<td>17.8</td>
</tr>
<tr>
<td>STAGE 4</td>
<td>1.6 (c)</td>
<td>1.2</td>
</tr>
</tbody>
</table>

(a) and (a) are significantly different (p=0.05)
(b) and (b) are significantly different (p=0.02)
(c) and (c) are significantly different (p<0.05)
Table (XXVIII) shows the means and standard deviations of the spindle-delta indices for sleep stages 2, 3, and 4. The spindle-delta index represents the ratio of epochs containing spindles to the percentage of delta activity in an epoch, (see section 7.5.6.).

TABLE XXVIII MEAN SPINDLE-DELTA INDEX FOR SLEEP STAGES 2, 3, AND 4, FOR ALL GROUPS.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Mean Spindle-Delta Index</th>
<th>S.D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>220.0</td>
<td>14.0</td>
</tr>
<tr>
<td>Stage 3</td>
<td>5.4</td>
<td>5.9</td>
</tr>
<tr>
<td>Stage 4</td>
<td>1.3 (a)</td>
<td>1.6</td>
</tr>
</tbody>
</table>

**CONTROL** (n=15)

<table>
<thead>
<tr>
<th>Stage</th>
<th>Mean Spindle-Delta Index</th>
<th>S.D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>165.5</td>
<td>135.1</td>
</tr>
<tr>
<td>Stage 3</td>
<td>3.6</td>
<td>3.4</td>
</tr>
<tr>
<td>Stage 4</td>
<td>0.7</td>
<td>1.2</td>
</tr>
</tbody>
</table>

**GOOD SLEEP + DISCOMFORT** (n=11)

<table>
<thead>
<tr>
<th>Stage</th>
<th>Mean Spindle-Delta Index</th>
<th>S.D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>212.9</td>
<td>167.7</td>
</tr>
<tr>
<td>Stage 3</td>
<td>4.0</td>
<td>4.9</td>
</tr>
<tr>
<td>Stage 4</td>
<td>0.18 (a)</td>
<td>0.31</td>
</tr>
</tbody>
</table>

(a) and (a>) are significantly different (p<0.05)
Table (XXIX) gives the means and standard deviations of percentage stage 1 sleep, sleep onset latency and sleep efficiency for all groups. Percentage sleep efficiency is calculated as the minutes of actual time asleep (sleep time minus interim wakefulness) divided by the sleep period, (from sleep onset to final awakening) multiplied by 100.

\[
\text{\% SLEEP EFFICIENCY} = \frac{\text{ACTUAL SLEEP TIME (mins)}}{\text{SLEEP PERIOD (mins)}} \times 100
\]

<table>
<thead>
<tr>
<th></th>
<th>% Stage 1 Sleep</th>
<th>Sleep Onset Latency (min)</th>
<th>% Sleep Efficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control Group (n=15)</td>
<td>13.4</td>
<td>13.9</td>
<td>91.5 (a)</td>
</tr>
<tr>
<td></td>
<td>(7.5)</td>
<td>(17.8)</td>
<td>(5.6)</td>
</tr>
<tr>
<td>Good Sleep</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>tDiscomfort (n=11)</td>
<td>13.5</td>
<td>29.0</td>
<td>88.0</td>
</tr>
<tr>
<td></td>
<td>(3.9)</td>
<td>(37.0)</td>
<td>(9.8)</td>
</tr>
<tr>
<td>Poor Sleep</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>tDiscomfort (n=4)</td>
<td>16.2</td>
<td>41.5</td>
<td>73.6 (a)</td>
</tr>
<tr>
<td></td>
<td>(5.2)</td>
<td>(58.0)</td>
<td>(21.5)</td>
</tr>
</tbody>
</table>

(a) and (a) are significantly different (p<0.02)

No significant differences were found for stage 1 sleep, or sleep onset latency.

The sleep stager output gives the number of individual episodes of each sleep stage and of movement and wakefulness for the total sleep period. In the following tables the number of episodes of each sleep stage has been divided by the actual sleep time to give the number of episodes per minute of actual sleep time; and then multiplied by 60 to give the number of episodes per hour of sleep.
Group means of episodes of wakefulness and episodes of movement per hour of sleep time are shown in table (XXX). Episodes of wakefulness are separated into episodes longer or shorter than 120 seconds, (as given on the sleep stager output).

<table>
<thead>
<tr>
<th></th>
<th>WAKEFULNESS</th>
<th>MOVEMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&gt;120sec</td>
<td>&lt;120 sec</td>
</tr>
<tr>
<td>CONTROL</td>
<td>0.28</td>
<td>1.8 (a)</td>
</tr>
<tr>
<td>s.d</td>
<td>(0.3)</td>
<td>(1.2)</td>
</tr>
<tr>
<td>GOOD Sleep</td>
<td></td>
<td></td>
</tr>
<tr>
<td>± Discomfort</td>
<td>0.36 (b)</td>
<td>1.44 (c)</td>
</tr>
<tr>
<td>s.d</td>
<td>(0.36)</td>
<td>(1.2)</td>
</tr>
<tr>
<td>POOR Sleep</td>
<td></td>
<td></td>
</tr>
<tr>
<td>± Discomfort</td>
<td>1.8 (b)</td>
<td>5.4 (a)(c)</td>
</tr>
<tr>
<td>s.d</td>
<td>(3.0)</td>
<td>(0.36)</td>
</tr>
</tbody>
</table>

(a) and (a) are significantly different (p<0.05)
(b) and (b) are significantly different (p<0.05)
(c) and (c) are significantly different (p<0.02)
Table (XXXI) gives the group means and standard deviations of episodes of sleep stage 1, 2, 3, 4, and REM per hour of actual sleep time. There were no significant differences.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Control Group</th>
<th>Good Sleep + Discomfort</th>
<th>Poor Sleep + Discomfort</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>8.4</td>
<td>7.8</td>
<td>12.0</td>
</tr>
<tr>
<td></td>
<td>(3.6)</td>
<td>(1.8)</td>
<td>(4.8)</td>
</tr>
<tr>
<td>2</td>
<td>13.2</td>
<td>12.6</td>
<td>15.0</td>
</tr>
<tr>
<td></td>
<td>(3.0)</td>
<td>(3.6)</td>
<td>(2.4)</td>
</tr>
<tr>
<td>3</td>
<td>10.2</td>
<td>10.8</td>
<td>9.6</td>
</tr>
<tr>
<td></td>
<td>(3.6)</td>
<td>(4.2)</td>
<td>(3.0)</td>
</tr>
<tr>
<td>4</td>
<td>2.4</td>
<td>2.4</td>
<td>1.2</td>
</tr>
<tr>
<td></td>
<td>(1.8)</td>
<td>(1.8)</td>
<td>(1.8)</td>
</tr>
<tr>
<td>REM</td>
<td>3.0</td>
<td>3.6</td>
<td>3.0</td>
</tr>
<tr>
<td></td>
<td>(1.2)</td>
<td>(1.8)</td>
<td>(0.6)</td>
</tr>
</tbody>
</table>

### 7.7. SUMMARY OF RESULTS OF SLEEP ANALYSIS.

There were no significant differences in mean percentages of sleep stages 2, 3, 4, SWS or REM sleep between the three subject groups.

**Control Group and Good Sleepers with Discomfort.**

There were no significant differences between the control group and good sleepers with discomfort in mean percentage alpha per epoch, mean percentage delta per epoch or mean percentage of epochs containing spindles for sleep stages 2, 3, and 4.

Comparison of the alpha-delta indices of NREM sleep stages 2, 3, and 4 between the two groups found that although the mean alpha-delta indices for the good sleepers with discomfort were greater than those of the control group, the large dispersion of values amongst the good
sleepers with discomfort resulted in the differences being statistically non-significant.

Although the control group were found to have larger mean values of NREM sleep spindle-delta indices, there were no statistically significant differences.

There were no significant differences in stage 1 sleep, sleep efficiency, the number of episodes of wakefulness and movement per hour of sleep, or the number of episodes of sleep stages 1, 2, 3, 4 and REM sleep per hour of sleep between the control group and good sleepers with discomfort.

**Control Group and Poor Sleepers with Discomfort.**

Poor sleepers with discomfort showed significantly greater means of alpha per epoch of sleep stages 2, 3 and NREM sleep than control subjects. Alpha-delta indices were also significantly greater for the poor sleepers with discomfort in sleep stages 2 and 3 but not 4. (All stage 4 means for the poor sleepers with discomfort are based on the 3 subjects exhibiting stage 4 sleep).

Poor sleepers with discomfort showed lower mean values of mean delta per epoch for NREM sleep stages 2 and 3, but none of these were significantly different from control values. The group of poor sleepers with discomfort consisted of four subjects with an age range of 21y to 49y. As delta activity decreases with age, the age differences may account for the large standard deviation observed for sleep stage 4.

A significant difference was observed for the mean percentage of spindles per epoch of sleep stage 4, with the poor sleepers with discomfort showing significantly less than the controls. This difference was also reflected in the significant difference observed between the spindle-delta indices of sleep stage 4.

There were no significant differences between percentages of stage 1 sleep, and episodes of sleep stages 1, 2, 3, 4, and REM per hour of actual sleep time. Poor sleepers with discomfort showed significantly more episodes of wakefulness, (<120 sec) per hour of sleep, and a significantly lower sleep efficiency than the controls, (73.6% and 91.5% respectively).
Good Sleepers with Discomfort and Poor Sleepers with Discomfort.

Poor sleepers with discomfort were found to have a greater mean percentage of alpha per epoch in sleep stages 2 and 3, NREM sleep and REM sleep compared to the good sleepers with discomfort, but mean alpha in sleep stage 4 was greater for the good sleepers with discomfort. However, only the difference in sleep stage 4 alpha-delta indices reached significance.

Good sleepers with discomfort showed more delta per epoch of sleep stages 2, 3, and NREM sleep compared to the poor sleepers with discomfort but only the difference in sleep stage 3 delta proved to be significantly different. Poor sleepers with discomfort had a greater mean percentage delta in stage 4 sleep, which was biased towards a particularly large amount of stage 4 delta shown by subject (E).

No significant differences were observed between mean percentage of epochs containing spindles, as these values were similar for the two groups. Stage 2 sleep mean spindle-delta index was greater for the poor sleepers with discomfort, although both groups showed a large distribution of values, and this difference proved to be non-significant.

Mean percentage stage 1 sleep was greatest, and sleep efficiency was less for the poor sleepers with discomfort, but these differences were non-significant. Significant differences were found for the mean number of episodes of wakefulness (<120 sec and >120 sec long) per hour of sleep, which were greater in the poor sleepers with discomfort. The mean number of episodes of movement per hour of sleep was greater for the good sleepers with discomfort, but this difference were non-significant. No significant differences were observed for mean number of episodes of sleep stages 1, 2, 3, 4 or REM between the two groups.
7.8. RESULTS OF CORRELATIONS.

Correlations of percentages of NREM sleep stages for each group are shown in table XXXII.

TABLE XXXII.

<table>
<thead>
<tr>
<th>SLEEP STAGE</th>
<th>STAGE 3</th>
<th>p</th>
<th>STAGE 4</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control Group (n=15)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STAGE 2</td>
<td>-0.61</td>
<td>2%</td>
<td>-0.87</td>
<td>0.2%</td>
</tr>
<tr>
<td>STAGE 3</td>
<td>-0.87</td>
<td>NS</td>
<td>0.48</td>
<td>NS</td>
</tr>
<tr>
<td>Good Sleep +</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Discomfort (n=11)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STAGE 2</td>
<td>-0.45</td>
<td>NS</td>
<td>-0.79</td>
<td>0.2%</td>
</tr>
<tr>
<td>STAGE 3</td>
<td>-0.79</td>
<td>NS</td>
<td>0.84</td>
<td>0.2%</td>
</tr>
<tr>
<td>Poor Sleep +</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Discomfort (n=4)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STAGE 2</td>
<td>-0.73</td>
<td>NS</td>
<td>-0.79</td>
<td>NS</td>
</tr>
<tr>
<td>STAGE 3</td>
<td>-0.79</td>
<td>NS</td>
<td>0.79</td>
<td>NS</td>
</tr>
</tbody>
</table>

Some relationship is to be expected between percentages of sleep stages, as they represent proportions of a night's sleep, and as the percentage of one sleep stage increases, another will decrease.

The next three tables (XXXIII, XXXIV and XXXV) give summaries of the statistically significant correlations of mean percentage alpha per epoch, mean percentage delta per epoch and percentage number of epochs containing spindles for NREM sleep stages and REM sleep within each individual group.
<table>
<thead>
<tr>
<th>VARIABLE 1</th>
<th>VARIABLE 2</th>
<th>r</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 3 Alpha</td>
<td>Stage 3 Spindles</td>
<td>0.51</td>
<td>5%</td>
</tr>
<tr>
<td>Stage 3 Alpha</td>
<td>Stage 4 Alpha</td>
<td>0.66</td>
<td>2%</td>
</tr>
<tr>
<td>REM Alpha</td>
<td>Stage 4 Spindles</td>
<td>0.71</td>
<td>0.2%</td>
</tr>
<tr>
<td>Delta</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 3 Delta</td>
<td>% Stage 2</td>
<td>-0.56</td>
<td>5%</td>
</tr>
<tr>
<td>Stage 3 Delta</td>
<td>% Stage 3</td>
<td>0.73</td>
<td>0.2%</td>
</tr>
<tr>
<td>Stage 4 Delta</td>
<td>% Stage 2</td>
<td>-0.82</td>
<td>0.2%</td>
</tr>
<tr>
<td>Mean NREM Delta</td>
<td>% Stage 3</td>
<td>0.56</td>
<td>5%</td>
</tr>
<tr>
<td>Mean NREM Delta</td>
<td>% Stage 2</td>
<td>-0.85</td>
<td>0.2%</td>
</tr>
<tr>
<td>Mean NREM Delta</td>
<td>% Stage 4</td>
<td>0.94</td>
<td>0.2%</td>
</tr>
<tr>
<td>Spindles</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 2 Spindles</td>
<td>Stage 3 Spindles</td>
<td>0.58</td>
<td>5%</td>
</tr>
<tr>
<td>Stage 2 Spindles</td>
<td>Stage 4 Spindles</td>
<td>0.75</td>
<td>0.2%</td>
</tr>
<tr>
<td>Stage 3 Spindles</td>
<td>% Stage 2</td>
<td>-0.54</td>
<td>5%</td>
</tr>
<tr>
<td>Stage 3 Spindles</td>
<td>% Stage 4</td>
<td>0.58</td>
<td>5%</td>
</tr>
</tbody>
</table>
TABLE XXXIV.
SIGNIFICANT INTER-CORRELATIONS OF PERCENTAGE ALPHA, DELTA AND SPINDLES OF SLEEP STAGES 2, 3, 4 AND REM, FOR GOOD SLEEPERS WITH DISCOMFORT.

<table>
<thead>
<tr>
<th>VARIABLE 1</th>
<th>VARIABLE 2</th>
<th>r</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Alpha</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>REM Alpha</td>
<td>Mean NREM Alpha</td>
<td>0.71</td>
<td>2%</td>
</tr>
<tr>
<td>REM Alpha</td>
<td>Stage 2 Alpha</td>
<td>0.84</td>
<td>0.2%</td>
</tr>
<tr>
<td>Stage 3 Alpha</td>
<td>Stage 4 Alpha</td>
<td>0.89</td>
<td>0.2%</td>
</tr>
<tr>
<td><strong>Delta</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 2 Delta</td>
<td>Stage 3 Delta</td>
<td>0.71</td>
<td>2%</td>
</tr>
<tr>
<td>Stage 2 Delta</td>
<td>Stage 4 Delta</td>
<td>0.74</td>
<td>2%</td>
</tr>
<tr>
<td>Stage 2 Delta</td>
<td>% Stage 4</td>
<td>0.84</td>
<td>0.2%</td>
</tr>
<tr>
<td>Stage 2 Delta</td>
<td>% Stage 3</td>
<td>0.89</td>
<td>0.2%</td>
</tr>
<tr>
<td>Stage 3 Delta</td>
<td>% Stage 3</td>
<td>0.84</td>
<td>2%</td>
</tr>
<tr>
<td>Stage 3 Delta</td>
<td>Stage 4 Delta</td>
<td>0.77</td>
<td>2%</td>
</tr>
<tr>
<td>Stage 4 Delta</td>
<td>% Stage 4</td>
<td>0.66</td>
<td>5%</td>
</tr>
<tr>
<td>Stage 4 Delta</td>
<td>% Stage 3</td>
<td>0.82</td>
<td>0.2%</td>
</tr>
<tr>
<td>Mean NREM Delta</td>
<td>% Stage 3</td>
<td>0.87</td>
<td>0.2%</td>
</tr>
<tr>
<td>Mean NREM Delta</td>
<td>% Stage 4</td>
<td>0.99</td>
<td>0.2%</td>
</tr>
<tr>
<td><strong>Spindles</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 2 Spindles</td>
<td>Stage 3 Spindles</td>
<td>0.75</td>
<td>2%</td>
</tr>
<tr>
<td>Stage 2 Spindles</td>
<td>Stage 4 Spindles</td>
<td>0.75</td>
<td>2%</td>
</tr>
<tr>
<td>Stage 3 Spindles</td>
<td>Stage 4 Spindles</td>
<td>0.71</td>
<td>2%</td>
</tr>
</tbody>
</table>

Figures (6) and (7) show correlations of percentage alpha per epoch of REM sleep with mean NREM sleep alpha (r=0.71) and sleep stage 2 alpha (r=0.84) respectively. Figure (8) shows the correlation of percentage alpha per epoch of sleep stages 3 and 4, (r=0.89). Figure (9) shows the correlation of stage 2 and stage 3 spindles, (r=0.75).

In the figures, GS+D is an abbreviation for good sleepers with discomfort.
Correlation of GS+D REM Alpha and Mean NREM Alpha
Correlation of GS+D Stage 2 Spindles and Stage 3 Spindles

- FIGURE 9 -

Correlation of GS+D Stage 2 Spindles and Stage 3 Spindles
Poor Sleepers with Discomfort.

Due to the small number of subjects in this group, and the absence of data where subjects did not exhibit a particular sleep stage, correlations were largely non-significant. Correlation coefficients of 0.9 were achieved in some cases, but inspection of the relevant scatter plots showed that a significant relationship between the two variables did not exist.

TABLE XXXV. CORRELATIONS OF SLEEP VARIABLES FOR THE POOR SLEEP WITH DISCOMFORT GROUP.

<table>
<thead>
<tr>
<th>VARIABLE 1</th>
<th>VARIABLE 2</th>
<th>r</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean NREM Alpha</td>
<td>Age (yrs)</td>
<td>0.72</td>
<td>NS</td>
</tr>
<tr>
<td>Stage 4 Alpha</td>
<td>% Stage 2</td>
<td>-0.92</td>
<td>10%</td>
</tr>
<tr>
<td>Mean NREM Delta</td>
<td>Age (yrs)</td>
<td>-0.93</td>
<td>10%</td>
</tr>
<tr>
<td>Stage 2 Spindles</td>
<td>Stages (3+4)</td>
<td>0.94</td>
<td>10%</td>
</tr>
<tr>
<td>Stage 2 Spindles</td>
<td>Stage 3 Spindles</td>
<td>0.95</td>
<td>5%</td>
</tr>
</tbody>
</table>

The next three tables (XXXVI, XXXVII and XXVIII), give the results of correlations between the alpha-delta indices of individual NREM sleep stages, and correlations between the spindle-delta indices of these stages for each group of subjects.
TABLE XXXVI. CONTROL GROUP: CORRELATIONS OF ALPHA-DELTA INDICES AND SPINDLE DELTA INDICES FOR NREM SLEEP STAGES.

(n=15).

ALPHA-DELTA INDICES.

<table>
<thead>
<tr>
<th>SLEEP STAGE</th>
<th>STAGE 3</th>
<th>p</th>
<th>STAGE 4</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>0.02</td>
<td>NS</td>
<td>0.02</td>
<td>NS</td>
</tr>
<tr>
<td>Stage 3</td>
<td></td>
<td></td>
<td>0.62</td>
<td>2%</td>
</tr>
</tbody>
</table>

SPINDLE-DELTA INDICES.

<table>
<thead>
<tr>
<th>SLEEP STAGE</th>
<th>STAGE 3</th>
<th>p</th>
<th>STAGE 4</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>0.48</td>
<td>NS</td>
<td>0.54</td>
<td>5%</td>
</tr>
<tr>
<td>Stage 3</td>
<td></td>
<td></td>
<td>0.32</td>
<td>NS</td>
</tr>
</tbody>
</table>

TABLE XXXVII. GOOD SLEEP WITH DISCOMFORT GROUP: CORRELATIONS OF ALPHA-DELTA INDICES AND SPINDLE-DELTA INDICES FOR NREM SLEEP STAGES.

(n=11)

ALPHA-DELTA INDICES.

<table>
<thead>
<tr>
<th>SLEEP STAGE</th>
<th>STAGE 3</th>
<th>p</th>
<th>STAGE 4</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>0.21</td>
<td>NS</td>
<td>0.07</td>
<td>NS</td>
</tr>
<tr>
<td>Stage 3</td>
<td></td>
<td>NS</td>
<td>0.88</td>
<td>0.2%</td>
</tr>
</tbody>
</table>

SPINDLE-DELTA INDICES.

<table>
<thead>
<tr>
<th>SLEEP STAGE</th>
<th>STAGE 3</th>
<th>p</th>
<th>STAGE 4</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>0.67</td>
<td>5%</td>
<td>0.84</td>
<td>0.2%</td>
</tr>
<tr>
<td>Stage 3</td>
<td></td>
<td></td>
<td>0.72</td>
<td>2%</td>
</tr>
</tbody>
</table>

Figure (10) shows the correlation of stage 3 and stage 4 alpha-delta indices for the good sleepers with discomfort.
Correlation of GS+D Stage 3 ADI and Stage 4 ADI
TABLE XXXVIII.

POOR SLEEP WITH DISCOMFORT GROUP:

CORRELATIONS OF ALPHA-DELTA INDICES AND SPINDLE-DELTA INDICES FOR NREM SLEEP STAGES.

(n=4)

ALPHA-DELTA INDICES.

<table>
<thead>
<tr>
<th>SLEEP STAGE</th>
<th>STAGE 3</th>
<th>STAGE 4</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>-0.63</td>
<td>NS</td>
<td>-0.95</td>
</tr>
<tr>
<td>Stage 3</td>
<td>-0.90</td>
<td>NS</td>
<td>0.98</td>
</tr>
</tbody>
</table>

SPINDLE-DELTA INDICES.

<table>
<thead>
<tr>
<th>SLEEP STAGE</th>
<th>STAGE 3</th>
<th>STAGE 4</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>0.91</td>
<td>NS</td>
<td>0.98</td>
</tr>
<tr>
<td>Stage 3</td>
<td>0.98</td>
<td>NS</td>
<td>0.98</td>
</tr>
</tbody>
</table>

(s) Inspection of the scatter plots of these variables found no apparent relationship, despite the high correlation coefficient.

Table XXXIX shows the correlations of mean percentage alpha per epoch of NREM sleep with indices of sleep disturbance, specifically wakefulness after sleep onset, episodes of movement and episodes of stage 1 sleep per hour of sleep, and sleep efficiency of each group.

TABLE XXXIX. CORRELATIONS OF PERCENTAGE ALPHA PER EPOCH OF NREM SLEEP WITH INDICES OF SLEEP DISTURBANCE FOR ALL GROUPS.

<table>
<thead>
<tr>
<th>MEAN PERCENTAGE NREM ALPHA (STAGES 2, 3, + 4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VARIABLE</td>
</tr>
<tr>
<td>WASO&gt;120 sec</td>
</tr>
<tr>
<td>WASO&lt;120 sec</td>
</tr>
<tr>
<td>Movement (eps)</td>
</tr>
<tr>
<td>% Stage 1</td>
</tr>
<tr>
<td>Stage 1 Episodes</td>
</tr>
<tr>
<td>Sleep Efficiency %</td>
</tr>
</tbody>
</table>

(WASO=Wakefulness After Sleep Onset)
(eps=Episodes)
Table (XL) shows the correlations of mean percentage of epochs containing spindles in NREM sleep with the same indices of sleep disturbance as described in table (XXXIX).

**TABLE XL.**

**CORRELATIONS OF EPOCHS WITH SPINDLES IN NREM SLEEP WITH INDICES OF SLEEP DISTURBANCE FOR ALL GROUPS.**

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>CONTROL GROUP</th>
<th>GOOD SLEEP +DISCOMFORT</th>
<th>POOR SLEEP +DISCOMFORT</th>
</tr>
</thead>
<tbody>
<tr>
<td>WASO&gt; 120 sec</td>
<td>0.50</td>
<td>-0.39</td>
<td>-0.33</td>
</tr>
<tr>
<td>WASO&lt; 120 sec</td>
<td>-0.02</td>
<td>-0.30</td>
<td>-0.10</td>
</tr>
<tr>
<td>Movement (eps)</td>
<td>0.01</td>
<td>-0.24</td>
<td>-0.17</td>
</tr>
<tr>
<td>Stage 1 Episodes</td>
<td>0.04</td>
<td>-0.17</td>
<td>-0.59</td>
</tr>
<tr>
<td>Sleep Efficiency %</td>
<td>-0.39</td>
<td>0.55</td>
<td>0.41</td>
</tr>
</tbody>
</table>

(WASO=Wakefulness After Sleep Onset)  
(eps=Episodes)

Table (XLI) shows the correlations of alpha-delta and spindle-delta indices of each NREM sleep stage with sleep efficiency for each group.

**TABLE XLI.**

**CORRELATIONS OF ALPHA-DELTA INDICES AND SPINDLE-DELTA INDICES WITH SLEEP EFFICIENCY FOR ALL GROUPS.**

<table>
<thead>
<tr>
<th>SLEEP EFFICIENCY %</th>
<th>CONTROL GROUP</th>
<th>GOOD SLEEP +DISCOMFORT</th>
<th>POOR SLEEP +DISCOMFORT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>-0.27</td>
<td>0.40</td>
<td>0.24</td>
</tr>
<tr>
<td>Stage 3</td>
<td>-0.14</td>
<td>0.25</td>
<td>-0.90</td>
</tr>
<tr>
<td>Stage 4</td>
<td>-0.41</td>
<td>0.34</td>
<td>-0.94</td>
</tr>
</tbody>
</table>

**ALPHA-DELTA INDICES.**

| Stage 2                   | -0.24         | 0.50                   | 0.41                   |
| Stage 3                   | -0.52         | 0.21                   | 0.64                   |
| Stage 4                   | -0.65         | 0.49                   | 0.56                   |

(∗)Significant at p<0.05
Table XLII shows the correlations of mean alpha per epoch of NREM sleep and mean alpha per epoch of REM sleep for each group.

**TABLE XLII.**

**CORRELATIONS OF MEAN PERCENTAGE ALPHA IN REM SLEEP AND MEAN PERCENTAGE ALPHA IN SLEEP STAGES 2, 3, 4, AND MEAN NREM SLEEP FOR ALL GROUPS.**

<table>
<thead>
<tr>
<th>Mean Percentage Alpha in REM Sleep</th>
<th>Control + Discomfort</th>
<th>Good Sleep + Discomfort</th>
<th>Poor Sleep + Discomfort</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>0.24</td>
<td>0.84 *</td>
<td>-0.13</td>
</tr>
<tr>
<td>Stage 3</td>
<td>0.03</td>
<td>0.46</td>
<td>-0.57</td>
</tr>
<tr>
<td>Stage 4</td>
<td>0.37</td>
<td>0.10</td>
<td>0.18</td>
</tr>
<tr>
<td>Mean (Stages 2,3,4)</td>
<td>0.33</td>
<td>0.71</td>
<td>-0.22</td>
</tr>
</tbody>
</table>

(*Significant at p<0.05

7.9. **SUMMARY OF RESULTS OF CORRELATIONS.**

Significant negative correlations were found between percentages of sleep stage 2 and 4 for both the control group and the good sleepers with discomfort. This relationship is predictable in view of the fact that more time is spent in stage 2 sleep when stage 4 sleep is absent or reduced.

A statistically significant correlation was observed between stage 3 sleep and stage 4 sleep (0.84) for the good sleepers with discomfort. The control group showed a less significant correlation (-0.61) between stage 2 and stage 3 sleep.

Correlations of percentage sleep stages for the poor sleepers with discomfort were statistically non-significant due to the small number of subjects.

For control subjects, mean percentage alpha per epoch of sleep stage 3 was found to be significantly correlated with that of sleep stage 4 (0.66), and with the mean percentage of epochs containing spindles in sleep stage 3, (0.51).
Alpha-delta indices of sleep stage 3 and 4 were also found to be significantly correlated in the control group, (0.62).

Good sleepers with discomfort showed a significant correlation (0.89) between mean percentage alpha of sleep stages 3 and 4. The correlation of the alpha-delta indices of these sleep stages was also statistically significant. In this group percentage alpha in REM sleep was significantly correlated with percentage alpha in NREM sleep (mean percentage alpha over sleep stages 2, 3, and 4), (0.71) and percentage alpha in stage 2 sleep, (0.84). These results suggest that there is some continuity both in the ratio of alpha to delta activity in sleep stages 3 and 4, and in the quantity of alpha activity found in REM sleep and NREM sleep.

In the control group, the mean percentage alpha in REM sleep and the mean number of epochs containing spindles in stage 4 sleep were found to be significantly correlated, (0.71), although this accounts for only 49% of the variation in these variables.

There were no significant correlations of mean percentage alpha with other sleep variables for the poor sleepers with discomfort.

A high negative but non-significant correlation was observed between age of the poor sleepers with discomfort and mean NREM percentage delta per epoch.

For both the control group and the good sleepers with discomfort, positive correlations of mean percentage of epochs containing spindles for NREM sleep stages suggest that there is some continuity in the quantity of spindle activity found through sleep stages 2, 3, and 4, with some subjects showing more NREM sleep spindle activity than others.

For the control group, correlations of the spindle-delta indices of NREM sleep stages 2 and 3 (0.48) and 2 and 4 (0.54) were just significant. For the good sleepers with discomfort however, correlations were more highly statistically significant, particularly those between sleep stages 2 and 4 (0.84) and sleep stages 3 and 4 (0.72).
Correlations of the spindle-delta indices of sleep stages 2 and 3 were also significant for this group.

No significant correlations were found between indices of sleep disturbance and mean percentage of alpha in NREM sleep or mean percentage of epochs containing spindles in NREM sleep.

However, a small (-0.65) but significant correlation was found between sleep efficiency and the spindle-delta index of sleep stage 4 in the control group.
7.10. FRONTAL-OCCIPITAL SLEEP EEG RECORDINGS.

The alpha component of the 'alpha-delta' sleep anomaly has been described by Sewitch et al, (1978), Scheuler et al (1983) and Weber et al, (1983) to be predominantly frontal and central in origin.

As we were interested in the nature and characteristics of the 'alpha anomaly' during NREM sleep, we employed this alternative montage in order to examine and compare frontal and occipital 'alpha-range' activity using Fourier Analysis. For comparison of waking and sleeping alpha frequencies, a sample of each subject's waking EEG was also obtained under various 'test' conditions.

Sleep EEG's were recorded from frontal and occipital electrode placements in addition to the C4-A2 recording.

7.10.1. METHOD.

Subject preparation for the 'frontal-occipital' recording was the same as for standard sleep EEG recordings. The equipment was also prepared in the same manner except that for a 'frontal-occipital' recording all channels of the C-120 tape were calibrated to 100μV.

Transverse, longitudinal and circumferential head measurements were performed to find electrode sites in accordance with the 10-20 system which were marked with water-soluble pen. Inter-electrode distances for frontal and occipital electrode pairs were kept equal to minimize amplitude differences. As two extra EEG's were recorded only one EOG channel was available and EMG recording was not possible. Electrode attachment procedure and subject instructions were the same as those described for the standard recordings.

Pre and post-sleep questionnaires were completed as for the standard sleep recordings.
7.10.2. **FRONTAL-OCCIPITAL RECORDING: WAKING TESTS**

The morning following the 'frontal-occipital' sleep EEG recording, subjects were asked to sit relaxed in a dimly lit (but not dark) room, and perform various waking mental tasks, during which the EEG was recorded. These were designed to evoke alpha and possibly kappa activity on the waking EEG:

1. Sit quietly relaxed, eyes shut, without thinking of anything. (10 mins).
2. Sit quietly relaxed, eyes open, without thinking of anything. (5 mins).
3. Sit quietly relaxed, eyes shut. Subtract 7 from 1000 consecutively. (5 mins).
4. Sit quietly relaxed, eyes open. Subtract 7 from 1000 consecutively. (5 mins).
5. Sit quietly relaxed with eyes shut, without thinking of anything. (5 mins).

7.10.3. **ANALYSIS OF RESULTS.**

Frequency (or spectral) analysis of each subject's frontal and occipital EEG, recorded using the 'frontal-occipital' montage, was carried out using a Hewlett Packard Fourier Analyser. Frequency analysis separates the EEG signal into an amplitude spectrum, producing a graph of amplitude as a function of frequency.

Prior to frequency analysis, the amplitudes of frontal and occipital channels were equalised using the 100μV calibration signals recorded at the beginning of each tape. Sleep recordings were played through the Oxford Medilog Page Mode Display, which allowed identification of each sleep stage.

Three minute epochs of sleep stages 2, 3, 4 and REM, were then analysed to identify the presence of activity within the alpha frequency band. As the playback device played tapes back at 20 times real time, observed frequencies were divided by 20. Random samples of unequivocal sleep stages were taken from the whole night recording. EEG's recorded during the waking tests were also
examined. Each graph was copied on to paper using an X-Y plotter.

Each graph was inspected for evidence of a peak of activity in the alpha range, and the results summarised in a table.

7.11. FOURIER ANALYSIS RESULTS.

Eight of the good sleepers with discomfort, (subjects B, C, D, F, K, L, M, and P), and nine of the controls (subjects 5, 6, 7, 10, 11, 12, 13, 14, and 15), underwent recordings using the alternative frontal-occipital electrode montage. Each subject also performed waking mental tests for analysis of waking alpha frequencies.

None of the poor sleepers with discomfort underwent this type of sleep montage, as it was difficult to obtain more than one recording from these subjects. One subject did agree to be recorded twice, but it was considered more important to get a good standard recording after a poor first night.

The results for each individual are given in appendix (VII) but a summary of the main findings is given below.

Tables (XLIII) and (XLIV) give a summary of the results of fourier analysis of frontal-occipital sleep EEG recordings taken from the control group subjects and subjects from the group of good sleepers with discomfort. The main object of this exercise was to look for evidence of frontal 'alpha' activity during sleep.

7.11.1. ALPHA ACTIVITY.

Frequencies and amplitudes of occipital alpha or frontal alpha-like activity for each subject are shown in the following tables. Amplitudes of occipital and frontal frequencies have been ranked for each subject, to enable comparison of the magnitude of each peak in each sleep stage for each subject. As different calibrations were used for each record, it would be erroneous to compare amplitudes between subjects.
Ranks of (1) to (4) are given for waking alpha and 'alpha-like activity' in sleep stages 2, 3, 4, and REM. (1) indicates the greatest amplitude observed.

**TABLE XLIII.**

**SUMMARY OF ALPHA FREQUENCIES AND RANKED AMPLITUDES OBSERVED FOR EACH SLEEP STAGE IN EACH SUBJECT.**

**CONTROL SUBJECTS (N=9).**

(Amplitude Ranks shown in parentheses: All frequencies in Hz).

<table>
<thead>
<tr>
<th>Sleep Stage</th>
<th>Waking</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>REM</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Subject 5</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital</td>
<td>10 (1)</td>
<td>8.6  (4)</td>
<td>7.4  (3)</td>
<td>-</td>
<td>9 (2)</td>
</tr>
<tr>
<td><strong>Subject 6</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital</td>
<td>11 (1)</td>
<td>8.6  (4)</td>
<td>9.4  (3)</td>
<td>-</td>
<td>8.2 (2)</td>
</tr>
<tr>
<td><strong>Subject 7</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital</td>
<td>8.8</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Subject 10</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital</td>
<td>9.2</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Subject 13</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital</td>
<td>7.6 (1)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>7.2 (2)</td>
</tr>
<tr>
<td><strong>Subject 11</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>-</td>
<td>8.8</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Subject 12</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>9.8 (5)</td>
<td>9 (3)</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital</td>
<td>11.0 (4)</td>
<td>-</td>
<td>8.2 (1)</td>
<td>-</td>
<td>9.4 (2)</td>
</tr>
<tr>
<td><strong>Subject 14</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>-</td>
<td>8.5 (2)</td>
<td>7.4 (2)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital</td>
<td>10.6 (1)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>9.8 (3)</td>
</tr>
<tr>
<td><strong>Subject 15</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>-</td>
<td>8 (3)</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital</td>
<td>8 (4)</td>
<td>7.4 (1)</td>
<td>-</td>
<td>-</td>
<td>8.8 (1)</td>
</tr>
</tbody>
</table>
Of the nine control subjects who underwent the 'frontal-occipital' montage recording, four showed evidence of frontal 'alpha-like' activity during NREM sleep.

In each of these four subjects, frontal 'alpha-like' activity varied between 8Hz and 9Hz, during sleep stage 2. Control subject (14) also showed an equal amplitude of frontal 'alpha-like' activity during sleep stage 3. In two of these subjects (12) and (14) frontal 'alpha-like' activity was slower than the observed occipital waking alpha activity.

Waking occipital alpha activity was seen in eight of the subjects and varied between 7.6Hz and 11Hz. Control subject (12) also showed a smaller amplitude of frontal 'alpha-like' activity during relaxed wakefulness, but this was slower than the occipital alpha activity.

During REM sleep, six subjects showed occipital alpha activity, of frequencies between 7.2Hz and 9.8Hz. No frontal 'alpha-like' activity was observed during REM sleep in the control group.

Table (XLIV) shows the results of Fourier analysis for the eight good sleepers with discomfort.
TABLE XLIV.

SUMMARY OF ALPHA FREQUENCIES AND RANKED AMPLITUDES
OBSERVED FOR EACH SLEEP STAGE IN EACH SUBJECT:
GOOD SLEEPERS WITH DISCOMFORT, (N=8).

(Amplitude Ranks shown in parentheses: All frequencies in Hz).

