BEHAVIOURAL AND PERFORMANCE CHARACTERISTICS OF SLEEP INERTIA FOLLOWING AWAKENING FROM NAPS

by

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Graduate Department of Community Health, University of Toronto

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ABSTRACT

This study examined a) time of night effects on sleep inertia upon awakening from naps and, b) whether subjective ratings are in concordance with cognitive performance measures of sleep inertia.

Thirteen healthy males (aged 20-35) completed two 34 hr protocols. Subjects were randomly assigned to a 90 min afternoon nap scheduled at either 1300h (protocol A) or 1600h (protocol B) followed by a series of four 60 min nap opportunities scheduled at 0000h, 0130h, 0300h, and 0430h (protocol A), and at 0300h, 0430h, 0600, and 0730h (protocol B). Cognitive performance and subjective ratings were derived from hourly test sessions (including immediately before and after the naps) of logical reasoning, serial reaction time, synthetic work (multi-task performance task), and self-reports of fatigue, sleepiness, drowsiness, and mood. Sleep inertia effects were determined by comparing pre- and post-nap measurements using repeated measures analysis of variance.

Afternoon naps did not result in any significant sleep inertia effects as measured by performance tasks. Significant sleep inertia effects (pre- vs post-nap

performance measures) occurred after naps scheduled at 0000h, 0130h, and 0300h in both protocols but not with naps at 0600h and 0730h in protocol B. After 15 min of wakefulness, deteriorated performance measured upon awakening always significantly recovered in protocol A but not after the 0430h and 0600h naps in protocol B. Therefore, differences between pre- and post-nap performances in protocol B became smaller resulting in apparent smaller sleep inertia effects. When only post-nap performances were compared between naps and protocols, no significant differences in sleep inertia effects were found. Sleep inertia effects were found for fatigue and sleepiness ratings after the 1300h and midnight naps in protocol A, and the 1600h nap in protocol B. Nocturnal subjective ratings did not show any significant improvements after 15 min spent awake.

In conclusion, the severity of sleep inertia effects on cognitive performances does not vary with time of night but the duration of impairments was longer following 0430h and 0600h naps. Subjective ratings of fatigue, sleepiness, drowsiness, and mood do not reflect sleep inertia effects as measured with cognitive performance tasks.

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CHAPTER I

INTRODUCTION

Performance of many services and work of high responsibility (medical and military personnel, transportation industries, search and rescue teams) requires individuals to perform and be attentive for prolonged and/or recurrent periods of time. In such circumstances, prolonged sleep restriction can result in a reduction of an individual's ability to carry out tasks, to judge situations effectively, and to make appropriate decisions. General decline in mood and motivation are also evident (Reviews: Horne, 1978; Krueger, 1989). In order to prevent or reverse the detrimental effects of sleep loss on mood and performance efficiency, adequate planning for sleep periods must be considered. Under most prolonged work environments, uninterrupted hours of sleep are difficult to obtain. Short sleep periods (naps), however, offer more flexibility in scheduling and have been found to be effective in terms of maintaining and recovering performance (Naitoh, 1981; Dinges *et al.*, 1981; 1985; Stampi, 1992). Although naps may be beneficial to performance several hours after awakening, the efforts to awaken are often accompanied by undesirable effects such as disorientation, confusion and less effective functioning immediately upon awakening. These negative effects are known as sleep inertia.

Because of the detrimental effects of sleep inertia on performance immediately upon awakening, napping during work periods (in Canada and in the US) has not been recommended, and sometimes even forbidden (Naitoh, 1992). There are still no guarantees that in certain situations people will have time to

completely wake up before starting to work. As a result, the beneficial effects of naps to relieve, or prevent fatigue in the workplace must be balanced against the adverse effects of sleep inertia. It is therefore relevant to investigate the relationship between the circadian placement of the nap and the severity of sleep inertia. Few studies have examined this question (Dinges *et al.*, 1985, 1987; Naitoh *et al.*, 1993; Mullington and Broughton, 1994). Moreover, sleep inertia was found either not to be influenced by the circadian placement or to be minimum around the circadian peak of body temperature. Therefore, it is uncertain whether sleep inertia is affected by the circadian placement of the naps. In addition, when subject's self-ratings of sleepiness was examined it did not reflect the severity of performance impairments caused by sleep inertia, especially from naps taken beyond 18 hours of wakefulness (Dinges, 1987). The relation between performance and subjective measures (fatigue, mood, and sleepiness) has also not been adequately addressed. When evaluating the effects of circadian placement of naps on sleep inertia, three other factors also need to be considered; the duration of the nap, prior wakefulness, and the sleep stage composition of the nap. These factors have previously been found to influence the severity of sleep inertia. Very short naps (< 30 min) and longer naps (> 80 min) resulted in less pronounced sleep inertia compared to intermediate nap lengths (Stampi, 1992). Naps taken after prolonged periods of wakefulness resulted in more serious and prolonged sleep inertia because the accumulated sleep debt was not fully discharged by the nap (Dinges, 1990). Finally, sleep inertia has been reported to be associated with the time spent in SWS or REM sleep during the nap (Dinges, 1990; Stampi, 1992; Naitoh *et al.*, 1993).

Research examining the time of day effects on sleep inertia would be useful in determining when to schedule naps in order to maximize their recuperative

value while minimizing detrimental effects (e.g., sleep inertia). Further evaluation of the sensitivity of subjective measures to sleep inertia effects would be helpful when other direct performance evaluations are difficult in order to estimate if an individual's performance is back to an acceptable level of performance. The primary purpose of this study was to determine whether performance and subjective measures of sleep inertia following awakenings from naps are influenced by time-of-day. Further, the relation between the subjective assessments (fatigue, mood and sleepiness) of sleep inertia and performance measures was also examined. A two protocol experiment was designed and conducted to compare two series of four one hour naps scheduled between 0000h and 0430h (protocol A), and 0300h and 0830h (protocol B). In an attempt to control prior wake time immediately preceding the night naps, a 90-min afternoon nap was scheduled to terminate 9.5 hr before the first night nap of each protocol. While sleep duration and prior wakefulness were kept constant, post-nap performance and subjective measures were evaluated in terms of nap sleep architecture, sleep stage upon awakening, and time of day. Performance tasks and subjective scales were selected either for their known sensitivity to the effects of sleep inertia or to evaluate their sensitivity to sleep inertia.

CHAPTER II

LITERATURE REVIEW

This chapter is divided into six sections. The first section (1.0) reviews how sleep is measured and describes the various stages of normal human sleep. Section 2.0 provides an overview of the theories of sleep function. Section 3.0 looks at the effects of total sleep deprivation on cognitive performance, subjective states and possible countermeasures. Section 4.0 summarizes the beneficial effects of napping on cognitive performance. Section 5.0 discusses the detrimental effects of sleep inertia following napping on cognitive performance. Finally, section 6.0 presents the rationale, predictions and hypotheses of the present study to determine the magnitude and time of variations of sleep inertia upon awakenings from naps scheduled at different time of the day.

1.0 HUMAN SLEEP BEHAVIOUR AND PHYSIOLOGY

The human sleep-wake cycle is a 24-hr circadian rhythm. Sleep normally occurs during the nocturnal period of the cycle. Sleep is defined as "a reversible behavioural state of perceptual disengagement from and unresponsiveness to the environment" and as "a very complex amalgam of physiological and behavioural processes" (Carskadon and Dement, 1989). The electroencephalogram (EEG) is the continuous recording of brain waves measured from scalp electrodes. EEG recordings show that the sleep period is characterized by distinct changes in brain electrical activity. Brain waves are described in terms of their amplitude (height) and frequency (number of waves

per second). A continuous electrophysiological record of sleep (polysomnogram) also includes the measurement of eye movements (electrooculogram or EOG), and submental muscle tone (electromyogram or EMG). The EOG and EMG in association with specific brain wave patterns are important for categorizing the various sleep stages. By visually scoring the polysomnogram according to standardized criteria proposed by Rechtschaffen and Kales (1968), wakefulness and five sleep stages (1, 2, 3, 4 and REM) can be identified (Figures 1 to 9 in Rechtschaffen and Kales, 1968).

Within sleep, two distinct types of sleep have been defined according to their physiological characteristics: non-rapid eye movement (NREM) and rapid eye movement (REM) sleep. NREM sleep is comprised of sleep stages 1, 2, 3, 4 generally representing a depth of sleep continuum. Based on EEG sleep responses to auditory stimuli, the highest arousal threshold is from stage 4 followed by stage 3, stage 2 and stage 1 (Bonnet, 1983). REM sleep is not divided into stages but sometimes into two classes of events: tonic events (low muscle tonus and desynchronized EEG) and phasic events (eye movements, twitching of the limbs and facial muscles) that are superimposed on the tonic background.

When the eyes are open, the waking EEG pattern is characterized by a relatively low voltage and mixed frequency. When the eyes are closed, the relaxed waking state shows alpha rhythm (7.5 to 11.5 cycles per second or cps). The EOG shows rapid eye movements and the EMG indicates a high level of muscle tone.

Stage 1 is a transition state between wakefulness and sleep of short duration. It is identified by a relatively low voltage, mixed frequency EEG. Bursts of high voltage activity in the theta (3 to 7 cps) range are commonly seen. High

amplitude vertex sharp waves may also appear during this stage. Muscle tone is reduced compared to wakefulness and the EOG shows slow, rolling eye movements. Stage 1 can also occur following body movements during sleep.

In stage 2, the eyes do not move, muscle tone is low compared to wakefulness, and EEG sleep spindles (1.5 to 2 seconds bursts of 12 to 14 cps activity) and K-complexes (slow negative EEG deflections followed immediately by a positive component lasting at least 0.5 seconds) occasionally appear.

Stage 3 is marked by the presence of high voltage slow wave activity covering approximately 20% to 50% of a given epoch (40 to 60 seconds worth of recording). The frequency of the waves are 2 cps or slower with an amplitude greater than 75 microvolts (μv) from peak to peak. The EOG registers high voltage slow wave activity and the EMG shows low muscle tension. Stage 4 is similar to stage 3 but with a greater density of slow waves exceeding 50% of the epoch. Stage 3 and 4 together constitute what is called "slow wave sleep" or "delta sleep".

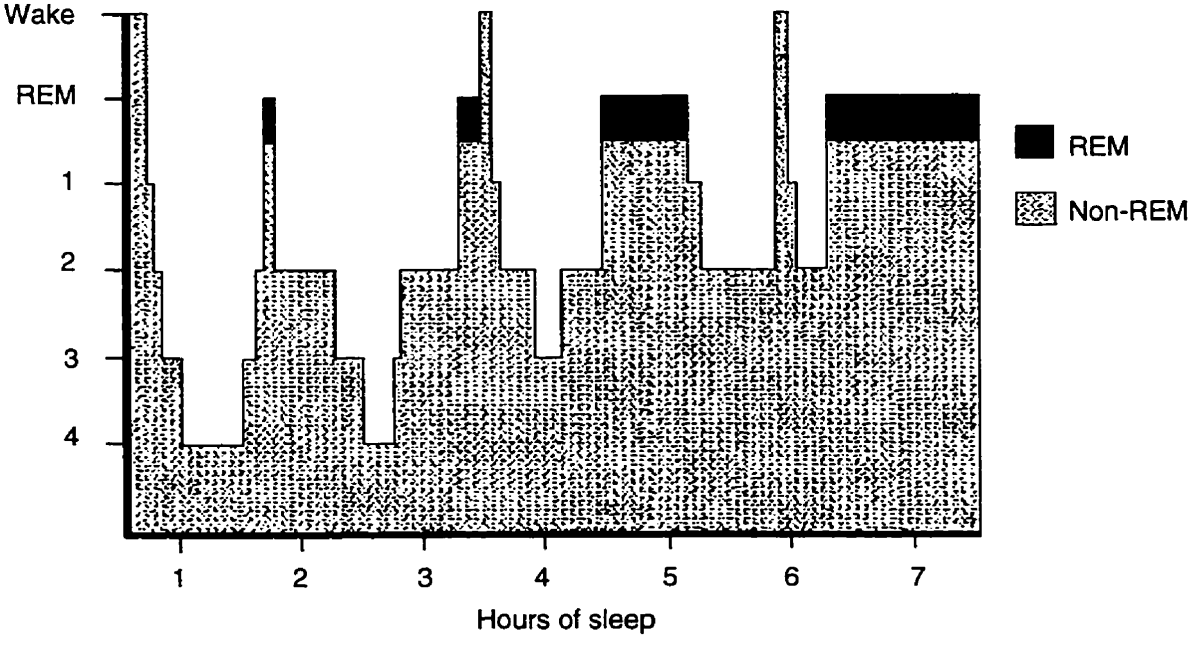
REM sleep is characterized by relatively low voltage, mixed frequency EEG activity resembling that described for stage 1 with the exception that there are no vertex sharp waves present. Also, distinctive triangular waves (4 cps) referred to as "sawtooth waves" may be seen. The EOG registers bursts of rapid eye movements after which this sleep stage was named. The EMG shows extremely low muscle tone. However some small sporadic muscular twitches of the face and extremities can be seen.

Normal Sleep Profile

The stages of sleep of a normal young adult occur in a predictable sequence throughout the night. A sequence of a NREM and a REM sleep period

constitutes a sleep cycle and a night's sleep is composed of 4 to 6 sleep cycles (Figure 1). Typically, sleep onset is defined as the point when stage 1, which generally persists from 1 to 7 min at the beginning of sleep (Rechtschaffen and Kales, 1968), is replaced by stage 2. As stage 2 progresses, there is a gradual appearance of high-voltage, slow wave activity in the EEG. Eventually this activity meets the criteria for stage 3. Stage 3 sleep usually lasts only a few minutes and transitions to stage 4. Stage 4 generally lasts for about 20 to 40 min in the first cycle. Usually a brief return to stage 3 or 2 occurs before the onset of the first REM period (1 to 5 min) starting 70 to 90 min after sleep onset. The average length of the NREM-REM sleep cycles is approximately 90 to 110 min (Carskadon and Dement, 1989). In the young adult, peak activity (acrophase) of SWS occurs within the first third of the night (i.e., mostly within one hour of sleep onset) while the duration of REM episodes progressively increases during the later part of the night peaking around 0730h.

Figure 1 Overnight sleep hypnogram for a young adult man



(from Moldofsky, 1996)

Other body functions show changes with the transition from wake to NREM sleep. There is a decrease in heart rate, respiration, arterial blood pressure, and total body oxygen consumption (Orem and Barnes, 1980). There is also a drop in body temperature. During REM sleep there are phasic changes in respiration and heart rate. Blood pressure is low but shows phasic fluctuations, thermoregulatory responses are lowered, but total body oxygen consumption is greater in REM sleep then in NREM sleep (Orem and Barnes, 1980).

SWS and REM Sleep Propensity

Sleep stages do not only occur in a predictable sequence during nocturnal sleep but SWS and REM sleep propensity (tendency to occur) show predictable changes in relation to the previous time awake, body temperature and time of day. This has been observed in studies examining sleep-stage composition (infrastructure) when sleep was taken during the daytime or in the form of naps.