<table>
<thead>
<tr>
<th>Sleep Stage</th>
<th>Waking</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>REM</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Subject D.</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital</td>
<td>10.6 (1)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>8.2 (2)</td>
</tr>
<tr>
<td><strong>Subject F.</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>-</td>
<td>7 (3)</td>
<td>7 (2)</td>
<td>10.4 (4)</td>
<td>-</td>
</tr>
<tr>
<td>Occipital</td>
<td>9 (1)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>7.8 (5)</td>
</tr>
<tr>
<td><strong>Subject C.</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>8.4 (6)</td>
<td>9 (2)</td>
<td>7 (1)</td>
<td>10 (3)</td>
<td>9.2 (5)</td>
</tr>
<tr>
<td>Occipital</td>
<td>10.2 (4)</td>
<td>-</td>
<td>-</td>
<td>9.4 (8)</td>
<td>9.4 (7)</td>
</tr>
<tr>
<td><strong>Subject K.</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>-</td>
<td>11 (3)</td>
<td>8.6 (4)</td>
<td>9.2 (6)</td>
<td>-</td>
</tr>
<tr>
<td>Occipital</td>
<td>10.4 (2)</td>
<td>8.4 (1)</td>
<td>8.8 (4)</td>
<td>-</td>
<td>10.4 (6)</td>
</tr>
<tr>
<td><strong>Subject L.</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>-</td>
<td>7.6 (3)</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital</td>
<td>10 (1)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>9.8 (2)</td>
</tr>
<tr>
<td><strong>Subject F.</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>9.8 (6)</td>
<td>7.8 (4)</td>
<td>7.4 (5)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital</td>
<td>9.4 (1)</td>
<td>8 (2)</td>
<td>-</td>
<td>9 (7)</td>
<td>8.8 (3)</td>
</tr>
</tbody>
</table>
Fourier analysis of sleep records from two good sleepers with discomfort, (subjects D and K) did not show any frontal alpha-like activity during NREM sleep.

Six of the eight good sleepers with discomfort exhibited frontal 'alpha-like' activity during NREM sleep. In three of the subjects the frontal 'alpha-like' activity was distributed throughout sleep stages 2, 3 and 4, (subjects B, C, and F). One of these subjects also showed frontal 'alpha-like' activity during REM sleep, and during relaxed wakefulness (subject C). Subject (F) also showed occipital alpha activity in sleep stages 2 and 3.

Subject (P) exhibited frontal 'alpha-like' activity during sleep stages 2 and 3 and occipital alpha activity during sleep stages 2 and 4. In subject (L) frontal 'alpha-like' activity was seen during sleep stage 2 only and in subject (K) it was seen during sleep stage 4 only. In both cases the frequency of frontal 'alpha-like' activity was slower than the frequencies of occipital alpha found during wakefulness and REM sleep.

In general the frequency of frontal 'alpha-like' activity varied between sleep stages. In four subjects it was slower than waking occipital alpha, (subjects C, K, L and P), but in subjects (F) and (B) it was faster during sleep stages 2 and 4 respectively. There was no consistent pattern detected in either amplitude or frequency of frontal 'alpha-like' activity between sleep stages for the group as a whole.

Occipital alpha activity during REM sleep was slower than waking occipital alpha in seven of the eight subjects studied. In seven subjects showing occipital alpha during NREM sleep, this activity was slower than waking occipital alpha.

Table (XLV) shows the NREM sleep stages in which alpha activity was observed in individual control subjects showing frontal 'alpha-like' activity, and gives the mean percentage of alpha found in individual NREM sleep stages of the sleep EEG recording of the same night. These percentages were taken from analysis of the central sleep EEG recorded on the night when the frontal-occipital montage was employed. Mean percentage alpha per epoch of REM sleep is not given as REM
sleep is not scored when the frontal-occipital montage is employed, (see method).

TABLE XLV.
RESULTS OF FOURIER ANALYSIS AND MEAN PERCENTAGE ALPHA PER EPOCH OF EACH NREM SLEEP STAGE FOR RESPECTIVE NIGHT: CONTROL GROUP.

<table>
<thead>
<tr>
<th>CONTROL SUBJECTS</th>
<th>11</th>
<th>12</th>
<th>14</th>
<th>15</th>
</tr>
</thead>
<tbody>
<tr>
<td>F-α Stage 2</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>-</td>
</tr>
<tr>
<td>O-α Stage 2</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Y</td>
</tr>
<tr>
<td>% Stage 2 Alpha</td>
<td>0.22</td>
<td>1.1</td>
<td>1.5</td>
<td>0.9</td>
</tr>
<tr>
<td>F-α Stage 3</td>
<td>-</td>
<td>-</td>
<td>Y</td>
<td>-</td>
</tr>
<tr>
<td>O-α Stage 3</td>
<td>-</td>
<td>Y</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>% Stage 3 Alpha</td>
<td>0.13</td>
<td>3.0</td>
<td>1.5</td>
<td>0.3</td>
</tr>
<tr>
<td>F-α Stage 4</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>O-α Stage 4</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>% Stage 4 Alpha</td>
<td>1.0</td>
<td>4.7</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td>F-α REM</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>O-α REM</td>
<td>-</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
</tbody>
</table>

F-α=Frontal alpha-like activity.
O-α=Occipital alpha.
*=No REM sleep scored by analyser on these nights.
Y=Alpha activity present.
(*=No Stage 4 recorded.

In control subjects showing frontal 'alpha-like' activity, this activity was not observed in the sleep stages showing the largest mean percentage of alpha per epoch. In control subjects (12) and (15), the individuals largest mean percentage of alpha was associated with the presence of occipital alpha activity, but in subjects (11) and (14), the largest percentage of alpha per epoch of sleep was found in sleep stage 4, during which neither frontal 'alpha-like' activity nor occipital alpha activity were observed.
Table (XLVI) gives a summary of the sleep stages of control subjects who did not show frontal 'alpha-like' activity. Mean percentages of alpha per epoch of each NREM sleep stage are also given.

### TABLE XLVI.

**OCCIPITAL ALPHA IN SUBJECTS NOT SHOWING FRONTAL ALPHA-LIKE ACTIVITY IN NREM SLEEP: CONTROL SUBJECTS.**

<table>
<thead>
<tr>
<th></th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>10</th>
<th>13</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-α Stage 2</td>
<td>Y</td>
<td>Y</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>% Stage 2 Alpha</td>
<td>4.5</td>
<td>2.7</td>
<td>6.2</td>
<td>0.5</td>
<td>3.5</td>
</tr>
<tr>
<td>0-α Stage 3</td>
<td>Y</td>
<td>Y</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>% Stage 3 Alpha</td>
<td>1.7</td>
<td>4.3</td>
<td>1.9</td>
<td>1.4</td>
<td>0.7</td>
</tr>
<tr>
<td>% Stage 4</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>O-α REM Sleep**</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

O-α=Occipital alpha present.
Y=Yes
*=No Stage 4 recorded during this night.
**=No REM sleep scored by analyser on this night.

In the two subjects (5 and 6) showing occipital alpha activity during NREM sleep, this activity was limited to sleep stages 2 and 3. Mean percentages of alpha per epoch for these stages were higher than those of sleep stage 4 in both subjects.
Tables (XLVII) and (XLVIII) summarise the sleep stages of good sleepers with discomfort who showed frontal 'alpha-like' activity and those who did not show frontal 'alpha-like' activity respectively. Mean percentages of alpha per epoch of each NREM sleep stage are also given.

**TABLE XLVII.**

**RESULTS OF FOURIER ANALYSIS AND MEAN PERCENTAGE ALPHA PER EPOCH OF EACH NREM SLEEP STAGE FOR RESPECTIVE NIGHT: GOOD SLEEPERS WITH DISCOMFORT.**

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>C</th>
<th>F</th>
<th>K</th>
<th>L</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>F-α Stage 2</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>-</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>O-α Stage 2</td>
<td>Y</td>
<td>-</td>
<td>Y</td>
<td>-</td>
<td>-</td>
<td>Y</td>
</tr>
<tr>
<td>% Alpha St 2</td>
<td>1.9</td>
<td>6.3</td>
<td>1.0</td>
<td>0.27</td>
<td>4.3</td>
<td>6.62</td>
</tr>
<tr>
<td>F-α Stage 3</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>-</td>
<td>-</td>
<td>Y</td>
</tr>
<tr>
<td>O-α Stage 3</td>
<td>-</td>
<td>-</td>
<td>Y</td>
<td>Y</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>% Alpha St 3</td>
<td>14.9</td>
<td>10.3</td>
<td>10.7</td>
<td>0.27</td>
<td>3.4</td>
<td>12.9</td>
</tr>
<tr>
<td>F-α Stage 4</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>O-α Stage 4</td>
<td>-</td>
<td>Y</td>
<td>-</td>
<td>Y</td>
<td>-</td>
<td>Y</td>
</tr>
<tr>
<td>% Alpha St 4</td>
<td>18.6</td>
<td>10.5</td>
<td>8.6</td>
<td>0.16</td>
<td>5.0</td>
<td>5.2</td>
</tr>
<tr>
<td>F-α REM</td>
<td>-</td>
<td>Y</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>O-α REM</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
</tbody>
</table>

Y = Frontal activity present.
F-α = Frontal alpha-like activity.
O-α = Occipital alpha activity.

In the three good sleepers with discomfort (subjects B, C and F), who showed frontal 'alpha-like' activity in sleep stages 2, 3, and 4, the highest percentages of alpha per epoch of sleep were observed in sleep stages 3 and 4. However, subject (K) showed both frontal 'alpha-like' activity and occipital alpha activity during sleep stage 4 which had the lowest percentage of alpha per epoch in this individual.

Subject (L) showed only frontal 'alpha-like' activity during sleep stage 2, which was associated with neither the largest or smallest alpha percentage for NREM sleep stages.
Subject (P) showed the largest mean percentage of alpha during sleep stage 3, in which frontal 'alpha-like' activity was observed. However both frontal 'alpha-like' activity and occipital alpha activity were observed during sleep stage 2, which had only half almost the same percentage of alpha as stage 3 sleep. Stage 4 sleep which had as much alpha as stage 2 sleep exhibited only occipital alpha activity.

TABLE XLVIII.

OCCIPITAL ALPHA IN SUBJECTS NOT SHOWING FRONTAL ALPHA-LIKE ACTIVITY IN NREM SLEEP: GOOD SLEEPERS WITH DISCOMFORT.

<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>D</th>
<th>M</th>
</tr>
</thead>
<tbody>
<tr>
<td>O-α Stage 2</td>
<td></td>
<td>Y</td>
</tr>
<tr>
<td>%Stage 2 Alpha</td>
<td>0.74</td>
<td>0.26</td>
</tr>
<tr>
<td>O-α Stage 3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>%Stage 3 Alpha</td>
<td>1.85</td>
<td>0.59</td>
</tr>
<tr>
<td>O-α Stage 4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>%Stage 4 Alpha</td>
<td>1.4</td>
<td>0</td>
</tr>
<tr>
<td>O-α REM Sleep</td>
<td>Y</td>
<td>Y</td>
</tr>
</tbody>
</table>

0-α=Occipital alpha
Y=Occipital alpha found during this sleep stage.

Amongst subjects not showing frontal 'alpha-like' activity, the largest percentages of alpha were observed in sleep stage 3, although subject (M) showed occipital alpha during sleep stage 2.
7.11.2. FRONTAL THETA (4 to <7Hz).

In addition to the observation of frontal 'alpha-like' activity, some subjects in both groups were seen to show frontal activity between 4 and 7Hz which occurred with or without frontal 'alpha'.

Control subject (5) showed frontal theta during sleep stage 2 only, whereas subject (10) showed it in sleep stages 2, 3, and 4, and three control subjects showed theta in stages 2 and 3 (subjects 12 and 14) or 3 and 4 (subject 11). Six good sleepers with discomfort showed frontal theta, of which subjects (B), (C), (F), (K), and (P) showed it in sleep stages 2, 3, and 4, and subject (K) showed it in stage 4 and REM sleep only.

Control subjects (11), (12), and (14), who showed frontal theta also showed frontal 'alpha-like' activity during NREM sleep. The presence of frontal 'alpha-like' activity was not necessarily coincident with the presence of frontal theta in a sleep stage, although these two activities were observed together in some cases. Control subjects (5) and (10) exhibited frontal theta but not frontal 'alpha-like' activity, whereas subject (15) showed frontal 'alpha-like' activity but not frontal theta.

In the case of the good sleepers with discomfort, four (B, C, F, and K) of the six subjects who showed frontal theta also showed frontal 'alpha-like' activity in exactly the same sleep stages, but subject (P) failed to show 'alpha-like' activity in sleep stage 4. Subject (M) showed frontal theta but not 'alpha-like' activity, whereas subject (L) showed frontal 'alpha-like' activity but not theta.
7.12. NEUROMAPPER RECORDING.

7.12.1. METHOD.

Subject (P) slept in the laboratory during an afternoon, allowing sleep frequency analysis using a Neuroscience Neuromapper temporarily loaned to the Sleep Laboratory.

Due to the limited availability of the equipment, it was used to study only one subject, and was of interest as a supplement to fourier analysis.

Frontal and occipital pairs of electrodes were placed as for frontal-occipital sleep recordings, in addition to a full electrode montage, (in accordance with the 10-20 system). Electrode fixing procedures were as for the main study:

This equipment produced a visual representation of the EEG frequencies produced during sleep and their origins on the scalp surface.

The Neuroscience Neuromapper breaks down the sleep EEG into it's frequency components, and displays the results in the form of colour maps. Distributions of various frequencies over the scalp can be shown in a single map, or maps of five different frequency bands can be viewed simultaneously, (multiple frame).

7.12.2. RESULTS.

Figures (11) to (13) show the results of the sleep EEG frequency analysis using the Neuroscience Neuromapper. Subject (P) showed sleep stages 2, 3, and 4, but no REM sleep.

The figures show a cross section of the head, which is shaded in patterns representing the frequency or amplitude of activity present in a particular area. The key to the shading is given on the right hand side of the figure. These figures have been reproduced from the original Neuromapper pictures due to problems encountered in colour copying.
FIGURE 11. CORTICAL SLEEP EEG FREQUENCY MAPPING OF SUBJECT (P). (1.)
FIGURE 12. CORTICAL SLEEP EEG FREQUENCY MAPPING OF SUBJECT (P). (2.)
FIGURE 13. CORTICAL SLEEP EEG FREQUENCY MAPPING OF SUBJECT (P). (3.)
Figures (11a) and (11b) show simultaneous activity in five frequency bands during two different four second epochs. Both delta and alpha activities are at their greatest amplitude frontally and at their lowest amplitudes occipitally.

Figure (12a) shows an epoch containing much higher amplitudes of frontal delta activity (>50μV). Two small foci of frontal alpha (30μV) and theta activity (~33μV) can also be seen frontally.

Figure (12b) shows a focus of 2Hz delta activity frontally, coincident with a focus of central 8Hz alpha activity (frame 9) which spreads transversely and then occipitally and frontally.

In figure (13) a band of delta activity can be seen frontally, whilst an occipital focus of alpha activity gradually spreads over the scalp.

7.12.3. SUMMARY

These results confirm the findings of fourier analysis of subject (P)'s sleep, in that 'alpha-like' activity was detected frontally during NREM sleep. Although the Neuromapper does not record the actual EEG (on to a disc), it can be assumed from the presence of delta activity, that this subject was in NREM sleep stages 2, 3 or 4, when the 'alpha-like' activity was identified.

The topographic mapping of the alpha frequency allowed the identification of the origin of this activity, and it was interesting to find that alpha activity also appeared centrally and occipitally in the presence of frontal delta activity. Fourier analysis of sleep stage 4 also identified low amplitude occipital alpha activity in this subject.
7.13. SUMMARY OF RESULTS OF PRE-SLEEP AND POST-SLEEP QUESTIONNAIRE ANALYSIS.

7.13.1. STANFORD SLEEPINESS SCALE SCORES.

For the first part of the following analysis, pairs of scores for each night recorded were used, rather than taking each subject's mean ratings over consecutive recordings. Due to occasional problems collecting questionnaires, there are four pairs of scores missing, one from the control group, and three from the good sleepers with discomfort.

Figure (14) illustrates the distribution of subjective pre-sleep SSS ratings for each group. Figure (15) shows the distribution of post-sleep SSS ratings.

A wide range (from (1) to (7)) of pre-sleep SSS scores were given by both the control group and the good sleepers with discomfort, but these were most frequently between (1) and (3). Ratings given by the poor sleepers with discomfort were between (2) and (5) inclusively.

More than half of the control group rated themselves as (3) on the SSS in the morning, (relaxed, awake, not at full alertness, responsive). Although there was a wide range of morning scores (from (1) to (6)) given by this group, the majority of these scores were at the 'more alert' end of the scale.

Post-sleep SSS scores given by the good sleepers with discomfort follow a roughly bimodal distribution, scores of (1) and (4) being most frequent. There is a broad range of scores within this group, with more at the 'sleepy' end of the scale (from (4) to (6)) compared to the control group.

Poor sleepers with discomfort gave scores of either (3) or (4) in the morning, of which (4) was most frequent.
Frequency Distribution of Pre-Sleep S.S.S Scores: All Groups

Legend:
- CONTROL
- GS+DISCOMFORT
- FS+DISCOMFORT

Stanford Sleepiness Scale Score: Pre Sleep
FIGURE 15.

Frequency Distribution of Post-Sleep S.S.S. Scores: All Groups

LEGEND:
- CONTROL
- GS+DISCOMFORT
- PS+DISCOMFORT

Stanford Sleepiness Scale Scores: Post-Sleep

Number

0 5 10 15 20

1 2 3 4 5 6 7
Table (XLIX) gives the means and standard deviations of pre-sleep and post-sleep ratings, and the mean evening to morning change in these scores for each group. These figures represent the means of all nights studied in subjects from each group. As some subjects underwent more recordings than others, these results may be biased towards the ratings given by subjects recorded more frequently. However, this analysis was performed to assess the individual overnight changes in scores, which would be lost by taking the means for each subject's group of recordings.

TABLE XLIX.

RESULTS OF INDIVIDUAL PRE-AND POST-SLEEP QUESTIONNAIRE ANALYSIS FOR EACH GROUP.

<table>
<thead>
<tr>
<th>CATEGORIES</th>
<th>PRE-SLEEP RATING</th>
<th>POST-SLEEP RATING</th>
<th>CHANGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control Group, (30 Nights)</td>
<td>2.7 (1.7)</td>
<td>2.9 (1.0)</td>
<td>0.2 (1.8)</td>
</tr>
<tr>
<td>Good Sleep + Discomfort, (27 Nights)</td>
<td>2.6 (1.5)</td>
<td>3.0 (1.5)</td>
<td>0.4 (1.1)</td>
</tr>
<tr>
<td>Poor Sleep + Discomfort, (6 Nights)</td>
<td>3.2 (1.2)</td>
<td>3.6 (0.5)</td>
<td>0.5 (0.8)</td>
</tr>
</tbody>
</table>

Mean pre and post-sleep ratings of the good sleepers with discomfort and the control group show little difference, although the mean overnight change is greater for the former. Mean ratings for the poor sleepers with discomfort were lower on the scale both before and after sleep, and the overnight change is greater than that of either of the other two groups.
Each pre-sleep score, post-sleep score and the difference between these (representing the overnight change) was compared to the mean NREM (mean over sleep stages 2, 3, and 4) alpha, delta and spindle values, and the actual sleep time for the respective night. Correlations were performed between groups of these values.

No significant correlations were found for the control group, but for the good sleep with discomfort group, there was a significant correlation between pre-sleep and post-sleep scores \((r=0.72, p<0.002)\). For the poor sleep with discomfort group, pre-sleep scores and mean NREM alpha showed the best relationship \((r=0.74, p<0.10)\).

For the next part of the analysis of SSS scores, the means of each individual's pre and post-sleep ratings were considered with respect to the means of other sleep variables, taken from the same recordings.

Results of this analysis showed very little difference to those described for individual nights. There was little difference between the means of pre-sleep and post-sleep ratings for the control group and the good sleepers with discomfort. Mean pre-sleep and post-sleep SSS ratings for the poor sleepers with discomfort were greater (more towards the 'sleepy' end of the SSS) than for either the good sleepers with discomfort or the control group.

Mean overnight change was greatest for the good sleepers with discomfort, who showed a drop in ratings from night to morning (+0.6). Poor sleepers with discomfort also showed an overnight drop (+0.3). The control group tended to show a small overnight improvement in ratings although group mean ratings were unchanged, (-0.03).
7.13.2. **SUBJECTIVE PRE-SLEEP RATINGS OF DAYTIME FEELINGS AND ACTIVITY.**

Pre-sleep ratings of general level of physical activity during the day and feelings over the majority of the day given by each group are shown in Table (L).

Mean number of recordings per subject was two for both the control group (max=3, min=1) and good sleepers (max=6, min=1) with discomfort. Two poor sleepers with discomfort underwent only one sleep recording whereas the other two were recorded twice.

**TABLE L.**

**RESULTS OF PRE-SLEEP QUESTIONNAIRE ANALYSIS:**

**PERCENTAGE PRE-SLEEP RATINGS OF DAYTIME FEELINGS AND ACTIVITY.**

<table>
<thead>
<tr>
<th></th>
<th>%CONTROL</th>
<th>%GOOD SLEEP</th>
<th>%POOR SLEEP</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No of nights</strong></td>
<td>30</td>
<td>27</td>
<td>6</td>
</tr>
</tbody>
</table>

**Phys. Activity**

- Greatly>Normal: 3 0 0
- Above normal: 20 15 17
- Normal: 54 70 33
- Below normal: 20 15 50
- Greatly<normal: 3 0 0

**General Feelings**

- Active+Cheerful: 55 18 0
- Alert+Contented: 32 48 67
- Tired+Relaxed: 9 30 33
- Lethargic+Uneasy: 5 4 0

Normal daytime activity was reported prior to the majority of recordings of both the control group and the good sleepers with discomfort, whereas half of the recordings of poor sleepers with discomfort were preceded by below normal daytime activity.
The majority of reports from poor sleepers with discomfort described feeling alert and contented during the preceding day. Amongst the two other groups, a third of reports from the good sleepers with discomfort described feeling tired and relaxed, but the majority were alert and contented. A small number of the control group reported daytime tiredness and lethargy prior to recordings.

7.13.3. POST-SLEEP SUBJECTIVE RATINGS OF SLEEP QUALITY, DIFFICULTY GETTING UP AND PHYSICAL CONDITION.

Table (LI) shows the percentage frequency of each rating prior to sleep recordings in each group of subjects.

| TABLE LI. RESULTS OF POST-SLEEP QUESTIONNAIRE ANALYSIS: PERCENTAGE POST-SLEEP RATINGS OF SLEEP QUALITY, DIFFICULTY GETTING UP AND PHYSICAL CONDITION. |
|---------------------------------|-----------------|---------------|
| %CONTROL GROUP | %GOOD SLEEP +DISC | %POOR SLEEP +DISC |
| No of Nights | 30 | 27 | 6 |
| Diff Getting up. | | | |
| Very difficult | 0 | 0 | 0 |
| Difficult | 23 | 41 | 33 |
| Moderate | 23 | 41 | 50 |
| Easy | 43 | 7 | 17 |
| Very Easy | 10 | 11 | 0 |
| Sleep Quality* | | | |
| Much better | 0 | 0 | 0 |
| Better | 3 | 7 | 17 |
| Normal | 50 | 63 | 17 |
| Worse | 47 | 26 | 66 |
| Much worse | 0 | 4 | 0 |
| Phys Condition* | | | |
| Better | 5 | 0 | 0 |
| No discomfort | 81 | 30 | 0 |
| Slight disc. | 9 | 55 | 83 |
| Aching+stiff | 5 | 15 | 17 |
| Worse | 0 | 0 | 0 |

(*) Compared to normal.
Following half of the sleep recordings, control subjects reported a normal quality sleep, and on more than half, the good sleepers with discomfort considered they had had a normal sleep. On other nights sleep quality was more often rated as worse than normal rather than better than normal for both these groups. Poor sleepers with discomfort generally described sleep as worse than normal during recordings, with only two of the six nights considered better than normal.

Morning discomfort was mainly reported in the good and poor sleepers with discomfort, although some of the controls reported some discomfort, which was attributed to previous exercise. Subjects in the group of good sleepers with discomfort did not always report discomfort, whereas but the poor sleepers with discomfort did.

On more than 80% of occasions, both discomfort groups reported some difficulty getting up, whereas control subjects who also had some difficulty, generally found getting up easy.
CHAPTER 8.

A STUDY OF THE RELATIONSHIP BETWEEN THE NREM SLEEP ALPHA ANOMALY AND MUSCULOSKELETAL DISCOMFORT IN A LOCAL POPULATION.

SUB-STUDIES.
8.1. SUB-STUDY 1: CONSISTENCY STUDIES.

These were carried out to examine the consistency of NREM sleep alpha over differing periods of time. Most of the sleep recordings were carried out at the individuals' convenience, consequently varying periods of time elapsed between consecutive recordings. It was therefore considered necessary to examine the consistency of NREM sleep alpha under normal circumstances, but due to social and work commitments few subjects were able to volunteer. For the few subjects studied, each sleep recording followed the same procedure as that described for the main study.

8.1.1. METHOD.

(i) Subject (P) who described good sleep and musculoskeletal symptoms agreed to undergo five consecutive nights of recordings. This subject had previously undergone two standard recordings, and was accustomed to the procedure and equipment.

Both standard and 'frontal-occipital' montages were used but the waking tests were carried out only once. As the 'frontal-occipital' montage does not allow REM sleep analysis, it was considered necessary to do standard recordings on alternate nights, whilst also studying frontal and occipital alpha characteristics.

(ii) Subject (C) who also reported good sleep with musculoskeletal discomfort, was studied on several occasions over a period of eighteen months. Both types of recordings were obtained during this time.

(iii) Although most subjects underwent successive recordings at varying time intervals, (maximum three months), we decided to look at the consistency of alpha, delta and spindle characteristics, in order to estimate the mean consistency for the group as a whole.
8.1.2. RESULTS OF SLEEP EEG ANALYSIS.

The mean difference between each mean NREM sleep alpha, delta and spindle value for consecutive records was computed for each subject. The mean differences for the three groups can be seen in table (LII).

**TABLE LII.**

**MEAN DIFFERENCE IN NREM SLEEP ALPHA DELTA AND SPINDLES BETWEEN CONSECUTIVE RECORDINGS FOR EACH GROUP.**

(Note n= No of subjects who underwent more than one recording).

<table>
<thead>
<tr>
<th></th>
<th>CONTROL GROUP (n=13)</th>
<th>GOOD SLEEP WITH DISCOMFORT (n=10)</th>
<th>POOR SLEEP (n=2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NREM Alpha</td>
<td>1.4</td>
<td>1.2</td>
<td>2.2</td>
</tr>
<tr>
<td></td>
<td>(1.0)</td>
<td>(1.4)</td>
<td>(0.7)</td>
</tr>
<tr>
<td>NREM Delta</td>
<td>4.8</td>
<td>5.1</td>
<td>2.9</td>
</tr>
<tr>
<td></td>
<td>(5.4)</td>
<td>(5.0)</td>
<td>(0.2)</td>
</tr>
<tr>
<td>NREM Spindles</td>
<td>3.1</td>
<td>2.5</td>
<td>2.3</td>
</tr>
<tr>
<td></td>
<td>(3.3)</td>
<td>(1.4)</td>
<td>(1.5)</td>
</tr>
</tbody>
</table>

Figures for the poor sleep with discomfort should be considered carefully, as these are based on two subjects only.

The majority of the control group and the good sleep with discomfort group underwent more than one recording. Mean differences in NREM sleep (mean over sleep stages 2, 3, and 4) alpha, delta and spindles between consecutive recordings are similar for the control and good sleep with discomfort group, although the good sleep with discomfort group show less difference in each case.

From the limited data on the poor sleep with discomfort group, there is a greater inter-recording difference in NREM sleep alpha than for either of the other two groups, but the mean difference in NREM sleep delta and spindles is less.
Subject (C) underwent three recordings. The first and second were three months apart, and the third recording was thirteen months after the second.

Figure (16) shows the percentages of sleep stages 2, 3, and 4, for each recording, and figures (17), (18) and (19) illustrate the variation in mean percentage alpha, delta and percentage of epochs containing spindles. Figure (20) shows the mean NREM sleep (sleep stages 2, 3, and 4) values for alpha, delta and spindles on each occasion. The means and standard deviations for each variable over three occasions are shown in table (LIII).

Mean percentage alpha per epoch of sleep stages 2, 3, and 4 varied over the three recordings, with the largest values observed in the second recording. Although the first recording showed slightly more stage 2 sleep alpha than the third recording, figure (17) shows that a general level of alpha activity is reflected across all the NREM sleep stages. On each of the three recordings, the highest percentages of alpha activity were seen during sleep stage 4, although there was little difference between sleep stages 3 and 4 in the second and third recordings.

Figure (18) shows stage 4 sleep showed the largest variation in percentage delta per epoch over the three recordings. Mean percentage of epochs containing spindles was greatest for sleep stage 2 on each recording occasion. The largest percentage was observed during the first night and was associated with the smallest percentage for sleep stage 3 and no spindles during sleep stage 4. Smaller percentages of epochs containing spindles during sleep stage 2 were coincident with increases in spindle activity during sleep stage 3 and 4. In the last recording of subject (C), sleep stage 2 spindle activity was less than half of that observed on the first and second recordings, whereas sleep stage 3 and 4 spindle activity were higher.
FIGURE 16.

Subject C: Consistency of Percentage Stage 2, 3, and 4

Legend
- 19.3.85
- 4.6.85
- 24.4.86
Subject C: Consistency of % Alpha in Stages 2, 3, and 4

Mean % Alpha per Epoch

Legend
- 19.3.85
- 4.6.85
- 24.4.86
Subject C: Consistency of % Delta in Stages 2, 3, and 4

Legend
- 19.3.85
- 4.6.85
- 24.4.86
Subject C: Consistency of Spindles in Stages 2, 3, and 4

Legend

- 19.3.85
- 4.6.85
- 24.4.86
FIGURE 20.

Subject C: Consistency of NREM Alpha, Delta and Spindles

Legend
- 19.3.85
- 4.6.85
- 24.4.86
Mean NREM sleep values of alpha, delta and spindle activity for each recording are shown in figure (20). Data for the first two nights indicates that an increase in NREM sleep alpha activity was accompanied by an increase in delta activity and a small decrease in spindle activity. However, in the third recording the same mean percentage of NREM sleep alpha as found on the first night was associated with the lowest percentages of both delta and spindle activity.

SUBJECT (P).

Subject (P) underwent five consecutive nights of recordings during a normal week's activity. Percentage sleep stages 2, 3, and 4 for each night are shown in figure (21).

Figure (22) shows that percentages of sleep stage 2 and 4 fluctuated over the five nights, with higher percentages of sleep stage 2 being associated with less stage 4 sleep. Stage 3 sleep showed very little variation.

Although this subject was accustomed to the sleep recording procedure, on the first night both sleep stage 3 and 4 were reduced compared to the second, third and fourth nights, suggesting this may have been a poor night's sleep.

Night to night consistency in mean percentage of alpha per epoch of each NREM sleep stage is shown in figure (22). Stage 2 sleep alpha was most consistent over the second, third and fourth nights, but reached it's lowest value on the first night, and it's largest on the fifth night. By comparison, percentage alpha per epoch of sleep stages 3 and 4 showed much less consistency from Monday to Friday. Except for the third night, in which sleep stage 2 showed the highest percentage of alpha per epoch, percentages of alpha were always greatest in sleep stage 3 and lowest in sleep stage 4.

Mean percentages of delta per epoch of sleep stages 3 and 4 (figure (23)) showed little night to night variation. However, delta in sleep stage 2 showed a pattern opposite to that of the percentage of stage 2 sleep, that is, time spent in stage 2 sleep was inversely related to the percentage of delta activity per epoch of sleep stage 2.
Subject P: Consistency of Percentage Stage 2, 3, and 4
Subject P: Consistency of Alpha in Stages 2, 3, and 4
Subject P: Consistency of Delta in Stages 2, 3, and 4

Legend
- Monday
- Tuesday
- Wednesday
- Thursday
- Friday
FIGURE 24.

Subject P: Consistency of Spindles in Stage 2, 3, and 4

Legend
- Monday
- Tuesday
- Wednesday
- Thursday
- Friday

Percentage of Epochs Containing Spindles

Stage 2
Stage 3
Stage 4
FIGURE 25

Subject P: Consistency of NREM Alpha, Delta and Spindles

Legend
- Monday
- Tuesday
- Wednesday
- Thursday
- Friday
No spindle activity was found in sleep stage 3 during the last three nights (figure 24), or in sleep stage 4 in the first three nights. On Wednesday night, spindle activity was found in stage 2 sleep only, whereas on Thursday night, stage 2 spindle activity reached its lowest percentage for the week, with a complete absence of spindles in sleep stage 3, although some spindles were seen in stage 4 sleep. Stage 4 and stage 2 sleep spindle activity both increased the following night, but spindles were still not found in stage 3 sleep.

Interestingly, the night to night pattern of stage 2 sleep spindle activity bears a close resemblance to the percentage delta activity in stage 2 sleep, except that on Thursday night, the relatively high percentage of delta activity is associated with the lowest amount of spindle activity. On all other nights, changes in the percentages of delta activity are similar to the pattern of spindle activity.

Mean percentage delta activity for NREM sleep shows the greatest variation over the five nights, compared to alpha and spindle activity, (figure (25)). However, the histogram indicates that the changes in percentages of alpha activity were the inverse of those in delta activity, with NREM sleep alpha increasing as NREM sleep delta activity decreased.

Table (LIII) gives the means and standard deviations of each variable for the consecutive nights recorded from subjects (C) and (P).
TABLE LIII.

MEANS AND STANDARD DEVIATIONS OF SLEEP VARIABLES FOR
CONSISTENCY STUDY OF SUBJECTS C AND P.

<table>
<thead>
<tr>
<th></th>
<th>SUBJECT C</th>
<th>s.d</th>
<th>SUBJECT P</th>
<th>s.d</th>
</tr>
</thead>
<tbody>
<tr>
<td>% Stage 2</td>
<td>54.7</td>
<td>(7.8)</td>
<td>44.0</td>
<td>(7.0)</td>
</tr>
<tr>
<td>% Stage 3</td>
<td>13.7</td>
<td>(1.0)</td>
<td>18.1</td>
<td>(1.0)</td>
</tr>
<tr>
<td>% Stage 4</td>
<td>4.2</td>
<td>(2.1)</td>
<td>12.5</td>
<td>(5.9)</td>
</tr>
<tr>
<td>% REM Sleep.</td>
<td>21.4</td>
<td>(4.0)</td>
<td>18.9</td>
<td>(0.0)</td>
</tr>
<tr>
<td>Stage 2 Alpha</td>
<td>7.0</td>
<td>(1.1)</td>
<td>8.6</td>
<td>(1.1)</td>
</tr>
<tr>
<td>Stage 3 Alpha</td>
<td>10.2</td>
<td>(2.5)</td>
<td>10.3</td>
<td>(1.8)</td>
</tr>
<tr>
<td>Stage 4 Alpha</td>
<td>11.1</td>
<td>(1.6)</td>
<td>4.8</td>
<td>(1.1)</td>
</tr>
<tr>
<td>REM Alpha</td>
<td>3.3</td>
<td>(1.6)</td>
<td>8.3</td>
<td>(1.1)</td>
</tr>
<tr>
<td>Stage 2 Delta</td>
<td>5.8</td>
<td>(1.2)</td>
<td>6.5</td>
<td>(2.7)</td>
</tr>
<tr>
<td>Stage 3 Delta</td>
<td>33.5</td>
<td>(1.1)</td>
<td>30.8</td>
<td>(1.5)</td>
</tr>
<tr>
<td>Stage 4 Delta</td>
<td>51.7</td>
<td>(5.7)</td>
<td>60.1</td>
<td>(1.7)</td>
</tr>
<tr>
<td>Stage 2 Spindles</td>
<td>10.0</td>
<td>(4.5)</td>
<td>0.9</td>
<td>(0.3)</td>
</tr>
<tr>
<td>Stage 3 Spindles</td>
<td>1.4</td>
<td>(0.5)</td>
<td>0.2</td>
<td>(0.3)</td>
</tr>
<tr>
<td>Stage 4 Spindles</td>
<td>0.5</td>
<td>(0.5)</td>
<td>0.1</td>
<td>(0.1)</td>
</tr>
</tbody>
</table>

8.1.3. PRE-SLEEP AND POST-SLEEP QUESTIONNAIRE RATINGS.

On each recording occasion, subject (C) rated his three
pre-sleep and post-sleep feelings as (2) on the SSS,
(functioning at high level but not at peak, able to
concentrate). Each recording was preceded by a day of normal
activity, feeling alert and contented. Subject (C) reported
normal sleep quality, with moderate difficulty and slight
discomfort on getting up.

Subject (P) completed a pre-sleep and post-sleep
questionnaire for each night recorded during the five
consecutive nights. The first night excepted, she
consistently rated her pre-sleep feelings as (1) on the SSS,
(i.e. she considered herself to have been 'active, vital,
alert and wide awake' for the majority of the day). On the
first night of the consistency study, she rated (3) on the
SSS, (i.e. 'relaxed, awake, not at full alertness,
responsive').
Post-sleep SSS ratings were (1) on each night except the third, after which she rated (3). Night to night changes in these ratings, and the mean NREM sleep alpha, delta and spindles for each respective night are shown in table (LIV). Subjective sleep quality was 'normal' on each night recorded.

There was only one report of slight morning discomfort, and this followed a night during which the highest percentage of NREM sleep alpha, and relatively low percentages (compared to other nights) of NREM sleep delta and spindles were found. Following this night, subject (P) reported moderate difficulty getting up and a decrease in her SSS rating, but there was no change in mood ratings or sleep quality compared to the other nights. Frontal 'alpha-like' activity was observed during sleep stages 2 and 3 on this night.
TABLE LIV.

NIGHT TO NIGHT CHANGES IN SSS RATINGS, NREM SLEEP ALPHA, DELTA AND SPINDLES, SUBJECTIVE SLEEP QUALITY AND PHYSICAL CONDITION FOR SUBJECT P DURING THE CONSISTENCY STUDY.

<table>
<thead>
<tr>
<th>NIGHT</th>
<th>1#</th>
<th>2</th>
<th>3#</th>
<th>4</th>
<th>5#</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Sleep SSS</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Post-Sleep SSS</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>NREM Alpha</td>
<td>7.5</td>
<td>6.6</td>
<td>7.9</td>
<td>6.9</td>
<td>7.4</td>
</tr>
<tr>
<td>NREM Delta</td>
<td>17.9</td>
<td>37.9</td>
<td>20.5</td>
<td>40.3</td>
<td>30.7</td>
</tr>
<tr>
<td>NREM Spindles</td>
<td>0.8</td>
<td>0.6</td>
<td>0.5</td>
<td>0.1</td>
<td>0.6</td>
</tr>
<tr>
<td>Actual Sleep (hrs)</td>
<td>6.3</td>
<td>5.4</td>
<td>6.4</td>
<td>6.5</td>
<td>6.4</td>
</tr>
<tr>
<td>Daytime Activity</td>
<td>BN</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Feelings over day</td>
<td>tr</td>
<td>ac</td>
<td>ac</td>
<td>ac</td>
<td>alc</td>
</tr>
<tr>
<td>Diff. getting up</td>
<td>E</td>
<td>E</td>
<td>M</td>
<td>VE</td>
<td>VE</td>
</tr>
<tr>
<td>Sleep quality</td>
<td>n</td>
<td>n</td>
<td>n</td>
<td>n</td>
<td>n</td>
</tr>
<tr>
<td>Phys. Condition</td>
<td>ND</td>
<td>ND</td>
<td>SD</td>
<td>ND</td>
<td>ND</td>
</tr>
</tbody>
</table>

* Frontal-Occipital montage used on these nights.

** Abbreviations used in table (LV):**

**Daytime Activity:**
BN= Better than Normal
N= Normal

**Feelings over day:**
tr= Tired and relaxed.
ac= Active and cheerful
alc= Alert and contented.

**Difficulty getting up:**
E= Easy
VE= Very easy
M= Moderate difficulty.

**Sleep Quality:**
n= normal

**Physical Condition:**
ND= No discomfort
SD= Slight discomfort.
8.1.4. **CONSISTENCY OF FRONTAL/OCCIPITAL ALPHA: FOURIER ANALYSIS.**

During the five consecutive nights of sleep recordings, subject (P) underwent three recordings using the frontal/occipital electrode montage.

**Night 1.**

Low amplitude frontal alpha-like activity (7.4Hz to 8.2Hz) was observed during stages 2 and 3 but was most apparent during stage 2. Occipital alpha of 8.6 to 8.8Hz was detected during REM sleep and some occipital alpha of 7.8Hz during stage 2 sleep.

**Night 2.**

Some low amplitude frontal alpha-like activity (7.4Hz to 8.2Hz) was detected during sleep stages 2 and 3, but frontal alpha-like activity was absent during REM sleep and stage 4. Occipital alpha activity of 8Hz was apparent during REM sleep.

**Waking Alpha: (Morning after Night 3).**

Occipital alpha was prominent during each waking condition, although of greater amplitude during conditions (1), (3) and (5), during which the eyes were closed.

During mental arithmetic, (conditions (3) and (4)), there was no evidence of any frontal alpha-like activity, but occipital alpha frequency was faster during these conditions compared to the relaxed state.

**Night 5.**

No frontal alpha-like activity was observed during any NREM sleep stage during this recording. Occipital alpha of 7.6Hz to 9.4Hz was seen during stage 2, and 8Hz during REM sleep.
8.1.5. **SUMMARY**

Mean differences in percentage alpha, percentage delta and percentage of epochs containing spindles between consecutive recordings showed little difference between the control group and the good sleepers with discomfort.

The two poor sleepers with discomfort showed the highest mean difference in alpha, but the lowest mean differences for delta and spindle activity.

Three recordings were carried out on subject (C). Percentages of alpha of sleep stages 2, 3, and 4 varied between recordings, but on each occasion, stage 4 sleep showed the highest percentage of alpha and stage 2 sleep the least. Delta varied between recordings but was most consistent in sleep stages 2 and 3.

Spindle activity showed the largest variation in sleep stage 2. High percentages of stage 2 sleep spindle activity were counterbalanced by lower percentages in sleep stages 3 and 4, and lower percentages in sleep stage 2 were compensated for by more spindle activity in sleep stages 3 and 4.

Subject (P) underwent home sleep recordings on five consecutive nights. On most nights alpha was greatest for stage 3 sleep, and least for stage 4 sleep, but on the third night, stage 2 sleep showed most alpha.

Sleep stage 2 delta activity showed least consistency between nights, but sleep stages 3 and 4 showed little variation. Night to night variation in spindle activity was observed in all stages, with spindles occasionally absent from either sleep stage 3 or 4 or both.

There was little variation in pre and post-sleep SSS ratings given by subject (P), the lowest post-sleep SSS rating accompanied the only reports of slight discomfort, and moderate difficulty getting up. The preceding night was characterised by the highest percentage of alpha per epoch of NREM sleep observed during the week, and relatively low levels of delta and spindle activity. Frontal alpha was identified during sleep stages 2 and 3 of this sleep recording.
8.2. **SUB-STUDY 2: SLEEP MENTATION QUESTIONNAIRE**.

8.2.1. **METHOD.**

This was designed to record details of 'dreaming' or 'thinking' during sleep immediately on awakening during the night or in the morning. Questions relating to sleep mentation were adapted from Arkin et al, (1978).

The questionnaires were completed for a week, during which subjects slept normally in their homes, and were not recorded.

The following questions relating to sleep mentation were included with the standard pre-sleep and post-sleep questionnaire:

**Pre-sleep.**

Subjects were asked to rate the activities which they had spent most or least time in during the day, for example if they had spent most time shopping, they were instructed to put (1) next to shopping. Other activities included, reading, talking, watching T.V. sleeping, eating, writing, working, exercising, and other, (describe).

**On awakening in the morning or during the night.**

(1) What time is it now? (Just after awakening).
(2) Were you aware of dreaming or thinking just prior to waking. If so which?
(3) If you were, what were you dreaming, or what was going through your mind? Please describe.
(4) Did you see pictures and/or did you think you could smell, hear or sense anything during this experience? Please describe.
(5) Did you experience any body sensations? Describe.
(6) Did the content bear any similarity or have any connection with current situations in your life? (Yes/ No: Describe).
(7) Were thoughts: Random and disconnected? or: Continuous?

(8) Did the content make sense? (Yes/ No).

(9) Would you describe your dream thoughts as any of the following?: (Answer Yes/ No).
   (a) Pleasant
   (b) Irritating.
   (c) Non-relevant.
   (d) Worrying.
   (e) Frightening.
   (f) Interesting.

(10) What caused you to wake up?
   (a) Noise.
   (b) Alarm.
   (c) Need to go to the toilet.
   (d) Thirst.
   (e) Dream/ thought content.
   (f) Worry.
   (g) Don't know.
   (h) Other. (Describe).

Post-sleep.

(1) Did anything happen yesterday to:
   (a) Cause you concern?
   (b) Make you happy/ content?
   (c) Make you worry?
   (d) Affect your ability to concentrate on things?
   (e) Surprise you, (pleasant)?
   (f) Shock you, (unpleasant)?
   (g) Change your opinions or ideas on anybody or anything?
   (h) Make you unhappy or depressed?
(Please give brief details).

(2) Please add any comments you think are relevant to your sleep or current state of mind.

(3) How would you interpret/ explain the content of your dreams or thoughts whilst you were asleep last night?
8.2.2. RESULTS.

Three good sleepers with discomfort, (subjects K, M and N), completed sleep mentation questionnaires, although five subjects initially volunteered to do so.

Some subjects said that when they awoke during the night, they fell asleep again before remembering to describe their thoughts, or else they were too sleepy to make the effort, and preferred to fall asleep.

An additional problem encountered was that subjects did not always have time to complete the questionnaire when they had to get up for work. Subjects (N) and (M) did not have to go out in the morning, thus it was easier for them to find time for the questionnaire. Subject (K) completed two nights over a weekend and two during weekdays.

SUBJECT (M).

Subject (M) completed the sleep mentation questionnaire on four consecutive nights.

Subject (M): Sleep Mentation Questionnaire 1.

During the day she had been concerned about her daughter, who was on a reducing diet, as she was anxious that she might not be getting a balanced diet on snack meals. She had also taken a sick friend to hospital for treatment and described her as "a different person when faced with fear". She considered these events had caused her concern and may have changed her opinions on some things. She had felt tired for the majority of the day. She was awoken at 0400hrs, when her daughter was getting up for work. She was aware of her legs and hips aching and remembered dreaming vividly although she was unable to recall the details. She described continuous visual imagery without sound or other sensations, and felt that the content of her dream may have been worrying, irritating and also relevant.

When she finally awoke at 0615hrs after what was considered a 'normal' night's sleep, she was sleepy, aching and stiff, and found it difficult to get up. Having recalled the events of the previous day she thought she may have dreamt about the long hours her daughter spent going to college and then working in a shop in the evening.
Subject (M); Sleep Questionnaire 2.

After a day of greater than normal activity, feeling tired and relaxed, she fell asleep at 2315hrs and awoke at 0620hrs with stomach pain and slight muscular discomfort. She could not remember if she had been dreaming or thinking prior to awakening. She rated her sleep as of normal quality but found difficulty getting up, feeling tired and sleepy.

Subject (M); Sleep Mentation Questionnaire 3.

Subject (M) fell asleep at 2345hrs after a day of less than normal activity, having been sleepy, struggling to remain awake and feeling lethargic and uneasy for the majority of the day. She described the events of the day as having made her very unhappy. Her doctor had told her to wear a collar during the day and this made her feel tired. When she retired to bed she had left the lights on for her daughter who was out, but as she slept deeply she did not hear her daughter coming in, consequently when she awoke finally she was worried that her daughter had not come back.

She awoke with the alarm at 0715hrs, and remembered having been dreaming about collecting fruit and vegetables from her mother's garden in the country. A small snake with a round tail had emerged from one of the big bowls of vegetables. The dream had been pleasant and interesting, containing continuous visual imagery, and she remembered being aware of smells of fruit and vegetables during the experience. The dream made sense to her as she often gathered produce at that time of year and had been watching a gardening programme prior to retiring. A large snake had featured on a television programme she had seen two nights previously.

She interpreted her dream as a concern that she would no longer be able to harvest fruit and vegetables at her mother's home in the country due to her own illness, and because she would have to sell her mother's home to meet her mother's nursing home fees.

She found it difficult to get up and her discomfort was worse than normal that morning, although she described her sleep as better than usual.

Subject (M); Sleep Mentation Questionnaire 4.

Subject (M) had a normal day's activity, but felt lethargic and uneasy most of the time. She had discovered that the owner of the house (for whom she was a live-in housekeeper) had not been passing on telephone messages for her, which was causing her embarrassment with her friends. She was considering moving out but knew that she couldn't cope with her own home under the circumstances of her health. This had made her unhappy and had caused her concern and anxiety. In addition she had suffered with neck and back pain from wearing the collar.

She awoke spontaneously at 0630hrs after a pleasant visual dream in which she visited her daughter's boyfriend's home with her sick friend. They had discussed central heating then her father had taken the younger children onto the roof. The previous evening, her daughter had spoken to her boyfriend's family on the telephone, a neighbour had had workmen on the roof, and the new central heating had been noisy during the night.
She got up with difficulty after a normal night's sleep, her legs more aching and stiff than usual.

SUBJECT (K).

Subject (K) completed the sleep mentation questionnaire on four nights.

Subject (K): Sleep Mentation Questionnaire 1.
Subject (K) fell asleep at 2359hrs after a normal day's activity, feeling active, alert and cheerful. He awoke at 0945hrs "aware of the time" and described having been thinking about "some type of trial" prior to awakening. There had been a room full of people passing strange objects around some of which had were associated with a smell of incense. This experience bore no connection to any current events nor did it make sense, however it was described as irritating and worrying, although quite interesting.

When he finally got up, subject (K) was aching and stiff, but described a normal night's sleep. He had spent the previous day walking and talking with his friends in Dove Dale.

Subject (K): Sleep Mentation Questionnaire 2.
He fell asleep at 2345hrs having been tired and 'let down' for most of the day, although he had spent a pleasant and happy time. He awoke at 0700hrs with slight discomfort but did not report any dreams or thoughts.

Subject (K): Sleep Mentation Questionnaire 3.
After a normal day at work, during which he had been shocked by a patient's death, subject (K) fell asleep at 2235hrs and awoke with the alarm at 0645hrs, aching and stiff. He described a normal night's sleep and reported thinking vague thoughts of University prior to awakening, seeing pictures of lecture theatres. This was vaguely linked to current events as he had recently had to cancel a blood transfusion session to be held at a University.

Subject (K): Sleep Mentation Questionnaire 4.
Subject (K) fell asleep at 2340hrs feeling lethargic and uneasy, and awoke at 0645hrs with the alarm. He felt worse than normal, found it difficult to get up, and could not recall having dreamt or having been thinking prior to awakening.
This lady also completed sleep mentation questionnaires on four nights during a normal week.

**Subject (N): Sleep Mentation Questionnaire 1.**

The day preceding her first night, subject (N) had felt lethargic and uneasy and had been less active than normal. She had expected a telephone call which she did not receive and had felt desperately tired with a headache. Her husband had recently been forced out of his job with the family business by his brother and she felt that this was affecting herself more than him.

She awoke at 0500hrs and reported that she had been dreaming. She had previously advised her daughter to seek advice about her grandson's persistent cough and was worried and frightened by the dream which had woken her up, although she could not remember the exact details of the experience. She finally awoke at 0830hrs aching, stiff and sleepy. She reported that she may have had a second dream about the same subject. She felt she had had a normal night's sleep, and described difficulty getting up.

**Subject (N): Sleep Mentation Questionnaire 2.**

Subject (N) spent a pleasant but tiring weekend with her daughter, but was shocked to find that her brother had changed the door locks at his works, thinking (she thought) that she had wanted to go in. She felt that the stress of her personal problems exhausted her, but she was unable to sleep for continually going over them.

She retired at 2220hrs but did not fall asleep until 0315hrs. She awoke at 0730hrs with the alarm, having dreamt that she was at a concert with her sister, looking at photographs of the coast. The dream had been random, disconnected and irrelevant but pleasant, interesting, clear and visual without any sound or smell. She was unable to interpret the content of this dream.

She felt she had had a worse than normal sleep, due to painful feet and an active mind. She found it difficult to get up eventually and was aching, stiff and sluggish.

**Subject (N): Sleep Mentation Questionnaire 3.**

Subject (N) had been visited by a close friend during the day which made her happy although she had felt tired. She retired at 2330hrs having been less active than usual, and awoke at 0145hrs having dreamt that her 51 year old sister was with her baby and was propping it up in the bath, although it had since turned into a doll. This dream had been clear and colourful, without sound or smell, random and disconnected and without sense. She described it as pleasant and interesting, although she was unable to interpret it.

She got up without difficulty feeling 'let down' and with some discomfort, but described her sleep as having been better than normal.
Subject (N): Sleep Mentation Questionnaire 4.

During a normal day she felt tired and below her full alertness. She had a better than normal night's sleep but reported no dreams or thoughts on awaking at 0730hrs with the alarm. When she got up she felt 'let down' and had slight discomfort.

8.2.3. SUMMARY OF RESULTS.

Due to problems in subject enrollment and participation in this sub-study, only three subjects, (M), (N) and (K) completed the sleep mentation questionnaires. Each subject returned four questionnaires.

All of the three subjects experienced daytime events which they found unpleasant or shocking, but dreams reported the following morning were not necessarily unpleasant.

In general, subjects reported dreams which were related to current events in their lives, but there was no apparent relationship between the content of their reported dreams and the magnitude of morning discomfort.

On three occasions, subject (M) reported that the contents of her 'dreams' were related to current events and anxieties. Following two of these nights she felt her discomfort was worse than normal on awakening.

Subject (K) reported 'dreaming' on two occasions prior to awakening in the morning, but on only one occasion was the content related to the previous day's events.

Subject (N) remembered dreaming in three of her four questionnaires, and on one occasion she described a disturbing dream which was related to her anxiety about her grandson. Two dreams were described as pleasant and interesting, although she was unable to interpret the content.
8.3. **SUP-STUDY 3: DAILY LOGS OF SUBJECTIVE WELL-BEING.**

8.3.1. **METHOD.**

Two good sleepers with discomfort, (subjects K and D) agreed to complete these logs half hourly every day for as many days as they could manage without too much inconvenience. In addition they completed pre and post sleep questionnaires for the intervening nights.

Daily logs consisted of the following scales.

SSS descriptions were used ranging from alert (1) to unable to stay awake, (7).

**Key to Stanford Sleepiness Scale Ratings.**

(1) Active, vital, alert and wide awake.
(2) Functioning at a high level, but not at peak. Able to concentrate.
(3) Relaxed, awake, not at full alertness, responsive.
(4) A little foggy, not at peak, let down.
(5) Fogginess, beginning to lose interest in staying awake, slowed down.
(6) Sleepiness, prefer to be back in bed, fighting sleep, woosy.
(7) Sleep onset soon, struggling to remain awake.

**Tense/calm scale ratings as follows:**

(1) Very tense.
(2) Moderately tense.
(3) Slightly tense.
(4) Neither tense nor calm.
(5) Slightly calm.
(6) Moderately calm.
(7) Very calm.
Ratings of pain/stiffness on the following scale:

1. No pain or stiffness.
2. Mild pain or stiffness.
3. Uncomfortable pain or stiffness.
4. Severe pain or stiffness.
5. Pain or stiffness as bad as it could be.

8.3.2. SUMMARY OF RESULTS.

For both subjects, patterns of alertness/drowsiness, tension/calmness and discomfort showed similar patterns for each of the days described on the logs.

Subject (K) experienced discomfort on each of the five days. He frequently suffered with discomfort for a few hours in the morning, and prior to retiring. Periods of increased tension generally occurred in the daytime, especially in the mornings, and occasionally during the late afternoon or evening. Ratings of alertness usually increased over the mornings, dropped slightly after lunchtime, then improved until late evening, when they fell again prior to retiring. SSS ratings of alertness often increased during periods of tension, and periods of uncomfortable pain were frequently, but not always, associated with feelings of calmness and drowsiness.

Prior to sleep on each of the five days, subject (K) described feeling either 'active and cheerful' or 'alert and contented' for the majority of the day, and always rated himself as (3) on the SSS. He slept between seven and eight hours on each night, and each time awoke with some discomfort, which varied in severity. He reported normal quality sleep on each occasion, but his SSS rating occasionally dropped to (4) in the morning, and never showed any improvement over his pre-sleep rating.

Although he usually had no difficulty getting up in the morning, he did have some difficulty the morning after being on call during the night, despite not having been called out. On this occasion he awoke with his legs aching and stiff, and continued to suffer with some discomfort until 2030hrs.
Subject (D)'s pre-sleep SSS ratings varied between (1) and (5) over the four days. Higher ratings (more alert) were associated with 'above normal' daytime activity, whereas lower ratings of (4) and (5) followed 'below normal' daytime activity, feeling 'tired and relaxed'.

He suffered with discomfort in the mornings, but this generally subsided within a few hours, and returned later in the evening. He became progressively more tense over the day, although this diminished slightly in the afternoon. In general, his tension increased as his discomfort diminished over the day. Alertness ratings also increased with his feelings of tension.

He described his sleep as 'normal' or 'better than normal' on each night, and frequently stayed in bed for an extra hour or more after six to eight hours sleep. On getting up, his SSS ratings varied between (2) and (5), and he always experienced some difficulty getting up, and was aching and stiff.
8.4. **SUB-STUDY 4: TEA AND COFFEE INTAKE.**

Due to the stimulating effects of caffeine, a small survey was carried out to see if patients with musculoskeletal discomfort consumed particularly large amounts of tea and coffee, which might interfere with sleep onset and contribute to sleep difficulties.

8.4.1. **METHOD.**

Tea and coffee intake was recorded for periods of one to two weeks depending on the subjects' memory and cooperation.

For each drink the following details were recorded:-

1) Date and time.
2) Size of cup or mug.
3) Quantity actually drunk.
4) Type of beverage, tea or coffee.
   (If tea; herbal, PG tips etc. If coffee; instant, decaffeinated, ground).
5) Strength, (strong, medium, weak).

8.4.2. **RESULTS.**

Four subjects (C), (K), (M) and (N) from the group of good sleepers with discomfort agreed to complete daily tea and coffee intake logs for a minimum of seven days.

**SUBJECT (C): Tea and Coffee Intake.**

Subject (C) completed the logs for thirteen days, during which time he worked at home and had free access to drinking facilities. Each day he considered he had drunk his usual amount of tea and coffee.

On average he drank approximately 36.9 fluid ounces (±18.5) of tea or coffee a day. Each mug was taken at intervals of approximately 3.5 hours. He drank mostly caffeinated coffee, (approximately 33.4 fl oz, ±18.4) and also had one mug of tea (~ 9 fl oz, ±0) on each of five days during this period.

Typically, he would have his first cup at breakfast time and his last after supper, at any time between 1800hrs and 2330hrs. There was no regular pattern of intake following his last meal at 1800hrs, but most drinks prior to
this were taken after mealtimes or during mid-morning or afternoon breaks.

SUBJECT (K); Tea and Coffee Intake

Subject (K) completed the intake logs for seven days of a normal week, on only one did he consider that he had consumed less than usual, and this days total fell short of the average daily intake by 11 fl. oz.

He drank approximately six mugs (~ 59.7 fl. oz, ±8.7) of tea or coffee per day, of which the majority (~ 44.1 fl. oz, ±10.6) was decaffeinated coffee, and the rest, weak Earl Grey tea (~ 17.5 fl. oz, ±4.1). He generally had his first drink at 0730hrs then at 1.5 to 3 hourly intervals following this. His last drink was usually decaffeinated coffee taken between 2100hrs and 2330hrs.

SUBJECT (M); Tea and Coffee Intake

Subject (M) was a live in housekeeper and had free access to tea and coffee facilities. However, as she was busy for most of the day, her drinks were taken at regular times on each day, the first at 0800hrs and the last at 1700hrs.

This lady measured the quantity of fluid held by each cup, and recorded all details precisely for eight days. On two days she drank more than usual, which exceeded her mean total intake (16.8 fl. oz, ±5.5) by 7 fl. oz and 16 fl. oz respectively. On each occasion she usually drank two cups of medium strength tea.

SUBJECT (N); Tea and Coffee Intake

This lady completed seven days of intake logs, during which she worked part-time or was at home. She claimed to usually drink tea, but consumed more coffee than tea on each of the seven days recorded. Her intake was irregular with two days recorded as less than usual, and three days recorded as more than usual, but this usually referred to whether she had had more coffee rather than the total intake.

Her mean intake of medium strength tea over seven days was 12.8 fl. oz (± 12.8), although on the four days that she drank tea she consumed from two to six cups a day. Her mean coffee intake for the week was 16.3 fl. oz (± 7.6), and her mean total intake of tea and coffee per day for this period was 14.5 fl. oz (± 10.7). Her last drink was taken no later than 2130hrs.

Five months previous to completing these logs, this lady was advised by her GP to cut down her tea and coffee intake, as a solution for her restless legs. This had proved effective, but had not relieved her morning discomfort and lethargy.
Table (LV) gives the mean daily intakes of tea and coffee for each of the four subjects, and the mean, maximum and minimum percentages of alpha per epoch of NREM sleep observed in each subject.

**TABLE LV.**

**MEAN TEA AND COFFEE INTAKES OF SUBJECTS C, K, M AND N AND INDIVIDUAL NREM SLEEP ALPHA CHARACTERISTICS.**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Daily Intake (fl. oz)</th>
<th>% NREM Alpha</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Mean</td>
</tr>
<tr>
<td>Tea</td>
<td>Coffee</td>
<td></td>
</tr>
<tr>
<td>Subject C.</td>
<td>9</td>
<td>33.4</td>
</tr>
<tr>
<td></td>
<td>(0)</td>
<td>(18.4)</td>
</tr>
<tr>
<td>Subject N.</td>
<td>12.8</td>
<td>16.3</td>
</tr>
<tr>
<td></td>
<td>(12.8)</td>
<td>(7.6)</td>
</tr>
<tr>
<td>Subject M.</td>
<td>16.8</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>(5.4)</td>
<td>(0.6)</td>
</tr>
<tr>
<td>Subject K.</td>
<td>17.5</td>
<td>44.1(d)</td>
</tr>
<tr>
<td></td>
<td>(4.1)</td>
<td>(10.6)</td>
</tr>
</tbody>
</table>

(d) This subject drank decaffeinated coffee only.

**Note.** Unless otherwise specified, one cup measured approximately 8 fl. oz and one mug measured approximately 9 fl. oz.

Subject (K) consumed the most tea per day, and also the most coffee, although this was decaffeinated. Subject (C) consumed the most caffeinated coffee, but the least tea.

Amongst the four subjects described, subject (C) showed the largest mean percentage of alpha per epoch of NREM sleep, whereas subject (K) showed the least.
8.4.3. SUMMARY OF RESULTS.

Four subjects completed daily logs of tea and coffee intake for between seven and thirteen days. Subject (C) who consumed the most caffeinated coffee per day, usually had his last drink before 2330hrs, immediately prior to retiring.

Subject (K) consumed the most tea per day, although this was described as 'weak Earl Grey'. He also consumed a large amount of decaffeinated coffee at regular intervals throughout the day and prior to retiring.

Subject (M) drank medium strength tea three times a day, but no coffee. Her last drink was usually taken at 1700hrs. Subject (N) also drank medium strength tea in addition to coffee, although she had been advised to cut down. Her last drink was taken at 2130hrs.

If decaffeinated coffee is ignored, then the mean daily intakes of coffee for each subject show some association with their mean percentages of NREM sleep alpha, although the alpha values were obtained at different times to the intake logs.

If the mean daily intakes of tea are considered, the relationship appears to be inverse, subjects who drink the most tea showing less NREM sleep alpha, (even if 'weak Earl Grey' is disregarded).

However, if the mean daily intakes of tea and coffee are added, to give the mean total daily intake, this shows some relationship to mean NREM sleep alpha. The results suggest that daily intake of caffeinated coffee may have some bearing on mean percentages of NREM sleep alpha, although a study of a larger number of subjects is required. It is also important to note that good sleepers with discomfort were used for this study, and as differences in percentages of NREM sleep alpha have been identified previously, it is possible that these subjects may show different alpha characteristics to control subjects.
CHAPTER 9.

A STUDY OF THE RELATIONSHIP BETWEEN THE NREM SLEEP ALPHA
ANOMALY AND MUSCULOSKELETAL DISCOMFORT IN A LOCAL
POPULATION.

DISCUSSION OF SLEEP EEG STUDY AND SUB-STUDIES.
9. DISCUSSION OF SLEEP EEG STUDY AND SUB-STUDIES.

Subjects who participated in the main sleep EEG study were seven males and eight females with musculoskeletal discomfort, who agreed to undergo sleep recordings following analysis of their preliminary questionnaires. Seven female and eight male control subjects also continued with the study.

The experimenter became well acquainted with all the subjects in the course of the study, and as a result gleaned additional information about each subject's personality and experiences.

Subjects with discomfort complained of daytime lethargy and musculoskeletal symptoms, and a tendency to periodic anxiety and depression. All subjects were outwardly cheerful and friendly, but eleven subjects described previous or current stressful experiences, and despite few reports of previous stress in the control subjects, five control subjects mentioned that they had been divorced or separated within the last five years.

Subjects with discomfort were divided into good sleepers and poor sleepers based on subjective evaluation of their sleep quality. Good sleepers with discomfort reported a variety of health problems, whereas poor sleepers with discomfort mainly reported anxiety and depression, rheumatism and migraine. One good sleeper with discomfort claimed to suffer with Raynauds syndrome, which was also diagnosed in 30% of patients at a fibrositis clinic, and in 9% of patients studied by Wolfe, (1986). Hay fever was the most common health problem in the control group and was reported by three of these subjects, but asthma and migraine were also reported by two control subjects respectively.

Two of the good sleepers with discomfort (subjects I and N), one poor sleeper with discomfort (subject J), and control subjects (5) and (7) smoked cigarettes. Soldatos et al, (1980) reported that sleep difficulties were more common amongst smokers than non-smokers, but the small number of smokers in the present study is unlikely to have affected the overall results.
Several types of non-CNS drugs interfere with sleep by antagonizing the sleep onset process and occasionally causing sleep interruptions or shortening of the sleep period, (Dement and Guilleminault, 1979). In the present study, the only one of these drugs used was the oral contraceptive. Three control subjects, and one of each of the discomfort groups reported current use of the 'pill', but their sleep characteristics did not appear to be different from other members of the group. None of the subjects regularly took any aspirin related medications although some subjects took paracetamol when they had headaches or colds.

Sleep anomalies were more frequently reported by the two discomfort groups than by the control subjects, most common were difficulty falling asleep, waking up early in the morning, difficulty staying awake in the day and a need to move the legs in bed. One good sleeper with discomfort experienced episodes of sleepwalking during the period of study. The most common sleep anomalies reported by the control subjects were sleeptalking and bruxism.

Subjects with musculoskeletal discomfort were generally physically active, but the effects of exercise on discomfort symptoms varied between individuals. Some found that exercise exacerbated their symptoms, whereas others found that lack of exercise often precipitated the problem. Strengthening exercises and gentle exercise brought relief to some subjects, including those whose discomfort was made worse by exercise.

In the fifty fibrositics studied by Yunus et al, (1981) symptoms were made worse by inactivity in 78%, and by overactivity in 62%, while they were improved by moderate activity in 82% of patients. Many patients reported that diversional activities (recreational or otherwise) made them feel better by distracting attention from their aches and pains. A sedentary state may worsen symptoms by the gelling phenomenon, whereas excessive physical activities probably worsen symptoms by producing fatigue and mechanical stress on ligaments, soft tissues and tendinous attachments, (Yunus et al, 1981).
Bennett, (1986) recommended that one of the ways in which patients with Fibrositis could help themselves, would be to remain active and to attain a greater level of physical fitness, as improved physical conditioning is believed by some to result in a worthwhile improvement of symptoms in this disorder. Mc Cain, (1986) reported that exercise may help fibrositics because of it's effects on SWS, as in non-fibrositics, spectral analysis of the EEG shows an increase in the power density of the delta band in response to exercise, (Torsvall, Akerstedt and Lindbeck, 1984).

According to Mc Cain, (1986) fibrositics who underwent a twenty week cardiovascular fitness training programme showed a bigger improvement in objective measurements of pain, when compared to subjects who underwent a placebo programme consisting of flexibility manouevers. However, despite a mean decrease in cardiovascular fitness, the placebo group did show some improvement on all pain measurements. The cardiovascular trained group initially had significantly more pain-reporting behaviour than subjects in the flexibility group, but it is possible that strenuous exercise may change pain-reporting behaviour.

Unfortunately, mood changes in relation to exercise were not reported by subjects in the present study, and other studies of the effects of exercise on mood have shown inconsistent and inconclusive results, (Kupfer et al, 1985).

Other factors which increased symptoms in the discomfort subjects were inactivity, tiredness, cold, wet weather, and weight gain, whereas fresh air, massage, rest, or gentle movement and warmth brought relief to different individuals.

Daily logs of subjective discomfort, alertness and tension, which were obtained from good sleep with discomfort subjects (K) and (D), suggested that in these individuals, musculoskeletal discomfort was most evident in the mornings and evenings. In most cases uncomfortable pain was associated with relative calmness and drowsiness. Fibrositics studied by Yunus, (1981) typically complained 'I am always tired', but this symptom usually occurred either
in the morning on arising, or in the evening on relaxing after a hard days work.

9.1. SLEEP CHARACTERISTICS.

According to Smythe (1979), patients with Fibrositis typically emphasize their physical complaints but minimize their sleep disturbance, although they describe a disabling exhaustion and arise feeling unrefreshed and more exhausted than the night before. However, Campbell et al. (1983) found that the frequency of complaints of difficulty falling asleep, waking frequently and early was the same for both fibrositics and control subjects.

In the present study, the eleven good sleepers with discomfort reported sleeping well or very well. Information supplied by subjects who underwent the main sleep EEG study and completed the second questionnaire showed that the majority of both the good sleepers with discomfort and the control subjects enjoyed their sleep, rarely awakening during the night, with few reports of difficulty falling asleep. The three poor sleepers described some problems with their sleep, with relatively more reports of longer sleep onset latency and increased nighttime awakenings, and this group showed the greatest mean sleep onset latency although there was a large variability in individual values. Control subjects had the shortest sleep latency, with the least variation within the group.

There were no significant differences in mean percentages of sleep stages 1, 2, 3, 4, SWS or REM between any pair of groups, but sleep stages 2 and 3 were found to vary most within the groups of good sleepers with discomfort and the control subjects, whilst stage 4 sleep, sleep efficiency and REM sleep showed the most inter-group variability for the poor sleepers with discomfort. This variation arose because although the poor sleepers with discomfort all reported poor and unrefreshing sleep, there were large individual differences in the magnitude of sleep disturbances observed.
Mean percentage of SWS was greatest for the good sleepers with discomfort (17%, ±10.5) and least for the poor sleepers with discomfort, (11.8%, ±10.2), although there was a wide range of mean percentage SWS within each group. In relation to the mean age of each group, poor sleepers showed less than the 13% quoted by Williams et al, (1974) for the mid-thirties, but the controls and good sleepers with discomfort fell within the 13% to 25% quoted for mid-twenties to mid-thirties age groups, (see section 1.5.).

Compared to normative sleep data of ten 17 to 30 year old subjects (Clausen, Sersen and Lidsky, 1974), and control night data of nine college students, (Langford, Meddis and Pearson, 1972) our subjects in all groups show less stage 2 and stage 4 sleep but more stage 1 and stage 3 sleep than subjects studied in their laboratories over four consecutive nights, or one control night respectively. Mean percentages of REM sleep are also less than the mean reported by Langford, Meddis and Pearson, (1972), but control subjects showed more REM sleep than the mean (21.3, ±4.5) reported by Clausen, Sersen and Lidsky, (1974). Unfortunately, in the present study, percentages of REM sleep could only be obtained from the standard sleep EEG montage recordings, and as these were generally the first recordings carried out, they tended to show more sleep disturbance as a result of unfamiliar sleeping conditions.

Although the group means indicate poorer sleep in each group in the present study, the means for each group do fall within the ranges observed for Clausen et al's subjects, and also reflect the group differences in reported sleep quality.

As found in the studies of fibrositic patients, most of the subjects in the discomfort groups awoke feeling unrefreshed and drowsy after a night's sleep. Most of the good sleepers with discomfort felt that tiredness on awakening, and prolonged sleep latency characterised a poor night's sleep, whereas the control subjects felt that waking a great deal and being aware of thinking all night were most important. Poor sleepers described waking a great deal, having a headache or feeling tired on awakening, but neither
of the discomfort groups reported that morning discomfort were important descriptors of a poor night's sleep.

In conjunction with the sleep recordings, all subjects completed pre and post-sleep questionnaires, giving their evaluations of subjective alertness and well being. Analysis of the mean overnight changes in the SSS ratings found that all groups showed a slight decrease in their alertness ratings from night to morning. Very little difference was found between the mean scores for the control group and the good sleepers with discomfort, but the poor sleepers with discomfort had the lowest alertness ratings both before sleep and in the morning.

Both discomfort groups reported some aching and stiffness on awakening after sleep recording nights, although good sleepers with discomfort subjects were occasionally free of complaints.

Subjective sleep quality was confirmed by the mean percentages of sleep stages and sleep efficiency shown by the three groups. Sleep stage characteristics of good sleepers with discomfort were similar to those shown by the control subjects, whereas the poor sleepers with discomfort showed more of sleep stages 1 and 2, less REM sleep and less of sleep stages 3 and 4, and SWS. Control subjects showed the greatest and least variable mean sleep efficiency, whereas the poor sleepers with discomfort showed the smallest mean sleep efficiency, and the most stage 1 sleep.

9.2. THE ALPHA SLEEP ANOMALY.

Analysis of the alpha content of NREM sleep stages followed the report by Moldofsky et al, (1975) who found a NREM sleep anomaly similar to 'alpha-delta' sleep, (Hauri and Hawkins, 1973) in patients conforming to the clinical criteria for fibrositis. Catesby Ware et al, (1986), also detected alpha intrusions in the NREM sleep of nine fibrositics and five depressed patients.

Smythe and Moldofsky, (1977,1978) suggested that the alpha anomaly may represent an internal arousal system, which competes with NREM sleep mechanisms thereby impairing the restorative role of SWS, resulting in the symptoms of
musculoskeletal discomfort and fatigue. Further to this, Moldofsky and Lue, (1980) reported that NREM alpha time and power were correlated with overnight increases in subjective pain, tenderness, and hostility.

More recent studies indicate that the alpha sleep anomaly is not consistently present in the sleep of fibrositic patients, and may be identified in normal healthy subjects. Weber et al, (1983) studied thirteen female fibrositic patients but found the alpha sleep anomaly in the sleep of only one of these patients, and in two patients with a non-restorative sleep syndrome. Scheuler, (1983) observed the alpha sleep pattern in 6 out of 44 subjects who were without somatic or psychological complaints, and in a later study, Scheuler (1986) claimed that 'alpha-delta' sleep is a common phenomenon, found in 15% of the normal population, and is not a causative factor in the appearance of fibrositic symptoms. In the largest survey reported, he found a significantly higher frequency of the 'alpha pattern' in 132 subjects who were without sleep disturbance, (20.5%) compared to 108 subjects with sleep disturbance, (8.3%), proving, he claimed, that there is no correlation of the alpha sleep pattern to sleep disturbances.

In our study, compared to the symptom-free control subjects, higher mean percentages of alpha activity were observed in the good sleepers with discomfort and poor sleepers with discomfort. The range of mean percentages of alpha for each sleep stage can be observed in the raw data given in appendix (XI). Mean percentages of alpha activity were obtained from computer detection of the percentage of alpha activity in each 20 second sleep epoch scored by the Oxford Sleep Stager. In this thesis the amount of alpha activity in each subjects sleep is described by the mean percentages of alpha per epoch of sleep for consecutive samples of each sleep stage.

Although good sleepers with discomfort showed greater mean percentages of alpha per epoch of sleep stages 2, 3, and 4, there was a wide range of values in the group as a whole. Some of these subjects actually showed less alpha in NREM sleep stages than members of the control group did.
Mean percentage NREM alpha for the controls was 2.0 (±1.1), four of the good sleepers with discomfort showed less than this, whereas one control subject showed a greater percentage of NREM alpha than the mean for the good sleepers with discomfort, (3.8, ±2.8).

Poor sleepers with discomfort showed the greatest mean percentages of alpha for sleep stages 2, and 3 and mean NREM sleep, and these were significantly different from the control group but not the good sleepers with discomfort. Mean REM sleep alpha was also greatest for the poor sleepers with discomfort and least in the control subjects.

In the group of good sleepers with discomfort, 45% of subjects showed their maximum NREM percentage of alpha in sleep stage 2, 27% showed the most in stage 3 sleep, and 18% in stage 4 sleep. Subject (K) showed more alpha in REM sleep than any NREM sleep stage.

Three poor sleepers with discomfort showed most alpha in stage 3 sleep, but subject (G) showed most in stage 2 sleep. Only 13% of controls showed most alpha in stage 4 sleep, with 46% and 40% showing most in stage 2 and stage 3 sleep respectively.

It is important not to label the alpha activity in NREM sleep as 'alpha-delta' sleep as this description was used by Hauri and Hawkins, (1973) for a mixture of 5 to 20% delta waves with alpha like rhythms, observed from a unipolar C4 sleep recording. The criteria of 5 to 20% delta waves and the total absence of sleep spindles in 'alpha-delta' sleep defines this sleep phenomenon as a unique sleep stage, and not a description for the presence of alpha frequency activity in slow wave sleep.