Under a normal sleep-wake schedule, SWS occurs mostly during the first 3 hrs of sleep. The amount of SWS is primarily dependent upon the duration of prior wakefulness within the normal bounds of waking time (Webb and Agnew, 1971; Borbély *et al.*, 1981). If sleep onset is delayed, the resultant increase in wakefulness (at least up to 16 hr) will be followed by more SWS (Dinges, 1989a) regardless of the time of day the sleep is taken (Broughton, 1989). However a second, less robust period of SWS has been observed in subjects extending their night sleep beyond 12 hr (Weitzman *et al.*, 1980; Gagnon and De Koninck, 1984). These studies showed that if night sleep is extended during the day the interval between the first SWS peak and the second one was approximately 12.4 hr. The second peak occurred in the early afternoon.

In a second study of extended sleep, Gagnon *et al.* (1985) delayed sleep onset by 4 hr (from 0000 to 0400h) to determine the times for the occurrence of the

second period of SWS. They showed three major SWS peaks occurring on the first day of shifted sleep. The first peak occurred after sleep onset, the second in the early afternoon as in their previous study (Gagnon and De Koninck, 1984), and the third one 12.5 hr after the first peak. The presence of the second peak was attributed to the influence from the sleep/wake schedule prior to the beginning of the shifted schedule. The second peak disappeared by the third day leaving only the initial peak after sleep onset and a second peak 12.5 hr later. Therefore, Gagnon and colleagues suggested that the delay observed in the second peak could not be explained by the amount of wakefulness after sleep onset but most likely by the influence of sleep dependent and circadian mechanisms.

Other reports substantiated further the existence of a second period of SWS. Afternoon naps, compared with those taken in the morning or evening hours, have been found to comprise higher amounts of SWS (Evans *et al.*, 1977; Webb and Agnew, 1971). This afternoon appearance for SWS has also been observed by Lavie and Scherson (1981) under their ultrashort sleep/wake schedule. They used a fast sleep/wake cycle consisting of 7 min sleep period and 13 min wake period. The data showed an equal distribution of stage 1 throughout the day but increased amount of stage 2 during the afternoon. Moreover, under a time-free environment where subjects were isolated from social and environmental time cues, naps showed a strong tendency to occur 10 hr prior to the onset of nocturnal sleep, in the middle of the waking period (Campbell and Zulley, 1989).

Again, prior wakefulness is insufficient to explain the appearance of SWS during afternoon naps compared to evening naps under a normal sleep/wake protocol or time-free environment. To explain both SWS peaks revealed in these studies, Broughton (1975), and Campbell and Zulley (1989) suggested

the presence of a 12-hr bimodal distribution of SWS propensity. Campbell and Zulley (1989) separated spontaneous naps taken under time-free environment studies into two groups on the basis of their circadian placement within the studies. Naps taken between 1400 and 1700h (middle of the nap distribution) showed twice the average percentage of SWS seen in the other naps. They concluded that these findings supported Broughton's hypothesis for a two per day regulation of SWS occurrence, with specific phase positions in the circadian day.

The amount of REM sleep varies as a function of the circadian phase, with higher occurrences around the morning hours during the rising phase of the body temperature curve (Czeisler *et al.*, 1980; Zulley, 1980). More evidence for this close relation between body temperature and REM sleep propensity was obtained in studies of night sleep extended into the later morning hours (Taub *et al.*, 1983; Gagnon and De Koninck, 1984) and in studies of naps taken at different times of the day (Webb and Agnew, 1967; Karacan *et al.*, 1970). Gagnon and De Koninck (1984) showed that the REM acrophase occurs around 0730h and REM sleep occurrence remains high throughout the morning period. The first REM sleep period appears to depend upon the accumulation of a critical amount of NREM sleep (Lavie, 1989). Under an ultrashort sleep-wake cycle, Lavie observed that the REM latency was approximately 90 min, identical to the one seen under uninterrupted nocturnal sleep period after the intervening wake episodes were subtracted from the accumulated NREM sleep. In contrast to REM onset, subsequent REM periods occurred at approximately 90 min intervals irrespective of the amount of intervening sleep (Lavie, 1987).

The occurrence of SWS following sleep deprivation will delay the latency for the first REM episode and shorten the REM episode (Broughton, 1989). Because of this mutual inhibition of SWS and REM sleep, Webb and Agnew (1971) and

Knowles *et al.* (1987) proposed that REM sleep propensity may be indirectly influenced by prior wakefulness. In other words, prior wake time is thought to influence SWS propensity which in turn will affect REM sleep occurrence. However, Jones and Oswald, (1968) and Mullaney *et al.* (1977) observed unusually short REM latencies and the usual proportion of REM sleep when sleep was curtailed (less than 6.5 hr) for prolonged periods of time. Moreover, selective REM deprivation during nocturnal sleep produced a REM rebound and a reduction in REM latency on the subsequent night of undisturbed sleep with little or no effect on SWS (Dement *et al.*, 1966; Agnew *et al.*, 1967; Moldofsky and Scarisbrick, 1976; Brunner *et al.*, 1990). These findings suggest that an homeostatic process maintains REM sleep within minimum obligatory limits (Dement *et al.*, 1966). Knowles *et al.*, (1987) concluded that "*Varying prior wakefulness appears to have an analogous but indirect effect, in that, at any phase of the rhythm of REM propensity, such variations will cause the values for REM % and REM latency to deviate (albeit modestly) from those expected after 16 hr of prior wake time, probably because of its relationship to slow-wave activity*".

Therefore, sleep propensity and architecture depend primarily on sleep circadian phase and prior wakefulness. Slow wave sleep intensity is clearly affected by increased prior waking but its occurrence is also influenced by the circadian timing of the sleep period. On the other hand, REM sleep within a certain limit, varies as a function of the circadian phase, but not with prior wakefulness.

2.0 THE FUNCTION OF SLEEP

Even though most individuals spend one-third of their life asleep and sleep can

be measured and classified, no one knows why we sleep. There are many theories about the function of sleep, but most fall into two categories. The "adaptive" theories consider sleep to occupy tedious hours of inactivity and may serve a purpose of energy conservation (Webb, 1974; Berger, 1984). The "restorative" theories are based on the concept that sleep is a state of enhanced tissue growth and repair (Adam, 1980; Oswald, 1980).

Webb (1983) describes sleep as an adaptive form of behaviour governed by an endogenous biological rhythm. Sleep pattern must have evolved in relation to the animal feeding habits, predatory pressures, and physiological capacities of each species (Webb, 1974). For example, large grazing animals (zebra, antelope) with high predator pressure tend to sleep for only a few hours in short episodes. On the other hand, hunting animals (feline) show highly flexible and longer sleep times. Thus sleep could be an enforced time out to conserve the energy needed for physical effort and as a result reduce the need for food (Berger, 1984).

If sleep has a function of energy conservation, Berger (1984) suggested that sleep and hibernation may have a common evolution. He believes that SWS evolved in parallel with temperature regulation as a periodic adaptation to conserve energy. Thermoregulatory adjustments that reduce body temperature and metabolism necessary to enter shallow torpor and hibernation are initiated during SWS. The occurrence of sleep, shallow torpor and hibernation depends on factors such as the environmental temperature, amounts of body fat, external food availability and the habitat (Berger, 1984). When food simply is not to be found during inclement seasons, large animals with large amount of body fat such as bears, will sleep to conserve their energy while waiting for food. Small animals such as squirrels or birds will enter shallow torpor or hibernate to conserve their energy and survive inclement seasons. The fact that shallow

torpor and hibernation are initiated during SWS provides some evidence that they are homologous energy conserving processes regulated in accordance with ecological factors affecting the energy intake and energy balance.

The concept that sleep provides optimal conditions for tissue growth and restoration in compensation for degradation processes occurring during wakefulness (Adam, 1980; Oswald, 1980) is not opposed to the adaptive theory. Adam (1980) and Oswald (1980) provided some evidence to support the proposition that sleep restoration depends on higher rates of protein synthesis and cellular divisions during the time of sleep. Adam (1980) proposed a model based on the 24-hr variations in cellular works to explain simultaneous variations in synthesis and degradation. During sleep, there is a decrease in cellular work as reflected in a lower metabolism and lower oxygen consumption. As cellular work decreases, there is an increase in the cellular energy charge (EC) represented by the relative intracellular concentration of adenosine triphosphate (ATP). High values of EC stimulate protein synthesis and cellular divisions and decrease degradative processes. Moreover, sleep is also associated with increased levels of growth hormone (GH) and other anabolic hormones whereas wakefulness is associated with increased secretion of catabolic hormones such as cortisol and catecholamines (Oswald, 1980).

Conditions of increased energy expenditure and catabolism (hyperthyroidism, starvation, and sleep deprivation) result in longer sleep, more SWS and GH secretion for compensatory synthesis (Oswald, 1980; Shapiro, 1982). SWS is thought to be more restorative because of its lower rate of cellular work, and as a consequence metabolism is at its lowest during SWS, as is the oxygen consumption. This means that the EC is higher during SWS than other sleep stages, resulting in further enhancement of synthetic processes (Adam, 1980).

In humans, oxygen consumption and metabolism are higher during REM sleep than during SWS. The intense brain activity during REM would lower EC levels and be incompatible with protein synthesis. Both Oswald and Adam have some difficulty in explaining how REM sleep is part of a restorative process.

Horne (1983, 1988) uses several arguments to contest the view that sleep is a condition of heightened restitution for both the brain and the body. Based on the results of several sleep deprivation studies, Horne concludes that of all the organs, only the brain showed some degradation, while the rest of the body seemed relatively unaffected (Horne, 1978). In humans, food intake and physical rest, rather than sleep, may well be all that is required for body restitution. More specifically, Horne argues that the night time fast would not support protein synthesis but more likely the opposite, tissue dissolution during sleep. The psychological and EEG effects measured during sleep loss are indicative of a brain restitution role for sleep. Horne points out that during wakefulness, the cerebrum is unable to rest and is always in a state of readiness. During sleep, the cerebrum enters a unique physiological state allowing some form of cerebral shut-down which may be needed for consolidation and restitution. Because SWS is influenced by the length of prior wakefulness (Webb and Agnew, 1971; Borbély *et al.*, 1981), SWS has the most cerebral shut-down, and takes priority over the other sleep stages. SWS, more than any other sleep stage, may have a restitutive role. Horne concludes that further technological advances are needed to clarify if any cerebral restitution, as measured in the form of neuronal/glial repair or protein turnover, takes place during sleep.

Horne (1988) observed that after sleep deprivation, only recovery of part of the sleep lost was needed for a complete return to normal psychological performance and normal behaviour. Recovery sleep comprises most of the

SWS lost and half the REM sleep lost. Also, sleep, given time for adaptation, can be reduced or extended within limits without adverse effects. These observations led Horne (1988) to suggest that only part of sleep seems obligatory and is oriented towards brain restitution (Core sleep) while the other part of sleep is more adaptable and can be extended or reduced without affecting daytime sleepiness (Optional sleep). Core sleep represents the first 5 to 6 hr of sleep and contains all of the nightly SWS and about 50% of REM sleep. Optional sleep occupies the remaining sleep and contains the other half of REM sleep and much of stage 2 sleep. Optional sleep seems to be under the influence of the sleep/wake circadian rhythm and motivational factors. Optional sleep may also be influenced by environmental protocols such as temperature and day length. For example, after Core sleep, Optional sleep would occupy the remaining hours of darkness until the wake up time regulated either by the increase of core body temperature or by environmental protocols such as an alarm, noises, etc. Interestingly, Horne's division of sleep into Core and Optional sleep appears to combine the two categories (adaptive and restorative) of sleep function and brings a different perspective to the typical classification of sleep into NREM and REM sleep.

In conclusion, the restorative and adaptive concepts provide some answers to the purpose of sleep. The restorative theories account for the results of sleep deprivation and the recovery effects of sleep. The adaptive theories recognize the behavioural advantages of sleep such as avoiding predators and/or conserving energy. Both theories have their limitations. The restorative theories have difficulty in defining the underlying mechanisms while the adaptive ones tend to ignore the apparent recovery effects of sleep after sleep loss. The two concepts, restorative and adaptive, are somewhat combined in the Core and Optional sleep theory. In this theory, the first 5 to 6 hr of sleep

represents the need/restorative component (Core sleep) and the remainder of the sleep represents a behaviourally and circadian-timed component (Optional sleep). In general, all sleep theories emphasize the importance of SWS, whereas the role of REM is less clearly defined.

3.0 SLEEP DEPRIVATION

There are practical consequences of sleep deprivation. They can lead to a reduction of an individual's ability to carry out tasks, to judge situations effectively, to make appropriate decisions, and to a lessened mood and motivation in general. Serious behavioural changes are rarely seen in normal subjects experiencing sleep deprivation, although some subjects undergoing prolonged sleep loss have experienced perceptual illusions, euphoria, confusion and feelings of persecution (Horne, 1985).

Sleepiness and Mood

Words like *tiredness*, *fatigue*, *drowsiness*, and *sleepiness* are often used interchangeably to describe the effects of sleep loss, but they may not describe the same thing (Dinges, 1989b). Their meaning will depend on the context under which the individuals are rating themselves. Subjects reporting being fatigued after performing a heavy physical exercise, mean that they are physically tired but not necessarily sleepy. Reports of intense fatigue or tiredness after or during sleep deprivation, may represent sleepiness or mental fatigue. The combination of sleep loss with intense physical or mental work creates further blurring of the meaning of fatigue, tiredness, drowsiness and sleepiness ratings (Dinges, 1989b). Moreover, emotional states or mood states (happy, angry, irritated, cheerful or tense) have also been found to vary

throughout sleep loss. Nevertheless, subjective impressions can be useful and sensitive indices of sleep loss if compared within subjects rather than between them. Various techniques are available to measure subjective sleepiness and mood, like checking a sentence that best describe one's state (Hoddes *et al.*, 1973) or ascribing ratings of agreement to a list of negative and positive mood adjectives (Harris *et al.*, 1971). Regardless of the type of scales used, declines in positive mood and increases in subjective reports of sleepiness and negative mood are increased with progressive sleep loss (Johnson and Naitoh, 1974; Angus and Heslegrave, 1983; 1985).

Froberg (1977) showed a clear circadian rhythm in subjective alertness superimposed on the expected decreasing trend caused by 72 hr of sleep deprivation in temporal isolation. Subjective alertness and body temperature showed identical acrophases (early evening) and nadirs (early morning), suggesting an influence of the temperature oscillator on subjective alertness (Froberg, 1977). However, studies of diurnal workers and shift workers (Monk and Embrey, 1981; Monk *et al.*, 1983) showed the occurrence of a second peak of alertness (at about mid-day) in addition to the evening peak. The presence of a 2-cycle per day component in alertness (or sleepiness) has also been reported in subsequent studies (Dinges *et al.*, 1988; Monk, 1987; Babkoff *et al.*, 1991). Monk's (1987) forced desynchronization experiment, in which the temperature oscillator was induced to run at a different period to that of the sleep/wake cycle by stretching the individuals sleep/wake cycle to last 26 hr while their temperature oscillator period was closer to 25 hr, was done to try to tease apart the influence of the temperature oscillator and sleep/wake cycle on subjective sleepiness. Monk found a minimum in sleepiness 6 hr after wake up time which corresponded to the afternoon peak in alertness observed in previous studies. These results indicated that this peak might be under the

influence of the sleep/wake cycle. Thus, these dual influences of the temperature oscillator and the sleep/wake cycle on subjective sleepiness, may explain the presence of only one peak in alertness when the sleep/wake cycle is interrupted during sleep loss (Monk, 1987).