In the present study, 'alpha-delta indices' were computed to indicate the ratio of percentage alpha activity to delta activity in each sleep stage. As with the mean percentages of alpha, mean percentages of delta in NREM sleep stages were similar in the control subjects and the good sleepers with discomfort, but whereas percentage alpha was greatest for the poor sleepers with discomfort, the group mean percentage NREM delta activity was the lowest observed. This arose because one poor sleeper with discomfort failed to show any stage 4 sleep, but when the
mean percentage delta was computed for the three subjects who showed stage 4 sleep, it was greater than for either of the other two groups. Subject (E), who was the youngest subject in this group, had a particularly high mean percentage of delta in stage 4 sleep, (74%), but subjects (J) and (H) showed mean percentages of delta just below the analyser's scoring criteria of 50% delta activity for stage 4 sleep.

All groups showed the highest ratio of alpha to delta activity in sleep stage 2, but in the group of poor sleepers with discomfort, there was more than twice as much alpha activity as delta activity in sleep stage 2. For both discomfort groups, the mean percentage of alpha per epoch of sleep stage 3 was approximately 18% of the amount of delta activity. Control subjects showed less than half of this percentage of alpha in proportion to their delta activity (7.2%).

In stage 4 sleep the good sleepers with discomfort showed the greatest mean alpha-delta index, which was approximately half that for stage 3 sleep, but the poor sleepers produced surprisingly low quantities of alpha per epoch of stage 4 sleep, compared to stage 2 and stage 3 sleep.

9.3. FRONTAL 'ALPHA' ACTIVITY.

More recent investigations into the nature of NREM sleep 'alpha' have employed a full electrode montage in order to determine the origin of this activity. Weber et al, (1983) found NREM sleep alpha activity was concentrated frontally and parasagitally on the scalp surface.

Scheuler (1986) described the 'alpha pattern' as a 'rhythmic activity in the alpha range, (amplitudes between 25 and 75μV) superimposed on regular sleep patterns, with an affinity for delta sleep stages 3 and 4...the alpha power has it's maximum in the frontal areas and decreases towards the temporal and occipital areas'.

Fourier analysis of the sleep EEGs of subjects in the present study found a greater incidence of frontal 'alpha-like' activity in the good sleepers with discomfort, (75%) although 44% of the control subjects also showed some frontal 'alpha-like' activity. In some cases frontal 'alpha-like' activity was slower than waking occipital alpha, but in others it was faster. More sleep stages were affected by this activity in the good sleepers with discomfort compared to the control subjects.

Neuromapping of sleep EEG frequencies in subject (P) detected a frontal focus of 'alpha-like' activity during NREM sleep, but alpha activity also appeared centrally and occipitally at different times. These results confirmed the fourier analysis results for this subject, who exhibited particularly high mean percentages of alpha activity during sleep stages 2 and 3. However, Buchsbaum et al, (1982), carried out topographical cortical mapping on four subjects also during an afternoon sleep, finding that although alpha activity diminished across the sleep stages, it tended to move to a more anterior position during sleep stages 3 and 4.

The appearance of frontal 'alpha' activity during NREM sleep is surprising considering that alpha has previously been considered as a predominantly occipital activity, characteristic of a state of relaxed wakefulness. It is therefore important to consider the waking characteristics of alpha activity before speculating on the nature of the frontal 'alpha-like' activity observed during NREM sleep.

9.4. CHARACTERISTICS AND ORIGINS OF WAKING EEG ALPHA ACTIVITY.

The 'alpha rhythm' of relaxed wakefulness occurs predominantly in the posterior half of the brain, especially in the parieto-occipital regions, whereas the anterior half of the brain is usually relatively silent. (Adrian and Matthews, 1934: cited by Kiloh et al, 1972). The rhythm is classically described as a 'bilateral posterior rhythm of substantially constant frequency, in the range of 8Hz to 13Hz, which is diminished primarily by eye opening, (this
effect is described as 'blocking' or 'desynchronisation'). A small minority of normal people have little or no alpha rhythm, ('Minimal' alpha types) whereas another small group have an alpha rhythm that persists even when the eyes are open, ('Persistent' alpha types) although the abundance of alpha is usually reduced. Most people are intermediate between these two extremes, and are known as 'Responsive' alpha types, (Kiloh et al, 1972).

It is interesting to note the higher incidence of persistent alpha producers in the good sleepers with discomfort studied here. Five of the eight good sleepers with discomfort (subjects B, L, K, Q and D), and three of the control subjects (6, 13 and 14) showed occipital alpha activity independent of the eye condition, although the amplitude of each peak was reduced with the eyes open. Two good sleepers with discomfort (subjects F and M) and five of the control subjects (5, 7, 10, 12 and 15) only showed occipital alpha in the eyes shut condition. Control subject (11) did not show any alpha activity during any waking condition.

A complex EEG pattern such as the alpha rhythm may be considered as a combination of two or more sinusoidal rhythms, in which case it is sometimes useful to analyse the pattern into components. The differentiation of components may be facilitated by spatial analysis, or by their responsiveness to stimuli, in which case the activity may be enhanced or attenuated, (Cooper et al, 1974).

In order to determine the spatial relationships between alpha generators, Inouye et al, (1986) recorded waking EEGs from eight electrode sites, F3, F4, C3, C4, P3 and P4, of twenty normal subjects. He then used 'Relative Power Contribution Analysis', (which allows the determination of the relative contributions of different sources to the power of a certain area at different frequencies) to divide the alpha activity into endogenous and exogenous components.

Endogenous alpha activity increased as the area was more posterior, with about 50% of occipital power arising in its own area. In the frontal and central regions, alpha activity originated mostly in the posterior regions of both hemispheres, with very little alpha originating anteriorly.
In most cases, alpha peaks occurring frontally or centrally were slower or faster than other areas. In the parietal areas, only twenty five per cent of alpha activity was endogenous, the remainder being generated occipitally.

Each area was considered to have its own alpha generator, which produced both endogenous and exogenous activity, of which the latter was influenced by generators in the other areas. Anterior alpha activity was more dependent on other areas and originated in more various areas than did posterior alpha activity. Although Nunez and Katzenelson (1981; cited by Inouye et al, 1986) found a slower alpha activity in the anterior regions and a faster alpha activity in the posterior regions, Inouye reported that in some subjects faster alpha frequencies were observed anteriorly.

Ozaki and Suzuki, (1987) identified two components of alpha activity corresponding to 'generalised' and 'localised' components, which were suggested to account for the major properties of rhythmic alpha activity. Whereas the 'generalised' component had its power maxima in the parieto-occipital chain over both lateral areas, the 'localised' component had its maxima over the anterior midline. Their results suggested that during wakefulness, a highly correlated alpha rhythm occurs over most of the scalp, except for the midline vertex area, while the characteristics of the 'localized' component support the idea of an anterior or vertex centred intrinsic activity.

Using spectral analysis of sleep and waking EEGs of dominant and non-dominant alpha subjects, Johnson et al, (1969) found no qualitative differences between high and low alpha subjects for sleep stages 2, 3, and 4, although there were differences during REM sleep and sleep stage 1. The primary quantitative difference was that high alpha subjects had a larger number of identifiable frequency peaks in their average spectrum. Furthermore, as waking delta, theta and alpha intensities predicted their counterparts during sleep, individual differences in waking were suggested to predict some individual differences during sleep. This was particularly evident for REM sleep, a high waking alpha subject will probably show alpha activity during REM sleep.
while a subject with low waking alpha would not. In addition, waking intensity of the alpha band never correlated with delta intensity during wakefulness or sleep.

In our study, control subject (11) was the only individual who showed frontal NREM 'alpha-like' activity but did not show any alpha during wakefulness or REM sleep. Each discomfort subject studied using the frontal-occipital montage showed alpha during wakefulness and REM sleep, but only six of the nine control subjects showed alpha during REM sleep, (of which three showed frontal 'alpha-like' activity during NREM sleep stages). The discomfort group may therefore have contained more 'high' alpha types than the control group, which could account for some of the differences in alpha observed in NREM sleep.

The apparent spread of waking alpha activity over the scalp may be from the anterior to posterior regions or bidirectional, (Inouye et al, 1983). However, during afternoon sleep, Buchsbaum (1982) observed that alpha activity moved to a more anterior position in sleep stages 3 and 4.

Due to the predominance of occipitally originating alpha activity during wakefulness, it is possible that alpha activity may spread anteriorly during nocturnal sleep, or that the characteristics and origins of NREM alpha activity may differ from those during wakefulness.

The frontal activity observed during NREM sleep may be a variant of alpha, within the same frequency range as alpha, but characterised by different functional properties.

9.5. THE 'KAPPA' RHYTHM, THETA ACTIVITY AND SLEEP MENTATION.

The 'kappa' rhythm is an alpha variant which is best recorded between anterior temporal electrodes on either side of the head. The presence of this alpha variant during sleep has been reported in a healthy twenty three year old man by Sewitch et al (1978) who consistently observed an anterior 9 to 11Hz sinusoidal activity superimposed upon delta activity in sleep stages 3 and 4.
The 'kappa' rhythm is a distinct fronto-temporal, 10Hz, cortical rhythm originally described by Kennedy et al, (1948), who demonstrated that kappa activity increased while subjects were engaged in intellectual tasks such as reading, discriminating, learning and mental arithmetic, requiring mental effort. Whereas classical waking alpha activity is attenuated during anxiety states, (Kiloh et al, 1972) waking kappa activity was found to be positively associated with anxiety estimates by subjects and experimenters studied by Chapman et al, (1962).

In this study, Chapman et al, (1962) recorded the waking EEGs of one hundred subjects who underwent various mental performance tasks. There was a tendency for hard tasks to decrease alpha activity, and increase kappa activity, whilst closing the eyes tended to increase both kappa and alpha. When 7 to 12Hz activity was recorded from fronto-temporal regions it showed different functional properties than when it was recorded occipitally. The kappa waveform appeared less uniform than that of alpha bursts, and the dominant frequency of kappa activity was lower than that of alpha activity.

Gibbs and Gibbs, (1952: cited by Sewitch, 1978) reported that kappa occurred in 11% of the normal population, whereas Kennedy et al, (1949b) reported a 30% occurrence. Chapman et al, (1962) explain that discrepancies may arise because of the relatively small amount of waking kappa activity time in the average subject and the small difference in increase in kappa from easy to hard tasks.

In our study we tried to provoke frontal kappa activity during the waking mental arithmetic tasks, (by subtracting seven from one thousand consecutively, with eyes shut and then open) but observed kappa in one subject only, (subject C), an incidence of 12% in this group. Kappa activity produced during mental effort with the eyes shut was 8.4Hz, but when the eyes were opened frequency decreased to 7.8Hz. Subject (C) also showed occipital alpha during mental arithmetic (8.4 to 10.2Hz), and during relaxed wakefulness with his eyes shut (8.4Hz), but not open. In the study by Chapman et al, (1962) 76% of subjects showed more kappa in the eyes closed condition, whereas 24% showed no difference.
In addition to frontal 'alpha-like' activity, subject (C) showed a particularly evident peak of frontal theta activity (4 to <8Hz) during all waking conditions, and in sleep stages 2, 3, and 4. During sleep the amplitude of theta activity was greater than frontal 'alpha' (or kappa) but less than occipital alpha.

When subjects are fully absorbed in mental performance tests, a rhythm of frontal mid-line theta may also be induced in the waking EEG. Frontal mid-line theta is defined as a burst of theta waves appearing in the bilateral frontal regions with the highest amplitude in the medial area.

In total, six good sleepers with discomfort (subjects B, C, F, K, M and Q) and five control subjects, (subjects 5, 10, 11, 12 and 14) were found to show theta activity during NREM sleep stages. Subjects (B) and (C), also showed theta during mental arithmetic, and control subject (12) showed some theta in the first period of relaxed wakefulness, but not in subsequent episodes.

As Kilch et al, (1972) defined the theta rhythm between 4 and less than 8Hz, and Hayashi et al, (1987) used a criteria of 6 to 7Hz activity lasting more than one second, in our study we defined theta activity as 4Hz to less than 7Hz.

Hayashi et al, (1987) examined the frontal and central sleep and waking EEGs of fifteen healthy subjects (aged 17 to 21y) in order to investigate the relationship between frontal mid-line theta and mental activity during sleep. Ninety nine subjects (14 to 19y) were awakened from stage 1 sleep or REM sleep during daytime naps, and asked to report their inner experiences before awakening.

Frontal mid-line theta appeared at different frequencies in different stages of sleep and wakefulness, and varied widely between subjects, but was most common firstly in REM sleep and then stage 1 sleep. Frontal mid-line theta was more common in sleep stage 2 than sleep stages 3 or 4, in which it was very rare or absent and it's presence in stage 2 sleep occurred just prior to REM sleep onset or spontaneous arousal.
Frontal mid-line theta occurred more frequently in the sleep of younger subjects who were more likely to show theta activity during waking mental tests.

Results of awakening subjects after sleep with or without theta indicated a close relationship of frontal mid-line theta to dream or sleep mentation during NREM sleep. Although dream or sleep mentation was not always accompanied by frontal mid-line theta in the sleep EEG, frontal mid-line theta was more frequent during REM sleep (in which dreaming and mentation are more common), than any stage of NREM sleep.

As mentioned previously, high alpha producers are more likely to show peaks of activity across the frequency spectrum, (Johnson et al, 1969) and this may explain some of the association between the appearance of 'frontal alpha-like' activity and frontal theta in the present subject groups. However, although subjects (B), (C), (K) and (P) and control subject (14) who showed frontal theta were amongst the high waking alpha group, subject (F), and control subjects (11) and (12) who also showed it, were not. Only three good sleepers (C, K and M) and three controls (10, 11 and 12) showed frontal theta during REM sleep.

The predominance of occipital alpha generators during wakefulness and the observation of frontal 'alpha-like' activity during NREM sleep suggests a change in the functional characteristics of anteriorly produced 'alpha' during sleep. The properties of waking 'kappa' activity and the observation of frontal kappa during NREM sleep, indicate that frontal NREM sleep 'alpha-like' activity may be 'kappa' activity. As waking 'kappa' is associated with mental effort and concentration, it's appearance during NREM sleep may be related to sleep mentation. The coincident appearance of frontal theta, (which is related to dreams and sleep mentation), in some subjects exhibiting frontal kappa supports this hypothesis.
9.6. SLEEP MENTATION AND PSYCHOLOGICAL PROFILE.

If sleep mentation processes underlie the appearance of frontal 'alpha-like' activity or 'kappa' in NREM sleep, then the difference between subjects exhibiting NREM kappa activity, and those not showing it, may be related to the presence of psychological factors which control sleep mentation processes.

NREM sleep mentation reports are generally less elaborate than REM sleep reports, and show a greater incidence of more conceptual thinking. They have less content involving emotional processes especially anxiety, hostility and violence.

Compared to REM sleep reports, NREM sleep reports contain less visual activity and less physical involvement. They tend to make use of more recent events in the subject's life and to represent a continuation of material reported on a previous awakening. They are more likely to be a realistic recreation of some recent event, and are more likely to 'make sense'. REM sleep reports are more unrealistic and subject to processes of displacement and condensation than NREM sleep reports.

Most 'thinking' reports come from NREM sleep, although most NREM sleep reports are of 'dreaming', (a visually hallucinated, dramatic episode). The typical NREM sleep report is less dreamlike than the typical REM sleep report but some NREM reports get very dreamlike indeed, and the time since the previous REM sleep period has some influence on NREM recall. Interestingly, Hauri and Hawkins, (1973) reported that sleep mentation during 'alpha-delta' sleep seems to be more 'dreamlike' than it usually is during NREM sleep.

Individual differences in mentation exist as the dreamlikeness reported differs amongst subjects, (Arkin et al, 1978). Although mentation becomes more intense later in the night, it cannot be explained as only a monotonic increasing function of EEG cortical arousal as reflected in conventional sleep stages, (Tracy and Tracy, 1974: cited by Arkin et al, 1978).
In the present study, three good sleepers with discomfort (subjects M, N, and K) recorded their 'dream' reports on awakening during or following their nocturnal sleep. All the subjects 'dreamt' about current events on one or more occasion, although when subjects experienced events which they found unpleasant or shocking, their 'dream' reports were not necessarily unpleasant. Fourier analysis of sleep on separate occasions found that subject (M) did not exhibit NREM sleep 'kappa', but did show frontal theta during sleep stages 2, 3, 4 and REM, and subject (K) showed both 'kappa' and theta during stage 4 and REM sleep. Subject (N) did not undergo a sleep recording using the frontal-occipital montage. Mean percentages of alpha from the central EEGs of subject (M) and (K) showed that these subjects were amongst the lowest NREM and REM sleep alpha producers of the good sleepers with discomfort.

The sleep mentation study would have benefitted from a larger number of subjects taken from each of the groups, and recordings of the sleep EEG prior to controlled awakenings from NREM and REM sleep. As it stands it is difficult to conclude whether subjects were reporting REM or NREM sleep mentation content.

The apparent psychological profiles of the subjects in the good sleep with discomfort group suggest that these subjects may be more likely to ruminate over current events and problems in their lives, and this may continue into sleep as NREM and REM sleep mentation. Subject (C) described being 'unable to get things out of my mind', and one of the most frequent reasons given by both the discomfort groups and control subjects to describe a poor night's sleep was 'I was aware of thinking all night'. Subject (K) reported some discomfort and difficulty getting up the morning after he had been on call during the night, despite being undisturbed. This could suggest that his awareness of being 'on call' resulted in him being unable to 'switch off' his mind should he have been required to act in an emergency during the night.
Crisp, (1986) suggests that similar mechanisms may operate in psychiatric illness and severe depression. If a subject's mind is active when he retires, he may find that it remains so for the first hour or two after retiring, and he may then awaken during the night and also early in the morning with the same thought association. People with severe depression awake to find they are overwhelmed with gloom and despair, because they have no solutions to problems surrounding them and from which they believe they were protected whilst asleep.

It is possible that sleep mentation, and frontal 'kappa' may be more commonly found in specific personalities such as the 'introversive' type described by Jung. An 'introversive' type tends to look within himself for his main sources of stimulation, interest and evaluation, and may rely primarily on thinking and intellectual processes for evaluating his own inner experience, (London, 1975).

Smythe, (1979) described fibrositic patients as 'demanding of themselves and others. They can be trying, but are often effective at work because of their dedication'. 'Their perfectionist demands make them dissatisfied with themselves and others, but contribute to their superior productivity'.

Other studies of fibrositic patients have suggested that these individuals are psychologically disturbed, and suffer from a disorder in which psychologic factors contribute significantly to their physical symptoms, (Payne et al, 1982). In a study of eighteen female fibrositics, Marks et al, (1983) reported that these subjects showed a psychological profile similar to somatizing and pain prone patients, and suggested that there was a convergence of physiologic and psychologic influences in production of the fibrositis syndrome.

Smythe and Moldofsky, (1975) reported that stressful experiences may trigger a perpetuating cycle of anxiety and depression, non-restorative sleep and musculoskeletal symptoms seen in 'Fibrositis' patients.
Goldenberg, (1986) described a study in which 71% of fibrositics were found to have a history of major depression, whereas 25% of patients were depressed at the onset of their symptoms, indicating some common psychobiologic association, but not necessarily a causal relationship.

Beecham (1979) outlined the importance of differentiating the aching and stiffness of the 'Fibrositis syndrome' from the aching of the anxious or depressed patient, (psychogenic rheumatism). Patients with psychogenic rheumatism were described as appearing 'tense, hostile, nervous, defensive or depressed' whereas the attitude of the fibrositic was more appropriate to the situation.

All of the discomfort subjects in the present study described a tendency to periodic anxiety and depression, and eleven of these reported a previous or current stressful experience such as separation, divorce, a past illness, and difficulties at work. In comparison, only seven of the control subjects described anxiety due to recent stressful experiences, and five of these subjects had also experienced a marital separation or divorce. Despite the greater incidence of reported depressive experiences in the discomfort subjects, none were clinically diagnosed as depressed, and mean REM sleep latency showed no significant differences between the three groups.

It is assumed that stress can act as a catalyst for depression in certain cognitively predisposed people. As a result of the continuous interaction between stress and specific cognitive processes, a person may develop a certain set of beliefs that he/she cannot cope. Individuals view stressors differently and a 'depressogenic stressor' for one person may not be so for another.

Beck, (1967: cited by Shaw, 1982) suggested that a negative view of oneself, one's experiences, and the future, was critical to the development of depression. This assumes that the predisposed person processes information about stressors under the influence of a factor called the 'self'. Beck's cognitive model assumes that the predisposed person may believe that he/she is inferior in some attribute, despite the fact that he/she is above the norm.
Beck, (1967; 1976; cited by Shaw, 1982) described three types of events which provoke enough stress to precipitate depression; situations that lower a person's self esteem, (for example failing an examination, being jilted by a lover); situations that involve a thwarting of goals or posing an insoluble dilemma, and physical disease or abnormality that activates ideas of personal deterioration or death.

Once a person has experienced depression, there is a high probability of recurrence within the next ten to twenty years, coupled with which is the increase in severity and chronicity of subsequent depressive episodes, (Shaw, 1982).

Processes of self-regulation, or 'coping' are normal features of stressful transactions, and are the key intervening processes in the causation and prevention of stress-related somatic disorders. In all likelihood, some will work, while others will not. A wide variety of coping processes are employed, depending on personal characteristics, environmental demands and contingencies, and the way our transaction with the environment is appraised. People who do not have a wide range of coping styles may be prone to psychological disorders including depression. In addition, stress emotions vary depending on the situation as well as personality characteristics, thus in the face of a heavy environmental demand, some individuals may experience, for example, mainly a sense of helplessness, anxiety and depression, while others might react with anger or some other emotional state, (Lazarus, 1977).

In addition to a possible psychological predisposition to depression, the 'coping' process may be one of the factors which differentiates the discomfort subjects from the control subjects in the present study. Whereas control subjects may be more outwardly demonstrative of their feelings and are able to cope with stressful events, discomfort subjects may be more likely to perceive the problem in relation to their 'selves', and in an attempt to cope, they conceal their anxieties, quietly 'ruminating' or 'brooding' alone. This characteristic may then affect their interpersonal relationships.
Under this hypothesis, NREM sleep mentation, (associated with EEG kappa activity) may be a characteristic of subjects who are psychologically or circumstantially predisposed to anxiety and depression or 'sadness', and who suppress their emotions. Control subjects who do not show the depression prone personality might still exhibit similar 'ruminating' tendencies when they are faced with a particularly personal stress such as the break up of a relationship.

It is interesting to find that the four control subjects who exhibited NREM sleep kappa activity, (control subjects 11, 12, 14 and 15), had all experienced a divorce or separation within the last five years, and especially in the case of control subjects (14) and (15), who had recently separated from their spouses, it is likely that they may still have been disturbed about the experience. These control subjects had mean NREM alpha percentages of 2.47 (±1.2), and 3.6 (±2.6) respectively. The control group mean of percentage NREM alpha was 2.0 (±1.1), indicating that these two control subjects were amongst the higher NREM sleep alpha producers of the control group. Control subjects (11) and (12) had smaller mean percentages of NREM sleep alpha than the whole group.

9.7. CHARACTERISTICS OF ALPHA ACTIVITY FOUND ON THE CENTRAL SLEEP EEG.

Webb, (1986) reported that the Cz or C3 electrode montage sites yield the maximal information about alpha, slow waves, sleep spindles and sleep onset. Mean percentages of these variables were obtained from A2 to C4 EEG recordings in the present study, but frontal kappa activity was observed from fourier analysis of the frontal EEG, hence the frontal 'alpha' activity may be independent of the alpha activity found centrally.

Discomfort and good sleep subjects (B), (C), (F), (K), (L) and (P) showed frontal kappa, whereas subjects (D) and (M) did not. Mean percentage of NREM sleep alpha for the whole group of good sleepers with discomfort was 2.0, (±2.3). Mean NREM percentages of alpha for subjects who
showed 'kappa exceeded the group mean except for subject (K) who showed very low percentages of central alpha. Subjects (D) and (M) showed less than half the group mean percentage of alpha in NREM sleep. There was no apparent relationship between reported severity of discomfort symptoms and the appearance of frontal kappa activity.

The results suggest that in most but not all cases, the incidence of frontal kappa is related to a higher percentage of NREM alpha on the central EEG. If the two are related, then the amount of 'alpha' activity detected frontally and centrally might also show some relationship. A direct comparison of the amplitude of these frequencies between subjects might clarify this but this was not possible in the present study.

The sub-study of daily tea and coffee intake suggested that caffeine consumption might be a contributory factor in determining central EEG NREM sleep alpha activity. Although only four good sleepers with discomfort were studied, (C, N, M and K), higher total mean daily intakes of tea plus coffee, but particularly coffee, were associated with higher mean percentages of NREM sleep alpha (although mean NREM sleep alpha percentage was obtained from recordings on other nights). This might be explained as a direct effect of caffeine on the brain mechanisms producing NREM sleep alpha, or that subjects with higher mean NREM alpha also have a tendency to consume more caffeinated drinks as a result of their personalities and chosen lifestyles, or the results may be spurious due to the small number of subjects. The study should also have accounted for drinks such as cola which also contain caffeine, although subjects reported that they had not drunk these during the period of the study.

Caffeine and other stimulants produce a 'stimulant' effect on the EEG, with some reduction in waking alpha activity, and an increase in low voltage fast activity, (Kiloh, 1972). Consequently, unless alpha activity shows different characteristics during sleep, it is unlikely that caffeine directly increases NREM sleep alpha, although it may contribute to reported sleep problems.
With sustained use of stimulants, taken in excess and too late in the day, there is poor nocturnal sleep, which may increase the tendency to take the stimulant during the day so as to maintain alertness. Ultimately the person becomes susceptible to sudden episodes of daytime sleepiness, and other symptoms which include anxiety, irritability, personality changes, and severe depression with suicidal potential, (Dement and Guilleminault, 1979).

If the percentage of alpha in NREM sleep stages is readily affected by external variables, then one might expect the percentage of alpha to vary over time. Scheuler, (1987) reported that in subjects who have been sleep recorded over several years, the alpha-sleep pattern demonstrates a remarkable stability, and presents as an individually constant characteristic.

The present study found that for the good sleepers with discomfort, there was a statistically significant positive relationship between percentage alpha over NREM sleep and REM sleep. Control subjects showed a similar association between stage 3 sleep alpha and stage 4 sleep alpha, but did not demonstrate the same significant relationship between mean NREM and REM sleep alpha. These results suggest that percentage alpha in NREM sleep stages and REM sleep of the good sleepers with discomfort is maintained at a level specific to each subject. For the control subjects, only sleep stages 3 and 4 were affected by a general level of alpha activity. Alpha-delta indices for sleep stages 3 and 4 also showed a significant positive correlation, which was highest for the good sleepers with discomfort.

There was no apparent relationship between age and mean percentages of alpha in any group. (Poor sleepers with discomfort showed a non-significant correlation of 0.7, based on only four subjects). The three oldest individuals in the study were subjects (N), (51y); (G), (49y); and (M), (43y), all other subjects were forty and under. Subject (N) showed the highest mean NREM alpha percentage in the discomfort groups, (9.4%), and subject (G) also showed a high NREM alpha, (5.7%), but subject (M)'s was particularly low, (0.9%). It is unlikely that alpha in NREM sleep is related to the effects of ageing processes.
There was very little difference in the inter-recording variation of percentage alpha between the good sleepers with discomfort, and the control subjects although the control subjects showed slightly more change.

Subject (C) was studied three times over sixteen months, and showed little variation in percentages of alpha in sleep stages 2, 3, 4 and REM. The most variation was seen in sleep stage 3. Subject (P) who was studied over five week nights also showed very little variation from her mean percentages of alpha, and also showed the most change in sleep stage 3 alpha, although there was less variation compared to subject (C).

In relation to the mean percentages of NREM alpha for each night recorded, pre and post-sleep questionnaires indicated that subject (P) experienced discomfort on only one morning, which was associated with an overnight deterioration in her SSSS score and the highest percentage of NREM alpha seen during the week. However, individual percentages of alpha in sleep stage 3 and REM sleep were the lowest of the week. This could suggest that NREM sleep alpha is related to morning symptoms, but a long term study is required to confirm this finding.

Moldofsky and Lue, (1980) reported that NREM sleep alpha power and time, and REM sleep alpha correlated with overnight increases in subjective pain and decreased energy in fibrositics, but changes in mood were correlated with NREM sleep alpha only. This could suggest that the general level of alpha activity during sleep is related to the mechanisms underlying subjective somatic symptoms, rather than NREM sleep alpha which may be related to mood. In the present study there was a significant positive correlation between NREM sleep alpha and REM sleep alpha for the good sleepers with discomfort, who also showed greater mean percentages of NREM sleep alpha than control subjects.

In the present study subjective mood ratings prior to sleep recordings indicated that a larger percentage of the good sleepers with discomfort and the poor sleepers with discomfort felt 'tired and relaxed' during the preceding day compared to the control subjects, but the majority of each group had been 'alert and contented' or 'active and
cheerful'. Unfortunately the phrases employed for describing mood pre-sleep were ambiguous and post-sleep ratings concentrated on sleepiness, difficulty getting up and discomfort symptoms.

However, questionnaire data revealed that a larger proportion of subjects with discomfort felt 'uneasy' or 'tense' and drowsy in the morning, compared to the controls who generally felt 'contented' or 'calm' and alert. Also, once subjects had got up, most controls felt lively/activated, whereas good sleepers with discomfort felt drowsy/tired, and poor sleepers with discomfort were idle/sluggish. As the two discomfort groups also showed higher mean percentages of NREM sleep alpha, mechanisms underlying NREM sleep alpha may be associated with those controlling mood, and therefore subjective experiences.

9.8. THE RELATIONSHIP OF NREM SLEEP ALPHA TO MUSCULOSKELETAL DISCOMFORT SYMPTOMS.

The exact relationship of the alpha sleep anomaly to morning symptoms of musculoskeletal discomfort remains unclear. The appearance in some control subjects of both frontal kappa and percentages of central alpha comparative to those of the discomfort groups indicates that alpha and/or kappa and musculoskeletal discomfort are not causally related. It is more likely that they are characteristics of the same underlying factor.

The 'alpha sleep anomaly' has been assumed to represent a sleep disturbance which interferes with the restorative role of SWS and is associated with symptoms of morning muscular discomfort, lethargy and mood changes, (Smythe and Moldofsky, 1977, 1978). Under this hypothesis, other sleep variables associated with the restorative role of SWS might be affected by the presence of NREM alpha.

Although the underlying neurophysiological mechanisms and the clinical and behavioural significance of spindle and delta activity are unknown, a study of spindle activity in cats suggested that spindle activity might preserve sleep continuity. Spindle density was highest among older cats who showed a significantly greater frequency of transient
arousals during sleep. Furthermore, as the incidence of transient arousals and low amplitude spindle activity was highly correlated in the young cats, but not aged cats, the authors suggested that spindle enhancement acts to preserve sleep in 'over aroused' animals, (Bowersox, Kaitin and Dement, 1985).

However, a later study of human sleep by Guazelli et al, (1986) found that the abundance, amplitude and duration of spindles were reduced in the elderly, indicating that age affects the temporal pattern and quantity of spindles. They also suggested that as the amount of waking in the elderly was not inversely correlated with spindle abundance, spindle abundance does not reflect the integrity of the systems that maintain the brain in NREM sleep.

In man, the benzodiazepine hypnotics are known to increase spindles, but decrease delta activity during NREM sleep. Johnson et al, (1983) indicate that of the two EEG measures, spindle rate appears to be the most sensitive to the benzodiazepine effect. This suggests that hypnotic induced spindle activity in man may preserve the sleep process.

In the present study there is little evidence to suggest that the quantity of delta or spindle activity is affected by the presence of alpha activity in the controls or good sleepers with discomfort, although in the poor sleepers with discomfort, reduced spindle activity was associated with a high mean percentage of alpha. These observations may be a function of the older mean age of the group, or of fragmented sleep due to movement and wakefulness, in addition to the alpha producing mechanisms. Unfortunately results from the poor sleepers with discomfort suffer from a profound lack of subjects.

Despite previous reports of the alpha sleep anomaly relating to morning symptoms in fibrositis (Moldofsky et al, 1975; Smythe and Moldofsky, 1977, 1978) and also rheumatoid arthritis, (Moldofsky, Lue and Smythe, 1983), in the present study, we can conclude that NREM sleep 'alpha' activity alone is not directly related to the appearance of musculoskeletal and other symptoms, as it may present in symptom free control subjects. Mechanisms producing frontal
'kappa' activity and alpha activity during sleep may have a psychological origin, which is more frequently found in the discomfort groups, but which may also occur in control subjects during periods of anxiety or stress.

The somatic symptoms associated with fibrositis have commonly been suggested to be of psychoneurotic origin, due to the difficulties encountered in finding an organic basis for the syndrome, and the typical personality profile of these patients. Payne et al, (1982) found that compared to rheumatoid arthritics, fibrositics scored higher on neurotic scales of hypochondriasis and hysteria, and on psychotic scales of paranoia and schizophrenia. It was suggested that fibrositics probably do not resemble each other to an unusual degree, except in their physical complaints and the fact that they are psychologically disturbed.

Modest improvement in fibrositic symptoms has been noted with use of tri-cyclic anti-depressants, in addition to reduction of stress, and rest and relaxation, (Wolfe, 1986). However, the doses of tri-cyclic anti-depressants found useful in Fibrositis were much lower and the response was much quicker than in patients with depression, (Goldenberg, 1986).

Monroe, (1967) reported that poor sleepers were more likely to subscribe to symptomatic complaints on the Minnesota Multi Phasic Personality Inventory and the Cornell Medical Index than were good sleepers who were more guarded. Adam, Tomeny and Oswald, (1986) found that poor sleepers were significantly more neurotic, had a higher level of trait anxiety and higher levels of obsessionality and somatic complaints, but differences in the MMPI were nonsignificant.

Hypochondriasis and functional somatic symptoms are different phenomena, although the distinction is not always clearly made. Hypochondriasis includes an unrealistic fear or belief of having a disease persisting despite medical reassurance and which causes impairment in social or occupational functioning. Functional somatic symptoms are somatic symptoms not caused by disease detectable by physical examination or routine laboratory investigations. These symptoms occur in normal persons, are common in
psychiatric patients and are not limited to patients with somatoform disorders. Psychosomatic diseases are physical diseases in which emotions can act as precipitating or aggravating factors, such as bronchial asthma, peptic ulcer and ulcerative colitis. Unlike functional symptoms, psychosomatic diseases can be life-threatening and usually require medical and sometimes surgical treatment, (Kellner, 1985).

It would be presumptuous and wrong to label the discomfort subjects in the present study as 'psychologically disturbed', or 'hypochondriacal' as these subjects have merely presented with a history of stressful events and anxiety in addition to musculoskeletal discomfort and daytime lethargy.

If the right questions were asked, approximately 30% of the population would report musculoskeletal symptoms, but further study is required to ascertain why some patients with musculoskeletal symptoms refuse to become patients whilst others do not. It may be that these patients are saying that they had this discomfort before but now there is something else in their life which is 'intolerable' and they cannot 'process' it successfully, (Hadler, 1986). At times of distress a person is less able to cope with somatic symptoms than at other times, or somatic symptoms may appear as more threatening, (Kellner, 1985).

Somatic symptoms are extremely common, but tend to be more prevalent in patients who are anxious. The correlation between self-rated anxiety and somatic symptoms is somewhat higher than between depression and somatic symptoms. Numerous theories have attempted to explain the transformation of emotions into somatic symptoms, and there are several known mechanisms by which these symptoms can be produced in the absence of disease or tissue trauma, for example overactivity of the autonomic nervous system in association with stress, (eg. irritable bowel syndrome) and increased tension in voluntary muscle, (eg. tension headaches).
In morbidly anxious patients complaining primarily of somatic symptoms, there is a high correlation of symptoms with physiologic changes, suggesting that the majority of somatic symptoms coexist with emotions, are consequences of emotions and can reinforce emotions. Subjects tend to have a fairly constant pattern of physiologic response to stress, (response stereotypy) and the same somatic symptoms tend to recur in some patients, but there are large differences between subjects in the ratios of emotional to somatic symptoms, (Kellner, 1985).

Musculoskeletal discomfort may therefore be partly attributed to psychological factors which affect a subject's perception of stressful experiences.

Although the alpha sleep phenomenon is not directly related to discomfort symptoms, other sleep characteristics may be contributory. The preliminary questionnaire survey showed that sleep problems were more frequently reported in poor sleepers and subjects with discomfort. Sleep complaints described by these groups, such as difficulties getting to sleep, frequent nighttime awakenings and premature morning arousal often arise as a result of psychological stresses and anxiety, and give rise to symptoms of daytime fatigue and malaise possibly by interrupting the restorative role of SWS. Similar symptoms to those described by the discomfort subjects are frequently reported during sleep deprivation, (Gulevitch, Dement and Johnson, 1966) and loss of stage 4 sleep, (Webb and Williams, 1967).

It is also interesting to note the predominance of 'evening types' in the discomfort subjects (who completed the main sleep study questionnaire), compared to the control group, of which only one described himself as 'more evening than morning type'. Subjects with discomfort and daytime lethargy could either consider themselves to be more 'evening type' than morning type as a result of their subjective feelings in the morning, or their 'evening type' might contribute to some of their experiences. For example, if subjects are more likely to retire to bed late but have to get up early, they are more likely to be tired during the day.
The next part of the thesis describes a small study of eight young, physically fit and discomfort free subjects who presented with subjective ratings of good quality sleep. These subjects were studied to investigate the incidence and consistency of NREM sleep alpha in normal healthy individuals.
10. A STUDY OF THE INCIDENCE AND CONSISTENCY OF NREM SLEEP ALPHA IN SYMPTOM-FREE GOOD SLEEPERS.

10.1. PRELIMINARY SLEEP QUESTIONNAIRES.

10.1.1. METHOD.

Posters were placed around the University departments requesting good sleepers to volunteer for home sleep recordings for which a small payment was offered.

Volunteers completed the 'Survey on Sleep' questionnaire, which was then examined for evidence of poor sleep, musculoskeletal and somatic symptoms. Where these were found, subjects were interviewed and asked to complete the 'Survey on Muscular Discomfort' questionnaire which was used to ascertain the severity and regularity of the symptoms. Subjects were then assigned to either the symptom-free group or the discomfort group of the first part of the study.

10.1.2. RESULTS.

Sleep questionnaires were examined by the experimenter and analysed using the BBC programs (appendix III). A summary of each individual's sleep characteristics is given in the subject profiles found in appendix (VIII). Other data from the preliminary questionnaires is given in the tables found in the results section.

Eighteen volunteers responded to the advertisements placed around the University. After completing the preliminary questionnaire, suitable subjects were asked if they would undergo home sleep recordings. Eight subjects who fulfilled the requirements of the study underwent more than one successful recording and the results of their recordings were used for analysis.

Ten subjects were not used for this study due to circumstances similar to those described for the main study.
SUBJECT PROFILES.

Details of the eight subjects completing the study were obtained from interview and from the preliminary and study questionnaires.

This group of eight subjects had a mean age of 21.1y (±1.45y) and consisted of six males and two females, all of normal weight and height.

Six subjects were approaching their final year degree examinations at the time of study, and two were coming up to their first year examinations. All data was collected in the spring term before examinations began. Two subjects (IM and SB) were also studied weekly for a month as part of a consistency study.

Profiles of each subject taken from personal interviews and preliminary questionnaire analysis can be found in appendix (VIII). A summary is given below.

SUMMARY OF SUBJECT PROFILES.

All subjects reported regular good quality sleep, and were physically fit and partook in sporting activities.

All were single, non-smokers, and free of sleep medication. One subject was prescribed inhalants for asthma, (SB).

Each subject reported good health, although four subjects occasionally suffered with asthma and hay fever. Other occasional health problems included headaches, eczema and allergies. Subject (JP) reported a past history of anxiety, depression, and breathing problems which had all resolved in the past year.

Three subjects (ML, SP, and RN) described occasional leg or back stiffness which was solely attributed to muscular exertion during exercise. Two subjects (ML and JP) occasionally awoke feeling lethargic and felt tired during the day, but this was thought to be a result of insufficient sleep (ML), or a morning characteristic of being an 'evening-type', (JP). Other subjects (IM, SP, and SB) described feeling tired on awakening.
Although subjects varied in their reported sleep latencies, (from one minute to an hour) only one subject reported lying awake worrying if he had family problems, (RN). None of the subjects reported any stress or current anxieties.

Two male subjects (ML and JP) described experiencing a great need to move their legs in bed, in addition to which subject (ML) reported teeth grinding, and subject (JP) reported regular snoring.

All subjects slept well for between seven and eight and a half hours per night, some awakening during the night as a result of noise or important events the next day, but most sleeping without interruptions.

10.2. SLEEP EEG STUDY.

10.2.1. PROCEDURE.

Subjects agreeing to further participation completed the sleep study questionnaire and underwent the same procedure as that described for the main study. Two of the eight subjects (SB and RN) failed to return their study questionnaires before leaving for their vacations. A summary of results for the six subjects is given below.

10.2.2. SUMMARY OF RESULTS OF SLEEP STUDY QUESTIONNAIRE.

Prior to sleep, three subjects felt 'drowsy', 'sluggish' or 'sleepy' but two felt 'stimulated' and one felt 'active'. This could imply that the three 'drowsy' subjects were more likely to be 'morning' types but four of the subjects felt that they were 'evening' types and only one felt he was a 'morning' type.

All six subjects said that they enjoyed their sleep, although they all reported awakening at least once or twice, but took less than ten minutes to fall asleep again. Four said that they experienced difficulty staying awake several times a month and one said he had this difficulty once a month or less. Half the group felt that their sleep quality
did not vary at all, but two felt it varied slightly, and one subject said his varied very much.

On awakening in the morning, four subjects felt either clear headed or alert, but two still felt moderately drowsy. Four were very or fairly refreshed and two felt fairly tired. Five subjects described agreeable feelings of being 'calm', 'peaceful' or 'contented' on awakening, but one felt 'uneasy'. Four subjects felt activated or aroused, and two felt drowsy.

Most likely causes of a poor night's sleep were, taking a long time to fall asleep, waking up a great deal and being aware of thinking all night, but the most important were feeling tired or dizzy on awakening.

Five of the subjects said that a poor night's sleep affected them the next day, and one subject felt the consequences both the next day and the day after. These effects were experienced in the morning by one subject, but five subjects felt them in the afternoon (3 subjects) or the evening (2 subjects). None of the subjects felt that a poor night's sleep affected their efficiency alone, but three felt it affected how they felt, and three felt it affected both how they felt and their efficiency. Three subjects expressed some concern about losing a nights sleep.

Three subjects (JD, ML and SP) said that current events were causing them concern, but none of the others had been concerned about any particular events in the last three months. The four subjects who reported lying awake worrying did so only once a month or less, and only two subjects said that their dreams occasionally caused them concern.
10.3. RESULTS OF STANDARD SLEEP RECORDINGS.

Eight subjects underwent sleep recordings using the standard sleep EEG montage. A full description of subject preparation and sleep recording procedures can be found in the main study method.

STANDARD RECORDINGS: ANALYSIS OF RESULTS.

Sleep recordings were analysed as described in the main study analysis of results section.

10.3.1. RESULTS OF SLEEP EEG ANALYSIS.

A minimum of two recordings was obtained from each subject in this study. The mean for each subject was obtained for all sleep variables, and the group mean calculated. Only good recordings were analysed, if a particularly poor night's sleep was recorded (as a result of the recording equipment) then this was discarded and other recordings collected.

The following tables give the mean group values of sleep variables obtained from sleep staging with the Oxford Sleep Stager.

Table (LVI) shows the group mean percentages of sleep stages 1, 2, 3, 4, SWS and REM.

TABLE LVI.

<table>
<thead>
<tr>
<th>Stage</th>
<th>MEAN %</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1</td>
<td>15.3</td>
<td>4.3</td>
</tr>
<tr>
<td>Stage 2</td>
<td>48.7</td>
<td>4.9</td>
</tr>
<tr>
<td>Stage 3</td>
<td>10.4</td>
<td>2.9</td>
</tr>
<tr>
<td>Stage 4</td>
<td>3.9</td>
<td>4.2</td>
</tr>
<tr>
<td>SWS</td>
<td>14.5</td>
<td>6.9</td>
</tr>
<tr>
<td>REM Sleep</td>
<td>20.2</td>
<td>5.8</td>
</tr>
</tbody>
</table>
Table (LVII) gives the group mean percentages of alpha per epoch of sleep stages 2, 3, 4, mean NREM sleep and REM sleep, the mean percentages of delta per epoch and the mean percentage of epochs containing spindles in sleep stages 2, 3, and 4.

**TABLE LVII.**

**MEAN PERCENTAGES OF ALPHA AND DELTA PER EPOCH AND PERCENTAGE OF EPOCHS CONTAINING SPINDLES IN NREM SLEEP STAGES.**

<table>
<thead>
<tr>
<th></th>
<th>Mean % Alpha</th>
<th>Mean % Delta</th>
<th>Mean % Epochs with Spindles</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stage 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>0.9</td>
<td>3.5</td>
<td>7.5</td>
</tr>
<tr>
<td>s.d</td>
<td>(1.0)</td>
<td>(1.5)</td>
<td>(3.1)</td>
</tr>
<tr>
<td><strong>Stage 3</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>1.1</td>
<td>30.0</td>
<td>2.0</td>
</tr>
<tr>
<td>s.d</td>
<td>(1.2)</td>
<td>(1.7)</td>
<td>(1.7)</td>
</tr>
<tr>
<td><strong>Stage 4</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>0.7</td>
<td>55.2</td>
<td>0.2</td>
</tr>
<tr>
<td>s.d</td>
<td>(0.5)</td>
<td>(6.4)</td>
<td>(0.2)</td>
</tr>
<tr>
<td><strong>NREM Sleep</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>0.8</td>
<td>15.1</td>
<td>5.6</td>
</tr>
<tr>
<td>s.d</td>
<td>(0.7)</td>
<td>(9.2)</td>
<td>(1.8)</td>
</tr>
<tr>
<td><strong>REM Sleep</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>0.8</td>
<td>$</td>
<td>$</td>
</tr>
<tr>
<td>s.d</td>
<td>(0.8)</td>
<td>$</td>
<td>$</td>
</tr>
</tbody>
</table>
Table (LVIII) shows the mean group alpha-delta and spindle-delta indices for sleep stages 2, 3, and 4.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Mean</th>
<th>s.d.</th>
<th>Mean</th>
<th>s.d.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>26.0</td>
<td>(13.8)</td>
<td>218.9</td>
<td>(59.9)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>3.6</td>
<td>(3.6)</td>
<td>6.6</td>
<td>(5.6)</td>
</tr>
<tr>
<td>Stage 4</td>
<td>1.3</td>
<td>(0.9)</td>
<td>0.4</td>
<td>(0.4)</td>
</tr>
</tbody>
</table>
Table (LIX) gives the group means of REM sleep latency, sleep efficiency, movement, sleep onset to stage 2 sleep, wakefulness and actual sleep time.

**TABLE LIX.**

**GROUP MEANS FOR REM SLEEP LATENCY, SLEEP EFFICIENCY, MOVEMENT, ONSET TO STAGE 2, WAKEFULNESS AFTER SLEEP ONSET, AND ACTUAL SLEEP TIME.**

<table>
<thead>
<tr>
<th></th>
<th>MEAN</th>
<th>S.D</th>
</tr>
</thead>
<tbody>
<tr>
<td>REM Latency (min)</td>
<td>106.1</td>
<td>(58.4)</td>
</tr>
<tr>
<td>Sleep Efficiency (%)</td>
<td>91.2</td>
<td>(3.6)</td>
</tr>
<tr>
<td>Movement (min)</td>
<td>11.7</td>
<td>(2.9)</td>
</tr>
<tr>
<td>Sleep Onset to Stage 2</td>
<td>12.6</td>
<td>(4.2)</td>
</tr>
<tr>
<td>WASO (&gt; 120 sec)</td>
<td>14.6</td>
<td>(10.2)</td>
</tr>
<tr>
<td>WASO (&lt; 120 sec)</td>
<td>9.2</td>
<td>(5.8)</td>
</tr>
<tr>
<td>Actual Sleep Time</td>
<td>435.2</td>
<td>(29.8)</td>
</tr>
</tbody>
</table>

WASO=Wakefulness after sleep onset (minutes)
10.3.2. RESULTS OF CORRELATIONS.

Pearson's product moment correlations which were performed on sleep variables gave the results shown in the following tables.

Table (LX) shows the results of correlations between mean percentages of sleep stages 1, 2, 3, 4 and REM sleep for the eight subjects.

TABLE LX.

CORRELATIONS OF PERCENTAGE SLEEP STAGES 1, 2, 3, 4 AND REM.

<table>
<thead>
<tr>
<th>SLEEP STAGE</th>
<th>Stage 2</th>
<th>Stage 3</th>
<th>Stage 4</th>
<th>REM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1</td>
<td>0.24</td>
<td>-0.84 (a)</td>
<td>-0.70 (c)</td>
<td>0.01</td>
</tr>
<tr>
<td>Stage 2</td>
<td>-*-</td>
<td>-0.45</td>
<td>-0.29</td>
<td>-0.38</td>
</tr>
<tr>
<td>Stage 3</td>
<td>-*-</td>
<td></td>
<td>0.71 (b)</td>
<td>-0.14</td>
</tr>
<tr>
<td>Stage 4</td>
<td></td>
<td></td>
<td></td>
<td>-*-</td>
</tr>
</tbody>
</table>

(a) Significant (p<0.01)
(b) Significant (p<0.025)
(c) Significant (p<0.05)

Table (LXI) shows the inter-correlations of percentage mean alpha per epoch of sleep stages 2, 3, and 4.

TABLE LXI.

CORRELATIONS BETWEEN PERCENTAGE MEAN ALPHA PER EPOCH IN SLEEP STAGES 2, 3, 4 AND REM.

<table>
<thead>
<tr>
<th>MEAN % ALPHA PER EPOCH</th>
<th>Stage 3</th>
<th>Stage 4</th>
<th>REM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>0.96 (a)</td>
<td>0.66</td>
<td>0.84 (a)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>-*-</td>
<td>0.77 (b)</td>
<td>0.75 (b)</td>
</tr>
<tr>
<td>Stage 4</td>
<td>-*-</td>
<td>-*-</td>
<td>0.54</td>
</tr>
</tbody>
</table>

(a) Significant (p<0.002).
(b) Significant (p<0.05).

Table (LXII) shows the inter-correlations of mean percentage delta per epoch of sleep stages 2, 3, and 4.
### TABLE LXII.

**CORRELATIONS BETWEEN PERCENTAGE OF DELTA PER EPOCH IN SLEEP STAGES 2, 3, AND 4.**

<table>
<thead>
<tr>
<th></th>
<th>Stage 3</th>
<th>Stage 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>0.56</td>
<td>0.75 (a)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>-</td>
<td>0.63</td>
</tr>
</tbody>
</table>

(a) Significant (p<0.05)

Table (LXIII) gives the correlations of mean percentage of epochs containing spindles in sleep stages 2, 3, and 4. None of these correlations were statistically significant.

### TABLE LXIII.

**CORRELATIONS BETWEEN PERCENTAGE OF EPOCHS CONTAINING SPINDLES IN SLEEP STAGES 2, 3, AND 4.**

<table>
<thead>
<tr>
<th></th>
<th>Stage 3</th>
<th>Stage 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>0.50</td>
<td>0.61</td>
</tr>
<tr>
<td>Stage 3</td>
<td>-</td>
<td>0.45</td>
</tr>
</tbody>
</table>
Table (LXIV) shows the correlations between mean percentage alpha per epoch of sleep stages 2, 3, and 4, mean percentage delta per epoch of sleep stages 2, 3, and 4, and mean percentage of epochs containing spindles in sleep stages 2, 3, and 4.

**TABLE LXIV.**
CORRELATIONS OF MEAN PERCENTAGE ALPHA PER EPOCH WITH DELTA AND SPINDLES FOR SLEEP STAGES 2, 3, 4, AND REM.

<table>
<thead>
<tr>
<th>MEAN % ALPHA PER EPOCH OF STAGE</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>REM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean % Delta</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 2</td>
<td>0.82 (b)</td>
<td>0.87 (a)</td>
<td>0.70</td>
<td>0.69</td>
</tr>
<tr>
<td>Stage 3</td>
<td>0.56</td>
<td>0.57</td>
<td>0.41</td>
<td>0.59</td>
</tr>
<tr>
<td>Stage 4</td>
<td>0.60</td>
<td>0.57</td>
<td>0.24</td>
<td>0.48</td>
</tr>
<tr>
<td>Mean % Epochs/Spindles</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 2</td>
<td>0.50</td>
<td>0.60</td>
<td>0.55</td>
<td>0.42</td>
</tr>
<tr>
<td>Stage 3</td>
<td>0.74 (c)</td>
<td>0.73 (c)</td>
<td>0.37</td>
<td>0.64</td>
</tr>
<tr>
<td>Stage 4</td>
<td>0.58</td>
<td>0.69</td>
<td>0.66</td>
<td>0.63</td>
</tr>
</tbody>
</table>

(a) Significant \(p<0.002\)
(b) Significant \(p<0.02\)
(c) Significant \(p<0.05\)
Table (LXV) gives the correlations of mean percentage delta per epoch of sleep stages 2, 3, and 4 with mean percentage of epochs containing spindles in sleep stages 2, 3, and 4.

**TABLE LXV.**

**CORRELATIONS OF MEAN PERCENTAGE DELTA PER EPOCH WITH SPINDLES FOR SLEEP STAGES 2, 3, AND 4.**

<table>
<thead>
<tr>
<th>Mean % Epochs/Spindles</th>
<th>Stage 2</th>
<th>Stage 3</th>
<th>Stage 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>0.80 (b)</td>
<td>0.50</td>
<td>0.52</td>
</tr>
<tr>
<td>Stage 3</td>
<td>0.88 (a)</td>
<td>0.58</td>
<td>0.84 (b)</td>
</tr>
<tr>
<td>Stage 4</td>
<td>0.65</td>
<td>0.49</td>
<td>0.12</td>
</tr>
</tbody>
</table>

(a) Significant (p<0.002)
(b) Significant (p<0.02)

Table (LXVI) shows correlations of percentage stage 1 sleep and percentage sleep efficiency with mean percentages of alpha and delta per epoch and mean percentage epochs containing spindles for sleep stages 2, 3, and 4.

**TABLE LXVI.**

**CORRELATIONS OF EPOCH ALPHA, DELTA AND SPINDLES WITH PERCENTAGE SLEEP STAGE 1 AND SLEEP EFFICIENCY.**

<table>
<thead>
<tr>
<th>PERCENTAGE</th>
<th>STAGE 1</th>
<th>SLEEP EFFICIENCY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2 Alpha</td>
<td>-0.50</td>
<td>0.41</td>
</tr>
<tr>
<td>Stage 3 Alpha</td>
<td>-0.64</td>
<td>0.40</td>
</tr>
<tr>
<td>Stage 4 Alpha</td>
<td>-0.52</td>
<td>0.65</td>
</tr>
<tr>
<td>Stage 2 Delta</td>
<td>-0.80 (a)</td>
<td>0.27</td>
</tr>
<tr>
<td>Stage 3 Delta</td>
<td>-0.78 (b)</td>
<td>0.12</td>
</tr>
<tr>
<td>Stage 4 Delta</td>
<td>-0.69</td>
<td>-0.27</td>
</tr>
<tr>
<td>Stage 2 Spindles</td>
<td>-0.80 (a)</td>
<td>0.29</td>
</tr>
<tr>
<td>Stage 3 Spindles</td>
<td>-0.74 (b)</td>
<td>0.06</td>
</tr>
<tr>
<td>Stage 4 Spindles</td>
<td>-0.64</td>
<td>0.65</td>
</tr>
</tbody>
</table>

(a) Significant (p<0.02)
(b) Significant (p<0.05)
10.3.3. **SUMMARY OF RESULTS**

The eight subjects studied here exhibited normal quantities of sleep stages 2, 3, 4 and REM. Although sleep stage 1 was slightly higher than normal, this could be attributed to some sleep disturbance as a result of the sleep recording equipment.

Stage 3 sleep showed the highest mean percentage of alpha per epoch, although there was very little difference between sleep stages 2, 3, 4 and REM. Stage 2 sleep showed the most spindle activity, whereas sleep stages 3 and 4 showed very little.

Mean values for alpha-delta indices reflected the lowest ratio of alpha to delta activity in sleep stage 4 (1.3%) and the highest in sleep stage 2, (26%). The largest dispersion of alpha-delta indices within the group was in stage 2 sleep, with less variation in indices for sleep stages 3 and 4.

Sleep stage 2 showed the highest mean spindle-delta index, indicating a large ratio of spindle to delta activity during stage 2 sleep. As delta activity increased in sleep stages 3 and 4, spindle activity was less prevalent.

Mean values of sleep onset latency, REM sleep latency, and indices of wakefulness and movement after sleep onset indicate that overall sleep quality during sleep recordings was good with little evidence of major sleep disturbances. Mean percentage sleep efficiency for the group was 91% (± 3.6).

Statistically significant correlations were found between mean percentages of alpha per epoch of sleep stages 2, 3, and 4, although the highest correlation was between mean percentage alpha of sleep stages 2 and 3. This suggests that the amount of NREM sleep alpha activity present during sleep (high or low) is reflected in both of these NREM sleep stages.

Mean percentage delta per epoch of sleep was significantly correlated for sleep stages 2 and 4, and there was a positive but non-significant correlation between delta in sleep stages 3 and 4. These results suggest there is an
overall level of delta activity throughout sleep stages 2, 3 and 4.

No significant correlations were found between percentage of epochs containing spindles in each of the NREM sleep stages.

Mean percentage of delta per epoch of sleep stage 2 was significantly correlated with percentage alpha in stage 2 (0.82) and in stage 3 sleep, (0.87). Mean percentage of epochs containing spindles in stage 3 sleep was also significantly correlated with percentage alpha in sleep stages 2 and 3, (0.74 and 0.73 respectively). Mean percentage of alpha in REM sleep was significantly correlated with percentage alpha per epoch of sleep stages 2 and 3.

Significant correlations were found between mean percentage delta per epoch of sleep stage 2 and spindle activity of stage 2, stage 3 and stage 4 sleep. Stage 4 delta and stage 3 spindles were also significantly correlated.

Mean percentages of alpha in sleep stages 2, 3, and 4 showed no statistically significant relationships to percentages of stage 1 sleep or sleep efficiency. However, it was interesting to find that percentages of alpha in sleep stages 2, 3, and 4 were negatively correlated with percentage stage 1 sleep, suggesting that alpha activity during these stages is not coincident with episodes of stage 1 sleep.

Stage 1 sleep was also negatively correlated with both delta and spindle activity in sleep stages 2, 3, and 4, but only those with stages 2 and 3 reached statistical significance. Sleep efficiency did not correlate significantly with any of the alpha, delta or spindle values for sleep stages 2, 3, or 4.
10.4. **SUMMARY OF RESULTS OF FOURIER ANALYSIS.**

Six subjects underwent sleep recordings using the 'frontal-occipital' montage as described in the main study.

A description of the results of each individual subject's fourier analysis is given in appendix (IX). A summary of the results is given below.

Table (LXVII) gives a summary of the frontal and occipital frequencies observed during sleep and relaxed wakefulness, in the six subjects studied. Each alpha-like frequency observed is ranked with regard to amplitude differences in that subject. A rank of (1) indicates the highest amplitude. Mean alpha for each NREM sleep stage is also given, but as REM sleep is not scored when the alternative montage is employed, mean REM alpha for the night is not given. All frequencies are in Hz.
TABLE LXVII.

ALPHA FREQUENCIES AND MEAN ALPHA FOR EACH SUBJECT AND STAGE.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Frontal-α</th>
<th>Occipital-α</th>
<th>Mean α</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n=6)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wake</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Subject IM</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal-α</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital-α</td>
<td>9.2 (1)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Mean α</td>
<td>-*</td>
<td>1.4</td>
<td>2.1</td>
</tr>
<tr>
<td>Subject JD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal-α</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital-α</td>
<td>9.8 (2)</td>
<td>8.6 (2)</td>
<td>8.8 (2)</td>
</tr>
<tr>
<td>Mean α</td>
<td>-*</td>
<td>0.2</td>
<td>0.2</td>
</tr>
<tr>
<td>Subject JP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal-α</td>
<td>-</td>
<td>11 (6)</td>
<td>-</td>
</tr>
<tr>
<td>Occipital-α</td>
<td>9.6 (1)</td>
<td>7.4 (3)</td>
<td>7.6 (2)</td>
</tr>
<tr>
<td>Mean α</td>
<td>-*</td>
<td>0.3</td>
<td>0.1</td>
</tr>
<tr>
<td>Subject SP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal-α</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital-α</td>
<td>9 (1)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Mean α</td>
<td>-*</td>
<td>0.2</td>
<td>0.04</td>
</tr>
<tr>
<td>Subject SB</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal-α</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital-α</td>
<td>10</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Mean α</td>
<td>-*</td>
<td>0.3</td>
<td>0.9</td>
</tr>
<tr>
<td>Subject MW</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal-α</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Occipital-α</td>
<td>11.4 (1)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Mean α</td>
<td>-*</td>
<td>0.2</td>
<td>0</td>
</tr>
</tbody>
</table>

Five of the six subjects did not show any frontal 'alpha-like' activity during NREM sleep stages 2, 3 or 4. One subject showed some frontal 'alpha-like' activity during sleep stage 2, but this did not appear consistently. Subjects (JD) and (JP) showed some occipital activity during NREM sleep stages 2, 3, and 4. Subjects (JD), (JP), (SP) and (MW) exhibited occipital alpha during REM sleep.
All subjects exhibited occipital alpha during relaxed wakefulness and during mental arithmetic. Waking occipital alpha frequencies varied between 9Hz and 11.4Hz.

Frontal theta activity (4 to 7Hz) activity was detected in stage 2 sleep of subject (IM), stage 3 sleep of subject (JD), and in sleep stages 3 and 4 of subject (SP).
10.5. SUMMARY OF RESULTS OF PRE-SLEEP AND POST-SLEEP QUESTIONNAIRE ANALYSIS.

A total of twenty seven nights were recorded from the eight subjects in this group. There was very little variation in pre-sleep and post-sleep SSS ratings, mean ratings for the twenty seven nights were 2.0 and 2.4 respectively. Mean overnight change indicated a drop from evening to morning in SSS ratings but this was less than one point on the scale. Group means taken from each subjects mean ratings gave similar results.

Individual pre and post-sleep ratings and overnight change showed no relationship to mean percentages of alpha, delta or spindles, or actual sleep time recorded on the respective nights.

Pre-sleep ratings of physical activity and feelings over the majority of the day, and post-sleep ratings of sleep quality, difficulty getting up, and physical condition, indicated that for the majority of the time subjects maintained their normal level of activity and were content and cheerful. Sleep during recorded nights was usually described as normal quality but occasionally worse than normal, or better than normal. There were only three reports of difficulty getting up, and one subject reported slight discomfort one morning, but this was attributed to previous exercise. There were no other reports of morning discomfort.
10.6. **CONSISTENCY STUDIES**

These were carried out to examine the consistency of NREM sleep alpha over differing periods of time. Sleep recordings of subjects in studies described previously were necessarily carried out at the individuals convenience, consequently varying periods of time elapsed between consecutive recordings. It was therefore considered necessary to examine the consistency of NREM alpha under normal circumstances, but due to social and work commitments few subjects were able to volunteer.

10.6.1. **METHOD**

For the few subjects studied, each sleep recording followed the same procedure as that described for the main study.

Two symptom-free subjects agreed to undergo sleep recordings on the same night for four weeks. Both standard and 'frontal-occipital' recordings were performed, in a different order for each subject. Waking tests were carried out only once.

As the 'frontal-occipital' montage does not allow REM sleep analysis, standard recordings were carried out on alternate weeks.

As in the main study, we also looked at the mean consistency of alpha, delta and spindle characteristics of the whole group.

10.6.2. **RESULTS**

Table (LXVIII) shows the mean difference in NREM sleep alpha, delta and spindles between consecutive recordings for each individual in the group. Standard deviations are shown in parentheses for subjects who underwent more than two recordings.
TABLE LXVIII.

MEAN DIFFERENCES IN NREM ALPHA, DELTA AND SPINDLES BETWEEN
CONSECUTIVE RECORDINGS FOR ALL SUBJECTS, (N=8).

<table>
<thead>
<tr>
<th>Subject</th>
<th>Alpha</th>
<th>Delta</th>
<th>Spindles</th>
</tr>
</thead>
<tbody>
<tr>
<td>IM</td>
<td>0.5 (0.5)</td>
<td>4.9 (3.7)</td>
<td>1.9 (0.8)</td>
</tr>
<tr>
<td>JD</td>
<td>0.9 (0.7)</td>
<td>13.0 (11.9)</td>
<td>2.9 (0.3)</td>
</tr>
<tr>
<td>JP</td>
<td>0.3</td>
<td>14.0</td>
<td>6.1</td>
</tr>
<tr>
<td>SP</td>
<td>0.6 (0.2)</td>
<td>13.1 (6.3)</td>
<td>6.3 (0.8)</td>
</tr>
<tr>
<td>RN</td>
<td>0.9</td>
<td>1.5</td>
<td>2.5</td>
</tr>
<tr>
<td>SB</td>
<td>0.8 (0.4)</td>
<td>3.7 (0.5)</td>
<td>6.0 (5.8)</td>
</tr>
<tr>
<td>MW</td>
<td>0.2 (0.1)</td>
<td>1.5 (0.8)</td>
<td>2.5 (0.4)</td>
</tr>
<tr>
<td>ML</td>
<td>0.8</td>
<td>1.7</td>
<td>0.3</td>
</tr>
<tr>
<td>Mean</td>
<td>0.6</td>
<td>6.7</td>
<td>3.5</td>
</tr>
<tr>
<td>s.d</td>
<td>0.2</td>
<td>5.3</td>
<td>2.1</td>
</tr>
</tbody>
</table>

The greatest differences in percentage of alpha per epoch of NREM sleep were seen in subjects (RN) and (JD), whereas the smallest differences were seen in subjects (MW) and (JP).

Subjects (JP), (SP), and (JD) showed the largest mean differences in delta, and subjects (JP), (SP), and (SB) showed the largest differences in percentage of epochs containing spindles in NREM sleep.

Two subjects, (IM and SB), underwent recordings on the same nights for four consecutive weeks of a calendar month. Figures (26) to (31) show the week to week differences in mean epoch alpha, delta and spindles for each sleep stage and subject.
Subject I.M.: Consistency of Alpha in Stages 2, 3, and 4

Legend
- Week 1
- Week 2
- Week 3
- Week 4
Subject L.M: Consistency of Delta in Stages 2, 3, and 4

Mean % Delta Per Epoch

Legend
- Week 1
- Week 2
- Week 3
- Week 4
Subject I.M.: Consistency of Spindles in Stages 2, 3, and 4

Legend
- Week 1
- Week 2
- Week 3
- Week 4
Subject S.B.: Consistency of % Alpha in Stages 2, 3, and 4

Legend
- Week 1
- Week 2
- Week 3
- Week 4
Subject S.B.: Consistency of Delta in Stages 2, 3, and 4

![Graph showing mean % Delta per epoch](image)

Legend:
- Week 1
- Week 2
- Week 3
- Week 4
Subject S.B.: Consistency of Spindles in Stages 2, 3, and 4

Legend
- Week 1
- Week 2
- Week 3
- Week 4
Table LXIX gives the mean and standard deviations of sleep variables for each subject over the four weeks.

**TABLE LXIX.**

**RESULTS OF CONSISTENCY STUDY:**

**MEAN PERCENTAGES OF SLEEP STAGES FOR EACH SUBJECT**

<table>
<thead>
<tr>
<th>SUBJECT (N=2)</th>
<th>IM</th>
<th>SB</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>s.d.</td>
</tr>
<tr>
<td>Stage 1</td>
<td>14.7 (2.8)</td>
<td>9.4 (0.3)</td>
</tr>
<tr>
<td>Stage 2</td>
<td>40.4 (5.6)</td>
<td>48.7 (3.2)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>12.9 (4.7)</td>
<td>14.0 (1.2)</td>
</tr>
<tr>
<td>Stage 4</td>
<td>2.6 (2.3)</td>
<td>3.0 (0.6)</td>
</tr>
<tr>
<td>REM sleep</td>
<td>28.6 (2.5)</td>
<td>24.5 (1.8)</td>
</tr>
</tbody>
</table>

Table LXX shows the overall means of sleep variables for each subject, over the four recordings.

**TABLE LXX.**

**RESULTS OF CONSISTENCY STUDY:**

**MEAN PERCENTAGE ALPHA AND DELTA PER EPOCH, AND PERCENTAGE OF EPOCHS CONTAINING SPINDLES IN STAGES 2, 3, 4, AND REM.**

<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>IM</th>
<th>SB</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>s.d.</td>
</tr>
<tr>
<td>Stage 2 Alpha</td>
<td>0.6 (0.4)</td>
<td>0.4 (0.1)</td>
</tr>
<tr>
<td>Stage 3 Alpha</td>
<td>1.0 (0.6)</td>
<td>1.6 (0.6)</td>
</tr>
<tr>
<td>Stage 4 Alpha</td>
<td>0.4 (0.1)</td>
<td>1.0 (0.4)</td>
</tr>
<tr>
<td>REM Alpha</td>
<td>0.4 (0)</td>
<td>0.1 (0.02)</td>
</tr>
<tr>
<td>Mean NREM Alpha</td>
<td>0.7 (0.4)</td>
<td>2.2 (2.6)</td>
</tr>
<tr>
<td>Stage 2 Delta</td>
<td>2.5 (1.5)</td>
<td>4.3 (0.8)</td>
</tr>
<tr>
<td>Stage 3 Delta</td>
<td>30.5 (1.1)</td>
<td>30.6 (0.6)</td>
</tr>
<tr>
<td>Stage 4 Delta</td>
<td>50.2 (2.0)</td>
<td>53.1 (1.2)</td>
</tr>
<tr>
<td>Mean NREM Delta</td>
<td>9.4 (3.7)</td>
<td>13.9 (1.7)</td>
</tr>
<tr>
<td>Stage 2 Spindles</td>
<td>3.7 (1.9)</td>
<td>12.8 (2.1)</td>
</tr>
<tr>
<td>Stage 3 Spindles</td>
<td>0 (0)</td>
<td>2.5 (0.9)</td>
</tr>
<tr>
<td>Stage 4 Spindles</td>
<td>0.3 (0.6)</td>
<td>0.6 (0.6)</td>
</tr>
<tr>
<td>Mean NREM Spindles</td>
<td>3.0 (1.6)</td>
<td>9.9 (1.5)</td>
</tr>
</tbody>
</table>
Table (LXXI) gives the alpha-delta and spindle-delta indices for sleep stages 2, 3, and 4 for subject (IM) on each of the four nights. Table (LXXII) gives these values for subject (SB) over the four nights recorded in the consistency study.

TABLE LXXI.

RESULTS OF CONSISTENCY STUDY: SUBJECT IM: ALPHA-DELTA AND SPINDLE-DELTA INDICES.

INDICES

<table>
<thead>
<tr>
<th></th>
<th>ALPHA-DELTA%</th>
<th>SPINDLE-DELTA%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2  3  4</td>
<td>2  3  4</td>
</tr>
<tr>
<td>Week 1</td>
<td>28.8 6.5 0.8</td>
<td>32.3 0 0</td>
</tr>
<tr>
<td>Week 2</td>
<td>30.7 1.5 0.2</td>
<td>923.0 0 2.6</td>
</tr>
<tr>
<td>Week 3</td>
<td>17.9 2.6 1.2</td>
<td>96.2 0 0</td>
</tr>
<tr>
<td>Week 4</td>
<td>29.5 2.2 1.0</td>
<td>294.9 0 0</td>
</tr>
<tr>
<td>Mean</td>
<td>26.7 3.2 0.8</td>
<td>336.6 0 0.6</td>
</tr>
<tr>
<td>s.d.</td>
<td>5.1 1.9 0.3</td>
<td>352.1 0 1.1</td>
</tr>
</tbody>
</table>

TABLE LXXII.

RESULTS OF CONSISTENCY STUDY: SUBJECT SB: ALPHA-DELTA AND SPINDLE-DELTA INDICES.

INDICES

<table>
<thead>
<tr>
<th></th>
<th>ALPHA-DELTA%</th>
<th>SPINDLE-DELTA%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2  3  4</td>
<td>2  3  4</td>
</tr>
<tr>
<td>Week 1</td>
<td>12.0 5.2 1.4</td>
<td>285.2 9.7 0</td>
</tr>
<tr>
<td>Week 2</td>
<td>8.9 3.1 2.1</td>
<td>278.6 8.3 2.4</td>
</tr>
<tr>
<td>Week 3</td>
<td>9.0 9.1 3.0</td>
<td>319.5 11.9 2.1</td>
</tr>
<tr>
<td>Week 4</td>
<td>11.3 4.1 0.9</td>
<td>309.3 3.3 0</td>
</tr>
<tr>
<td>Mean</td>
<td>10.3 5.4 2.2</td>
<td>298.1 8.3 1.1</td>
</tr>
<tr>
<td>s.d.</td>
<td>1.3 2.3 0.6</td>
<td>16.8 3.1 1.1</td>
</tr>
</tbody>
</table>

The largest mean alpha-delta indices were seen in stage 2 sleep and the lowest were seen in stage 4 sleep for both subjects. Spindle-delta indices were also greatest in stage 2 sleep, but subject (IM) failed to show any spindle activity in stage 3, although spindles were found in stage 4 sleep in the second recording.
Alpha-delta and spindle-delta indices in stage 2 and 4 were both greater for subject (IM) than they were for subject (SB), but subject (SB) showed greater alpha-delta and spindle-delta indices in sleep stage 3.

Both subjects showed their largest mean percentage of NREM sleep alpha per epoch in sleep stage 3, but only subject (SB) showed any spindle activity in this sleep stage. Subject (SB) showed the greatest mean percentages of alpha per epoch of NREM sleep and REM sleep, and also the most spindle activity in each NREM sleep stage.

**FOURIER ANALYSIS DURING CONSISTENCY STUDY.**

**SUBJECT (IM): Sleep.**

(IM) underwent recordings using the alternative montage on the first and third weeks.

On both occasions, neither frontal alpha-like activity or occipital alpha were observed during any NREM sleep stage. However, small peaks of 9Hz activity were seen occipitally during REM sleep.

**Wakefulness.**

A prominent (70.4μV) of 9.2Hz appeared during relaxed wakefulness with the eyes shut. This went completely when the eyes were opened. A smaller (60.8μV) of 9.4Hz was observed during mental arithmetic with the eyes shut, but this also went when the eyes were opened.

**SUBJECT (SB).**

(SB) underwent recordings with the alternative montage on the second and fourth weeks.

**Subject (SB): Sleep.**

Neither frontal alpha-like activity nor occipital alpha were found in NREM sleep stages 2, 3, and 4 or REM sleep in this subject on either night.
Wakefulness.

A small (31.8 μV) peak of 10 Hz occipital alpha activity which was seen during relaxed wakefulness with the eyes shut, was completely absent when the eyes were open. During mental arithmetic with the eyes shut, a small peak (20.3 μV) of 10.2 Hz activity was seen, and this also was absent when the eyes were open.

PRE-SLEEP AND POST-SLEEP QUESTIONNAIRE RATINGS.

Tables (LXXIII) and (LXXIV) show the ratings given by subjects (IM) and (SB) on the pre-sleep and post-sleep questionnaires for each recording night of the consistency study.

**TABLE LXXIII.**

RESULTS OF CONSISTENCY STUDY: RESULTS OF PRE- AND POST-SLEEP QUESTIONNAIRES:

SUBJECT IM

<table>
<thead>
<tr>
<th>WEEK</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Sleep SSS</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Post-Sleep SSS</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Daytime Activity</td>
<td>n</td>
<td>n</td>
<td>n</td>
<td>n</td>
</tr>
<tr>
<td>Feelings over day</td>
<td>AlC</td>
<td>AcC</td>
<td>AcC</td>
<td>AcC</td>
</tr>
<tr>
<td>Difficulty getting</td>
<td>mod</td>
<td>mod</td>
<td>mod</td>
<td>eas</td>
</tr>
<tr>
<td>Sleep Quality</td>
<td>n</td>
<td>btn</td>
<td>n</td>
<td>n</td>
</tr>
<tr>
<td>Discomfort</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>no</td>
</tr>
<tr>
<td>Actual Sleep Time (hrs)</td>
<td>6.3</td>
<td>6.6</td>
<td>6.8</td>
<td>6.9</td>
</tr>
<tr>
<td>Mean NREM Alpha</td>
<td>1.5</td>
<td>0.21</td>
<td>0.47</td>
<td>0.64</td>
</tr>
<tr>
<td>Mean NREM Delta</td>
<td>15.8</td>
<td>7.6</td>
<td>6.1</td>
<td>8.1</td>
</tr>
<tr>
<td>Mean NREM Spindles</td>
<td>1.0</td>
<td>3.8</td>
<td>2.1</td>
<td>5.2</td>
</tr>
</tbody>
</table>
Summary of Abbreviations used in Tables (LXXIII) and (LXXIV).

n=normal
btn= Better than normal.
Alc=Alert and contented.
AcC=Active and cheerful.
mod=Moderate.
eas=easy.
no=No discomfort.

TABLE LXXIV.

RESULTS OF CONSISTENCY STUDY:
RESULTS OF PRE- AND POST-SLEEP QUESTIONNAIRES:
SUBJECT SB.