Subjective rating of sleepiness showed greater degradation when completed during or at the end of performance sessions rather than prior to it (Angus and Heslegrave, 1985; Dinges *et al.*, 1987; Babkoff *et al.*, 1991). Dinges *et al.* (1987) suggested that as sleep loss increases, subjective ratings of sleepiness become more dependent upon the stimulating effects provided by the task or the environment. However, the amount of prior wakefulness did not explain Babkoff's lack of difference between the ratings before and after cognitive testing at 1800h on the third day of sleep loss; whereas the same ratings at 0400h on the first day were significantly different. In fact, the potentiating effects of cognitive performance on sleepiness ratings were influenced more by the phase of the circadian cycle than by the amount of prior sleep loss (Babkoff *et al.*, 1991). Babkoff and colleagues concluded that the most appropriate explanation, also considered by Angus and Heslegrave (1985), was "that cognitive performance unmasks the physiological sleepiness and that this effect is more manifest at the nadir of the circadian cycle".

Performance

Generally performance efficiency declines with increasing sleep deprivation (Johnson and Naitoh, 1974). The effect of sleep loss on performance appears to be additive to the well established presence of circadian rhythms in human performance (Kleitman, 1963; Hockey and Colquhoun, 1972). Therefore, performance shows a linear decline under sleep loss superimposed on a circadian rhythm resulting in greater decrements during night-time than during

the day. Kleitman (1963) originally believed that this circadian fluctuation of performance corresponded closely to the body temperature rhythm. However, the location of the peaks and troughs of the circadian components of human performance were not always coincident with the peaks and troughs of body temperature and were therefore not always easy to predict (Hockey and Colquhoun, 1972). In addition, the exact pattern of diurnal variation depended on the nature of the task (Smith, 1992). Tasks involving a memory component (short term memory, card sorting) may result in the best performance in the early morning while perceptual-motor tasks (vigilance, reaction time) may peak in the evening (Hockey and Colquhoun, 1972). Angus and Heslegrave (1983; 1985) observed minimal increases in performance following the overnight decline; instead daytime performance plateaued. It was suggested that the interaction of sleep loss with continuous testing reduced the reserve capacity to improve performance during the daytime (Angus and Heslegrave, 1985). However, this plateauing may have resulted from the balancing effects of circadian improvements with the detrimental effects of continued wakefulness (Horne, 1985).

The degree to which performance is impaired by sleep loss also depends upon task variables such as duration, difficulty and complexity, pacing, knowledge of results, proficiency level, and memory requirements (Johnson, 1982). Tasks which take a long time to complete (10 min or more) or require sustained concentration or effort are especially affected by sleep loss (Donnell, 1969; Wilkinson, 1968; Wilkinson and Houghton, 1982). Short duration tasks can be sensitive to sleep loss when workload protocols are high and continuous (Heslegrave and Angus, 1985) or if the tasks call for sustained attention, high response rates, reasoning, or creativity (Dinges and Kribbs, 1991). In general, more cognitively demanding tasks are more sensitive to sleep loss than simple

tasks (William and Lubin, 1967; Wilkinson, 1968). However, monotonous tasks such as simple reaction time tasks can show profound and early sleep loss decrements (Dinges and Kribbs, 1991). During sleep loss, accuracy on "subject paced" tasks is maintained by slowing down, and for "externally paced" tasks accuracy is compromised (Broadbent, 1953). When feedback (or knowledge of results) is provided, task performance is more resistant to the effects of sleep loss, especially if the feedback informs the subject of his failure or success, rather than giving information on speed and accuracy (Williams *et al.*, 1959). The notion of failure or success may have more impact on the compensatory effort from the subjects than speed and accuracy (Dinges and Kribbs, 1991). Complex tasks requiring sequences of mental operations or short term memory utilization may not be sensitive to sleep deprivation if the interest for the task counteracts the effect of complexity (Dinges and Kribbs, 1991).

The control and/or measurement of motivation is critical to any study using performance tasks. Highly stimulating tasks, and environment, or the use of incentives will improve motivation and consequently minimize performance decrements due to sleep loss or even dampen the circadian oscillations (Dinges and Kribbs, 1991). Horne and Petit (1985) found that the positive effects of incentives on performance only last a day or two at the most. However it is important to note that no amount of interest, motivation or personal effort of any kind will alleviate completely the effects of sleep loss on performance (Johnson, 1982). Furthermore, individual factors (age, prior experience with sleep deprivation) may also influence motivation, circadian rhythms, and performance deterioration during sleep loss (see Colquhoun, 1982; Johnson, 1982; Smith, 1992, for reviews).

The degree of degradation in nocturnal performance will vary with the testing frequency and the ratio of the work-to-rest periods of the experimental protocol.

For example, Haslam (1982), using a testing frequency of three times per 24 hr, found a degradation in logical reasoning performance to 71% of baseline values after one night of sleep loss and 35% after two nights. Cognitive testing limited to once a day showed either no significant drop in performance after one night of sleep loss (Percival *et al.*, 1982) or a 10 to 15% drop in performance over 90 hr of wakefulness (Opstad *et al.*, 1978). Alluisi (1972) compared performance impairments between three groups of subjects following different work-rest schedules during sleep loss. One group followed a 4 hr on, 4 hr off schedule, a second group worked on a 4 hr on, 2 hr off schedule and the last group worked continuously for 48 hr. Performance dropped to about 70% of baseline in both the 4 hr on, 2 hr off and the continuous work groups after 40 and 48 hr of sleep loss respectively, whereas performance fell only to about 90% of baseline in subjects on the 4 hr on, 4 hr off work-rest schedule after 44 hr of sleep deprivation. Studies using continuous, demanding cognitive work conditions (Angus and Heslegrave, 1983; 1985) have reported decreases in reaction time performance to 76% of baseline values after one night of sleep loss, and a further 43% after two nights; logical reasoning performance decreased to 57% and 26% of baseline respectively, over the same period. In addition, substantial performance decrements (about 30% of baseline values) in serial reaction time, logical reasoning and encode/decode tasks were seen after only 18 hr of sustained work (Angus and Heslegrave, 1983; 1985).

It is also important to evaluate how subjective ratings can predict performance ability since subjective ratings may be the only possible means available to assess sleepiness and determine when the individual should discontinue work to avoid errors or accidents. The relation between performance and subjective sleepiness has been investigated in a number of studies and the results are somewhat conflicting. Glenville and Broughton (1979) found significant

correlations between performance measures (vigilance and choice reaction time tasks) and sleepiness ratings on the Stanford Sleepiness Scale (SSS) after one night of sleep loss. However, the SSS ratings did not reflect the individual's capacity to maintain performance following partial sleep deprivation even though the SSS was sensitive to the cumulative sleep reduction (Herscovitch and Broughton, 1981). Johnson *et al.* (1990, 1991) found that subjective ratings were marginally related to performance under a normal sleep/wake schedule whereas objective sleepiness was not significantly related to any performance tasks. Greater sleepiness ratings were associated with poorer performance on a 4-choice reaction time task. Nevertheless, subjective ratings were not related to decreases in performance during a tapping task. Moreover, significant differences in the time of the afternoon dip in alertness as measured by the sleep latency on the multiple sleep latency test (MSLT) were found in comparison to the subjective ratings (SSS and Visual Analog Scale). Objective sleepiness (MSLT) showed an increase in sleepiness at 1300h whereas subjective ratings showed an increase at 1500h. Johnson and colleagues suggested that their results support the belief that all measures of sleepiness do not measure the same thing. More recently, Gillberg *et al.* (1994) found a strong relationship between subjective ratings of sleepiness and performance during one night of sleep loss. Higher correlations between subjective sleepiness and performance were found in ratings obtained immediately after performance tasks. Similar results were reported by Glenville and Broughton (1979). Gillberg *et al.* (1994) suggested that the subjects may better evaluate their sleepiness after performing a task because they used their perceived quality of performance on the task to rate their sleepiness and, because the task protocol controlled external influences on alertness. Hence, these results suggested that the prediction of performance from subjective ratings may only be valid when a large variation in sleepiness is expected due

to the combination effects of prior wakefulness and circadian timing.

In summary, declines in mood and increases in subjective reports of sleepiness intensify with progressive sleep loss, and a circadian trend is apparent with poor ratings in the early morning which improve slightly in the evening. Subjective sleepiness and mood states need to be studied and evaluated because they appear to be positively related to performance level especially under sleep loss protocols and they may be important determinants in the decision to go to sleep (Dinges, 1989b; Johnson *et al.*, 1990; Gillberg *et al.*, 1994). In general, the level of functioning under protocols of sleep deprivation has been shown to be a function of various factors interacting with each other: the amount of prior wakefulness, the nature and type of tasks, circadian rhythms, testing frequency and work-to-rest ratio, individual, motivational and environmental variables. In general, performance shows a monotonic decrease under sleep loss superimposed on a rhythmic (circadian) pattern which can vary with the type of task. The specificity of each task (difficulty, pace...) will determine its sensitivity to sleep loss and the factors affected such as speed or accuracy. In addition, continuous and demanding conditions result in earlier and greater decrements in performance. Highly motivated subjects and stimulating tasks will reduce performance impairments but will not alleviate completely the effects of sleep deprivation. Finally, since subjective ratings of sleepiness may not always reflect and/or predict objective measures of sleepiness they should be used in addition but not necessarily as a substitute for objective performance measures of sleepiness.

4.0 NAPPING: A COUNTERMEASURE

Although the generally accepted upper limit for continuous and intensive work

under total sleep loss is between 2 to 3 days, some detrimental effects may appear within the first 24 hr of sleep deprivation (Stampi, 1992). In view of the detrimental effects of sleep deprivation on mood and performance efficiency mentioned in the previous section, two types of strategies that may enable performance to be maintained at a higher level during continuous work or emergency situations, have emerged from the literature.

Characteristic of the first type, are strategies that use alerting substances to extend wakefulness and counteract performance degradation (Stampi, 1991). When Bonnet *et al.* (1995) compared the effects of caffeine versus naps as possible countermeasures to 48 hr of sleep deprivation, they reported that naps provided longer and more evenly graded increases in performance and alertness than caffeine. Caffeine showed a dose response effect; one large dose of caffeine (400 mg) per 24 hr caused a peak in performance and alertness which dissipated by 6 hr. Small repetitive dose of caffeine (150 or 300 mg) every 6 hr maintained performance and alertness better than a large single dose. Both nap and caffeine effects were more effective during the first 24 hr than the next 24 hr. Another recent study compared the effects of two stimulants (modafinil and dextro-amphetamine) versus a placebo on cognitive performance during 64 hr of sleep deprivation (Pigeau *et al.*, 1995). Both drugs ameliorated or restored performance by about 25 to 30% of their baseline values when compared to performance of the placebo group. The placement of the first two drug treatments matched the placement of two 2 hr naps used in previous studies (Angus *et al.*, 1992) as a countermeasure for sleep loss and conducted by the same laboratory. Both naps scheduled at 2200 and 0400h had been found to be effective in maintaining or recuperating impaired cognitive performance (Angus *et al.*, 1992). When the results from the nap studies are compared with those of the drug study, it seems that both drugs

were able to maintain performance for a longer period after the 2200h treatment compared to the nap scheduled at the same time. However, drugs and naps had similar recuperative effects on performance when scheduled between 0400 and 0600h after 46 hr of wakefulness. Pharmacological stimulants are of relatively short term use because of well known deleterious effects including the development of tolerance, dependency, increase in heart rate and blood pressure, and withdrawal effects (Lagarde and Batejat, 1994; Bonnet *et al.*, 1995).

The second type of strategy uses the only non-pharmacological procedure to restore performance efficiency, sleep. Different approaches are offered in obtaining sleep under around-the-clock work environments. A minimum of 4.5 to 5.5 hr of sleep per day is necessary to maintain an acceptable level of performance (60 to 70 % of baseline level) but not necessarily mood for acute periods of time (Johnson *et al.*, 1977; Dinges, 1989). However, under most sustained work environments, an uninterrupted 5-hr block of sleep might be difficult to obtain because it may be in conflict with work demands (Stampi, 1991). In these cases, short periods of sleep, namely naps, have been found to be disproportionately effective in terms of recovery functioning and offer more flexibility in scheduling (Stampi, 1989). The term nap can take on different meanings depending on the context and user. Before examining the different napping strategies, a brief summary of the main definitions of the term nap in the sleep literature will be presented.

The Oxford English Dictionary (1971) defines a nap as "a short or light sleep, especially one taken during the day". Nap, in the sleep literature, is mainly used for any sleep period shorter than the habitual nocturnal sleep period. More specifically, four distinct conditions for napping can be delineated:

1) The most commonly usage for 'nap' is considered to be a short sleep episode that supplements the usual full length nocturnal sleep. Typically, these naps are taken during daytime, often in the afternoon. Dinges (1989b) observed a remarkably consistent nap length among a variety of napping studies, ranging from 0.58 to 1.60 hr with an overall mean of 1.21 hr. In general, naps were not shorter than 15 min or longer than 2 hr.

2) In the context of prolonged wakefulness and sustained work operations, naps are considered brief opportunities (less than 4 hr) to overcome sleepiness. Naps, in these cases, occur after prolonged sleep deprivation and may be the only sleep permitted in order to enable the individuals to continue their duties.

3) Repeated short sleep periods may be used to replace a continuous episode of nocturnal sleep. These naps may be scheduled at regular and irregular intervals throughout the day or night. Nap durations in this category are usually shorter than 4 hr but longer than 10 min and will result in the accumulation of 8 hr of bed time over 24 hr (Stampi, 1992).

4) Naps may be involuntary sleep episodes of patients suffering from excessive daytime sleepiness. Naps taken by individuals suffering from a variety of sleep disorders, such as narcolepsy and sleep apnea, can be subdivided into two types: voluntary or partially voluntary sleep episodes that occur during the daytime, and involuntary naps that are irresistible sleep episodes or sleep attacks over which the person has no control.

In summary, any sleep period with a duration of less than 50% of an individual's average nocturnal sleep is considered to be a nap (Dinges *et al.*, 1987). This definition has the advantage of including all previous definitions of naps. This thesis will use the term "nap" to denote any sleep period less than 4 hr in

duration taken during the day or night.

Napping Strategies

Recovery and prophylactic napping: The typical way of obtaining some sleep under sustained operations is to sleep whenever possible (Naitoh, 1992; Stampi, 1992). Angus *et al.* (1987) showed that a 2-hr evening nap (2200h to 2400h) given prior to the second night of sleep deprivation (after 40 hr of wakefulness) prevented the drop in performance that usually occurs during the second night of sleep loss. In a second group of subjects given a 2-hr nap between 0400h and 0600h on the second night after 46 hr of wakefulness, the nap restored performance to the level of the previous day even after performance had already started to drop. Dinges *et al.* (1985; 1987) were especially interested in the beneficial effects of napping in anticipation of sleep loss compared with napping after some sleep debt accumulation. *Prophylactic* napping or napping in advance of sleep loss was originally proposed by Orne (Evans and Orne, 1976) but not appropriately considered because it contradicted the view that 'sleep could not be stored'. Subjects in Dinges' study were given a single 2-hr nap opportunity placed at one of five times (after 6, 18, 30, 42 and 54 hr of prior wakefulness) during a 56-hr sleep deprivation period. The results showed that naps taken prior to profound sleep loss (after 6 or 18 hr of wakefulness) had a stronger and longer lasting positive effect on performance than later naps (Dinges *et al.*, 1987). In contrast, none of the naps had much effect on mood parameters (Dinges *et al.*, 1988) suggesting that even though prophylactic naps improved the subjects ability to perform, they continued to report feeling fatigued and sleepy, regardless of the timing of the nap as if they were not aware of the nap benefits.

Polyphasic sleep/wake patterns: In order to systematically prevent the

accumulation of sleep loss during continuous work scenarios, it may be possible to fragment sleep into short periods and disperse them throughout the 24-hr period. Such sleep strategies called polyphasic and/or ultrashort sleep schedules, will reduce the waking interval between one nap and the following one and prevent or at least postpone sleep debt accumulation (Stampi, 1992; Dinges *et al.*, 1980). Polyphasic sleep schedules can be divided in two groups, one which allows the subjects a total time in bed of 8 hr, whereas the second involves sleep reduction. However, polyphasic sleep patterns resulting in approximately 8 hr in bed every 24 hr are not appropriate for sustained operations. Polyphasic sleep schedules involving various levels of sleep reduction could represent feasible and preferable strategies in adapting to continuous work scenarios and reducing fatigue and performance deterioration (Stampi, 1989; Naitoh, 1992).

Effects of naps

Data on the benefit of naps to capture some sleep under continuous work periods have revealed two major concerns: the sleep efficiency of the nap; and sleep inertia upon awakening (Naitoh, 1992). Multiple napping strategies appear to reduce sleep efficiency (total sleep time as a proportion of total time in bed) in comparison to continuous nocturnal sleep with an efficiency of the order of 90% or higher (Carskadon and Dement, 1989). In the studies of Moses *et al.* (1975) and Lubin *et al.* (1976), subjects had the opportunity to nap for 60 min every 160 min across a 40 hr study for a total bed time of 6.5 hr every 24 hr. Sleep efficiency was 47.5%. Other studies providing 8 hr of total bed time per 24 hr (Carskadon and Dement, 1975; 1977; Weitzman *et al.*, 1974) found an average sleep efficiency of about 50%. Such a decrease in sleep efficiency is usually attributed to longer sleep onset and/or unconsolidated nap sleep. However, a lower nap sleep efficiency may also be a result of sleep duration

rather than sleep onset or consolidation. For example, if the subjects take ten minutes to fall asleep in a 30 min nap, sleep onset may represent a third of the available sleep period resulting in a low sleep efficiency. If the same subjects had slept 60 min, their sleep efficiency would be higher even though they still took 10 min to fall asleep and did not wake up during the nap. Thus, the longer the nap is, the higher its efficiency can be. Moreover, Stampi (1992) suggested that nap sleep efficiencies would improve once the subjects have adapted to the new schedule. It appears that nap sleep efficiency is related to the opportunity and/or motivation of subjects to adapt to the polyphasic sleep schedules. Naitoh (1992) suggested the use of biofeedback and autogenic relaxation techniques or to use quick-acting hypnotics to induce rapid sleep onset and improve sleep efficiency.

Sleep inertia is characterized by a reduction in performance capability immediately following abrupt awakening from sleep relative to pre-sleep status (Naitoh and Angus, 1989). Sleep inertia is usually brief under normal circumstances (Wilkinson and Stretton, 1971; Dinges *et al.*, 1980) and more prolonged during sleep deprivation (Naitoh, 1981; Dinges, 1990). Napping characteristics (sleep duration, timing and architecture) and their effects on sleep inertia will be discussed in detail in Section 5.0.

One methodological detail that complicates the comparison of findings in the napping literature is the characteristics (type, duration and pace) of the task used to measure performance. Some tasks may be more sensitive to sleep inertia effects than others and after sleep inertia has dissipated, some tasks show more improvement as a result of a nap than others. Although no task seems exempt from sleep inertia effects (Dinges *et al.*, 1985), tasks which require a higher level of cognitive skills (tachistoscope threshold recognition and 'clock reversal') are more prone to deterioration upon awakening than a

simple reaction time task (Scott, 1969). In Hartman & Langdon's study (1965), a multi-dimensional pursuit test (MDP) and a complex behavior simulator task (CBS) were compared. Both tasks showed strong sleep inertia effects, but performance recovery at the end of the 10 min testing indicated only a minor trend toward improved performance in the MDP test compared to approximately 25% improvement in response time in the CBS task. The MDP test appeared to be more demanding in terms of concentration and neuromuscular coordination. Two studies (Dinges *et al.*, 1988; Stampi, 1992) showed that performance decrements measured by the Descending Subtraction Test (DST) were more pronounced and did not recuperate as much as performance measured by the Memory and Search test (MAST). After severe sleep loss, Haslam (1982) observed that subjects were unable to start a logical reasoning test (LRT) within 5 min of awakening at 0545h. Moreover, Wilkinson and Stretton (1971) found that the addition and the coordination tasks were more affected by arousal towards the end of the night, whereas reaction time was more affected by early awakenings.

Most investigators have used performance tasks as a probe to investigate sleep inertia but some studies have also tried to use electroencephalographic (EEG) indices to document sleep inertia (Pigeau *et al.*, 1987; Dinges, 1990). Dinges (1990) reported no differences in the EEG obtained during pre- and post-sleep awakening whereas the DST performance showed profound sleep inertia effects. However, Pigeau *et al.* (1987) found that sleep inertia is characterized by an EEG resembling an early phase of sleep (stage 1). Differences between the two electroencephalographic studies might be attributed to the fact that in Dinges' study, pre-sleep EEG recordings were not made while performing the DST as in the post-sleep recordings, whereas pre- and post-sleep recordings in Pigeau's study were both taken during a 4-min eyes closed relaxation period.

Sleep inertia seems to more systematically affect higher level cognitive functioning or mental processing tasks than others.

Seminara and Shavelson (1969) found an inverse relationship between the length of time required to perform a task and sleep inertia. They suggested that subjects were more alert in the performance of longer tasks: in other words, the sleep inertia effects were wearing off. Since sleep inertia is a transient phenomena, investigators often use short duration tasks (5 min or less) which are repeated several times (Balkin & Badia, 1988; Balkin *et al., 1989*) or longer tasks later analyzed by dividing the performance period into short blocks (Tassi *et al.*, 1992). Tasks do not need to be of long duration or embedded in long sessions of continuous work to be sensitive to the effects of sleep inertia.

Based on sleep loss effects, sleep inertia is expected to reduce work rate on self-paced tasks in order to maintain accuracy, whereas an increase in the number of errors will occur in externally-paced tasks compromising accuracy (Dinges, 1992a). Nevertheless, a reduction in accuracy and work rate was found immediately upon awakening in a self-paced addition task (Balkin & Badia, 1988) and subtraction task (Dinges *et al.*, 1981). Furthermore, when subjects from Dinges' study slept in a 'sleep alerting environment' (i.e., room with light on and background noises such as footsteps, paging door closing, etc.), a reduction in work rate was obtained but accuracy was maintained.

Another interesting aspect of sleep inertia is how subjects perceive and estimate their fatigue, sleepiness and mood. Following a nap taken after a sustained period of wakefulness, the magnitude of performance impairment during severe sleep inertia is often misjudged or denied by the subject. This difficulty in evaluating how sleepy one feels does not occur if the nap is taken before sleep deprivation (Dinges, 1989b, 1990; Balkin & Badia, 1988). Dinges

(1990) reported a clear dissociation between subjects rating of sleepiness (improved) and their performance (deteriorated) upon awakening from a nap taken beyond 18 hr of wakefulness. Finally, Davidson (1987) found no deterioration in subjective ratings immediately following a 2-hr nap taken after 40 hr of sleep deprivation.

As sleep inertia dissipates, performance following a nap will return to or exceed pre-nap levels (Dinges *et al.*, 1980). Lubin *et al.* (1976) found that 1-hr naps taken every 220 min helped maintain performance on vigilance and addition tasks over 48 hr but failed to maintain word memory performance and subjective sleepiness. Naitoh (1982) observed that 3-hr naps taken after 20 hr of wakefulness reduced performance deterioration on a four choice reaction time task but failed to restore or maintain performance on other tests (alphanumeric visual vigilance, logical reasoning, tapping, mood and fatigue). Over 42 hr, one hour naps were permitted every 7 hr and resulted in performance impairment on an addition task, performance improvement over time on a tracking task, and performance maintenance on a pattern memory task (Mullaney *et al.*, 1983). Webb (1985) reported performance improvement in addition attempts, vigilance (percent hits) and word detection (false alarms) in three groups allowed either two 2-hr naps or one 4-hr nap over 3 days. No improvements were observed in any other measure (word memory, reaction time, digit-symbol, object-uses, reasoning, long term memory, anagrams, visual search, line judgment, sleepiness and mood). It is apparent that the type of measurement chosen will affect subsequent performance improvement.

In the situations where the naps are taken prior to prolonged wakefulness (Gillberg, 1984; Godbout & Montplaisir, 1986; Dinges *et al.*, 1987; 1988), performance enhancement may not always be observed immediately following the naps but sometimes it is apparent only hours later (1.5 to 10 hr later). Two

hour naps taken once sleep deprivation was present (after 18 to 42 hr of wakefulness) showed beneficial effects on performance within an hour after the nap (Dinges et al, 1987, 1988). Positive effects lasted anywhere between 6 and 30 hr post-nap (Dinges *et al.*, 1987). Even though napping in advance of or during sleep loss appeared to enhance subsequent performance, naps had no effect on self-report measures of mood and sleepiness (Dinges, 1992b). Most studies (Dinges *et al.*, 1988; Gillberg, 1984; Naitoh, 1982) report that subjects in the laboratory are not aware of any performance improvement subsequent to a nap taken during sleep deprivation.

In brief, prophylactic naps are beneficial for preventing performance from further decrements, whereas naps taken during sleep loss (recovery naps) will help recovery from already degraded performance levels. Under sleep reduction circumstances, it is clear that naps help performance but do not fully restore the subjects ability to perform at initial baseline levels, nor do they substantially improve mood ratings. The benefits of multiple napping strategies to delay or reverse the deleterious effects of sleep loss on performance need to be balanced against two important drawbacks, reduced nap sleep efficiency and the effects of sleep inertia. It also appears that as sleep loss accumulates, the probability of waking up from a nap with severe sleep inertia increases. Moreover, the nature of the task performed will influence its sensitivity to sleep inertia and subsequent potential for recovery.

5.0 SLEEP INERTIA

A period of disorientation, confusion and inferior task performance referred to as 'sleep drunkenness' (Broughton, 1968; 1973), 'postdormital disorientation' (Association of Sleep Disorders Centers, 1979) and 'sleep inertia' (Lubin *et al.*,

1976) occurs a few minutes after awakening from sleep. The term sleep inertia has been recently applied to describe awakening impairments in healthy persons (Dinges, 1989b). It is characterized by poor performance immediately after awakening compared to pre-sleep performance. Performance returns to normal following approximately 15 min of continuous wakefulness (Dinges *et al.*, 1985). The largest decrements are observed during the first 3 to 4 min after awakening (Wilkinson & Stretton, 1971). Following extended wakefulness, sleep inertia impairments upon awakening from a nap can be longer lasting and more severe than the degrading effect of sleep deprivation on performance i.e., the performance degrading effect of sleep inertia is additive to performance degradation induced by sleep loss (Dinges, 1990; Naitoh *et al.*, 1993).

Several other factors influence the severity of sleep inertia and need to be considered: sleep duration, time of napping, prior wakefulness, sleep architecture and sleep stage at awakening. Each of these factors will be discussed separately in the following sections, although it is clear that they interact in any given situation.

Sleep duration

Very few studies have compared the effects of different nap durations on the degree to which performance is impaired by sleep inertia (sleep inertia intensity or severity). Stampi (1989) described how solo sailors on long distance voyages were more concerned with the duration of the nap rather than the timing of their sleep episodes. Each individual tried to sleep for the number of minutes (or hours) he/she had found ideal for producing maximum restoration and minimum impairment immediately upon arousal (sleep inertia effect). They also reported avoiding oversleeping during stable environmental conditions because it would produce unpleasant feelings of drowsiness and increase the

sleep inertia effects. Sailors with the best performance in the race adopted mean sleep episode durations between 20 and 60 min. Based on the "solo sailors" results, Stampi (1992) performed an experimental study utilizing napping strategies comprised of 4 hr of continuous night sleep and another 4 hr of sleep in the form of naps (20, 50, and 80 min) distributed regularly throughout the remaining 20 hr of the day. Stampi compared performance on a memory and search test (MAST) and descending subtraction task (DST) measured immediately upon awakening from the naps. Performance decrements (measured by the MAST) upon arousal were greater following the 80 min naps, and least following the 20 min naps. However, Stampi reported greater sleep inertia effects (measured by the DST) following the 50 min naps as compared to 20 min naps. Sleep stage at arousal was considered the major factor responsible for the performance decrements. Duration of the naps had an indirect effect on the sleep architecture and stages from which the subjects were awakened. Shorter naps did not allow enough time to proceed into 'deeper' sleep stages (SWS) whereas longer naps permitted enough time to return to 'lighter' sleep stages (1, 2, and REM) (Stampi, 1992). Similar results were found by Mullington and Broughton (1994) with narcoleptics patients who showed pronounced sleep inertia for both the descending subtraction test and the reaction time task following short naps (30 min) taken at 0950, 1245, 1540, 1835, and 2130h compared to a long afternoon nap (120 min) taken at 1540h.

Thus, very short naps (< 30 min) and longer naps (> 80 min) result in less pronounced sleep inertia compared to intermediate nap lengths (> 30 min and < 80 min). Sleep architecture and stage upon awakening will vary in relation with nap duration. Sleep inertia effects appeared to be more severe if sleep comprises some SWS, and if SWS occurs in close proximity to sleep termination. Intermediate nap lengths (> 30 min and <80 min) appear to be

long enough to initiate SWS, but did not provide enough time to return to 'lighter' sleep stages, resulting in more SWS awakening and consequently more sleep inertia.