<table>
<thead>
<tr>
<th>WEEK</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Sleep SSS</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Post-Sleep SSS</td>
<td>3</td>
<td>1</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Daytime Activity</td>
<td>n</td>
<td>n</td>
<td>n</td>
<td>n</td>
</tr>
<tr>
<td>Feelings over day</td>
<td>AlC</td>
<td>AcC</td>
<td>AlC</td>
<td>AcC</td>
</tr>
<tr>
<td>Difficulty getting u</td>
<td>mod</td>
<td>eas</td>
<td>eas</td>
<td>mod</td>
</tr>
<tr>
<td>Sleep Quality</td>
<td>n</td>
<td>n</td>
<td>n</td>
<td>n</td>
</tr>
<tr>
<td>Discomfort</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>no</td>
</tr>
<tr>
<td>Actual Sleep Time (hrs)</td>
<td>7.6</td>
<td>5.8</td>
<td>7.2</td>
<td>8.0</td>
</tr>
<tr>
<td>Mean NREM Alpha</td>
<td>0.87</td>
<td>0.5</td>
<td>1.0</td>
<td>0.57</td>
</tr>
<tr>
<td>Mean NREM Delta</td>
<td>14.3</td>
<td>10.8</td>
<td>15.1</td>
<td>12.0</td>
</tr>
<tr>
<td>Mean NREM Spindles</td>
<td>12.6</td>
<td>9.2</td>
<td>9.2</td>
<td>8.6</td>
</tr>
</tbody>
</table>
10.6.3. **SUMMARY OF RESULTS OF CONSISTENCY STUDY.**

In the eight subjects studied, NREM sleep delta showed the greatest mean difference between recordings, whereas alpha showed less difference. For each subject, the mean difference in alpha between recordings was less than 1%, but mean differences in percentage delta ranged from 1.5 to 14%. Percentages of epochs containing spindles varied by up to 6.3%, but some subjects showed very little change in mean NREM spindle activity.

Subjects (IM) and (SB) underwent two standard recordings and two frontal-occipital recordings over a four week period. Subject (IM) showed the most variation in percentages of each sleep stage, with sleep stages 2 and 3 showing the least consistency. For subject (SB), sleep stages 2 and REM varied most.

Subject (SB) had the greatest mean percentages of alpha for NREM sleep, for individual stages 3 and 4, and for REM sleep. She also showed higher mean percentages of delta and spindle activity during each NREM sleep stage.

Both subjects showed consistently low alpha-delta indices for the four recordings. Spindle-delta indices were greatest in sleep stage 2, and subject (SB) showed least variation.

Fourier analysis of sleep recordings taken two weeks apart did not reveal any differences in the appearance of alpha activity in subject (IM) or (SB).

Subject (IM) consistently rated his sleep as 'normal' on his post-sleep questionnaires. Pre-sleep SSS ratings were either (1) or (2) on each occasion. These did not show any improvement overnight, as they were either the same or one to two points lower the following morning. There were no reports of morning discomfort. The largest percentage of NREM sleep alpha was found following the first recording, and this was associated with the highest percentage of NREM delta, but the lowest percentage of spindle activity.
Subject (SB) also rated her sleep quality as normal, and did not report any morning discomfort. Pre-sleep SSS ratings varied from (2) to (3), and did not show any consistent pattern of overnight improvement or deterioration. However, her highest percentage of NREM sleep alpha occurred during the night after which she showed a drop in her SSS score from her pre-sleep rating. This was also associated with the highest mean percentage of NREM delta.
10.7. DISCUSSION.

This group of eight subjects responded to advertisements requesting good sleepers, and all were students approaching their end of year examinations at University. Six were coming up to their final year examinations, and two were about to sit their first University examinations. All subjects described that they were under pressure with revision and projects at the time of study, but they all regularly participated in sporting and social activities and were happy to volunteer for sleep recordings.

Mean pre and post-sleep scores on the SSS indicated that subjects generally felt alert and active prior to retiring and after getting up in the morning. In general there was very little change in ratings overnight although post-sleep scores tended to be lower on the alertness scales than pre-sleep scores were. There were no reports of morning discomfort other than stiffness as a result of previous exercise, and the majority of subjects found no difficulty getting up after recordings.

These subjects showed some sleep disruption as a result of the sleep recording procedure, and while some nights were considered to be normal sleep quality, some were considered to be worse than normal. Although mean percentages of sleep stages for the group were within normal ranges, and sleep efficiency and REM sleep latency were both comparable with those of the control subjects in the previous study, percentage stage 1 sleep was greater and percentage REM sleep were less than those found for the control subjects. Mean percentage of SWS (14.5, ±6.9) was lower than that described by Williams et al, (1974) for the mid-twenties age group, and also non-significantly less than that of the control group from the previous study.

Mean percentages of alpha per epoch of sleep stages 2, 3, 4 and REM were all lower than those found in the control subjects or discomfort subjects from the previous study. Differences between mean NREM alpha of the eight subjects and the control subjects were significant at p<0.05, and at
p<0.02 and p<0.002 compared to good sleepers and poor sleepers with discomfort respectively.

62% of the group showed their highest percentage of alpha in sleep stage 3, whereas 25% showed their highest in sleep stage 4 and one subject showed the most in stage 2 sleep. In the previous study, 40% of the fifteen control subjects showed most alpha in sleep stage 3, and 46% showed most in stage 2 sleep, with the remainder showing the most in stage 4 sleep.

Significant positive correlations between percentages of alpha in sleep stages 2, 3 and 4, and between sleep stages 2 and 3 and REM sleep indicate that as found in the previous study, each subject showed an individual level of alpha activity during sleep which was reflected in these NREM and REM sleep. Group means of alpha in NREM sleep stages and REM sleep ranged between 0.7 and 1.1%, with sleep stages 2 and 3 showing the greatest standard deviations. These results support our previous findings, and also the report by Scheuler, (1986) that the 'alpha-sleep pattern' presents as an individually constant characteristic.

Mean percentages of delta activity in sleep correlated significantly between NREM sleep stages 2 and 4 (0.75), but correlations between stages for spindle activity were non-significant.

There appeared to be some relationship between delta and spindle activity in this group of subjects, and significant positive correlations were found between percentages of alpha in sleep stages 2 and 3 and the mean percentage of delta in sleep stage 2, and spindles in stage 3 sleep.

Correlations of these variables within a particular sleep stage have to be considered with caution, because each represents a percentage of an epoch, and as the percentage of one type of activity increases, another will decrease. If there is a general level of alpha activity in each individual then there is likely to be some relationship of alpha with delta and spindle activities in different sleep stages.

In addition there may be other factors influencing the quantity of each activity in sleep.
Age is known to affect both the amplitude and frequency of delta waves (Feinberg, 1974; Smith, Karacan and Yang, 1977) and spindle activity (Bowersox, Kaitin and Dement, 1985), however, Scheuler (1986) reported that the alpha-sleep pattern is not age dependent, and the results of our previous study also suggest this. Other unknown factors may be operative, although it is important to consider the small number of subjects when discussing these correlations.

Bowersox, Kaitin and Dement, (1977) suggested that the function of sleep spindles may be related to sleep continuity, and it is interesting to note that the mean percentage of epochs containing spindles for the eight subjects (who showed very little alpha activity) was similar to that shown by both discomfort groups in sleep stage 2 and sleep stage 4, but more than any other group for sleep stage 3.

Only one of the eight subjects (JP) (12.5% in this part of the study) showed any frontal 'alpha-like' activity or kappa during NREM sleep, which was during sleep stage 2. In addition, this subject and also subject (JD) showed occipital alpha during NREM sleep stages 2, 3, and 4, and in REM sleep.

Each of the six subjects showed occipital alpha during REM sleep and all six subjects showed waking occipital alpha between frequencies of 9 to 11.4Hz, but none showed any frontal kappa during the mental arithmetic condition. Alpha activity was attenuated in three subjects (SP, MW and JP) when the eyes were opened, suggesting that these subjects may be higher waking alpha producers than the subjects whose alpha disappeared completely.

Subject (JP), one of the higher waking alpha producers, showed frontal 'alpha-like' activity which was more than 1Hz faster than his waking alpha frequency of 9.6Hz observed occipitally, and also showed occipital alpha during sleep stages 2, 3 and 4. However, despite this, he had a mean percentage of NREM sleep alpha which was amongst the lowest found in this group, although it showed the most consistency. Hence the presence of frontal 'alpha-like' activity did not bear any direct relationship to the percentage of alpha shown on the central sleep EEG. It is
possible that central alpha represents true NREM sleep alpha activity which is consistent within the subject whereas frontal 'kappa' represents processes of sleep mentation, which vary according to psychological factors.

Although there was a low incidence of reported stress and anxiety in the group as a whole, subject (JP) did report previous anxiety and depression and was the one subject who exhibited frontal 'alpha-like' activity during NREM sleep. Although, subject (JP) described waking during the night if he had an important event the following day, he claimed to never lie awake worrying. He reported snoring and having a great need to move his legs in bed, and occasionally awoke with lethargy and tiredness but attributed this to being a 'definite evening type'.

In the previous study frontal theta was frequently associated with the presence of frontal 'kappa', but subject (JP) did not show any frontal theta during his recording.

However, frontal theta activity was detected in stage 2 sleep of subject (IM), stage 3 sleep of subject (JD), and in sleep stages 3 and 4 of subject (SP). All three subjects were final year students who were free of anxiety but expressed some concern over their forthcoming examinations and job interviews. It is possible that these 'pressures' may have been 'on their minds' at the time of study, and may have been incorporated into NREM sleep mentation, but as there are no reports of this, it is pure speculation, as other subjects who were in the same situation did not show frontal theta, (subjects SB, MW and ML) or frontal kappa. However it is also true that individuals have different attitudes towards examinations and employment, with some being more likely to show concern than others. It is also likely that sleep characteristics such as sleep mentation may vary over time, relating to specific events and anxieties which are influenced by the individuals personality.

Consistency studies of subjects (IM) and (SB) found that mean percentages of alpha in NREM sleep and REM sleep were most consistent from week to week for both subjects, compared to mean percentages of delta activity and
percentage of epochs containing spindles in each sleep stage.

Mean percentage alpha for each sleep stage varied by less than 1% but was greatest in sleep stage 3 and least in REM sleep for both subjects. Mean percentage delta showed the most variation in sleep stage 4 and the least in stage 3 sleep. Whereas subject (IM) did not show any spindle activity in sleep stage 3 on any night, and none in stage 4 except on the second recording, subject (SB) showed most consistency in spindle activity in sleep stage 4, but most variation of spindle activity in stage 2 sleep.

Of the eight subjects studied, subjects (JD) and (RN) showed the least consistency in mean NREM sleep alpha, whereas subject (JP) showed the most. Subjects, (JP), (SP), and (JD) showed the greatest mean difference in consecutive percentages of mean NREM delta, and subjects (JP), (SP), and (SB) showed the most variation in spindles.

Compared to subjects in the previous study, the eight young subjects showed less inter-recording variation in percentages of NREM sleep alpha, but more variation in delta and spindle activity than either the control or discomfort groups. Subjects (IM) and (SB) showed less variation in mean percentages of alpha in sleep than subject (P) who was studied every night for a week, and less than subject (C) who was studied three times over sixteen months. Mean percentages of delta showed most inconsistency for subject (C) in sleep stage 4, as did spindle activity.
CHAPTER 11.

OVERALL CONCLUSIONS FROM STUDIES OF NREM SLEEP ALPHA ACTIVITY.
OVERALL CONCLUSIONS FROM STUDIES OF NREM SLEEP ALPHA ACTIVITY.

Good sleepers with discomfort and control subjects were found to exhibit few statistically significant differences in sleep variables, which supported their subjective ratings of good sleep quality. Although mean percentages of alpha per epoch of sleep were greater in the discomfort subjects, the variation within both groups indicated that the distribution of percentages of alpha found in both NREM sleep and REM sleep overlapped between the two groups. This indicates that the percentage of alpha activity in epochs of NREM sleep is not directly related to the appearance of musculoskeletal symptoms and daytime lethargy, but may be related to other characteristics which are common in individuals reporting these symptoms.

Within each group of subjects, there was no consistency in the NREM sleep stage which showed the highest percentage of alpha activity. Although more good sleepers with discomfort showed most alpha in stage 2 sleep, and more poor sleepers with discomfort showed most in stage 3 sleep, both sleep stages 2 and 3 showed most alpha in the control group subjects.

Correlations of NREM and REM sleep alpha suggest that each individual might have a 'general' level of alpha activity which varies between sleep stages but is generally consistent over NREM sleep and REM sleep.

The group of eight symptom-free good sleepers described in chapter 10 showed lower mean percentages of NREM sleep alpha than the groups of older control subjects or subjects with discomfort in the previous study. However, according to the information collected the eight symptom-free subjects differ from the three groups of subjects studied previously in the following ways:

These subjects presented as a more homogenous group, they had more in common with one another in their lifestyles, current situations, health and fitness, than subjects in the control group and good sleep with discomfort studied previously. All of these factors may affect sleep variables in different ways.
As a group the eight subjects presented as a younger, fitter and healthier sample of subjects than those studied previously. There was a lower incidence of reported health problems, sleep anomalies and sleep difficulties.

They also showed an inter-recording consistency of NREM sleep alpha which was greater than that of delta or spindle activities, and greater than that found in good sleepers with discomfort.

It is possible that NREM sleep alpha found in our studies may be a normal phenomenon which varies between individuals, but shows a higher prevalence in subjects with musculoskeletal discomfort and lethargy. The mechanisms which influence the quantity of NREM sleep alpha may be related to those determining waking alpha activity, and may also be associated with the psychological profile of the individual.

Scheuler (1986) suggested that the 'alpha sleep anomaly' may be genetically determined, and this has also been suggested for waking alpha characteristics, (Kiloh et al, 1972).

Frontal 'alpha-like' activity or 'kappa' activity, was observed predominantly in the subjects with discomfort but also in control subjects. It is suggested that this activity may relate to NREM sleep mentation processes particularly evident during periods of emotional stress, but also apparent in subjects who tend to ruminate over current anxieties. The sub-study of sleep mentation reports suggested that the four good sleepers with discomfort frequently incorporated current events into their sleep mentation content, although without a concurrent sleep EEG, it was difficult to determine whether the 'dreams' reported took place during NREM sleep or REM sleep.

From our studies, it is not clear to what extent the central sleep EEG alpha activity is related to the appearance of frontal 'alpha-like' or kappa activity, although personality type may influence both.

NREM sleep alpha activity on the central EEG appears to be an individually constant characteristic, whereas frontal 'alpha-like' activity may be more related to events or circumstances evoking psychological stress or anxiety. Such
processes may be more active in subjects with a particular personality profile, who show an increased tendency to reports of anxiety and depression.

Despite more reports of sleep difficulties in the discomfort groups, there were no significant correlations between indices of sleep disturbance and NREM sleep alpha. Subjects may be more aware of sleep difficulties when they are under stress or have things 'on their minds', which they want to escape from by sleeping.

Musculoskeletal symptoms and daytime lethargy are not uncommon in the normal population, although they occur with less frequency and may receive less attention from the individual. Questionnaire analysis showed that subjects with discomfort reported a higher incidence of anxiety and depressive symptoms, health problems and sleep anomalies. It is possible that this group of individuals may represent a section of the normal population who are more psychologically predisposed to anxiety, rumination, and somatic complaints than other subjects who are better able to 'cope' with problems encountered in daily living.

They may simply represent a variation in human personality type and 'coping' styles, which is frequently associated with the same characteristics that underly the quantity of NREM sleep alpha activity.

My hypothesis is that the amount of NREM sleep alpha and the incidence of frontal kappa may be influenced by the same underlying factors, the effects of which vary between individuals in the normal population. The amount by which characteristics such as NREM sleep alpha and frontal kappa change over longer periods of time cannot be determined without further studies.
11.1. SUGGESTIONS FOR FUTURE STUDIES.

The studies described suffer from a relatively small number of subjects and a lack of clinical and psychological diagnoses of individuals, which are obviously required for more detailed analyses of each subject's health and psychological profile.

1. In order to determine whether frontal NREM kappa is related to NREM sleep mentation, and whether central EEG alpha is associated with these processes, it would be interesting to record laboratory sleep using a full EEG montage, and to wake subjects up on the appearance of frontal 'alpha' (or kappa) and central alpha. Simultaneous tape recordings would allow Fourier analysis of both EEGs to ascertain the concordance between frontal and central alpha activities.

2. It would be interesting to conduct a 'blind' investigation of the relationship between personality types and the incidence of frontal kappa. Psychological characteristics might be ascertained by psychological tests and interview with a 'blind' psychiatrist or psychologist, and the results of these revealed following sleep analysis.

3. Similarly it would be interesting to conduct a longitudinal study using two large groups of subjects with and without musculoskeletal discomfort symptoms. An independent psychologist would maintain regular contact with these subjects and evaluate their psychological well-being, subjective ratings of stress, and somatic complaints at different periods over several years. Sleep recordings would be collected and analyzed for frontal kappa, 'blind' of the current psychological and symptomatic state of the individual.

4. It would also be interesting to determine whether there were any differences in the nature of REM sleep 'dreams' when frontal kappa is present in NREM sleep. A more detailed analysis of REM sleep episodes and periodicities might be easier to perform using paper write outs of the EEG compared to visual displays from tape recordings.
5. To investigate individual characteristics of high and low NREM sleep alpha producers, a large number of subjects (more than 200 for example) could be studied to evaluate the relationship of NREM sleep alpha to age, sex, psychological and somatic variables, exposure to occupational and personal stress, and subjective evaluations of achievements, interpersonal relationships and personal life histories. It would also be helpful to define the exact relationship between waking and sleeping alpha characteristics in subjects.

6. It would also be worthwhile to do a long-term study of the effects of increasing fitness on sleep alpha activity, and examine each subject for changes in psychological factors and discomfort symptoms.
CHAPTER 12.

DISCUSSION OF SOME METHODOLOGICAL POINTS IN THE THESIS.
12. **DISCUSSION OF SOME METHODOLOGICAL POINTS IN THE THESIS.**

This thesis described two investigations into different aspects of SWS, the first was a laboratory based study of the effects of passive heating on subsequent SWS and the influence of aspirin. The second study involved the use of home sleep recordings and investigated the relationship of the alpha sleep anomaly to musculoskeletal discomfort in a local population, and the incidence of this anomaly in a small group of young and healthy symptom-free subjects.

In the laboratory based experiment, we studied six students who were paid for their services and who were willing to cooperate with the conditions and requirements of the experiment as instructed. Although the environment of the sleep laboratory was strange to them they soon adapted to sleeping there for four nights a week and soon found the relaxed and informal atmosphere enjoyable and conducive to sleep.

More practical difficulties were encountered using home sleep recordings as subjects were unpaid volunteers from the local community. Individuals were generally happy to complete questionnaires, but were less willing to commit themselves to sleep recordings, and to restrict their normal habits, and the reasons for this were usually understandable. The difficulties encountered in getting subjects to undergo sleep recordings meant that an adaptation night was not possible. Several subjects were 'put off' by the first recording and did not want to undergo further recordings, so it was decided to record subjects on each occasion and discard recordings if they were unacceptable. Subjects were encouraged to undergo further recordings with the hope that they would be more familiar with the procedure and equipment.

It is difficult to determine the exact magnitude of the effect of the sleep recording equipment on each individual's sleep, compared to their normal sleep without the equipment.

Home recordings have the advantage over laboratory recordings in as far as the subjects are in their usual sleep environment, and this should remove the unseen stress of the laboratory atmosphere. However, subjects sleeping in
the laboratory might be more relaxed than they are at home, if their home environment is associated with stresses and anxieties, or disturbances such as noise, or temperature extremes.

Subjects in the laboratory study became very relaxed while they were having their electrodes attached, and the females likened this to the relaxing effect of being at the hairdressers. This effect was also reported by the subjects in the other study, but in this case, electrode glue was dried using a hairdryer rather than compressed air, which had an additional effect of warming the top of the head, which subjects found soporific.

Sleep EEGs recorded using the Oxford Medilog recording system were analysed automatically by the Oxford Sleep Stager according to the recommendations of Rechtschaffen and Kales, (1968), which were also employed for visual sleep scoring in the passive heating study. Comparison of visual and automatic sleep staging in the passive heating study showed 87% agreement between the two methods, which improved to 90% when total SWS (stage 3 plus stage 4 sleep) was considered.

Using the Medilog 9000 recording system and Oxford Sleep Stager, Crawford, (1986) compared manual and automatic scores in each 40 second epoch, and found an 83% (±3.8) agreement. Sleep stages which were clearly indicated, (for example, well definable spindles in sleep stage 2, and almost continuous delta activity in sleep stage 4), showed the most agreement between the two methods. Disagreements were highest for wakefulness, which was frequently scored as stage 1 sleep by the analyser, and REM sleep which was occasionally scored by the analyser during relaxed wakefulness. The sleep stager was considered to be an accurate method for the estimation of delta activity and slow wave periods, and for illustrating the details of individual sleep architecture. Our own results showed the highest agreement between the two methods for sleep stage 2, SWS and REM sleep.
Appendix (X) shows a repeatability test of the sleep stager results with and without changes in calibration prior to analysis. The best repeatability was found for sleep stages, whereas sleep stage latencies were less consistent between each analysis. Recalibration produced larger mean differences in sleep latencies, but very small differences in sleep staging results.

In our study, automatic analysis of sleep presented some limitation on visual inspection of the sleep EEG activities. Although the analyser gave percentages of each EEG activity in each sleep epoch, it would have been useful to have had a paper write out of the EEG by which to inspect and compare the characteristics shown by each subject more carefully.

12.1. QUESTIONNAIRES AND PRE AND POST-SLEEP SCALES OF SUBJECTIVE VARIABLES.

In each of the studies reported we employed the Stanford Sleepiness Scale in order to obtain pre and post-sleep ratings of subjective sleepiness. This seven point scale has been proved to be a sensitive and reliable indicator of levels of sleepiness, which shows high correlations with performance on the Wilkinson Addition test and the Wilkinson Vigilance test which are sensitive to moderate amounts of sleep loss, (Hoddes et al, 1973).

Other questions on the pre-sleep questionnaires related to daytime activity levels and subjective well being. In the latter, subjects were asked to rate their well being using one of four phrases. This met with some comment as some subjects found that the two adjectives used in each phrase did not adequately describe their feelings, for example the phrase 'active and cheerful'; subjects may have had an active day, but were not necessarily cheerful. On retrospect these ratings should have been more clearly defined and less ambiguous.
The preliminary questionnaires used in the Surveys on Sleep and Muscular Discomfort, did not encounter many obvious problems except that the answers given were sometimes vague or ambiguous, or the questions had been misunderstood or omitted. It is also possible that the sleep anomalies and health problems described may have provoked over-reporting by suggestion. However, this method allowed the experimenter to look at the incidence of particular problems such as anxiety and depression which may have been ommitted by subjects in reporting their state of health.

The questionnaire given to subjects in the main sleep EEG study (appendix II) gave multiple choice answers to questions, in addition to short Yes/No answers. This was designed to reduce the ambiguity encountered in the preliminary questionnaires. This worked well except that questions 44 to 46 at the end of the questionnaire were frequently omitted, probably because the answers required a little more thought.

These problems will be taken into consideration when designing experimental procedures for future studies.
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APPENDIX I.
## CONFIDENTIAL

### SURVEY ON SLEEP

<table>
<thead>
<tr>
<th>Name</th>
<th>Ethnic origin</th>
</tr>
</thead>
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<tr>
<td>Age</td>
<td>Married/Single</td>
</tr>
<tr>
<td>Sex</td>
<td>Recreational activities</td>
</tr>
<tr>
<td>Height</td>
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<tr>
<td>Weight</td>
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<tr>
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</table>

<table>
<thead>
<tr>
<th>Ethnic origin</th>
<th>Cigarettes smoked/day</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Occupation</td>
</tr>
<tr>
<td></td>
<td>Distance to work</td>
</tr>
<tr>
<td></td>
<td>How do you get to work</td>
</tr>
</tbody>
</table>

1. **How well do you feel you usually sleep at night?** (Please tick)
   - Very Well ____  Well ____  Poorly ____

2. **How long does it usually take you to fall asleep at night?**

3. **How much alcohol do you drink in the evenings?**

4. **Do you lie awake worrying at night?**

5. **How many times do you wake up during the night, and why?**

6. **What time do you usually wake up in the morning?**

7. **Do you have difficulty waking up in the mornings?**

8. **What time do you usually get up in the morning?**
9. How do you usually feel when you wake up in the morning? (Please tick)
   Active and alert ___  Relaxed, awake ___  Tired and sleepy ___

10. Do you regularly wake up with any of the following:-
   Aching ____________  Where? ________________
   Pain _______________  Where? ________________
   Stiffness ____________  Where? ________________
   Tenderness ___________  Where? ________________
   Cramps _______________  Where? ________________
   Headache _____________
   Bad Temper ____________
   Lethargy ______________

11. How many hours do you usually sleep at night, during the:
   Week ____  Weekend ____  Holidays ____

12. How many hours do you think you yourself need?

13. Do you regularly feel very tired or sleepy during the day?

14. Do you take sleeping pills? If so, which, and for how long have you been taking them?

15. Are you currently taking any prescribed pills or medicines? If so, which ones and for what?
16. Have you ever worked night shifts? How well did you sleep during the day?

17. Do you worry about your health?

18. Do you regularly do any of the following:

- Sleepwalk
- Sleeptalk
- Grind your teeth while asleep
- Take naps
- Have nightmares
- Wake up with a jerk
- Wake up early in the morning
- Have difficulty falling asleep
- Have a great need to move your legs in bed
- Have difficulty staying awake during the day
- Find you are unable to move when you wake up
- Have weakness in your limbs and/or trunk when you laugh or get excited?

19. Have you ever suffered with any of the following:

- Asthma
- Hay Fever
- Eczema
- Other allergies
- Thyroid problems
- Migraine
- Breathing problems
- Undue anxiety
- Stammering
- Difficulty reading or writing
- Arthritis
- Rheumatism
- Gout
- Depression
- Heart problems
- Stomach problems
20. Do you regularly take aspirin, codeine or paracetamol for any reason?

21. Do you have any recent history of stress or anxiety at home or at work?

Please add any comments you think may be relevant.
CONFIDENTIAL

SURVEY ON MUSCULAR DISCOMFORT

<table>
<thead>
<tr>
<th>Name</th>
<th>Ethnic Origin</th>
</tr>
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<tbody>
<tr>
<td>Age</td>
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<tr>
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<td></td>
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<tr>
<td>How do you get to work</td>
<td></td>
</tr>
</tbody>
</table>

1. Please shade in the areas where you have discomfort

2. Please tick the word which best describes the pattern of your pain
   - Continuous
   - Periodic
   - Brief
3. Is your discomfort worse when you wake up in the morning?

4. How would you describe the discomfort? (e.g. aching, stiffness, tenderness, etc.)

5. What kind of things increase the discomfort?

6. What kind of things relieve the discomfort?

7. What do you think is the cause of the discomfort?

8. Do any of your family have a similar problem?

9. When did you first notice the discomfort? (Year/month)

10. Do you worry about the discomfort?

11. Do you worry about your health?

12. How well do you feel you usually sleep at night? (Please tick)
    Very Well _____    Well _____    Poorly _____

13. How long does it usually take you to fall asleep at night?

14. How much alcohol do you consume in the evenings?
15. Do you lie awake worrying at night?

16. How many times do you wake up during the night, and why?

17. Do you have difficulties waking up in the morning?

18. What time do you usually wake up in the morning?

19. What time do you usually get up in the morning?

20. How do you usually feel when you wake up in the morning? (Please tick)
   Active and alert ___  Relaxed, awake ___  Tired and sleepy ___

21. How many hours do you usually sleep at night, during the:
   Week ____  Weekend ____  Holidays ____

22. How many hours do you think you yourself need?

23. Do you regularly feel very tired or sleepy during the day?

24. Do you take sleeping pills? If so, which, and for how long have you been taking them?

25. Are you currently taking any prescribed pills or medicines? If so, which ones and for what?
26. Does the discomfort start at any other time of the day? If so, when?

27. Have you ever worked night shifts? If so, did you have the same discomfort on awakening during the day?

28. Do you regularly do any of the following:

- Sleepwalk
- Snore
- Sleeptalk
- Take naps
- Have nightmares
- Grind your teeth while asleep
- Wake up with a jerk
- Wake up early in the morning
- Have difficulty falling asleep
- Have a great need to move your legs in bed
- Have difficulty staying awake during the day
- Find you are unable to move when you wake up
- Have weakness in your limbs and/or trunk when you laugh or get excited?

29. Have you ever suffered with any of the following:

- Asthma
- Stammering
- Hay Fever
- Difficulty reading or writing
- Eczema
- Arthritis
- Other allergies
- Rheumatism
- Thyroid problems
- Gout
- Migraine
- Depression
- Breathing problems
- Heart problems
- Undue anxiety
- Stomach problems
30. Do you regularly take aspirin, codeine or paracetamol for any reasons?

31. Do you have any recent history of stress or anxiety at home or at work?

Please add any comments you think may be relevant.
CONFIDENTIAL

SLEEP QUESTIONNAIRE

NAME ........................................
AGE ........................................... SMOKER? NO/DAY .................
MARITAL STATUS: MARRIED ............... RECREATIONAL ACTIVITIES ......
(Please tick) SINGLE ......................... HRS/WK.
DIVORCED OR SEPARATED ................. HRS/WK.
WIDOW OR WIDOWER ................. HRS/WK.

PRESENT OR MOST RECENT OCCUPATION? (Please be as specific as you can)

Please read each question carefully and put the letter corresponding to your answer, in the right hand box. If answer to question is 'Yes' or 'No' please circle the appropriate word ('Yes', or 'No')

1. How well do you normally sleep at night?
   a. Very well.  
   b. Satisfactorily.  
   c. Some problems.  
   d. Poorly.

2. How much do you enjoy sleep?
   a. Very much.  
   b. Moderately.  
   c. Not much.  
   d. Not at all.

3. How refreshed do you normally feel in the mornings?
   a. Very refreshed.  
   b. Fairly refreshed.  
   c. Fairly tired.  
   d. Very tired.
4. How concerned are you about losing a night's sleep?
   a. Very concerned. 
   b. Fairly concerned. 
   c. Fairly unconcerned. 
   d. Not concerned at all.

5. Do you have any medical or other reason which prevents you from getting a good night's sleep regularly?
   Yes    No
   If Yes please tell us about the reason.

6. Have you any special technique or habit etc which you use to give you a good night's sleep?
   Yes    No
   If yes please tell us what it is.

7. Are you taking sleeping pills at the moment?
   Yes    No
   If yes how often do you take them?
   a. Every night. 
   b. Most nights a week. 
   c. Several times a month. 
   d. Once a month or less.

8. How long does it usually take you to fall asleep after turning out the light?
   a. Less than 10 minutes. 
   b. Up to 30 minutes. 
   c. No more than 1 hour. 
   d. More than 1 hour. 
   e. Longer (please specify) ............... 

9. What is your usual time of settling down to go to sleep?
   1. During the week.
      ........................................
   2. At the weekends (i.e. Friday/Saturday night)
      ........................................
10. How regular are these times from day to day?

   During the week - within ..... minutes
   At weekends (Friday/Saturday night - within ..... minutes.

11. How many times per night do you wake up, on average?
   a. Once or twice.
   b. No more than 5 times.
   c. Between 5 and 10 times.
   d. More than 10 times (please specify) ..... 

12. If you wake up during the night, how long does it usually take you to go back to sleep?
   a. Less than 10 minutes.
   b. Up to half an hour.
   c. No more than 1 hour.
   d. Longer than 1 hour (please specify) ..... 

13. When in bed can you hear loud noises from:-
   a. A busy main road.
   b. A busy main railway line.
   c. Any other sources outside the bedroom (please specify) ..... 

14. What type of mattress do you prefer to sleep on?
   a. Hard.
   b. Fairly hard.
   c. Fairly soft.
   d. Soft.
   e. No mattress.

15. What type of mattress do you actually sleep on?
   a. Hard.
   b. Fairly hard.
   c. Fairly soft.
   d. Soft.
   e. No mattress.

16. How clear headed do you usually feel after getting up in the morning?
   a. Still very drowsy indeed.
   b. Still moderately drowsy.
   c. Fairly clear headed.
   d. Alert.
   e. Very alert.
17. Do you ever have difficulty staying awake during the day?
   a. Never.
   b. Most days per week.
   c. Several times a month.
   d. Once a month or less.

18. At what time does the sleepiness start and how long does it last?
   Starts about (time) ........ Until about (time) ........

19. Is there usually a good reason for this?
   YES NO
   If Yes please give details
   ............................................................................

20. Do you have any medical condition which you are currently receiving treatment for?
   YES NO
   If Yes please give details ..............................................................

21. Sometimes one hears about people who 'feel best in the morning' or who 'feel best in the evening'. Which of these two types do you think you are?
   a. Definitely a 'morning' type.
   b. Rather more 'morning' than 'evening'.
   c. Neither one nor the other.
   d. Rather more 'evening' than 'morning'.
   e. Definitely an 'evening' type.
22. This question is concerned with how you decide that you have had a poor night's sleep. Below are some statements about sleep. Imagine that you had a poor night's sleep then ......

1. In the first column, put a tick by the 4 phrases that would most describe your sleep.

   I moved a lot during the night          1
   I took a long time to fall asleep       2
   My dreams made me anxious
   I had a headache on waking up
   I woke up a great deal
   I had many dreams
   I felt dizzy on waking up
   I was aware of thinking all night
   I felt very tired when I woke up finally
   Parts of me ached when I woke up

2. Now put another tick in Column 2 to show which of your 4 choices is the most important in your eyes.

23. If you have had a poor night's sleep, does it affect:–
   a. How you feel
   b. Your efficiency
   c. Both?

24. If a poor night's sleep affects you, when do you feel the consequences?
   a. The next day
   b. The day after
   c. Both

25. At what time of day?
   a. Throughout the day
   b. Mainly in the morning
   c. Mainly in the afternoon
   d. Mainly in the evening.

26. At what time of day do you have your last meal?
   a. Between 4pm and 6pm
   b. Between 6pm and 8pm
   c. Between 8pm and 10pm
   d. Later (please indicate the approximate time of finishing your meal).
27. How much does the quality of your sleep vary from one night to the next?
   a. Very much
   b. Moderately
   c. Slightly
   d. Not at all.

28. Which of the following best describes how you feel in the morning?
   a. Peaceful.
   b. Contented.
   c. Calm.
   d. Tense.
   e. Uneasy.
   f. Distressed.

29. How would you describe your general level of wakefulness once you have got up?
   a. Active/Energetic.
   b. Vigorous/Alert.
   c. Lively/Activated.
   d. Stimulated/Aroused.
   e. Drowsy/Tired.
   f. Idle/Sluggish
   g. Sleepy/Passive.

30. Do you lie awake worrying at night?
   a. Every night.
   b. Most nights a week.
   c. Several times a month.
   d. Once a month or less.
   e. Never.

31. Do you worry about your health?
   YES    NO
   If Yes, what aspect in particular? .............................................
   .................................................................
32. If you wake up during the night, what usually causes this?
   a. I don't know. Awake spontaneously.
   b. Nervous tension, worries.
   c. Need to pass urine.
   d. Shortness of breath or coughing.
   e. Pain in the chest.
   f. Pain in the stomach.
   g. Pain in the legs.
   h. Noise.
   i. Dreams or nightmares.
   j. Other causes (please specify).

33. Do your dreams cause you concern?
   a. Never.
   b. Occasionally.
   c. Frequently.
   d. Always.

34. How would you describe your level of wakefulness prior to going to bed?
   a. Active.
   b. Alert.
   c. Lively.
   d. Stimulated.
   e. Drowsy.
   f. Sluggish.
   g. Sleepy.

35. Have you had any illness in the past for which you have had time off work?
   YES     NO
   If Yes, please give details ..........................................................
   ........................................................................................................

36. Have you ever suffered a severe injury, or had any operations?
   YES     NO
   If Yes, please give details ..........................................................
   ........................................................................................................
37. Have any events you can think of caused you particular concern or anxiety?
   a. More than 2 years ago.
   b. More than 3 months ago.
   c. Less than 3 months ago.
   d. Current situations.

38. Do any of your relatives suffer with depression or anxiety?
    YES    NO
    If Yes, please state relationship of the person to yourself.

39. Have you ever worked night shifts?
    YES    NO

40. Have you ever noticed any change in the number of dreams you recall in the morning?
    YES    NO
    If Yes, please indicate when you noticed the change ...

41. Do you regularly take any pills or medicines from the chemist or prescribed by your doctor?
    YES    NO
    If Yes, please give details ...

42. Do people consider you to be a nervous person?
    YES    NO

43. Do you frequently dream about particular things?
    YES    NO
    If Yes, please give details of what you most often dream of ...

..........................................................
44. At what times of the day are you most likely to experience any of the following feelings. (Please enter appropriate letter against the times in the column below). For example, if you feel active, alert and wide awake at 7am to 9am enter 'a' in the column by 7am to 9am.

a. Active, alert and wide awake.

b. Functioning at high level but not at peak.
   Able to concentrate.

c. Relaxed, awake, not at full alertness, responsive.

d. A little foggy, not at peak, let down.

e. Fogginess, slowed down, starting to lose interest in remaining awake all day.

f. Sleepiness, preferred to be resting, fighting sleep, woozy.

g. Almost unable to stay awake, struggling to remain awake.

<table>
<thead>
<tr>
<th>Time</th>
<th>Letter</th>
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<tbody>
<tr>
<td>7am to 9am</td>
<td></td>
</tr>
<tr>
<td>9am to 11am</td>
<td></td>
</tr>
<tr>
<td>11am to 1pm</td>
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<tr>
<td>1pm to 3pm</td>
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<td>3pm to 5pm</td>
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<td>5pm to 7pm</td>
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<td>7pm to 9pm</td>
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<tr>
<td>9pm to 11pm</td>
<td></td>
</tr>
<tr>
<td>11pm to 1am</td>
<td></td>
</tr>
</tbody>
</table>

45. If you were to compare your feelings at home to your feelings at work would you describe yourself as being 'more' or 'less' of any of the following? Please tick under 'more' or 'less' against the appropriate words.

<table>
<thead>
<tr>
<th></th>
<th>'More'</th>
<th>'Less'</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tense</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bothered</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Up-tight</td>
<td></td>
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<tr>
<td>Distressed</td>
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<tr>
<td>Relaxed</td>
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<td>Contented</td>
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<tr>
<td>Comfortable</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calm</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
46. If you were to compare your feelings at home to your feelings when out socialising, would you describe yourself as being 'more' or 'less' of any of the following? Please tick under 'more' or 'less' against the appropriate words.