Temporal placement of naps

Studies evaluating the effects of temporal placement of a nap on sleep inertia have placed naps of constant duration at different times of the day and compared post-nap performance. Naitoh (1981) found that a 2-hr nap taken at noon caused less sleep inertia than a 2-hr nap taken at 0400h even though the afternoon nap followed a longer sleep deprivation period (53 hr compared to 45 hr in the early morning nap). The results of Dinges and colleagues (1985) are consistent with Naitoh's findings. In the Dinges *et al.* (1985) "quasi continuous work" study, each of five groups were given a 2-hr nap occurring either near the circadian peak (1500h) or near the trough (0300h) of body temperature and preceded by a different amount of prior wakefulness (6, 18, 30, 42 and 54 hr). Naps taken at the circadian trough of body temperature produced immediate post-nap cognitive decrements nearly identical to naps taken 12 hr later near the circadian peak despite relatively equivalent sleep patterns (stages and amount). Conversely, naps taken near the circadian peak produced less immediate performance decrements than trough naps taken 12 hr later. Dinges *et al.* (1985) findings suggest an additive influence from circadian timing and sleep deprivation on immediate post-sleep performance. Thus, it appears that circadian influences at the circadian trough increase sleep inertia effects whereas at the peak they add little or even attenuate post-nap impairments. In Lavie and Weler's (1989) resisting sleep paradigm, the subjects were required to resist sleep repeatedly at frequent interval throughout the day after one night of total sleep deprivation. The subjects followed a schedule of 7 min awake in bed with eyes closed (resisting sleep) and 13 min awake outside the bedroom,

for 24 hr. The ultradian schedule was interrupted at 1500h for a 2-hr nap during the first experimental period. At least 10 days later, the same subjects followed a second experimental period identical to the first one except for the timing of the 2-hr nap, which occurred this time at 1900h. This resisting sleep paradigm showed less sleep inertia effects on sleepiness (measured by the minutes of sleep in a subsequent 7 min resisting sleep period) and mood ratings after the 1500h nap compared to the 1900h nap. Thus, the authors speculated that if the end of an afternoon nap coincides with the 'forbidden zone for sleep' (early evening period of decreased propensity for sleep), it would help to lower the sleep inertia effects upon awakening.

On the other hand, several studies have shown no significant difference in sleep inertia attributable to the circadian placement of the naps. Angus and coworkers reported finding similar degrees of sleep inertia occurring after a 2-hr evening nap (2200h) following 40 hr of sleep loss and after a 2-hr morning nap (0400h) taken after 46 hr of sleep loss (Naitoh and Angus, 1989). In another 64 hr sleep deprivation study, Naitoh *et al.* (1993) also found that sleep inertia measured with the logical reasoning task upon awakening from 20 min naps taken at 6 hr intervals, did not show any circadian variation. However, they recommended that scheduling of naps around the circadian trough of body temperature should be avoided because of unpleasant psychological reactions to nap awakenings experienced by some subjects. Naitoh called this phenomenon "nap avoidance or aversion".

In contrast, Tassi *et al.* (1992) found more sleep inertia after an early 1-hr nap (0000h) than after a later 1-hr nap (0300h) where greater performance decrements were expected because of the additive influence of circadian timing and sleep loss. Patients with narcolepsy showed greater and more persistent sleep inertia after naps taken at 1540h and 1835h compared to naps at 0950h,

1245h and 2130h (Mullington and Broughton, 1994). These authors suggested that in situations with little or no sleep deprivation, the sleep stage upon awakening (SWS), rather than the circadian placement of the nap, influences the amount of sleep inertia early during the night or in the afternoon.

Another interesting aspect is how subjects' estimations of sleepiness are influenced by the circadian timing of the naps and their correspondence to sleep inertia effects on performance measures. During four nights of sleep disruption/restriction, Balkin & Badia (1988) observed a stable sleep inertia effect on the number of addition problems correctly completed when awakened at 0040, 0140, 0240, 0340, 0440, and 0540h each night. However, self-rated sleepiness measured at the same times tended to increase across the night. Similarly, Dinges (1990) reported that subjects' self-ratings of sleepiness and electroencephalographic measures did not relate to the severity of sleep inertia seen in the performance measures. Interestingly, Balkin and Badia's subjects overestimated their sleepiness ratings at the end of the night whereas Dinges' subjects underestimated their sleepiness ratings in comparison with their performance measures.

In brief, various studies to date have shown no consistent relationship of sleep inertia effects and circadian placement of naps. The variable effects reported may be the results of the differing procedures, subjects, and methodologies that were used in the studies for evaluating sleep inertia. In addition, previous studies have often simply compared napping at different phases of the circadian cycle following varying amounts of prior sleep loss, thereby confounding time of day with prior wakefulness. Finally, sleepiness ratings were not related in any obvious way to the circadian timing of the naps and in fact, sleep inertia seems to affect the subjects capacity to adequately appreciate how sleepy they are.

Prior wakefulness

As sleep loss accumulates the probability of waking up from a nap with severe sleep inertia increases (Dinges *et al.*, 1985; Balkin & Badia, 1988). This is because after prolonged sleep loss the accumulated sleep debt is not fully discharged by the nap. The longer the sleep loss, the greater is the sleep pressure (propensity for falling asleep), and the greater the depth of nap sleep (amount of SWS), the more severe is the nap sleep inertia (Dinges *et al.*, 1985). Dinges and colleagues observed that naps taken after only 6 or 18 hr of wakefulness contained lighter sleep than later naps which had greater amounts of SWS because more sleep loss had accumulated (30, 42, and 54 hr). Similarly, Balkin and Badia (1988) found a reduction in performance on an addition test and a tendency to obtain more SWS across the four consecutive nights of sleep disruption/restriction. Rosa & Bonnet (1985) examined performance upon repeated awakenings during baseline sleep and across two recovery nights following 40 or 64 hr of wakefulness. They reported that performance measures upon awakening showed greater decrements during recovery sleep compared to baseline. They suggested that deeper recovery sleep (as measured by sensory arousal thresholds) caused by sleep deprivation, amplified sleep inertia effects on performance. In contrast, Tassi *et al.* (1992) showed more intense sleep inertia after an early 1-hr nap taken at 0000h than at 0300h. In this case, the amount of prior wakefulness could not account for the smaller sleep inertia effects upon awakening from the 0300h nap. The difference in sleep inertia intensity between the naps was explained by the higher prevalence of stage 4 awakenings after the midnight nap.

In general, it appears that naps taken after prolonged sleep loss result in more serious and prolonged sleep inertia. In fact, prior wakefulness might have an indirect influence on the intensity of sleep inertia via the sleep architecture of

the nap. As Dinges (1989b) explains "*Since sleep depth is a function of sleep pressure resulting from prior wakefulness and circadian phase, nap sleep inertia will vary in relation to these factors, as well as the duration of the nap*".

Sleep architecture and stage at awakening

The sleep composition and structure of naps are subject to the same rules that govern sleep (see section 1). In brief, the shorter the nap, the greater the amount of NREM sleep, REM sleep appearing only after a complete NREM sleep cycle. Morning naps will have higher REM percentages than afternoon or evening naps. However, increasing prior wakefulness will result in greater pressure for SWS, and hence the higher the percentage of SWS in the nap. Thus, nap architecture depends on its duration, circadian timing, and prior wakefulness. These same factors, as discussed previously, influence sleep inertia. In fact, these factors may directly affect sleep architecture and consequently have an influence on post-nap performance.

Several authors have reported that the amount of SWS during sleep is related to cognitive decrements upon awakening, that is, the greater the amount of SWS, the more severe the decrements (Dinges *et al.*, 1981; Dinges *et al.*, 1985; Lavie and Weler, 1989; Stampi, 1992). In addition, early morning naps containing REM sleep (and very little SWS) resulted in very little or no sleep inertia effects (Mullington and Broughton, 1994). Equivalent amounts of SWS produced comparable increases in daytime sleepiness measured by an MSLT even though subjects were awakened either from stage 2 or from REM sleep (Glovinsky *et al.*, 1990). Dinges (1990) suggested that the severity of sleep inertia is a function of depth of sleep. Sleep depth has been associated with a shorter latency to SWS onset (Dinges, 1986), high amounts of SWS (Dinges, 1986; Feinberg *et al.*, 1987; Webb and Agnew, 1971), failure to respond to an

alarm (Rosa and Bonnet, 1985), and a rapid return to sleep following an awakening (Bonnet, 1978). However, some studies have shown that SWS and REM are unrelated to sleep inertia. During frequent sleep disruption over several nights, subjects showed severe performance decrements upon awakening despite being denied most of their SWS and REM sleep (Bonnet, 1985; Downey and Bonnet, 1987). In fact, the severity of sleep inertia may be related to the individual's need to sleep (sleep pressure). The intensity of sleep pressure is normally associated with the amount of SWS sleep in the nap.

Severity of sleep inertia is often assumed to be directly related to the preawakening stage of sleep (Dinges, 1990). Tassi *et al.* (1992) suggested that the difference in sleep inertia intensity seen between the midnight nap and the 0300h nap was the result of more stage 4 awakenings in the early naps than in the later ones. Awakening from SWS exacerbated the severity of sleep inertia with respect to logical reasoning (Naitoh *et al.*, 1993), stroboscopic flashes (Feltin & Broughton, 1968), descending subtraction tasks (Stampi, 1992), and short and long term memory performance (Bonnet, 1983). Moreover, arousals from REM (Feltin & Broughton, 1968; Stones, 1977) or stage 2 (Stampi, 1992) produced the least performance decrements. In a study looking at the effects of SWS versus REM sleep arousal on a stroboscopic performance test, Feltin and Broughton (1968) reported that subjects awakening from REM sleep were initially more alert, but soon tended to lack interest (nonmotivated test) and fall asleep whereas subjects arousing from SWS were initially worse, but progressively awakened with time. Thus, one might expect performance improvement and motivation to vary according to which sleep stage subjects are awakened from. In contrast, sleep stage upon awakening failed to predict performance impairments in studies where subjects were awakened from the same sleep stage but resulted in different performance

impairments. Instead, performance impairments in these studies were found to vary as a function of time of night (Rosa *et al.*, 1983), to be dependent on the amount of sleep deprivation (Akerstedt & Gillberg, 1979; Rosa *et al.*, 1983), or on the frequency of awakenings throughout the night (Downey & Bonnet, 1987).

In summary, the severity of sleep inertia is not only associated with sleep stage upon awakening but also, with the nap architecture. Sleep inertia increases with sleep depth and more specifically with the preawakening depth of sleep making the stage upon awakening an indicator of the degree of performance impairments upon arousal.

In conclusion, published research to date suggests that:

1) Napping prior to sleep loss can prevent performance deterioration whereas naps taken during sleep loss can help maintain or restore already degraded performance levels. One of the most important factors in the design of an effective napping strategy is to find a sleep schedule that provides maximal recuperation with the minimal sleep inertia effects particularly in situations involving multiple naps, because with each nap there is a potential for long and/or severe sleep inertia which would reduce the benefits of napping.

2) Sleep inertia intensity will also vary as a function of sleep pressure. Sleep tendency will depend on prior wakefulness, prior sleep duration and circadian placement of sleep. Prior sleep loss will increase the pressure for sleep causing deeper sleep which as a result exacerbates the mean performance deficits and alertness upon awakening. Variations in the circadian timing and duration of the naps will alter their sleep architecture

and consequently the stage at awakening. It is not yet clear whether the circadian timing of the naps has any direct influences on the subsequent severity of sleep inertia.

3) It has been shown that profound sleep inertia can occur even if the subjects have judged themselves to be awake and relatively alert. This suggest that subjects are not always able to judge accurately their ability to perform certain tasks based on their subjective feelings of sleepiness.

4) Another key factor in using napping strategies is the ability of the individuals to fall asleep quickly and at almost any time of day. Shorter sleep latencies would improve sleep efficiencies and the capacity to adapt to the schedule, and consequently enhance or optimize the benefits of napping strategies.

Finally, there are still no guarantees that in certain situations people will have time to completely wake up before starting to work. Understandably, more information is needed on the relationship between the circadian placement of the nap and the severity of sleep inertia. Judicious timing of the naps may permit maximum performance benefits with minimum debilitating effects from sleep inertia.

6.0 OBJECTIVES AND HYPOTHESES FOR THIS STUDY

The main objective of the present study was to investigate the time of night effect on sleep inertia upon awakening from naps, while minimizing the confounding effects of sleep deprivation.

This objective was examined in a two protocol (A and B) experiment by varying the circadian placement in the normal sleep period of two series of four 1 hr naps and comparing the severity of sleep inertia effects on cognitive performance and subjective measures immediately upon awakening. One hour naps in protocol A were scheduled in the first half of the normal sleep cycle to begin between 0000h and 0430h, and in the latter half of the sleep cycle at 0300h and 0830h in protocol B. During both protocols, each nap was separated from the following one by 30 min of wakefulness to assess cognitive performance and subjective measures. To minimize the confounding effect of sleep deprivation that would result from the 3 hr difference in prior wake time preceding the night naps between the two protocols, a 90 min afternoon nap was introduced and scheduled to terminate 9.5 hr before the first night nap of each protocol.

Because of the additional 3 hr spent awake after an 8-hr nocturnal sleep period prior to the afternoon nap in protocol B (1600 to 1730h) compared to protocol A (1300 to 1430h), the sleep architecture of the former nap will demonstrate a higher amount of SWS as compared to the afternoon nap in protocol A (1300 to 1430h). With respect to cognitive function, afternoon naps will help maintain cognitive performance prior to the first nighttime nap at comparable levels in both protocols A and B.

The following hypotheses were proposed:

1. With respect to cognitive functioning and subjective ratings immediately upon awakening, significant difference will be found between measures taken before and after the naps i.e., significant sleep inertia effects will be found.

2. More specifically, sleep inertia effects are not expected to differ among naps taken at a different time of day i.e., no time of night effects on sleep inertia will be found.

3. Cognitive functioning impairments found immediately upon awakening from a nap will recover to initial level of performance prior to the subsequent nap.

4. Subjective assessments of fatigue, sleepiness, mood and drowsiness will demonstrate sleep inertia effects upon awakening from nighttime naps in concordance with sleep inertia effects measured with cognitive performance tasks.

CHAPTER III

EXPERIMENTAL METHODOLOGY

1.0 SUBJECTS

Fourteen healthy male non-smokers ranging between 20 and 35 yrs of age (mean age 26 years) volunteered for this study (summary of subjects profile in appendix B). Female subjects were excluded from the study because of a recent finding by Moldofsky *et al.* (1995) showing a delayed and reduced onset of SWS and concomitant natural killer (NK) cell activities during the high progesterone phase of the menstrual cycle in comparison to the low progesterone phase. Until more research is conducted to elucidate possible gender differences with regard to sleep and the immune system, it was preferable to study one gender at this time. Each subject's level of health was assessed with medical and psychiatric interviews, and a physical examination. Volunteers were asked to complete the Cornell Medical Health Index questionnaire (Weider *et al.*, 1948), the Derogatis SCL-90-R psychological questionnaire (Derogatis, 1977), a physical symptoms inventory (Wahler, 1973), a morningness-eveningness questionnaire (Horne & Ostberg, 1976), and the Toronto Western Hospital sleep-wake questionnaire (Moldofsky & Hefez, 1984). Criteria for exclusion from the study were: irregular sleep habits, average bedtime length greater than 8.5 hr or less than 6 hr, problems with falling asleep or staying asleep, frequent drug or alcohol usage, or major medical or psychiatric disorders. All subjects were informed about the general purpose of the experiment and the procedures to be employed, with the understanding that they were free to withdraw from the experiment at any time.

The subjects were required to complete the University of Toronto Human Review approved consent form (appendix A) before participating in this research, which was approved by the Human Review Committee of the University of Toronto. Volunteers were required to abstain from the use of alcohol and caffeine during the study, and were instructed to maintain a normal sleep/wake schedule during the week preceding the study and to refrain from napping during the daytime. Subjects chosen were not frequent nappers (less than once a week) and had no known difficulty to nap in the afternoon. All subjects successfully completed the study, and no adverse medical or psychological problems were reported during the entire experiment. One subject's data were not used in the statistical analysis because he had sleep deprived himself prior to the second protocol. After observing that the subject fell asleep at inappropriate times, he was asked why he was so sleepy. The subject admitted that on the night prior to the beginning of the study he did not sleep even though he had been reminded to keep regular sleeping habits.

2.0 PHYSIOLOGICAL MEASURES

Throughout the experiment all subjects wore a Medilog MR-90 ambulatory cassette recorder capable of storing 8 channels of physiological variables on a standard 120 min audio cassette (low noise) for approximately 24 hr. The recorder generated and recorded time on a separate channel which could integrate the information from a manually activated event marker. Electrophysiological data recorded on the Oxford portable MR-90 cassette recorder were played back using the Oxford Medilog 9000 display system. The MR-90 continuously recorded input from central (C3/A2, C4/A1) and occipital (Oz/A1) electroencephalograms (EEG), 2-lead electrocardiogram (ECG), submental electromyogram (EMG), left and right electroocculograms (EOG).

Core body temperature was recorded continuously on a portable Oxford MR-90 recorder during both protocols by rectal probes (Yellow Spring Instrument #401) inserted approximately 10 cm into the rectum. The thermistor had a resolution of 0.01°C and a temperature range of 35 to 40°C. Temperature data were digitized and stored every min. Sleep EEG's were also recorded during sleep periods on a Grass model 78D or 8-24D polygraph to minimize the loss of data. Electrode placement and scoring (30 sec epochs) were done according to standardized criteria (Rechtschaffen and Kales, 1968). The latency to sleep onset (Lat.SO) was calculated in minutes from the start of sleep recording at lights out to the first epoch of any stage of sleep. Latency to consolidated sleep (Lat.CS) is the elapsed time from "lights out" to the first epoch of 5 min of sleep (stages 1, 2, 3, 4 or REM) not interrupted by wake or movement time. Sleep period time (SPT) is the time in bed less the period of wakefulness prior to onset of EEG sleep. Sleep efficiency is the time spent asleep as a function of total time spent in bed (TIB), expressed as a percentage. Latency to SWS, stage 2, and REM sleep were calculated from sleep onset to the first min of these stages. Each sleep stage was analyzed by measuring its total duration, number of episodes, mean episode durations and proportion of total sleep time. For the control nights, the first sleep cycle was defined by the first min of stage 1 or 2 until the end of the first episode of REM sleep. The following sleep cycles extended from the end of one REM sleep episode to the end of the subsequent one. Two REM sleep episodes differed when they are separated by more than 15 min. SWS and REM sleep stability indices represent the percentage of actual SWS and REM sleep within SWS and REM sleep episodes.

Blood samples were collected during both protocols with an IV catheter inserted into the subject's non-dominant forearm. For the first protocol, IV catheters were inserted around 1000h in the morning following the control night. For the

second protocol, IV catheters were inserted around 2000h prior to the control night. Results on the immune and endocrine parameters on the samples will be presented elsewhere.

2.0 NAP PROTOCOL

During the week preceding the experiments, the subjects were instructed that after their 8 hr overnight sleep period they would be given an afternoon nap in their bedroom. In addition they were informed that their sleep would be interrupted several times during the second night. Information pertaining to timing and length of naps was not given nor was the exact time of the end of the experiment. They were told that they had to perform 15 min test sessions as soon as they were awakened and on a continual basis during the study. Also during that week, subjects received verbal instructions and training on how to complete the various cognitive tasks which they practiced again several times a few hours prior to the start of the study. This training was given so that the subjects would be performing these tasks near the asymptotic level of their learning curve before the start of the study. The asymptotic level was achieved for all subjects in all task. Performance level during the synthetic work task was judged on the composite score (a measure of overall performance).

The experiment was conducted in a self-contained sleep laboratory at the Western Division of the Toronto Hospital. Each pair of subjects resided in the laboratory for 36 hr on two separate occasions. All test sessions and sleep periods were given simultaneously to both subjects. The subjects worked and slept independently in 3 x 4 m experimental rooms with no windows. Each room was equipped with a desk and a personal computer placed on a cart beside their bed. Bedrooms were also equipped with an intercom system which allowed two-way communication with the experimenter in the adjacent control

room. Close-circuit video cameras were used to visually monitor the subjects to alert observers against any sleeping behaviour at inappropriate times. If the subjects fell asleep at inappropriate times, an experimenter would call his name on the intercom; this procedure was sufficient to wake up the subjects. The environment was temperature controlled and there were no clocks present; window blinds from the corridor were shut; radio, television, and phone calls were not permitted; and staff refrained from discussing time. This was done to prevent any anticipation of the schedule by the subjects during the protocols. There was no expectation that the subjects would lose track of time.

Subjects arrived at the laboratory at approximately 2000h on day 1. If necessary, they changed into comfortable clothing. They were equipped with EEG, ECG, EOG, EMG, intravenous line (IV) and rectal temperature recording equipment. Subjects then practiced the test session until 2300h and went to bed for 8 hr. Just prior to sleep the subjects completed their first study test session. This full night of sleep was used to screen out candidates who did not fulfill all inclusion and exclusion criteria and provided an opportunity for adaptation to the sleep laboratory. Immediately upon awakening at 0700h, the subjects began a test session, which was repeated every hour until the scheduled afternoon nap. Since the main objective of this experiment was to evaluate time of night effects of sleep inertia upon awakening from nocturnal naps, the introduction of afternoon naps was made in an attempt to better control for prior wake time preceding the night naps, prior wakefulness being another important factor affecting nap architecture and sleep inertia. The placement of these naps was based on the fact that spontaneous naps generally occur in the afternoon (Campbell and Zulley, 1985, 1987) and that under a normal sleep/wake schedule, afternoon naps have been found to comprise higher amounts of SWS (Webb and Agnew, 1971; Evans *et al.*, 1977)

compared to evening and morning naps. The 90 min nap length was chosen because naps of this duration would be very likely to contain both NREM and REM sleep (Dinges, 1989a; Stampi, 1992). Consequently, 90 min afternoon naps were scheduled to terminate 9.5 hr before the first night nap of protocols A and B. Test sessions were held prior to and after the afternoon nap scheduled at 1300h in protocol A and at 1600h in protocol B. Hourly test sessions then continued until the night time naps. Subjects were then allowed to sleep in four 1-hr naps distributed either between 0000h and 0430h (protocol A) or between 0300h and 0730h (protocol B). Test session always occurred immediately prior to and after each nap in both protocols (figure 2).

Protocol A: In this protocol, the first 1-hr nap occurred at 0000h. Immediately upon being awakened after each nap, subjects performed a 15 min cognitive test session, followed by another 15 min test session. The subsequent nap started as soon as the second test session was finished (i.e., 30 min after the end of the first nap). This pattern was repeated until they had completed their four 1-hr naps scheduled at 0000h, 0130h, 0300h, and 0430h (figure 2). During the rest of the night until the end of the study at 0900h, subjects were kept awake and completed test sessions scheduled at the same time as in protocol B.

Protocol B: In this protocol, subjects were tested at the same time as in protocol A until their first scheduled nap started at 0300h and terminated an hour later. As soon as they woke up, they were subjected to two 15 min test sessions followed by a second nap of 60 min. This sequence was repeated until the four 1-hr naps scheduled at 0300h, 0430h, 0600h, and 0730h were completed (figure 2).

In both protocols subjects stayed in the laboratory until 0900h of day 3.

Breakfasts were given at 0900h, lunches at 1200h, dinners at 1800h, and snacks were offered at 2230h. Substances containing caffeine were not available. The starting order of the protocols was counterbalanced for each pair of subjects and their second protocol occurred 3 to 5 weeks after the first. In between protocols, the subjects were asked to complete a 2-week sleep diary (Moldofsky & MacFarlane, 1990). These diaries provided self-report information on each subject's sleep/wake habits over each 24 hr period. Awakenings of the subjects from each nap were accomplished by an experimenter walking into the room and calling the subject's name. On average, it took 45 seconds for the subjects to sit up on the edge of their bed and start performing the tasks. The subjects completed 31 test sessions of cognitive tasks all scheduled at the same time during both protocols (A or B). Between test sessions, subjects would either read or write in their bedrooms. Subjects were constantly visually monitored to ensure that they remained awake between sleep periods.

Test Session

Psychological tests performed by the subjects were generated by a VAX-II work station on a personal computer (PC); subjects responded by typing answers on individual keyboards. The computers were equipped with a hard disk drive, 12" color screen, ATI Stereo F/X Sound Blaster card and a Microsoft mouse. Each test session included two self-paced performance tasks, one multi-task, three self-report scales, and a relaxation period presented in a standardized sequence.

Several tasks of short durations were chosen because this allowed the examination of time of night effects of sleep inertia on different types of task. The logical reasoning task involves more cognitive processes such as short-term memory and immediate processing whereas the serial reaction time task involves more perceptual and motor processes. The synthetic work task

(synwork), on the other hand, requires one to simultaneously attend to more than one task at a time. Self-report scales of fatigue, sleepiness and mood were chosen because of their frequent use under laboratory and field conditions to evaluate whether one can properly fulfill a task.

Because of their known sensitivity to sleep inertia, the logical reasoning and serial reaction time tasks were performed by the subjects within the first 5 min of awakening. These tasks were immediately followed by the self-report scales in order to examine their sensitivity to sleep inertia effects and compare them with previous cognitive performance measures. The new synwork task was placed last in the test session after the relaxation period because little was known about its sensitivity to different stressors such as sleep inertia and sleep deprivation.

Performance tasks (Appendix C)

Logical reasoning (LR): This task was adapted from Baddeley's (1968) reasoning test based on grammatical transformations. In this experiment, the task consisted of individual presentations of 16 sentences each of which indicate the order of A and B with respect to each other (e.g., "A is preceded by B"). This would be followed by the pair of letters (either "AB" or " BA") resulting in 32 possible combinations. The subjects were required to indicate whether the statement correctly described the order of the letters by typing T (true) or F (false). The 32 combinations were presented in random order without replacement until all 32 combinations had been completed. The duration of the trial was 2 min. Response times and number of correct and incorrect responses were recorded by the computer for all sessions.

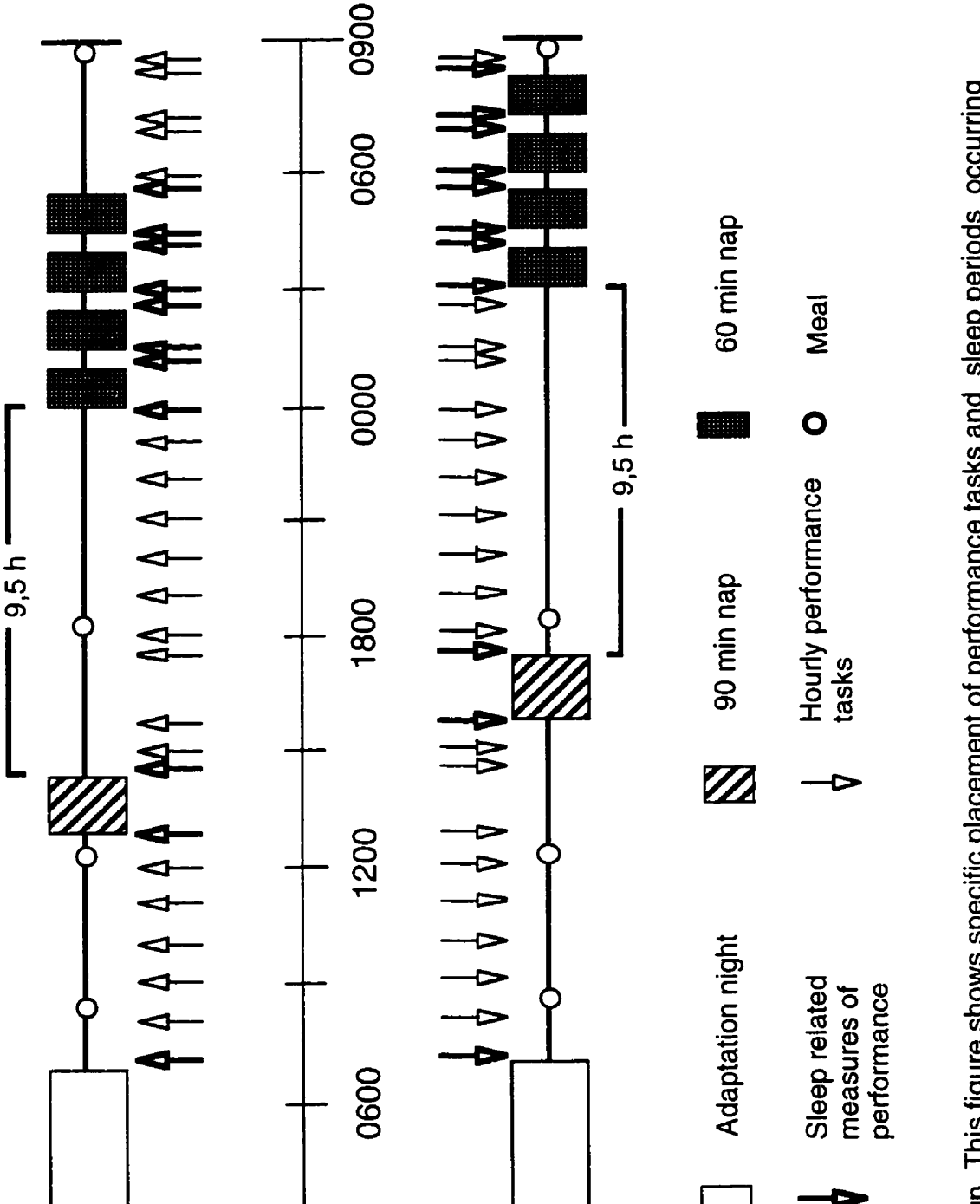


Fig. 2: Experimental design. This figure shows specific placement of performance tasks and sleep periods occurring during protocols A and B.