<table>
<thead>
<tr>
<th>Feeling</th>
<th>More</th>
<th>Less</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tense</td>
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<tr>
<td>Calm</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
47. Please indicate if you have experienced any of the following using the code below
   In the past - P
   At present - N
   Regularly - R

   Asthma
   Hay Fever
   Eczema
   Allergies
   Thyroid problems
   Migraine
   Breathing problems
   Undue anxiety
   Stammering
   Sleepwalking
   Loud snoring
   Nightmares
   Teethgrinding
   (while asleep)
   Difficulty reading or writing
   Arthritis
   Rheumatism
   Fibrositis
   Gout
   Depression
   Heart problems
   Stomach problems

   Waking up with a jerk
   Waking up early in the morning
   Difficulty falling asleep
   Difficulty staying awake during the day
   Being unable to move when you wake up
   Weakness in your limbs and/or trunk
   when you get excited
   Stress or anxiety at home or work
APPENDIX III
LOAD "NEW/SNEW/S"

10*DRIVE 1
20*FIL1,0
30CLS:loc1=1:4FF7
40DIM DR(5),C 40
50CLOSE EO
60REM SLEEP QUESTIONNAIRE ANALYSIS
70REM SLEEP PROBLEM DATA
80VDU 12
90REM CREATE A NEW FILE FOR EACH SET OF DATA
1000MODE 4
110PROC MENU_OPTIONS
120CLS
130IF opt=1 THEN PROC NEW_FILE
140IF opt=2 THEN PROC OLD_FILE
150IF opt=3 THEN PROC ADD TO_FILE
160IF opt=4 THEN PROC CHANGE
170PROC MENU_CONT
180VDU 12
190IF contal THEN RUN
2000OR E
210END
220
230
240
250DEF PROC NEW_FILE
260LOCAL I, Z
270VDU 12
280INPUT "NEW FILE NAME " NF$.
290PRINT "Please put disc in drive l" "Press any key when done"; G=S$ET
300CLS:OPEN OUT NF$.
310INPUT "NUMBER OF NEW DATA "; I
320FOR I=1 TO I
330PROC INFO
340NEXT
350CLOSE E
360ENDPROC
370
380DEF PROC INFO
390PRINT "ENTER DATA FOR EACH SUBJECT"
400PRINT "**************
410PRINT "SUBJECT NO." I
420PRINT "SUBJECT INITIALS" I
430PRINT "**************
440INPUT n.me$
450INPUT "SEX (Kl OR (F) s
460INPUT "AGE (yrs)" age
470INPUT "HEIGHT (5') = 1, (5') =2, (5') =3"; height
480INPUT "WEIGHT,(<65kg)=1, (>65kg)=2, (>75kg)=3"; weight
500IF weight>3 THEN GOTO 490
510INPUT "SMOKER,(no/yes)"; smoke
520INPUT "MARRIED(MA), SINGLE(SI), OR DIVORCED(DI)"; status$
530PRINT "ACTIVITY LEVEL"; "1=NO EXEVTION"; "2=LIGHT EXERCISE"; "3=REGULAR EXERCISE"; "4=REGULAR VIGOROUS SPORTS"
540INPUT "sport"
550INPUT "WORK CLERICAL(CL), MANUALL(MA), Non-MANUAL (NM), work$
560PRINT "USUAL SLEEP RATING"; "1=POOR","2=WELL","3=VERY WELL"
570INPUT "qual$
580PRINT "USUAL SLEEP LATENCY"
590PRINT "1=1St impr=1, >1hr=2hrs=
600INPUT "lat$
610PRINT "ALCOHOL CONSUMED PER NIGHT"
620INPUT "NO. OF DRINKS"; alc
630INPUT "LIE AWAKE WORRYING AT NIGHT,(Y/N/S)"; worr$
640INPUT "NUMBER OF AWAKENINGS"; ain
650INPUT "TIME OF WAKING IN MORNIN=1 before 6am", "2=6am to 8am","3=8am to 10am"
"=4=after 10am"; tk
660INPUT "ANY DIFFICULTY MAKING,(Y/N/S)"; diff$
670INPUT "TIME OF GETTING UP"; "1=6am", "2=6am to 8am","3=8am to 10am"
"4=10am"; getu
680PRINT "FEELINGS ON WAKING"
690INPUT "ALERT(3), RELAXED(2), OR TIRED(1)"; fil
700REM HOURS SLEPT DURING WEEK, WEEKEND, AND HOLIDAYS
710INPUT "AVERAGE NO. OF HOURS SLEPT"; hrs
720INPUT "HOURS THOUGHT TO BE NEEDED"; req
730PRINT "SYMPTOMS ON WAKING"
750PRINT "Bad Temper & Headache=BTH", "Bad Temper & Lethargy=BLT", "1 type discomfort=A", "2 types discomfort=B", "3 types discomfort=C", "Symptom free=SF"
760INPUT symp$
770INPUT "TIRED OR SLEEPY DURING THE DAY,(Y/N)"; tdd$


INPUT "SLEEPING PILLS (Y/N)"

INPUT "FILLS OR MEDICINES (Y/N)"

INPUT "REASONS FOR MEDICATION (Musculoskeletal MS" "Circulatory CL" "Gastrointestinal GI" "Genito-urinary GU" "Respiratory R"

"Psychiatric PS" "Nerve Disorder ND" "Endocrine GL"

INPUT "Allergies AL" "Skin SE" "Heart HE\" "Others OT\" "Two or more of above TOH"

INPUT "NIGHT SHIFTS, FAST OR PRESENT (Y/N)"

INPUT "WORRY ABOUT HEALTH (Y/N)"

INPUT "NO. OF SLEEP ANOMALIES"

INPUT "STRESS AT HOME OR WORK"

INPUT "NO. OF HEALTH PROBLEMS"

IS INFORMATION CORRECT Y/N

PROC_OLD_FILE
PROC_I$NU_OUTPUT
PROC_I$NU_OPTION6

PROC_MAIN

SUBJECT No
SUBJECT NAME
SEX
AGE
HEIGHT
WEIGHT
CIGARETTES SMOKED/DAY
MARITAL STATUS
ACTIVITY LEVEL
TYPE OF WORK
NUMBER OF AWAKENINGS
SYMPTOMS ON WAKING/AM
SLEEPING PILLS
TIME ON SLEEPING PILLS
FILLS OR MEDICINES
REASONS FOR MEDICATION
STRESS AT HOME OR WORK
USUAL SLEEP QUALITY
SLEEP LATENCY
ALCOHOL DRUNK/NIGHT
WORRY DURING NIGHT
DIFFICULTY AWAKENING AM.
TIME OF GETTING UP AM.
FEELINGS ON AWAKENING AM.
AVE. HOURS SLEPT/NIGHT
HOURS THOUGHT NECESSARY
DAILY SLEEPINESS
NIGHT SHIFTS
WORRY ABOUT HEALTH
NO. OF SLEEP ANOMALIES
NO. OF HEALTH PROBLEMS
DAYTIME SLEEPINESS
NIGHT SHIFTS
WORRY ABOUT HEALTH
NO. OF SLEEP ANOMALIES
NO. OF HEALTH PROBLEMS

PRINT "SUBJECT No"
PRINT "SUBJECT NAME"
PRINT "SEX"
PRINT "AGE"
PRINT "HEIGHT"
PRINT "WEIGHT"
PRINT "CIGARETTES SMOKED/DAY"
PRINT "MARITAL STATUS"
PRINT "ACTIVITY LEVEL"
PRINT "TYPE OF WORK"
PRINT "NUMBER OF AWAKENINGS"
PRINT "SYMPTOMS ON WAKING/AM"
PRINT "SLEEPING PILLS"
PRINT "TIME ON SLEEPING PILLS"
PRINT "FILLS OR MEDICINES"
PRINT "REASONS FOR MEDICATION"
PRINT "STRESS AT HOME OR WORK"
PRINT "USUAL SLEEP QUALITY"
PRINT "SLEEP LATENCY"
PRINT "ALCOHOL DRUNK/NIGHT"
PRINT "WORRY DURING NIGHT"
PRINT "DIFFICULTY AWAKENING AM.
PRINT "TIME OF GETTING UP AM.
PRINT "FEELINGS ON AWAKENING AM.
PRINT "AVE. HOURS SLEPT/NIGHT"
PRINT "HOURS THOUGHT NECESSARY"
PRINT "DAILY SLEEPINESS"
PRINT "NIGHT SHIFTS"
PRINT "WORRY ABOUT HEALTH"
PRINT "NO. OF SLEEP ANOMALIES"
PRINT "NO. OF HEALTH PROBLEMS"

PRINT "PRESS SPACE BAR TO CONTINUE"

PROC_MAIN
1600 IF S$="" THEN PROC_MENU_CONT
1620 VDU12
1670 LOCAL D#
1640 D$="" ""CONTINUE"
1650 D$="" ""GO TO MAIN MENU"
1660 D$="" ""END PROGRAM"
1670 cont=FN_MENU(2)
1680 ENDPROC
1690
1700 DEF FN_MENU (OPT)
1710 LOCAL F.A,1,X,LAST1
1720 VDU23,1,0;0;0;
1730 X=LEN(D$)+1/2
1740 CLS;PRINT "TAB(20-X-2);"
1750 I=0;REPEAT:I=I+1;PRINT "*";UNTIL I=LEN(D$)+4
1760 PRINT "TAB(20-X-2);I=I+1;PRINT "*";UNTIL I=LEN(D$)+4
1770 PRINT "TAB(20-X-2);I=I+1;PRINT "*";UNTIL I=OPT
1780 PRINT "TAB(4);OPTIONS:"
1790 I=0;REPEAT:I=I+1;PRINT TAB(10,9+2*I);I=I+1;PRINT "*";UNTIL I=OPT
1800 VDU28,0,31,39,25;COLOUR(2)\ VDU12;COLOUR(0)
1810 PRINT "TAB(6);<SPACE> Move to next option:" TAB(6);"<RETURN> Select the option:" VDU20,26
1820 I=1;LAST1=I
1830 FOR I=1 TO OPT
1840 COLOUR(0);PRINT TAB(10,9+2*I);#
1850 NEXT
1860 COLOUR(2);PRINT TAB(8,9+2*I-LAST1);" 
1870 IF I>OPT THEN I=1
1880 K=0
1890 IF K>1 THEN K=0
1900 COLOUR(2);IF K=0 THEN PRINT TAB(8,9+2*I); "ELSEPRINT TAB(8,9+2*I);""
1910 LAST1=I
1920 A=INKEY(50);IF A=I THEN K=K+1;GOTO 1890
1930 IF A=20 THEN I=I+1;GOTO 1860
1940 IF A=80 THEN GOTO 1960
1950 VDU27;GOTO 1920
1960 FX9.25
1970 FX10.25
1980 VDU20,23,1,1;0;0;0;CLS=1
1990
2000 DEF PROC_ADD_TO_FILE
2010 LOCAL1,I,
2020 VDU12
2030 INPUT "FILE NAME ";OF$
2040 INPUT "NUMBER OF NEW DATA ";I
2050 CM=OPENUP OF$#
2060 OPCODE=EXT CX%
2070 FOR K=1 TO Z
2080 PROC_INFO
2090 NEXT
2100 CLOSE
2110 ENDPROC
2120
2130 DEF PROC_MENU_OUTPUT
2140 VDU12
2150 LOCAL D#
2160 D$="" ""OUTPUT"
2170 D$="" ""SCREEN"
2180 D$="" ""PRINTER"
2190 out=FN_MENU(2)
2200 ENDPROC
2210
2220 DEF PROC_CHANGE
2230 VDU12
2240 INPUT "FILE NAME ";OF$
2250 INPUT "SUBJECT No ";I
2260 D$=OPENUP OF$#
2270 IF D$=0 PROC_NO_FILE1 GOTO 2260
2280 CM=OPENUP "TEMP"
2290 IF I PROC_FIND_REC
2300 PROC_INFO
2310 PROC_END_FILE
2320 CLOSE
2330 CLOSE
2340 VY=DIV256\ YOC DIV256
2350 DCM="DELETE ";OF$
2360 CALL asc11
2370 asc11="RENAME TEMP ";OF$
2380 CALL asc11
2390 ENDPROC
2400 DEF PROC_FIND_REC
DEF PROC_FIND_REC
LOCAL K
FOR K=1 TO 1-1
INPUT CDX, name$, sex$, age, height, weight, smoke, status$, sport, work$, aid$, sympf$, sed$, med$, medr$, stress$, qual
IF EOF$CDX GOT0 2610
INPUT CDX, lat$, alc$, worr$, mak$, difw$, getu$, felw$, wk$, req$, ldd$, osp$, nsf$, worf$, spr$, hist
PRINT CDX, name$, sex$, age, height, weight, smoke, status$, sport, work$, aid$, sympf$, sed$, med$, medr$, stress$, qual
NEXT K
ENDPROC

DEF PROC_END_FILE
IF EOF$CDX GOT0 2610
INPUT CDX, name$, sex$, age, height, weight, smoke, status$, sport, work$, aid$, sympf$, sed$, med$, medr$, stress$, qual
PRINT "READING"
INPUT CDX, lat$, alc$, worr$, mak$, difw$, getu$, felw$, wk$, req$, ldd$, osp$, nsf$, worf$, spr$, hist
PRINT CDX, lat$, alc$, worr$, mak$, difw$, getu$, felw$, wk$, req$, ldd$, osp$, nsf$, worf$, spr$, hist
PRINT CDX, lat$, alc$, worr$, mak$, difw$, getu$, felw$, wk$, req$, ldd$, osp$, nsf$, worf$, spr$, hist
NEXT K
ENDPROC

DEF PROC_NO_FILE
CLS
PRINT TAB(5,10) "FILE NOT ON THIS DISC" "CHANGE DISC AND PRESS ANY KEY"
GET
ENDPROC
I ur,D

"S.S"H:'l~"

'-L •

1 (lt1nnE7
:".'"Uf\ I '-!E0
3'.lCL(ISEfft

.q',)I NPlIT"r-r;:J IJ rOl IT";' t VIN) ": pr; ,,1'
!:i(IF'RI NT1AB (5,1 (I' "St.EEP QUEST IOt.,...,AlPE ANAL VS)S": rR INTTAB ~5. J J) ..... '" •••••••••••
.................... :PRINTTA9(1 ,2',,'''REAIW WIlH FlLENAME AND Nn.UF PATIENTS7'" '''PRES3 Ii
bOG=GET: VOU'3: CLS: IFprf I ,:t"" .. V" THENVOU2:
-:'1)) t.WUT"F ILE...,At1E"; -I i I e~
BOINPUT"NUMBER Or- PATIENTS ON FILE";~~
qOPRINT"=-============-="'===-=="=z===="
l(~'\.'DU~: 0..5: IFpri n~="Y"TI£N'''OU2
1 J-:-I1'100E7
12(I_oRIVEl
J :>(lC%=Or'EN
f i 1 eS
14QIFC7.-0 PROC_tlO_FILE:GOTOJ30
J SOol I'll (35) : DJ t'1nellref" (35) : 01 Mse::' (35) : 01 M... ye (~5) : 0 I Mhei ght (35) z DJ Mwej ght (:;~) : DJ
Hsrnol:e (3:::;': 0lMslatu ... t (35)
16(1[lH1Spc..,. l (:::5) : VI Mworl;J; {~~) : DIMej n C3~) : 0] M~ympt: (35) : D I M!iedS (3S) : 0 I Mmerlf: (:;~, : 0
(35) : 01 NIt; .( wl" ~ :~5) : D 1MIJetu (05) : 0 I I'1f eJ w (35) : DJ Mwl: (35)
1700 IMr-eq (35, : DJ Mtddr (3~) : D 1Musp (35) : DJ t'1nst' ('35) : D I Mwort' (;:;5) : OIt1spr (~5) : D] Nhi ~t

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(3~)

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(I:Pnea='OrPnor-O:PVuI ... O:P.le... O:Prel ....O:Ptir ...O
PI'!"P~~': Pr@'c=O:P'lre""'('I:Pmto=(1:Pmtt::(l:Pp! 1 =('1: P'lpi 1 =(1
260Pmus""-(l: Phea=Or Pres"'O, rYllt=(I: Pal J =0: Ppsy=O: Pvun=C'= Pder ...O: Pbl o=a(t: PQl aDO: Pnudc
/):F'e15=OIPngh ... O:Pnns=O:P... ah=('i:Pnwl'I ...O:Psah ...O:Ps .... H=O
27~'F'usr ... \':r"5r=O: Pvsr=O: P'lsl"'O: Pm-:.l=O: PI 51 =0: Pvs) =0: Pm.) e-O: F'fechi "'(1: Phl'll Q(I: ph",
bt=Q:P}a~t"'O:Poom ... O:Ptm=O:Pthr"'O:F'ok=O
290A=~··ge(]):M.ge%"'A/trl

::O(l} F!"p.:: $ <1 ) ..... F .. THEN of' p.mi ""f emi. J lPof' emi"" I NT ( ( (of' emi INi:) .·1 (0) *100+0. :i) f 1 OOELSEn,illl
e=mal e.1 : Pmal e=J NT ( ( (mal e/N"l.) _ 1 (1(1) "100+0.5) 1100
:::JOH=H.heiQhtll)zMhite%=H/N7.
320W=W~wp.iQht(J):MwAte7.=W/NX

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:o:.40IFstBtusS (l) ... ··SI "THENsi nasi n~J: Psi n=INT e( <si n/Ni:) .1(0) .100+0. 5) / JOOELSE350
35(1JFslatu~., (I) D"DI"nfENdi v=di v" 1 :Pd; v=INT « <di v/NX,) .10(1) .100+0. '5) 1100
36n IF smC'l:e(]»O THEN S=S+l:Smo7.... (SIt~7.).IOO
37(1]F BftIOl:.{]')(1 TIEN T~fttok .. TSCItOI.+smol{e(l) :MBmok7.c::Tsml'I~/S
3S0JF lIlcelJ)O THEN D=[l+I:Dri7.=CO/NX).IOO
40f'"JIFaport (], --I Tf-{E:Nnup-nue+l. Pnu_... INT « (nue/N%' .1(0) .100+0.5) IlooaSF..q 10
41(1 r rspo,'l Cl) -:2Tf-fE:Nl eJo: IEJ P-': ..... PI e.:- U"T ( ( (J Pr. INX)" t (to) • 100+0 .. 5) Il(lOELSEII20
~Z(llF!:oport (I) "':;T,--fENre>c",,.e:: + J: Pre)(I;"INT ( «re): IN7.) -1(0).1')0+(1. 5) IIOOELSE4~
~3(1) Ff'por·t (' )=4THE"N{ v'!"'=fvs+ I: Pfvo;:=INT ( «(vs/m) "S(IO) -1100+0. 5) 1100
44(1 J Fworl:.t (] ) ~"CL "THENeJ er=cl er" 1 : Pc 1 er=I NT { ( (cl er-/NY.) of! 1(JO) .1(10+0. S) 11 OOELSE45
4~(!1

r-wC'r I,' (I) ="MA"THEr"'m .. J,=lItllln" J ; rfllane 1NT ( ( ('"an/N"l.) _ 1(.10). 11.10+'). !j) 11 ClOtLSE4ltr..'
46(1' Fwo, 1':1" ( 1 ) Q "Nt1"THENnmiil=nIlUlI+ 1 : Pnmll=-] NT ( e (nma/NY.) • J 00) .1 (10+(1. 5) 1100
47·.. 1 Fq... IIl (I, .. J TlIE'Nt.,srausr-' 1 : Pusr-="'.4 r ( < (Llsr It.,~~) • J (1) 1"" J 0(1+(1. S) 11 OOELSE4EtO
~13(t1 Fqlutl (I' =2THE"N... ~r""wsr·+ll P..,sr"'INT ( ( (wsr/N7.'.' 00,., '.10+0. 5) / JOOELSE~90
49" J r qual ( J ) ""~THENgsrl;"gs, of 1 : Pvsr= INT· f ( (gsr IN~~' • 10(1).100.0.5) 11 (10
~I)(IJ Fl at' I ' el THENusl "ulil +1: PusI =- HIT < ( lust IN7., •• 00)'" HIO+O. 3) 1I (lOELSE51 (I
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990*NEW_FILE NAME "NF"
1000 PRINT "Allergies AL" "Skin DE" "Heart HE" "Others OTH" "Two or more of above MT" 1010 INPUT med$ 1020 INPUT "NIGHT SHIFTS, PAST OR PRESENT (Y/N)" ; ns$ 1030 INPUT "WORRY ABOUT HEALTH (Y/N)" ; wor$ 1040 INPUT "NO. OF SLEEP ANOMALIES" ; jspr 1050 INPUT "STRESS OR ANXIETY, (HOME OR WORK)" ; stress$ 1060 INPUT "NO. OF HEALTH PROBLEMS" ; hist$ 1070 PRINT "IS INFORMATION CORRECT Y/N " 1080 CHECK = GET$ 1090 IF CHECK = "N" THEN GOTO 420 1100 PRINT CC$ . name$ . sex$ . age. . height. . weight. . smoke. . status$. . sport. . work$. . sads. . pat$. . ins$. . tyd. . msds. . rel$. . cods$. . fps$ 1110 PRINT CC$ . find. . wad$. . qual. . lat. . al$. . wor$. . sin. . wak. . dsf. . getu. . felw. . wk. . req. . tsdf$ . edf$. . osp. . med$. . meed$. . ns$. . wor$. . spr$. . stress$. . hist$ 1120 ENDPROC 1130 1140 DEF PROC_old_FILE 1150 LOCALI 1160 VDU12 1170 INPUT "OLD FILE NAME " ; OF$ 1180 VDU12 1190 PROC_MENU_OUTPUT 1200 CC$ = OPENIN OF$ 1210 IF CC$ = 0 THEN GOTO 1200 1220 REPEAT 1230 = 1+1 1240 INPUT CC$ . name$ . sex$ . age. . height. . weight. . smoke. . status$. . sport. . work$. . sads. . pat$. . ins$. . tyd. . msds. . rel$. . cods$. . fps$ . find. . wad$. . qual. . lat. . al$. . wor$. . sin. . wak. . dsf. . getu. . felw. . wk. . req. . tsdf$ . edf$. . osp. . med$. . meed$. . ns$. . wor$. . spr$. . stress$. . hist$ 1250 IF out$ = 1 THEN VDU 14 ELSE VDU2 1260 OUT$ = 1 1270 OCLS 1280 PRINT TAB (10) ; "NUMBER " ; I 1290 PRINT TAB (10) ; name$ . TAB (15) . sex$ . TAB (30) . age 1300 PRINT TAB (10) ; "MA/SL" ; status$. TAB (12) . "HT" ; height . TAB (27) . "WT" ; weight 1310 PRINT TAB (10) ; "SMOKE" ; smoke$ . TAB (12) . "AL" ; sport$ . TAB (26) . "JOB" ; work$ 1320 PRINT TAB (10) ; "S/PIL$" ; sads$ . TAB (12) . "yr$" ; oas$ . TAB (26) . "stress$" 1330 PRINT TAB (10) ; "DRINK" ; jalc$ . TAB (11) . "MED$" ; med$ . TAB (26) . "FOR$" ; medr$ 1340 PRINT "USUAL SLEEP QUALITY " ; qual$ 1350 PRINT "SLEEP LATENCY " ; lat$ 1360 PRINT "WORRY DURING NIGHT " ; wor$ 1370 PRINT "TIME OF MAKING AM" ; getu$ 1380 PRINT "NUMBER OF AWAKENINGS" ; sin$ 1390 PRINT "DIFFICULTY MAKING AM" ; getu$ 1400 PRINT "TIME OF GETTING UP AM" ; getu$ 1410 PRINT "FEELINGS ON MAKING AM" ; felw$ 1420 PRINT "AVERAGE HOURS SLEPT/NIGHT" ; wk$ 1430 PRINT "HOURS THOUGHT TO NEED ☃️ " ; req$ 1440 PRINT "DAYTIME SLEEPINESS " ; tds$ 1450 PRINT "NIGHT SHIFTS " ; insf$ 1460 PRINT "WORRY ABOUT HEALTH " ; wor$ 1470 PRINT "AREAS WHERE DISCOMFORT " ; sads$ 1480 PRINT "PATTERN OF PAIN " ; patent$
<0x0>
1900 PRINT "No pills" : "sleep" : "3 months" : "noI%" : "1 year" : "need%" 
1 to 3 years" : "toB%" : "5 years" : "toB%" 
1970 PRINT "DIFFICULTY AWAKENING": PRINT "Yes" : "yes%" : "No" : "nsw%" 
1980 PRINT "Sometimes" : "yes%" : 
2000 PRINT "USUAL SLEEP LATENCY": PRINT "15 mins" : "use%" : "<1 hr" : "sm%" 
3050 PRINT "1 hr" : "sl%" : "2 hrs" : "sll%" 
2090 PRINT "Tired": "tir%" 
2040 PRINT "reason for medication": 
2060 PRINT "On medication": "ypr%" : "No medication": "npr%" 
2070 PRINT "Musculoskeletal": "paw%" : "Gastrointestinal": "gut%" : "Circulation": "bl%" 
2080 PRINT "Psychiatric": "ps%" : "Respiratory": "r%" : "Nerve Disorders": "nd%" : "Genito-urinary": "gun%" 
2090 PRINT "Endocrine": "end%" : "Allergies": "al%" : "Cardiac": "car%" 
2100 PRINT "Skin": "sk%" : "Other": "ot%" 
2110 PRINT "Two or more": "tpr%" 
2090 PRINT "Next": "next%" 
2120 PRINT "Sleepy during day": "psl%" 
2130 PRINT "Ave.no health problems": "pr%" 
2140 PRINT "Who worry about health": "wp%" 
2150 PRINT "Ave. no sleep anomalies": "an%" 
2160 PRINT "Night shifts (past or present)": "nsh%" 
2170 PRINT "Stress": "st%" 
2180 PRINT "Home": "hm%" 
2190 PRINT "Work": "w%" 
2200 PRINT "Do you wish a repeat of the results (Y/N)" 
2210 IF Rep$ = "Y" THEN GOTO 1660 
2220 IF Rep$ = "N" THEN CLS: GOTO 2230 
2230 IF Proc_NO_FILE = 1 THEN GOTO 1660 
2260 PRINT "FILE NOT ON THIS DISC" 
2270 GOTO 1660 
2280 IF Proc_NO_FILE = 0 THEN GOTO 2290 
2290 END
APPENDIX IV.
APPENDIX IV.

RESULTS OF A COMPARISON OF FIVE AND FIFTEEN SAMPLES OF SLEEP STAGE PERIODS.

SAMPLING METHOD.

For computation of mean percentages of alpha and delta per epoch, and percentage of epochs containing spindles in each sleep stage of a sleep EEG record, periods of uninterrupted sleep stage 2, 3, and 4 were chosen from the sleep stage analysis of each epoch. Means were then computed from the respective results of each analysis, (alpha, delta and spindles).

Comparison of analyses using five or fifteen periods of each sleep stage gave the following results.

TABLE A.

COMPARISON OF FIVE AND FIFTEEN SAMPLES IN ANALYSIS OF MEAN ALPHA AND DELTA PER EPOCH, AND PERCENTAGE OF EPOCHS CONTAINING SPINDLES.

<table>
<thead>
<tr>
<th></th>
<th>Number of Samples Used</th>
<th>Percentage Agreement</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5</td>
<td>15</td>
</tr>
<tr>
<td><strong>MEAN % ALPHA</strong></td>
<td></td>
<td></td>
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<tr>
<td>Stage 2</td>
<td>2.5</td>
<td>2.1</td>
</tr>
<tr>
<td>Stage 3</td>
<td>10.5</td>
<td>6.0</td>
</tr>
<tr>
<td>Stage 4</td>
<td>5.8</td>
<td>6.0</td>
</tr>
<tr>
<td>Mean NREM</td>
<td>5.2</td>
<td>4.4</td>
</tr>
<tr>
<td><strong>Mean % Agreement (Alpha)</strong></td>
<td></td>
<td></td>
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<tr>
<td><strong>MEAN % DELTA</strong></td>
<td></td>
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<tr>
<td>Stage 2</td>
<td>7.1</td>
<td>7.1</td>
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<tr>
<td>Stage 3</td>
<td>33.3</td>
<td>31.4</td>
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<tr>
<td>Stage 4</td>
<td>70.5</td>
<td>74.6</td>
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<tr>
<td>Mean NREM</td>
<td>42.4</td>
<td>40.7</td>
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<tr>
<td><strong>Mean % Agreement (Delta)</strong></td>
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<td></td>
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<tr>
<td>% EPOCHS WITH SPINDLES</td>
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<td></td>
</tr>
<tr>
<td>Stage 2</td>
<td>10.2</td>
<td>6.8</td>
</tr>
<tr>
<td>Stage 3</td>
<td>1.3</td>
<td>1.8</td>
</tr>
<tr>
<td>Stage 4</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>Mean NREM</td>
<td>4.0</td>
<td>3.1</td>
</tr>
<tr>
<td><strong>Mean % Agreement (Spindles)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean % Overall Agreement</td>
<td></td>
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APPENDIX V.
A STUDY OF THE RELATIONSHIP BETWEEN THE NREM SLEEP ALPHA ANOMALY AND MUSCULOSKELETAL DISCOMFORT IN A LOCAL POPULATION.

SUBJECT PROFILES.

Subject (A).
Subject (A) was a 34 year old male physical education teacher, who for five years had suffered with periodic widespread stiffness around his thighs, ankles and lower back, which was made worse by exercise but relieved by more exercise, although it persisted during rest periods. Discomfort was generally worse on awakening, but also started when he resumed physical exercise after long periods of inactivity.

He claimed to sleep very well for seven hours each week night. However he reported difficulties awakening, tiredness on awakening and a need for eight to nine hours sleep. He cycled thirty miles to work everyday and was generally very active and fit. Although he appeared confident and relaxed his questionnaire revealed that he was more likely to be tense and up-tight when at work and out socialising than at home.

Subject (B).
Subject (B) was a 23 year old male student who reported periodic musculoskeletal discomfort in the back, neck, shoulders and knees, sometimes worse on awakening, but without a specific pattern. His discomfort was made worse by running, heavy weight bearing and long periods of standing, but eventually relieved by strengthening exercises. He claimed that the discomfort had started three years previously, at which time he described he had been experiencing problems and anxiety whilst settling into University life, which were still present.

He also described sleeping well, feeling relaxed and alert on awakening after his habitual eight hours sleep, but was tired and sleepy during the afternoon. He sometimes lay awake worrying at night and also found some of his dreams quite disturbing.

Subject (C).
Subject (C) was a 23 year old male researcher, who described periodic stiffness in the back of his thighs and ankles, which had started four years previously and was worse when he was tired or 'run down'. He thought the discomfort might result from a lack of regular exercise as it increased with inactivity, and was relieved by exercise and fresh air. The discomfort had started during a period of illness (possibly glandular fever) whilst he was under intense academic pressure.

He reported sleeping well, although occasionally taking up to two hours to fall asleep, and feeling sluggish on awakening. He did not lie awake worrying but described difficulty 'getting things out of my mind' especially the next days work, feeling irritable if things didn't go his way, and suffering with bouts of depression and anxiety. He
later reported frequent episodes of sleepwalking during periods of work-related anxiety.

**Subject (D).**

Subject (D) was a 20 year old physically active male student, who described periodic stiffness in the lower back, calves and ankles over the last four years. This was worse in the morning, although it often started at the end of a long day. It was increased by bending forward, and sometimes relieved by massage. He attributed his discomfort to persistent misuse during tennis and lacrosse.

He was an habitually long sleeper (average 10hrs), but lay awake worrying most nights, although he generally slept very well. He reported 'extreme difficulties' waking up, feeling distressed and very tired and sleepy during the day. He described occasional whole body 'spasms' whilst dozing, a great need to move his legs in bed and occasionally found he was unable to move when he woke up. Although he was outwardly cheerful, he mentioned that he was worried about his teeth as he was concerned about going to see the dentists due to his fear of injections.

**Subject (E).**

Subject (E) was a 21 year old male student who reported poor sleep. Although he habitually slept nine hours, he complained of difficulties awaking, daily persistent tiredness and sleepiness, discomfort, lethargy and headaches, for which he took paracetamol several times a week. He also reported sleeptalking, nightmares and weakness when he laughed or got excited, undue anxiety and depression. He was a quiet and pleasant person who was physically active, health conscious and a vegetarian.

**Subject (F).**

This 22 year old male reported periodic generalised heaviness and aching on awakening which was exacerbated by tiredness. He slept well, although he occasionally lay awake worrying, had problems falling asleep and difficulties waking up. He felt tired and sleepy on awakening when he regularly experienced bad 'temper' and lethargy. He was concerned about his health, particularly diet, and was also a vegetarian.

**Subject (G).**

This 49 year old married lady had recently undertaken a voluntary redundancy due to her previous working conditions, and commenced a degree course away from home. She reported poor sleep, lying awake worrying, prolonged sleep latency and awakenings during the night. In the mornings she described difficulties awakening, tiredness and sleepiness, aching, pain and stiffness in her hands, hips, back and neck, headache and lethargy. About once a month she took a short acting sleeping pill, to overcome her problems getting to sleep. She was generally in good health but had suffered with migraines, arthritis, rheumatism, heart problems, undue anxiety and depression.
Subject (H).

Subject (H) was a 37 year old female student who reported poor sleep, and regularly awoke with backache, neck and shoulder stiffness and lethargy. Although she slept seven hours per night, she took up to two hours to fall asleep, often lying awake worrying, and awakening two or three times during the night. She found difficulty awakening in the morning and regularly felt tired throughout the day. She had been divorced two years before and had recently left her job due to stress. Following her divorce she was prescribed tranquillisers for her sleeping problems but was no longer using them.

She worried about her health, in particular she was concerned she may have inherited heart disease and bronchitis from her parents. She reported occasional attacks of anxiety and depression, and had had time off work due to stress related anxiety.

Subject (I).

This 22 year old student complained of very intense lower back pain, which occurred periodically during the day, and was made worse by lifting and moving heavy objects. She thought that the pain was due to her left leg being 2cm shorter than her right, causing her spine to bend, which had affected her for several years. Ponstan (analgesic tablets) had been prescribed, but she tried not to take these too often.

She reported sleeping very well, and quite deeply for about eight hours per night, although she awoke two or three times during the night and always felt tired on awakening and throughout the day.

She had been knocked down by a car in 1981, and sacked from her job in 1982 after which she had become 'almost suicidal' and suffered severe depression. Following this, in 1984 her marriage broke up after nine months. She was fairly content at the time of completing the questionnaire but described bouts of depression when she was tense or tired, and occasional nightmares.

Subject (J).

This 40 year old woman complained to her general practitioner of continuous low back pain and periodic sacral pain, which was increased by immobility. She had suffered with this discomfort for many years although the cause was unknown, but had been labelled as 'Fibrositis' by one doctor.

She slept poorly, taking up to two hours to fall asleep, and awaking several times during the night due to back pain, but she reported no difficulties waking up and only occasional daytime tiredness. She had had business problems two years previously and had suffered with a peptic ulcer, but had since given up work and spent the day doing very little. As her husband was away she usually retired when she became anxious about being alone, and sometimes lay awake worrying at night. Her back pain and continuing stomach problems caused her particular concern. She was known to snore loudly, especially after alcohol.
Subject (K).
This 27 year old doctor complained of periodic aching and stiffness in the backs of his thighs and calves, increased by tiredness and relieved by elevating his legs. This discomfort was worse in the mornings and evenings and had first been noticed about five years ago.

He reported sleeping very well, falling asleep very quickly, and awakening during the night only when he was on call or to visit the bathroom. He regularly slept seven and a half hours per night, but felt tired and sleepy during the day.

He had recently experienced financial problems, in addition to anxieties and depression regarding his job and medical examinations. He also occasionally suffered with unilateral migraines, irritability, stomach problems and shortness of breath due to cold or exercise.

Subject (L).
This 35 year old lady complained of periodic generalised stiffness, aching and tenderness of unknown cause which affected her lower back, shoulders and limbs. This was made worse by cold, wet weather and periods of inactivity, and relieved by gentle movement and warmth. She first noticed the discomfort thirteen years before but also described 'growing pains' as a child. The discomfort generally began whenever she felt tired or sleepy and was not always worse in the morning.

She reported sleeping very well for eight hours per night, with a very short sleep latency, and few nighttime awakenings. However she described difficulties waking up (usually between 0430hrs and 0600hrs), with tiredness and sleepiness during the day.

This lady lived alone with her young daughter, since the father had left after a long period of domestic disharmony. She had always suffered with a form of endogenous depression and found that various past events had contributed to this, although she was reasonably happy at the time of the study.

Subject (M).
This 43 year old lady described continuous pain in her lower limbs and left shoulder blade, periodic stomach and chest discomfort and brief sharp pains in her hands and feet. In general, the discomfort was exacerbated by coldness, dampness and inactivity, and relieved by warmth, rest and elastic leg hose supports. She attributed the problems to Raynauds Disease, mild arthritis and poor muscles. The symptoms had been evident since 1958, and she found them frustrating as they restricted her activity.

She reported sleeping very well and very deeply, falling asleep quickly unless in pain, but having difficulties awakening in the morning, feeling tired and sleepy. She felt she needed about ten hours sleep, although she habitually slept about seven per night. She described a 'whole body tiredness' after doing very little work, which was particularly bad in the morning.

This divorced lady was a live-in housekeeper for a middle aged man, who also allowed her two daughters to live with her. She described feeling frustrated about the current living situations and the limitations imposed upon her by
her discomfort, and was also worried about her 19yr old daughter who had a heart condition which was influencing her employment prospects. She had previously received treatment for an underactive thyroid, which was discontinued as the doctors disagreed on the diagnosis.

**Subject (N).**

This 50 year old lady complained of aching in the neck and the lower legs particularly aggravated by strain and tiredness at the end of the day. Her leg discomfort deterred her from activity, and she found that she became tired after doing very little work. She gained partial relief by taking hot baths and keeping warm, but her lack of vitality concerned her.

She reported sleeping well, although she took up to an hour to fall asleep, and experienced a great need to move her legs in bed, as well as leg 'twitching'. She had difficulties waking, feeling tired and sleepy and often waking with headaches and bad temper, and taking a long time to get going mentally and physically.

There had recently been family disagreement over financial inheritances and this had created bad feeling between her and her sister in law, which caused her anxiety. In addition her husband was experiencing problems in the family business and during the period of study was sacked by his brother. Previous to these events her father had suddenly died of a heart attack and her daughter had been seriously ill.

**Subject (P).**

Subject (P) was a 31 year old nurse who gave up her community midwife job to attend University. She described periodic tenderness in her lower back and lateral aspect of her right thigh, which was aggravated by exercise and weight gain, (due to hormonal imbalance). This was sometimes worse in the morning but also occurred after long periods of sitting down. She attributed her discomfort to unstable joints and attended a chiropractor for manipulation, which she could also perform herself to some extent.

She reported sleeping very well, with no difficulties awakening, and no complaints of daytime tiredness except after a heavy meal. However due to her unstable joints she woke up during the night when she turned over, but immediately went back to sleep. Although she did not report any recent stressful experiences, she occasionally suffered with anxiety and depression, particularly pre-menstrually.

**CONTROL SUBJECTS PROFILES.**

**Control Subject (1).**

This subject was a 22 year old undergraduate engineer, who was physically fit and reported sleeping deeply for at least eight hours per night. He was approaching his final year examinations at the time of study and expressed some anxiety with regard to this but described no other anxieties, stress or depression. He was free of health problems and described no sleep anomalies.
Control Subject (2).
This 24 year old postgraduate student was physically fit, very active and without health problems. She described habitually undisturbed good sleep of seven to eight hours duration, was free of medication, stress or anxiety.

Control Subject (3).
This twenty two year old student reported good sleep and was healthy and free of medication. She was moderately fit, participating in sporting activities at least four times a week. She described sleeping seven to eight hours per night and was free of anxiety and depression, although she was under some academic stress at the time of study as she was approaching her final year examinations.

Control Subject (4).
This student was also 22 years old and reported good sleep. She was healthy and moderately physically active but at the time of study was worried about her work and achievements in life-saving examinations. She was outwardly lively and happy but appeared to worry unduly about things.

Control Subject (5).
This subject was a 23 year old postgraduate student, who smoked ten cigarettes a day, and occasionally played squash. He described sleeping well, but occasionally lay awake worrying at night, although he rarely awoke during the night. He reported occasionally awaking with lethargy, but had no daytime tiredness or sleep problems. He suffered from asthma and a recent development of skin disorders but also described current anxiety and mild depression, attributed to recent emotional stress from a break up of a relationship.

Control Subject (6).
This 20 year old student smoked twenty cigarettes a day and was moderately physically active. She slept well, sometimes worrying at night but was without any other sleep difficulties. She described lethargy on awakening, but was free of daytime tiredness. She reported regularly sleepwalking, waking with a jerk and having a great need to move her legs in bed. Although she was in good health she occasionally suffered with hay fever and migraines. She had no anxiety or depression and did not describe any recent stressful events.

Control Subject (7).
This was a 25 year old postgraduate student who slept well for seven to eight hours a night and reported a very short sleep latency (2 min). He was free of sleep problems and was in good health although he regularly suffered from asthma and hay fever, but was free of stress, anxiety or depression.

Control Subject (8).
This subject was a 21 year old undergraduate student who regularly participated in strenuous sports. He slept well but sometimes lay awake worrying at night. He occasionally experienced a great need to move his legs in bed, but was generally free of sleep problems. He reported having eczema and occasional bouts of undue anxiety and
depression. He described a recent short stay in hospital for a knee operation as stressful as it was during term-time in his final year of study.

**Control Subject (9).**
This was a 22 year old student who was not very physically active. She slept well for about eight hours a night, and was occasionally awoken during the night although she returned to sleep within a few minutes. She was free of health problems, but reported grinding her teeth whilst asleep. She reported no stress or anxiety.

**Control Subject (10).**
This subject was a 38 year old technician, married with one child. He described sleeping well, occasionally being awoken by the child but generally without any problems. He was free of sleep anomalies, stress and anxiety, but suffered with stomach problems which were described as not being related to stress but attributed to too long periods between meals. He was not taking any medication for this.

**Control Subject (11).**
This subject was a 31 year old divorcee, whose recreational activities included spiritual sight-seeing, meditation and artistic creativity. She described sleeping very well, without any problems or symptoms on awaking. She regularly took acupuncture treatment for general health maintenance and reported only occasional stomach problems. She reported no recent stressful experiences, anxiety or depression. At the time of study she had decided to give up her job and return to studying, and expressed some excitement at having made this decision.

**Control Subject (12).**
This subject was a 40 year old divorced lecturer who lived alone with occasional visits from his two children. He was physically active and described sleeping well, but occasionally lying awake worrying at night, although he rarely experienced any sleep problems. He occasionally awoke with a headache but was free of sleep anomalies, health problems, stress and anxiety.
APPENDIX VI.
APPENDIX VI.

SUMMARY OF MAIN RESULTS OF THE SLEEP EEG STUDY QUESTIONNAIRE: COMPLETED BY SUBJECTS PARTICIPATING IN THE SLEEP EEG STUDY.

Table (B) shows the information regarding sleep characteristics obtained from nine good sleepers with discomfort, three poor sleepers with discomfort and six control subjects.

<table>
<thead>
<tr>
<th></th>
<th>Good</th>
<th>Poor</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Sleep+Disc</td>
<td>Sleep+Disc</td>
<td>Group</td>
</tr>
<tr>
<td></td>
<td>(n=9)</td>
<td>(n=3)</td>
<td>(n=6)</td>
</tr>
<tr>
<td><strong>Sleep quality:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very well</td>
<td>5</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Satisfactorily</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Some problems</td>
<td>2</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Poorly</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td><strong>Enjoy Sleep?</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very much</td>
<td>5</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Moderately</td>
<td>4</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Not much</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Not at all</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Sleep Onset</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 10 mins</td>
<td>5</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Up to 30 mins</td>
<td>2</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>&gt; 1 hr</td>
<td>2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>&gt; 1 hr</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>No. of awakenings.</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Once or twice</td>
<td>7</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>No &gt; 5 times</td>
<td>0</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>5 to 10 times</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>&gt; 10 times</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Quality of sleep vary?</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very much</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Moderately</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Slightly</td>
<td>5</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Not at all</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>
Table (C) summarises the feelings on awakening reported by subjects in each group.

**TABLE C. RESULTS OF THE SLEEP EEG STUDY QUESTIONNAIRE: FEELINGS ON AWAKENING.**

<table>
<thead>
<tr>
<th>Feeling</th>
<th>Good Sleep+Disc (n=9)</th>
<th>Poor Sleep+Disc (n=3)</th>
<th>Control Group (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Refreshed in the morning?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Fairly</td>
<td>1</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Fairly tired</td>
<td>5</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Very tired</td>
<td>3</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Clear headed in the morning?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very drowsy</td>
<td>4</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Mod. drowsy</td>
<td>2</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Fairly C/H</td>
<td>1</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Alert</td>
<td>0</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Very alert</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Feelings in the morning?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peaceful</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Contented</td>
<td>2</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Calm</td>
<td>1</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Tense</td>
<td>1</td>
<td>1*</td>
<td>0</td>
</tr>
<tr>
<td>Uneasy</td>
<td>4</td>
<td>1*</td>
<td>0</td>
</tr>
<tr>
<td>Distressed</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>General level of wakefulness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Once up</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Active/Energetic</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Vigorous/alert</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Lively/Activated</td>
<td>0</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Stim’tld/Aroused</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Drowsy/tired</td>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Idle/Sluggish</td>
<td>1</td>
<td>2*</td>
<td>0</td>
</tr>
<tr>
<td>Sleepy/Passive</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

(*) One subject omitted this question.
The following tables summarise the results to question (21) in the sleep EEG study questionnaire. Subjects were asked to tick the four most likely reasons they would give to describe their sleep after a poor night's sleep. Table (D) shows the results of this question. Table (E) shows the results when subjects were asked to tick the one statement, which they considered was most important in their eyes.

**TABLE D.** RESULTS OF THE SLEEP EEG STUDY QUESTIONNAIRE: FREQUENCY OF STATEMENTS USED TO DESCRIBE A TYPICAL POOR NIGHT'S SLEEP IN EACH GROUP.

<table>
<thead>
<tr>
<th>Statement</th>
<th>Good Sleep+D (n=9)</th>
<th>Poor Sleep+D (n=3)</th>
<th>Control Group (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I moved a lot during the night</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>I took a long time to fall asleep</td>
<td>7</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>My dreams made me anxious</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>I had a headache on waking up</td>
<td>2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>I woke up a great deal</td>
<td>4</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>I had many dreams</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>I felt dizzy on waking up</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>I was aware of thinking all night</td>
<td>4</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>I felt very tired when I awoke</td>
<td>7</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Parts of me ached when I woke up</td>
<td>6</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

**TABLE E.** RESULTS OF THE SLEEP EEG STUDY QUESTIONNAIRE: MOST IMPORTANT REASONS FOR A POOR NIGHT'S SLEEP.

<table>
<thead>
<tr>
<th>Statement</th>
<th>Good Sleep+D (n=9)</th>
<th>Poor Sleep+D (n=3)</th>
<th>Control Group (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I moved a lot during the night</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>I took a long time to fall asleep</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>My dreams made me anxious</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>I had a headache on waking up</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>I woke up a great deal</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>I had many dreams</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>I felt dizzy on waking up</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>I was aware of thinking all night</td>
<td>1</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>I felt very tired when I awoke</td>
<td>4</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Parts of me ached when I woke up</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Table (F) shows the effects of a poor night's sleep as reported by the three groups in the main sleep study questionnaire.
### TABLE F. 
**RESULTS OF THE SLEEP EEG STUDY QUESTIONNAIRE:**
**DAYTIME FEELINGS.**

<table>
<thead>
<tr>
<th></th>
<th>Good Sleep+D (n=9)</th>
<th>Poor Sleep+D (n=3)</th>
<th>Control Group (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A poor night’s sleep affects.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>How you feel</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Your efficiency</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Both</td>
<td>0</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>When?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>The next day</td>
<td>5</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>The day after</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Both</td>
<td>2</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>At what time of day?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Throughout the day</td>
<td>5</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Mainly in the morning</td>
<td>3</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Mainly in the afternoon</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Mainly in the evening</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Difficulty staying awake in day?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>1</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Most days per week</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Several times a month</td>
<td>3</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Once a month or less</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

Table (G) shows the most likely reasons chosen by the three groups for being awoken during the night.

### TABLE G. 
**RESULTS OF THE SLEEP EEG STUDY QUESTIONNAIRE:**
**REASONS FOR AWAKING DURING NIGHT.**

<table>
<thead>
<tr>
<th></th>
<th>Good Sleep+D (n=9)</th>
<th>Poor Sleep+D (n=3)</th>
<th>Control Group (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Don’t know: wake spontaneously</td>
<td>4</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Nervous tension: worries</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Need to pass urine</td>
<td>1</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Shortness of breath/ coughing</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Pain in the chest</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Pain in the stomach</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Pain in the legs</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Noise</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Dreams or nightmares</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Other</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Table (H) shows the frequency of worries and anxieties reported by the three groups.
TABLE H. RESULTS OF THE SLEEP EEG STUDY QUESTIONNAIRE:
FREQUENCY OF WORRIES AND ANXIETIES.

<table>
<thead>
<tr>
<th></th>
<th>Good Sleep+D (n=9)</th>
<th>Poor Sleep+D (n=3)</th>
<th>Control Group (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Do you lie awake worrying?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Every night</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Most nights a week</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Several times a week</td>
<td>3</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Once a month or less</td>
<td>4</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Never</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Have any events caused you anxiety?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>More than 2 years ago</td>
<td>2*</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>More than 3 months ago</td>
<td>2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Less than 3 months ago</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Current situations</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Do your dreams cause you concern?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>4</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Occasionally</td>
<td>4</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Frequently</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Always</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

(*) One subject omitted this question.

Table (J) gives the frequency of reported sleep anomalies from the preliminary questionnaires completed by all subjects in all groups.

TABLE J. RESULTS OF PRELIMINARY QUESTIONNAIRE ANALYSIS:
FREQUENCY OF REPORTS OF SLEEP ANOMALIES.

<table>
<thead>
<tr>
<th></th>
<th>Good Sleep+D (n=11)</th>
<th>Poor Sleep+D (n=4)</th>
<th>Control Group (n=15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleepwalking</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Sleepwalking</td>
<td>2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Nightmares</td>
<td>0</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Bruxism</td>
<td>2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Wake up with a jerk</td>
<td>4</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Wake up early (morning)</td>
<td>3</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Difficulty falling asleep</td>
<td>4</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Need to move legs in bed</td>
<td>4</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Difficulty staying awake (day)</td>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Unable to move on waking</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Limb/trunk weakness</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>(when laughing or excited).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Snoring</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Napping</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

Table (K) shows the frequency of reported medical anomalies taken from the preliminary questionnaires of all study subjects. (The anomalies are generally based on subjective evaluation of experiences, and not on medical diagnoses).
TABLE K. RESULTS OF PRELIMINARY QUESTIONNAIRE ANALYSIS:
FREQUENCY OF MEDICAL ANOMALIES.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Good Sleep +Discomfort (n=11)</th>
<th>Poor Sleep +Discomfort (n=4)</th>
<th>Control Group (n=15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asthma</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Hay Fever</td>
<td>3</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Stammering</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Eczema</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Allergies</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Thyroid Problems</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Migraine</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Breathing Probs</td>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Difficulty reading/writing</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Arthritis</td>
<td>3</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Rheumatism</td>
<td>2</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Gout</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Depression</td>
<td>6</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Heart Problems</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Stomach Problems</td>
<td>4</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Anxiety</td>
<td>3</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

Table (L) gives the subjective evaluation of morningness/eveningness taken from the study questionnaire.

TABLE L. MORNING AND EVENING TYPES IN MAIN SLEEP STUDY SUBJECTS:
SUBJECTIVE EVALUATION.

<table>
<thead>
<tr>
<th>Subjective Evaluation</th>
<th>Good Sleep (n=11)</th>
<th>Poor Sleep (n=3)</th>
<th>Control Group (n=5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definitely 'morning' type</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>More 'morning' than 'evening'</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Neither one nor other</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>More 'evening' than 'morning'</td>
<td>3</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Definitely 'evening' type</td>
<td>4</td>
<td>2</td>
<td>0</td>
</tr>
</tbody>
</table>
APPENDIX VII.
APPENDIX VII
RESULTS OF FOURIER ANALYSIS.

GOOD SLEEPERS WITH DISCOMFORT.

SUBJECT (B): Sleep.
This subject showed frontal alpha-like activity in sleep stages 2, 3, and 4, and occipital alpha activity during stage 2 sleep and REM sleep.

The frequency and amplitude of frontal alpha-like activity during stage 2 sleep was 7Hz and 34.7µV, and 7Hz and 37.6µV during sleep stage 3 but during sleep stage 4 the frequency ranged between 7.4Hz and 10.4Hz (33.8µV).

During REM sleep some 7.8Hz alpha activity was observed occupitally, although the amplitude was small (21.2µV).

Wakefulness.
The occipital alpha frequency during relaxed wakefulness with eyes closed was 9Hz (205µV) increasing to 9.2Hz (167µV) when the eyes were opened. During mental arithmetic, with eyes shut, occipital alpha frequency continued to be 9.2Hz, (137µV) increasing again to 9.4Hz (94.6µV) during mental arithmetic with the eyes open. Frontal alpha-like activity was not seen during waking conditions.

SUBJECT (C): Sleep.
This subject showed frontal NREM alpha-like activity during sleep stages 2, 3, and 4. He also showed some frontal alpha-like activity during REM sleep, in addition to occipital alpha.

Frontal alpha-like activity during sleep ranged from 9Hz (287µV) during stage 2, to 7Hz (283µV) during stage 3 sleep. In stage 4 sleep a peak of frontal alpha-like activity of 10Hz (142µV) was seen. Frontal REM sleep alpha frequency ranged between 7.4Hz (87.8µV) and 9.2Hz (87.8Hz) whereas the occipital alpha frequency was 8.4Hz (33.8µV). Some (7.4 to 9.4Hz, 29.9µV) activity was also observed occupitally during stage 4.

Wakefulness.
During relaxed wakefulness, with eyes shut, occipital alpha was prominent at 10 to 10.2Hz (101µV) and there was evidence of some frontal activity at a frequency of 8.4Hz (77.2µV). Frontal alpha-like activity of 7.8Hz (58.99µV) appeared when the subject relaxed with his eyes open. Both frontal (8Hz, 71.4µV) and occipital (10.2Hz, 117µV) activity were found during mental arithmetic with the eyes open, but both frontal and occipital activities were attenuated and slowed when the eyes were closed. Frontal and occipital activity was then found at a frequency of 8.4Hz.

SUBJECT (D): Sleep.
Subject (D) showed very low amplitude (29.9µV) occipital 8.2Hz alpha activity during REM sleep, but no occipital alpha during NREM sleep stages 2, 3, and 4.

Wakefulness.
Alpha activity (10.6Hz) during relaxed wakefulness was also diminished in amplitude (43.4µV) compared to that seen in most subjects. Occipital alpha activity completely disappeared when the eyes were opened, but reappeared during
the mental arithmetic eyes shut condition (10.4Hz), when its amplitude (58.9μV) slightly exceeded that seen during relaxed wakefulness. Occipital alpha was diminished (25μV) when the eyes were opened during mental arithmetic but was still evident at a frequency of 9.8Hz.

**SUBJECT (F): Sleep.**

Frontal NREM alpha-like activity was found in sleep stages 2 and 4 of this subject's sleep. No frontal alpha activity was seen during stage 3 sleep or REM sleep.

During sleep stage 2, alpha-like activity in the frequency range of 9.4 to 11Hz (max 70.4μV) was seen frontally, in addition to some occipital activity at 8.4Hz (95.5μV). During stage 3 sleep, some 8.6Hz activity was evident frontally and 8.8Hz occipitally, but both of these were of very small amplitude, (29.9μV max).

Stage 4 sleep analysis showed peaks of frontal activity in the range 7.4 to 9.2Hz (max 22.2μV). During REM sleep, there was evidence of some occipital activity at 10.8Hz (22.2μV).

**Wakefulness.**

Relaxed wakefulness with eyes closed produced occipital alpha of 10.4Hz (71.4μV), which went when the eyes were opened. During mental arithmetic with eyes shut, a large peak (70.4μV) of 10.8Hz occipital alpha was evident, which went completely when the eyes were opened.

**SUBJECT (K): Sleep.**

Some alpha-like activity (7.2Hz, 64.7μV) was detected frontally during stage 4 sleep in this subject. Some occipital activity of 8.8Hz (90.7μV) was also observed during stage 4 sleep, and stage 3 sleep, (8.6Hz, 50μV).

During REM sleep, there were peaks of occipital activity in the alpha range from 7.6 to 10.4Hz, (max 90.7μV).

**Wakefulness.**

Peaks of 9.8 to 11Hz, (87.8μV) occipital alpha activity were observed during relaxed wakefulness with eyes shut, but decreased in amplitude (41.5μV) when the eyes were opened. During mental arithmetic with eyes shut, occipital alpha activity of 10.6Hz (127μV) was observed, and this diminished in frequency (to 9.8Hz) and amplitude (45μV) when the eyes were opened.

**SUBJECT (L): Sleep.**

A small rise in amplitude was observed frontally during sleep stage 2, at a frequency of 7.6Hz, (33.3μV) but none during sleep stages 3 or 4.

During REM sleep, occipital alpha activity of 8 to 9.8Hz frequency (max 63.7μV) was seen.

**Wakefulness.**

Relaxed wakefulness with eyes shut produced a large peak of occipital 10Hz activity, (139μV) which reduced in amplitude (37.6μV) but increased in frequency to 10.8Hz when the eyes were open. The same pattern of frequency change was seen during mental arithmetic (eyes shut and eyes open).
SUBJECT (M): Sleep.
Subject (M) showed 7.2Hz (93.6μV) occipital alpha activity during stage 2 sleep, and low amplitude (36.7μV) 8Hz occipital activity during REM sleep.

Wakefulness.
During relaxed wakefulness, subject (M) showed a small amplitude (28μV) peak of occipital 7.4Hz activity, which disappeared when the eyes were opened. No peaks were observed during mental arithmetic.

SUBJECT (P): Sleep.
Subject (P) exhibited frontal alpha-like activity during sleep stages 2 (7.8Hz, 38.6μV) and 3 (7.4Hz, 36.7μV), and occipital alpha activity during sleep stage 2 (8Hz, 47.3μV). Some occipital alpha activity of low amplitude was also detected during stage 4 sleep (9Hz, 25.1μV) on the second recording, but not during the first.

A peak of occipital alpha activity was found during REM sleep at a frequency of 8.8Hz (45.4μV). On other nights REM sleep alpha was found at a frequency of 8Hz (86.9μV), and at 7.6Hz (51.1μV).

Wakefulness.
During relaxed wakefulness with eyes shut, this subject showed a peak of occipital alpha (139μV) at 9.4Hz, which slowed to 9.2Hz and diminished in amplitude (to 69.5μV) when the eyes were opened. A small increase (to 29.9μV) in frontal activity in the range 4.8Hz to 9.8Hz was also evident when the eyes were shut..

During mental arithmetic with eyes shut, an occipital alpha peak at 9.6Hz (179μV) was apparent, and this also slowed to 9.4Hz and diminished in amplitude (97.5μV) during mental arithmetic with the eyes open.

CONTROL SUBJECTS: RESULTS OF FOURIER ANALYSIS.

CONTROL SUBJECT (S): Sleep.
This subject showed occipital alpha during sleep stage 2, (8.6Hz, 45.4μV) and 3 (7.4Hz, 56.9μV), but none during sleep stage 4. During REM sleep, prominent occipital alpha peaks between 8.4Hz 93.6μV) and 9Hz (78.2) were seen.

Wakefulness.
A large peak (243μV) of occipital alpha activity (10Hz) was seen during relaxed wakefulness with the eyes shut, but disappeared completely when the eyes were opened. During mental arithmetic with the eyes shut, a smaller amplitude alpha peak (18μV) was seen at the same frequency, and this diminished in amplitude (to 51.1μV) and frequency (to 11Hz) when the eyes were opened.

CONTROL SUBJECT (6): Sleep.
Occipital alpha activity of 7.8Hz (64.7μV) to 8.6Hz (60.8μV) was found during sleep stage 2, whereas occipital alpha of 9.4Hz (64.7μV) was seen during sleep stage 3, but none during stage 4 sleep. During REM sleep, a large (71.4μV) peak of occipital alpha (8.2Hz) was present.

Wakefulness.
A large peak (280μV) of 11Hz alpha activity was prominent during relaxed wakefulness with eyes shut, but disappeared when the eyes were opened. Mental arithmetic with the eyes shut produced a peak (169μV) of 10.8Hz.
Occipital alpha, which was reduced in amplitude (to 61.8μV) and frequency (10.2Hz) when the eyes were opened.

CONTROL SUBJECT (7): Sleep.
A small peak (27μV) of 8.8Hz occipital alpha was seen during REM sleep.
Wakefulness.
Relaxed wakefulness with eyes shut and mental arithmetic with eyes shut produced occipital alpha (8.8Hz) at amplitudes of 36.7μV and 31.8μV respectively. When the eyes were open during mental arithmetic, 9Hz activity of 27μV was found occipitally.

CONTROL SUBJECT (10): Sleep.
No obvious peaks of occipital alpha were found during any NREM sleep stage or during REM sleep.
Wakefulness.
Small occipital peaks (64.7μV) of 9.2Hz alpha were seen only during relaxed wakefulness, with the eyes shut, and during mental arithmetic with the eyes shut, (47.3μV, 9.6Hz).

CONTROL SUBJECT (11): Sleep.
A small (27μV) peak of frontal 8.8Hz alpha-like activity was apparent during sleep stage 2, but no peaks of frontal or occipital activity were seen during sleep stages 3, 4 or REM.
Wakefulness.
No peaks of occipital alpha activity were visible during relaxed wakefulness with the eyes shut, although a small rise was detected at about 10Hz in the eyes open condition, and during mental arithmetic.

CONTROL SUBJECT (12): Sleep.
7.4Hz (41.5μV) and 9Hz (34.7μV) frontal alpha-like activity was seen during fourier analysis of sleep stage 2, whereas stage 3 analysis showed occipital alpha activity of 7.4Hz (44.4μV) to 8.2Hz (42.5μV). During REM sleep, an occipital peak in amplitude at 9.4Hz (40.5μV) was clearly visible.
Wakefulness.
During the first condition of relaxed wakefulness with the eyes shut, occipital alpha activity of 11Hz (26.1μV) was apparent. Small rises in amplitude were also seen frontally at 7Hz (~30μV) and 9.8Hz (~25μV). During other conditions, peaks of alpha activity were not found.

CONTROL SUBJECT (13): Sleep.
Alpha activity of 7.2Hz (64.7μV) was observed during REM sleep, but no occipital alpha activity was seen during any NREM sleep stage.
Wakefulness.
During relaxed wakefulness, with eyes shut, an occipital peak of 7.6Hz (217μV) was seen, but this was absent when the eyes were opened. Mental arithmetic with the eyes shut produced a similar peak (220μV) of the same frequency.
CONTROL SUBJECT (14): Sleep.

Frontal alpha-like activity was present at 7.6Hz to 8.5Hz (max ≈45μV) during sleep stage 2, and 7.4Hz (50μV) during stage 3 sleep. None was detected during sleep stage 4. During REM sleep, prominent increases in amplitude of occipital activity were found at 8.4Hz (43.4μV) and 9.8Hz (46.3μV).

Wakefulness.

During relaxed wakefulness with the eyes shut, a peak (93.6μV) of 10.6Hz activity was seen, which changed to 10.8Hz (40.5μV) when the eyes were opened. Mental arithmetic with the eyes shut produced an occipital peak at 11Hz (56.9μV) and some occipital 8.2Hz activity was also present.

When eyes were open during mental arithmetic, 10.6Hz activity appeared occipitally (37.6μV).

CONTROL SUBJECT (15): Sleep.

Some (51.1μV) occipital 7.4Hz alpha activity was seen during sleep stage 2, accompanied by frontal alpha-like activity of a smaller amplitude, (7 to 8Hz, ≈40μV). During REM sleep, peaks of alpha activity were seen occipitally at 8Hz (38.6μV) and at 8.8Hz (51.1μV).

Wakefulness.

Occipital alpha activity of 8Hz (38.6μV) seen during the relaxed wakefulness with eyes shut condition, disappeared when the eyes were opened. During mental arithmetic, with eyes shut, occipital activity of 9.6Hz (36.7μV) was evident, and also disappeared during the eyes open condition.
APPENDIX VIII.
APPENDIX VIII.

A STUDY OF THE INCIDENCE AND CONSISTENCY OF NREM SLEEP ALPHA IN SYMPTOM-FREE GOOD SLEEPERS.

SUBJECT PROFILES.

SUBJECT (IM).

This twenty one year old student reported sleeping well for eight and a half hours per night. His average sleep latency was fifteen minutes, but he occasionally awoke during the night due to noisy neighbours. Although he often felt tired and sleepy on awakening, he did not have any difficulties awakening, and was symptom free and without stress. His recreational activities included running, circuit training and hillwalking and he was physically fit and in good health.

SUBJECT (JD).

(JD) was a twenty one year old female who regularly slept well for eight hours per night. She rarely awoke in the night, and generally felt active and alert in the morning. She reported occasional difficulties falling asleep, but resolved this by waiting until she was exhausted before retiring. This subject described occasional headaches, but was otherwise physically fit, healthy and symptom free, and did not report any stress.

SUBJECT (JP).

This subject was a nineteen year old student who slept well for approximately eight and a half hours per night. He regularly took ten to twenty minutes to fall asleep, but never lay awake worrying. He described waking during the night before an important day, or for no apparent reason, and feeling tired and sleepy on awakening. He regularly woke with lethargy and felt that he did not 'wake up' until the afternoon or evening, describing himself as a definite 'evening type'.

Although he was generally in good health, physically fit and active and without any symptoms of muscular discomfort, he reported that he had suffered with asthma, hay fever, anxiety and depression and breathing problems over a year ago, but these had reduced over the past year. He reported experiencing a great need to move his legs in bed and snored regularly.

SUBJECT (SP).

(SP) took only ten minutes to fall asleep and slept very well for eight hours per night. On awakening in the morning he was tired and occasionally experienced leg stiffness, but this was generally attributed to exertion the previous day. His recreational activities included running, swimming and hillwalking, and he was moderately physically fit. He was without any stressful experiences and in good health, but occasionally suffered with hay fever and eczema.
SUBJECT (RN).

Subject (RN) was a nineteen year old ex-army undergraduate student who was very physically fit and in good health. He regularly partook in vigorous sporting activities (weight training, judo and fencing), and felt that this was responsible for his occasional leg and back stiffness on awakening.

He reported sleeping very well, taking up to twenty minutes to fall asleep. Sometimes he lay awake worrying at night if there were family or work problems, but he had no recent history of stressful events at home or work. He regularly slept continuously for seven to eight hours a night, and felt relaxed after waking in the morning.

SUBJECT (SB).

This twenty three year old student slept well for eight hours per night, but felt tired and sleepy on awakening. She usually took up to fifteen minutes to fall asleep, rarely awakening during the night unless affected by asthma, (for which inhalers were prescribed). She also periodically suffered with hay fever, eczema and some allergies, but was generally fit and in good health.

SUBJECT (ML).

(ML) was a twenty three year old student who ran sixty miles a week as part of his training for cross country events. He also participated in regular circuit and weight training, and played squash.

He described sleeping very well, usually for seven to eight uninterrupted hours per night, and taking only ten seconds to fall asleep. He reported some leg and back stiffness and lethargy on awakening in the morning, but attributed the former to the impact of roadrunning and lack of stretching for training, and the latter to insufficient sleep.

He was free of health problems, but reported grinding his teeth, and experiencing a great need to move his legs in bed, and described himself as a 'heavy' sleeper, not easily awoken.

SUBJECT (MW).

This twenty two year old student took up to forty five minutes to fall asleep, but did not lie awake worrying. He usually slept very well for eight uninterrupted hours per night and considered this to be quite sufficient.

On awakening in the morning, he felt relaxed and was free of discomfort or lethargy. He was physically active, his health was good although he suffered with hay fever and allergies, and he was free of anxiety or stress.
RESULTS OF QUESTIONNAIRE ANALYSIS.

A summary of the data obtained from the preliminary questionnaire analysis is given in the following tables.

Table (M) summarises the marital status and habits of the eight subjects.

**TABLE M.**

**CHARACTERISTICS OF SUBJECTS.**

<table>
<thead>
<tr>
<th>Feature</th>
<th>No. of Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>6</td>
</tr>
<tr>
<td>Females</td>
<td>2</td>
</tr>
<tr>
<td>Single</td>
<td>8</td>
</tr>
<tr>
<td>Smokers</td>
<td>0</td>
</tr>
<tr>
<td>Using Sleeping Pills</td>
<td>0</td>
</tr>
<tr>
<td>Take regular exercise</td>
<td>6</td>
</tr>
<tr>
<td>Do frequent vigorous sports</td>
<td>2</td>
</tr>
</tbody>
</table>

Table (N) gives the sleep characteristics of the subjects.

**TABLE N.**

**SLEEP CHARACTERISTICS OF SUBJECTS.**

<table>
<thead>
<tr>
<th>Feature</th>
<th>No. of Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>USUAL SLEEP LATENCY.</strong></td>
<td></td>
</tr>
<tr>
<td>&lt; 15 mins</td>
<td>5</td>
</tr>
<tr>
<td>&lt; 1 hour</td>
<td>3</td>
</tr>
<tr>
<td>&gt; 1 hour</td>
<td>0</td>
</tr>
<tr>
<td>&gt; 2 hours</td>
<td>0</td>
</tr>
<tr>
<td><strong>USUAL SLEEP RATING.</strong></td>
<td></td>
</tr>
<tr>
<td>Very well</td>
<td>4</td>
</tr>
<tr>
<td>Well</td>
<td>4</td>
</tr>
<tr>
<td>Poor</td>
<td>0</td>
</tr>
<tr>
<td><strong>LIE WORRYING AT NIGHT.</strong></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>0</td>
</tr>
<tr>
<td>Sometimes</td>
<td>1</td>
</tr>
<tr>
<td><strong>AWAKE DURING NIGHT.</strong></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>2</td>
</tr>
<tr>
<td>Sometimes</td>
<td>1</td>
</tr>
<tr>
<td>No</td>
<td>5</td>
</tr>
<tr>
<td><strong>DIFFICULTY AWAKENING (am).</strong></td>
<td></td>
</tr>
<tr>
<td><strong>TIME OF AWAKING (am).</strong></td>
<td></td>
</tr>
<tr>
<td>6am to 8am</td>
<td>8</td>
</tr>
<tr>
<td><strong>TIME OF GETTING UP (am).</strong></td>
<td></td>
</tr>
<tr>
<td>6am to 8am</td>
<td>6</td>
</tr>
<tr>
<td>6am to 10 am</td>
<td>2</td>
</tr>
<tr>
<td><strong>FEELINGS ON AWAKENING.</strong></td>
<td></td>
</tr>
<tr>
<td>Alert</td>
<td>2</td>
</tr>
<tr>
<td>Relaxed</td>
<td>3</td>
</tr>
<tr>
<td>Tired</td>
<td>4</td>
</tr>
<tr>
<td><strong>SLEEPY DURING DAYTIME.</strong></td>
<td>2</td>
</tr>
</tbody>
</table>
Table (P) summarises the number of subjects who reported discomfort and lethargy, stress and use of medications.

**TABLE P. HEALTH AND DISCOMFORT CHARACTERISTICS OF SUBJECTS.**

<table>
<thead>
<tr>
<th>NO. OF SUBJECTS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td><strong>SYMPTOMS ON AWAKING.</strong></td>
</tr>
<tr>
<td>Stiffness</td>
</tr>
<tr>
<td>Lethargy</td>
</tr>
<tr>
<td>Discomfort and lethargy</td>
</tr>
<tr>
<td>Symptom free</td>
</tr>
<tr>
<td><strong>MEDICATION OR TREATMENTS.</strong></td>
</tr>
<tr>
<td>Respiratory</td>
</tr>
<tr>
<td>Others</td>
</tr>
<tr>
<td><strong>WORRY ABOUT HEALTH</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>STRESS.</strong></td>
</tr>
<tr>
<td>Home</td>
</tr>
<tr>
<td>Work</td>
</tr>
</tbody>
</table>

(*)Attributed to exercise
RESULTS OF FOURIER ANALYSIS.

Six of the eight subjects studied underwent at least one recording using the alternative electrode montage, and performed the waking tests outlined in the main study.

SUBJECT (JM).

The results of this subject's fourier analysis are described in the consistency study. No frontal alpha-like activity was observed during NREM sleep or during REM sleep.

SUBJECT (JD). Sleep.

Some occipital alpha activity (8.6Hz, 50µV) was observed during stage 2 sleep, and during sleep stages 3 and 4. However, no frontal alpha-like activity was seen during any stage. During REM sleep, a prominent peak of 8.6Hz (111µV) was observed occipitally.

Wakefulness.

During relaxed wakefulness, a 50µV peak of occipital alpha activity (9.8Hz) was found, which was absent when the eyes were open. A smaller peak (38.6µV) of 11.2Hz activity was then found during mental arithmetic with the eyes shut, but not when the eyes were open.

SUBJECT (JP). Sleep.

During stage 2 sleep, a 22.2µV peak of 11Hz activity was found frontally, but this was not found consistently in all stage 2 sleep samples. No frontal alpha-like activity was detected in stages 3 or 4. Some occipital alpha was also present during stage 2 sleep, (7.4Hz, 40µV), stage 3 sleep (7.6Hz, 49.9µV), and stage 4 sleep (7.6Hz, 38.6µV). During REM sleep, occipital activity between 8.6Hz (28µV) and 9.8Hz (28µV) was observed.

Wakefulness.

A peak of 9.6Hz (125µV) activity was found occipitally during relaxed wakefulness with the eyes shut, which changed to 10Hz (37.6µV) when the eyes were open. During mental arithmetic a prominent peak (134µV) of 9.8Hz occipital activity appeared and increased to 10Hz (34.7µV) when the eyes were open.

SUBJECT (SP). Sleep.

No frontal alpha-like activity or occipital alpha activity was seen during any stage of NREM sleep, but during REM sleep, a 9Hz (45.4µV) peak of occipital alpha was apparent.

Wakefulness.

A 75.3µV peak of 9Hz activity was found during relaxed wakefulness with the eyes shut. This decreased in amplitude to 19.3µV when the eyes were open. With the eyes shut, mental arithmetic produced a 60.8µV peak of occipital alpha, which was absent when the eyes were open.
SUBJECT (SB): Sleep
The results of this subject's fourier analysis are described in the consistency study. No frontal alpha-like activity or occipital alpha was observed during NREM sleep or during REM sleep, but occipital alpha was found during relaxed wakefulness with eyes shut, \(10\text{Hz}, 31.8\mu\text{V}\).

SUBJECT (MW): Sleep.
A small \((31.8\mu\text{V})\) peak of \(9.8\text{Hz}\) occipital alpha was found during REM sleep, but no frontal alpha-like activity or occipital alpha was seen during stages 2, 3 or 4.

Wakefulness.
A peak \((47.3\mu\text{V})\) of \(11.4\text{Hz}\) activity was found occipitally during relaxed wakefulness with the eyes shut, and decreased to \(11.2\text{Hz}\) \((20.3\mu\text{V})\) during the eyes open condition. Mental arithmetic with eyes shut produced an occipital peak of \(11.2\text{Hz}\) \((55\mu\text{V})\), which was completely absent when the eyes were open.
APPENDIX X.

REPEATABILITY OF SLEEP STAGER RESULTS.

PROCEDURE EMPLOYED.

1. The playback device and Oxford Sleep Stager were switched on half an hour prior to starting analysis on each occasion, to allow the machines to 'warm up'.
2. Following calibration of the sleep stager, the beginning of the EEG recording was identified, and the recording start time entered on to the playback clock.
3. The 'run' button was pressed to start the analyser, and when the results title had been printed, the 'play' button was pressed to start the tape.
4. The tape was switched off immediately the end of tape recording (1) had finished, the clock time was recorded and the analyser 'report' button was pressed to obtain the results print out.
5. This procedure was repeated using the same sleep recording, without any alteration of calibration.
6. Tape (2) was then analysed using the same procedure, but requiring a new calibration.
7. Tape (2) was reanalysed without a recalibration, using the same procedure.
8. After recalibration, tape (1) was reanalysed using the same procedure.
9. After recalibration, tape (2) was reanalysed using the same procedure.
10. Results of each analyses were compared for each tape, and the differences computed.
TAPE 1. RESULTS.

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<th>ANALYSIS 1</th>
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<th>ANALYSIS 3</th>
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Analysis (1) refers to the first analysis of each tape. Analysis (2) refers to the reanalysis without recalibration. Analysis (3) refers to reanalysis after recalibration.
## Results

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### Sleep Time

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### REM

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APPENDIX XI.

RAW DATA.

INDIVIDUAL MEANS OF PERCENTAGE ALPHA PER EPOCH OF SLEEP STAGES FOR SUBJECTS IN ALL GROUPS.

A STUDY OF THE RELATIONSHIP BETWEEN THE NREM SLEEP ALPHA ANOMALY AND MUSCULOSKELETAL DISCOMFORT IN A LOCAL POPULATION.

RAW DATA

TABLE S.  
CONTROL SUBJECTS.

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TABLE T.  
DISCOMFORT SUBJECTS.

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<th>REM</th>
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POOR SLEEP+DISC. SUBJ.  
E | 3.8 | 4.4 | 1.4 | 6.1 |
G | 10.4 | 2.4 | 0 | 2.0 |
H | 4.8(0.7) | 5.8(1.6) | 0.6 | 3.0 |
J | 2.7(0.2) | 12.8(9.3) | 1.5 | 0.5(0.2) |
A STUDY OF THE INCIDENCE AND CONSISTENCY OF NREM SLEEP ALPHA IN SYMPTOM-FREE GOOD SLEEPERS: RAW DATA

<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>REM</th>
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<td>0.1(0.06)</td>
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<td>0.3(0.1)</td>
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