Four-Choice Serial Reaction Time (SR): This task was based on a task described by Wilkinson and Houghton (1975). Their task required subjects to cancel an illuminated light by pressing a spatially corresponding button as quickly as possible. Another light would be illuminated as soon as the prior one was extinguished until the end of the trial. In a militarized version of the task (Angus and Heslegrave, 1985), there was no spatial correspondence between the four stimuli and the response buttons. One stimulus was randomly selected from the total set and displayed on the terminal screen. The subject's task was to press the appropriate corresponding key, which removed the stimulus from the screen, immediately followed by replacement with another stimulus. The response keys were organized in a 2 x 2 matrix pattern. The duration of the trial was 2 min. Response time and number of correct responses were recorded by the computer for all sessions.

Self-report scales (Appendix C)

Fatigue : The scale used was the US. Air Force School of Aerospace Medicine Subjective Fatigue Checklist (Harris, Pegram, & Hartman, 1971). In this scale, subjects were presented with 10 sequential statements, such as "very lively" or "petered out", and rated themselves as "better than", "same as", or "worse than" each of these statements. Scoring was on a scale from 0 to 20, with lower scores indicating greater subjective fatigue.

Sleepiness : Sleepiness was measured with the Stanford Sleepiness Scale (Hoddes, Zarcone, Smythe, Phillips and Dement, 1973). Subjects chose a rating from 1 to 7, based on their immediate subjective level of sleepiness. The scale ranged from "wide awake" (1) to "almost in reverie" (7).

Mood : Mood was assessed by the U. S. Naval Health Research Center's (NHRC) Mood Scale (Johnson & Naitoh, 1974). Subjects were sequentially

presented with mood-related words (e.g., active, tense, happy). For each word they rated themselves on a 4-point scale, from "not at all" to "extremely", relative to each word. Nineteen positive and ten negative mood words were presented so that a separate positive score (57-point range) and negative score (30-point range) were calculated. A larger score indicated a more extreme positive mood or a more negative mood.

Three min eyes closed relaxation period : This was a shorter version of the original 4 min eyes closed relaxation period designed by Pigeau et al (1988). On their video display terminal, subjects were instructed to relax in a seated position with their eyes closed. At the end of the 3 min the terminal bell rang and subjects were asked to choose from a list of sentences and type in the sentence number best describing how they felt while their eyes were closed.

Multi-task performance (Appendix C)

Synthetic Work Environment (Synwork) : The multi-task performance developed by Elsmore (1991) of the Office of Military Performance Assessment Technology (OMPAT) was composed of four tasks which appeared simultaneously on the PC screen divided into four windows. A composite score for all tasks was displayed in a small window in the middle of the screen. The subjects were instructed to perform the tasks repeatedly for the duration of the session (4 min) in order to maximize the composite score. A mouse was used as an input device for each task. The task comprised the Sternberg Memory task, an Arithmetic task, a Visual Monitoring task, and an Auditory Monitoring task.

Specific task characteristics:

A. *Sternberg Memory task*: Subjects were presented with a list of upper case letters ("positive list") chosen from the alphabet at the beginning of each

session. An equal sized list of letter was also selected and called the negative list. The positive list was displayed for only 5 sec in a box at the top of the left window, and was then replaced by the words "RETRIEVE LIST". The positive list could be examined temporally at any time by clicking the mouse on the list box. At various times, a sample letter from one of the lists appeared in the decision box in the center of the window. The subject's task consisted of deciding if the letter was a member of the positive list or not. Then, they would click the mouse on the appropriate box (YES or NO) at the bottom of the window. Ten points were awarded for each correct response, and deducted for each error or retrieval of the positive list.

B. *Arithmetic task*: An addition task presented two randomly selected numbers lower than 1000, with an initial answer represented by zeros in a response box. The correct answer was built by using the mouse to click on "+" or "-" boxes below each digit column. Clicking with the mouse on a box labeled DONE at the bottom of the upper right window signified completion and resulted in the presentation of a new problem. Ten points were awarded for each correct answer and subtracted for each error.

C. *Visual Monitoring task*: In the lower left window a pointer moved from the center on a horizontal scale towards either end at a rate of 200 msec per step. At this speed, 20 sec were required to reach the end of the scale. The subject's task was to prevent the pointer from reaching the end of the scale by clicking the mouse on a RESET box at the top of the window to reset the pointer to the center. Points were awarded depending on the distance from the center (i.e., the closer to the extremities the greater the number of points up to a maximum of 10). Ten points were deducted for each second the pointer remained at either end of the scale.

D. *Auditory Monitoring task*: Two tones at different frequencies (low, 5000 Hz; high, 7000 Hz) were randomly presented to the subjects every 5 sec. If the tone was identified as "high frequency", the subject was required to click on the HIGH TONE REPORT box. No action was needed following a low sound. The probability of a high tone was set at 20%. Ten points were given for each correct response following a high tone, prior to the next scheduled tone and 10 points were deducted for each error.

3.0 STATISTICAL ANALYSES

Analysis of variance (ANOVA) for repeated measures using the general linear model (GLM) procedure in SAS was used in evaluating within-subjects 1- or 2-factor designs (all repeatedly measured). Two 1-factor ANOVA (for the overnight and afternoon sleep periods), and a 4 by 2 (nighttime nap by protocol) analyses of variance were performed to compare sleep composition between protocols and sleep period of similar duration. A 2 by 31 (protocol by test session) analysis of variance was employed to examine overall differences between protocols across the study on measures of performance, fatigue, sleepiness, mood and drowsiness. In order to investigate sleep inertia effects upon awakening from the naps in each protocol, a 2 by 5 (pre-post by nap) ANOVA was used for each measure. The Greenhouse-Geisser correction (Geisser and Greenhouse, 1958) was adopted to correct for statistical violations of the assumptions of sphericity. However, this procedure did not allow for missing data and any missing data had to be substituted by averaging the adjacent data points for the missing values. When overall F-ratios were significant, the *post-hoc* Tukey test for multiple comparisons was used at the 0.05 level. Only comparisons regarding the hypothesized sleep inertia effects

were looked at. All associations between sleep stage durations and related performance were evaluated using Pearson Product Moment correlation coefficients adjusted for the number of correlations conducted with Bonferroni test (Keppel, 1982). Statistical significance will be indicated on figures and tables with asterisks and arrows.

CHAPTER IV

RESULTS

Section 1.0 of this chapter presents detailed analyses of sleep composition of the overnight, afternoon and night naps including specific comparisons between protocol A and B. Core body temperature measurements are also reported in Section 1.0. Section 2.0 looks at cognitive performance and subjective state changes occurring in each protocol, between protocols, and more specifically at the effects of sleep inertia upon awakening from the naps. Finally, Section 3.0 presents the cognitive performance measures upon awakening from the naps in relation to prior sleep composition.

Overall, subjects appeared to adapt well to the unusual napping schedules of the protocol. Interestingly, even though the subjects had slept the previous night, they were all able to fall asleep and maintain sleep during the 90 min afternoon naps. This was particularly important since these naps were introduced to control for the wake time (9.5 hr) between the termination of the afternoon naps and the first night naps of each protocol. Without a successful afternoon nap to alleviate some of the "tiring" effects of the additional 3 hr spent awake since the previous night's sleep in protocol B, subjects would have started the series of night naps in protocol B feeling more sleep deprived than in protocol A. Although this strategy has not been used in previous studies, it is noteworthy that it proved to be successful in this study. Cognitive performance measures were similar prior to the first night nap (0000h) in protocol A and protocol B (0300h). Similar performance levels during the day allowed us to compare the circadian placement of the naps and their effects

on the resultant sleep inertia upon awakening. The variations in cognitive performance measures obtained are clustered around the naps in both protocols and that performance returns to similar levels by the end of both protocols.

1.0 SLEEP COMPOSITION AND CORE TEMPERATURE VARIATIONS

Sleep composition during the overnight sleep periods and the various naps in both protocols are shown in table 1 to 3. Hypnograms of afternoon and nocturnal naps of two subjects are shown in appendix D. To facilitate comparisons between sleep periods and protocols, wakefulness, SWS and REM sleep are also presented in figure 3 as a proportion of total sleep time (TST).

Control sleep

No significant differences in the mean sleep composition of the overnight sleep were found between protocol A and protocol B. Sleep efficiency was greater than 90% with approximately 17% of sleep time spent in rapid eye movement (REM) sleep and 15% in slow wave sleep (SWS). The latencies to sleep onset and to consolidated sleep were in the 10 to 20 min range. Since each subject slept during the first control night (either in protocol A or B) without an IV catheter and the second control night (the remaining protocol) with an IV catheter, the effects of the IV catheter on sleep composition were evaluated (see table in appendix E). The analysis showed that the 7 subjects who slept with an IV catheter in protocol A had a shorter sleep latency (IV: 5 min, noIV: 18 min) at the 0.02 level [$F(6,5)=12.08$], and spent less time (IV: 70.5 min, noIV: 78.2 min) in REM sleep [$F(6,5)=12.38$, $p < 0.009$] than when they slept without the IV catheter in protocol B. No differences were found in the 6 subjects who slept

with an IV catheter in protocol B compared to their control night without the IV catheter in protocol A.

Afternoon nap

The afternoon nap taken at 1600 h in protocol B had significantly greater amounts of stage 4 and total SWS in minutes compared to the afternoon nap at 1300h in protocol A [$F(1,12)=12.8$, $p < 0.005$]. On average, subjects accumulated 7.4 min more stage 4 when napping at 1600h after 9 hr of prior wakefulness than they did in the 1300h nap after 7 hr of prior wakefulness. Significant differences were also obtained when SWS [$F(1,12)=14.45$, $p < 0.003$] and stage 4 [$F(1,12)=14.28$, $p < 0.003$] were expressed as a percentage of TST. In addition, the mean duration of SWS episodes were greater in the 1600h nap than in the 1300h nap [$F(1,12)= 10.32$, $p < 0.008$]. No other sleep stage comparisons between protocols reached significance (table 1).

Night naps

Comparisons between protocols by order of presentation (nap 1 to 4): To examine the extent to which the infrastructure of the night naps was linked to their circadian timing, the sleep composition of the naps was compared between the first night nap (0000h) in protocol A (nap 1A) and the first night nap (0300h) in protocol B (nap 1B), nap 2A with nap 2B and so on. No significant differences were found between the two protocols for any of the sleep variables (table 2 and 3). When the sleep measures of nap 1 to 4 were combined between the two protocols, the nap periods showed a significant difference in the amount of REM in minutes [$F(3,36)=3.58$, $p < 0.03$] between the first (1.7 min) and last nap (15.5 min). The number of REM sleep episodes were significantly smaller [$F(3,36)=6.96$, $p < 0.002$] in the first nap (0.20) than in the second (0.43), third (0.54) and last nap (0.59).

Table 1: Sleep measures for nocturnal control nights and afternoon naps for protocol A and B (mean ± s.d.)

Sleep measures	Baseline sleep - A		Baseline sleep - B		Afternoon nap - A		Afternoon nap - B	
Time in bed	453.04	(20.1)	452.1	(20.6)	84.09	(3.2)	85	(3.9)
Stage changes	70.2	(18.4)	70.1	(20.3)	14.6	(3.3)	16.2	(6.5)
Sleep period time (min)	441.9	(22.7)	444.8	(25.2)	77.1	(3.9)	81.1	(3.2)
Total sleep time (min)	429.6	(20.5)	433.5	(24.3)	71.8	(5.5)	81.3	(3.6)
Lat. to sleep onset (min)	12.3	(15.1)	11.3	(10.1)	7	(10.3)	3.9	(2.5)
Lat. consolidated sl. (min)	19.5	(18.5)	18.5	(9.9)	8.8	(5.9)	7.5	(5.0)
Sleep efficiency (%)	93.3	(4.2)	91.2	(6.4)	87.92	(15.4)	86.93	(12.4)
Wakefulness (min)	21.3	(9.7)	30.2	(23.8)	5.3	(8.9)	7.2	(10.2)
Stage 1 (min)	12.9	(10.2)	15.6	(8.7)	7.3	(6.3)	7.4	(5.1)
Stage 2 (min)	261.6	(25.9)	254	(43.5)	41.7	(17.5)	32.9	(10.3)
Stage 3 (min)	35.3	(17.9)	27	(20.9)	5.5	(7.8)	5.2	(3.4)
Stage 4 (min)	33.5	(25.1)	42.9	(29.8)	6.4	(8.4)	19.3 *	(15.2)
SWS (stage 3+4, min)	68.8	(29.8)	69.9	(23.3)	11.9	(11.6)	24.4 *	(16.9)
REM sleep (min)	74.6	(28.8)	74.8	(27.0)	13.6	(11.4)	9.3	(10.1)
Wakefulness/SPT (%)	4.86	(2.3)	6.82	(5.4)	6.01	(9.4)	6.92	(12.0)
Stage 2/TST (%)	62.3	(7.1)	61.2	(9.9)	55.45	(19.6)	46.46	(19.3)
SWS/TST (%)	16.23	(6.5)	16.87	(5.8)	14.6	(14.2)	31.32 *	(20.6)
REM sleep/TST (%)	16.54	(8.3)	17.47	(6.8)	17.66	(14.7)	12.1	(13.1)
Number of SWS episodes	4.15	(1.7)	3.84	(1.6)	1.08	(0.9)	1.07	(0.5)
Duration of SWS episodes	19	(7.5)	21.9	(8.7)	11.4	(10.0)	13.49 *	(11.5)
SWS latency (min)	34.06	(18.4)	27.8	(8.1)	24.29	(18.3)	18.5	(10.1)
SWS stability index	80.4	(17.3)	85.7	(14.6)	80.3	(19.9)	92.17	(15.2)
Number of REM sleep episodes	3.54	(1.1)	3.76	(1.2)	0.83	(0.6)	0.69	(0.5)
Duration of REM sleep episodes	23.5	(11.9)	28.8	(23.8)	13.48	(11.7)	10.23	(11.4)
REM latency (min)	128.3	(74.1)	160.8	(62.1)	36.69	(26.4)	41.38	(33.1)
REM sleep stability index	87.2	(15.7)	89	(22.2)	97.81	(4.0)	94.53	(13.4)

* Significant difference between protocols in the afternoon naps ($p < 0.05$)

Table 2: Sleep measures for nocturnal naps for protocol A (mean ± s.d.)

Sleep measures	Nap 1 (0000h)		Nap 2 (0130h)		Nap 3 (0300h)		Nap 4 (0430h)	
Time in bed	54.46	(5.5)	58.15	(2.8)	58.15	(1.5)	57.6	(1.9)
Stage changes	10.46	(5.0)	7.77	(2.9)	9.23	(2.6)	10.4	(6.2)
Sleep period time (min)	41.9	(8.0)	46.35	(12.4)	47.7	(6.9)	46.5	(8.5)
Total sleep time (min)	37.3	(13.7)	43.55	(13.3)	44.45	(8.9)	45.3	(8.5)
Lat. to sleep onset (min)	12.5	(8.6)	11.8	(9.6)	8.9	(7.9)	11.1	(7.7)
Lat. consolidated sl. (min)	23.2	(17.5)	20.6	(15.8)	13.6	(9.7)	16.3	(10.5)
Sleep efficiency (%)	69.5	(25.2)	70.8	(23.1)	80.8	(16.5)	87.4	(14.6)
Wakefulness (min)	4.4	(8.1)	2.8	(2.8)	1.9	(3.3)	1.2	(1.2)
Stage 1 (min)	6.3	(4.9)	5	(4.4)	4.3	(4.4)	7.4	(6.5)
Stage 2 (min)	15.9	(8.7)	19.1	(5.3)	22.9	(8.6)	22.3	(12.3)
Stage 3 (min)	5.9	(5.8)	2.5	(3.3)	5.9	(6.1)	4	(4.0)
Stage 4 (min)	9.3	(7.3)	4.7	(7.7)	7	(7.2)	2.3	(7.2)
SWS (stage 3+4, min)	15.2	(9.4)	7.1	(10.3)	12.8	(10.2)	6.3	(8.5)
REM sleep (min)	0	(0.0)	8.1	(11.0)	6.5	(13.1)	13.3	(16.0)
Wakefulness/SPT (%)	2.36	(3.1)	4.08	(4.5)	3.35	(7.1)	2.78	(3.0)
Stage 2/TST (%)	38.54	(18.0)	54.69	(23.1)	50.83	(19.5)	50.5	(25.5)
SWS/TST (%)	37.75	(20.5)	15.59	(21.7)	26.84	(19.7)	12.74	(17.1)
REM sleep/TST (%)	0	(0.0)	18.13	(24.2)	12.33	(23.4)	24.21	(31.3)
Number of SWS episodes	1	(0.4)	0.54	(0.5)	0.77	(0.4)	1.41	(2.4)
Duration of SWS episodes	13.49	(11.5)	7.12	(10.3)	14.18	(12.3)	7.13	(9.7)
SWS latency (min)	19.79	(11.9)	15.1	(15.9)	19.1	(12.7)	18.5	(14.1)
SWS stability index	93.32	(12.2)	100	(0.0)	93.86	(8.3)	84.93	(21.8)
Number of REM sleep episodes	0	(0.0)	0.385	(0.5)	0.462	(0.5)	0.5	(0.5)
Duration of REM sleep episodes	0	(0.0)	8.42	(11.6)	7.12	(13.6)	13.1	(16.9)
REM latency (min)	0	(0.0)	7.19	(11.6)	15.12	(21.3)	16.2	(14.7)
REM sleep stability index	0	(0.0)	97.2	(5.2)	99.53	(0.7)	94.65	(7.2)

Table 3: Sleep measures for nocturnal naps for protocol B (mean ± s.d.)

Sleep measures	Nap 1 (0300h)		Nap 2 (0430h)		Nap 3 (0600h)		Nap 4 (0730h)	
Time in bed	56.84	(2.8)	56.77	(2.9)	58.07	(2.1)	58.15	(3.1)
Stage changes	12.5	(5.4)	10.2	(4.7)	10.9	(6.1)	10	(3.8)
Sleep period time (min)	50.04	(8.1)	51.97	(5.5)	55.37	(2.4)	55.87	(4.8)
Total sleep time (min)	49.8	(7.1)	51.8	(3.3)	55.7	(2.8)	55.8	(3.7)
Lat. to sleep onset (min)	6.8	(5.2)	4.8	(2.8)	2.7	(1.9)	2.3	(1.4)
Lat. consolidated sl. (min)	15.1	(12.3)	10.1	(9.1)	7.8	(3.6)	8.3	(5.7)
Sleep efficiency (%)	82.3	(13.6)	87	(11.1)	93.5	(3.6)	93.7	(4.1)
Wakefulness (min)	3	(3.9)	2.7	(5.2)	1.2	(1.4)	1.5	(1.0)
Stage 1 (min)	6.6	(5.8)	4.8	(5.1)	3.2	(3.2)	5.3	(4.8)
Stage 2 (min)	23.5	(10.8)	22.7	(11.8)	13.3	(13.3)	24.8	(16.2)
Stage 3 (min)	5.2	(2.8)	3.2	(3.1)	3	(3.0)	1.4	(2.2)
Stage 4 (min)	8.1	(7.8)	7.9	(11.5)	4.8	(7.9)	4.3	(8.2)
SWS (stage 3+4, min)	13.3	(7.7)	11.1	(12.9)	8.2	(9.3)	5.7	(10.1)
REM sleep (min)	3.4	(7.2)	10.5	(15.3)	16.8	(19.2)	18.5	(18.6)
Wakefulness/SPT (%)	4.34	(5.5)	3.78	(5.8)	1.8	(2.1)	2.04	(1.6)
Stage 2/TST (%)	50.31	(21.0)	43.82	(20.9)	45.34	(25.1)	48.97	(30.3)
SWS/TST (%)	26.85	(18.1)	26.1	(24.9)	15.59	(17.7)	9.87	(17.4)
REM sleep/TST (%)	11.18	(19.9)	19.13	(29.0)	30.15	(30.9)	32.01	(32.4)
Number of SWS episodes	0.923	(0.5)	0.692	(0.5)	0.692	(0.5)	0.385	(0.5)
Duration of SWS episodes	15.03	(11.3)	14.53	(13.0)	10.31	(10.8)	5.77	(10.1)
SWS latency (min)	20.5	(12.5)	16.19	(12.5)	20.7	(16.9)	10.57	(16.7)
SWS stability index	80.03	(22.9)	86.02	(21.7)	79.86	(23.3)	92.95	(14.7)
Number of REM sleep episodes	0.385	(0.7)	0.462	(0.5)	0.615	(0.5)	0.692	(0.5)
Duration of REM sleep episodes	6.54	(11.3)	11.08	(15.6)	17.84	(19.4)	19.5	(18.0)
REM latency (min)	7.08	(14.1)	13.58	(17.0)	13.65	(18.7)	14.08	(16.8)
REM sleep stability index	99.88	(0.2)	89.96	(13.2)	93.62	(14.6)	94.67	(7.2)

SWS in min [$F(3,36)=3.25$, $p < 0.03$] or as a percentage of TST [$F(3,36)=3.1$, $p < 0.05$] was significantly greater in the first nap (14.2 min, 32.2%) compared to the last nap (5.9 min, 11.2%). The mean duration of REM sleep episodes [$F(3,36)=6.95$, $p < 0.002$] in the last nap (14.9 min) were significantly greater than the first (3.3 min), second (9.7 min), and third nap (12.5). Latencies to sleep onset [$F(3,36)=5.48$, $p < 0.03$] and consolidated sleep [$F(3,36)=7.02$, $p < 0.001$] were shorter in the third (5.8, 10.7 min) and fourth (6.1, 11.5 min) naps compared to the first one (9.6, 19.1 min). In turn, significantly greater sleep efficiencies [$F(3,36)=7.14$, $p < 0.001$] were seen in the third (87.1%) and fourth (86.0%) naps compared to the first one (75.9%). In summary, subjects fell asleep quicker and slept more efficiently in both protocols towards the end of their respective nap series.

Comparisons between naps taken at the same clock time (0300: N3-A vs. N1-B & 0430h: N4-A vs. N2-B):: Under the specific protocols of this experiment, two night naps from each protocol were scheduled at the same clock time, nap 3A and 1B taken at 0300h and, nap 4A and 2B taken at 0430h. This comparison did not reveal any significant differences between the naps taken at 0300h in protocol A and B for any of the sleep variables. However, a significantly greater latency to sleep onset [$F(1,12)=4.76$, $p < 0.05$] was found in protocol A (10.1 min) compared to protocol B (4.8 min) in the nap taken at 0430h. No other comparisons reached significance.

Comparisons of accumulated sleep in each protocol

To estimate how successful the two protocols were at providing sleep, the cumulative amounts of sleep after the 4 night naps and after all naps including the afternoon naps were calculated and compared between the two protocols.

Cumulated sleep in nocturnal naps only : The number of stage changes was significantly greater [$F(1,12)=4.84$, $p < 0.05$] in protocol B (10.9) than protocol A (9.5). Protocol B had shorter latencies to sleep onset [$F(1,12)=11.31$, $p < 0.006$] and consolidated sleep [$F(1,12)=6.24$, $p < 0.03$] than protocol A. Sleep efficiency was affected in the same direction with an average of 89.1% in protocol B compared to 74.7% in protocol A [$F(1,12)=9.25$, $p < 0.01$]. The accumulated sleep spent in REM was significantly greater [$F(1,12)=7.57$, $p < 0.02$] in protocol B (49.2 min) compared to protocol A (25 min). No other sleep variables reached significance. Thus, subjects in protocol B fell asleep quicker, slept more efficiently, and had more REM sleep than in protocol A.

Cumulated sleep in afternoon and nocturnal naps: Sleep onset latency [$F(1,12)=9.88$, $p < 0.009$] and consolidated sleep latency [$F(1,12)=6.15$, $p < 0.03$] were significantly shorter in protocol B compared to protocol A. Sleep efficiency was greater in protocol B than protocol A [$F(1,12)=11.9$, $p < 0.005$] No significant difference between the two protocols could be seen in the accumulated amount of SWS and REM sleep or any other sleep stage. REM sleep showed a trend ($p < 0.08$) towards greater amounts in protocol B compared to protocol A. When the afternoon naps were included in the comparison of the subjects' sleep composition between the two protocols, the rapidity and efficacy of sleep were still better in protocol B but the difference in REM sleep was reduced below significance.

Sleep expressed in percentage of total sleep time (TST)

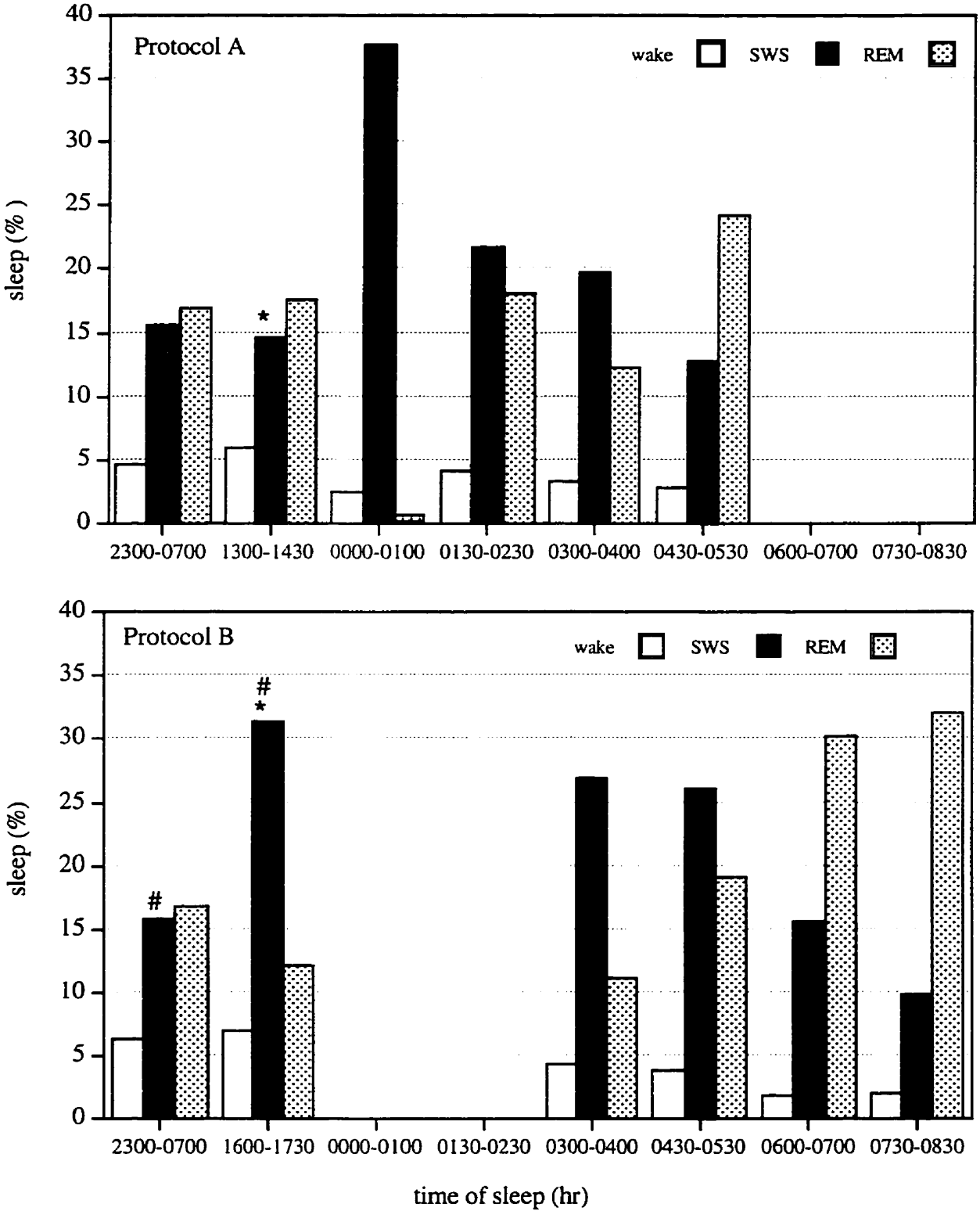
The amount of SWS, and REM sleep were expressed as a percentage of TST, and for wakefulness (within sleep) as a percentage of sleep period time and represented in figure 3 to facilitate comparisons between sleep periods and protocols. The proportions of sleep spent in SWS, wake, and REM sleep during

the control nights were similar between the two protocols. However, the percentage of TST spent in SWS was significantly greater in the 1600h nap in protocol B (31.3%) compared to the 1300h nap in protocol A (14.6%) as well as the overnight period in condition B (15.7%).

Overall, the percentage of SWS and REM sleep displayed a different pattern between the two protocols. Nocturnal naps in protocol A were characterized by alternations between a nap with more REM sleep (0130h and 0430h) and a nap with less REM sleep (0000h and 0300h) whereas naps in protocol B showed a consistent increase in the percentage of REM sleep from the first to the fourth night nap. Both protocols showed a consistent decrease in the percentage of SWS from the first to the last nap. No REM sleep was found in the first night nap in protocol A compared to the first nap in protocol B where 11.1% of TST was spent in REM sleep. A comparison of the naps taken at 0300h and 0430h in both protocols showed no significant differences in the percentage of REM sleep and SWS. Only a trend to greater percentage of SWS in protocol B and to greater percentage of REM sleep were observed in the 0430h naps.

In summary, subjects in protocol B spent more time in SWS in the afternoon nap than in protocol A. Different patterns of sleep composition were found between the two protocols. The changes in sleep composition in the sequence of naps of protocol A where more characteristic of the progression of changes in sleep composition observed during the first part of a normal night of sleep. The first hour of sleep was characterized by the predomination of SWS and the absence of REM sleep, SWS progressively decreased in the following naps and REM sleep alternated between naps with a higher and a lower proportion of REM sleep. Nocturnal naps in protocol B were characterized by a progressive decrease in SWS and a progressive increase in REM sleep.

Fig. 3: The amount of SWS and REM sleep are expressed as a percentage of total sleep time (TST) and wakefulness (within sleep) as a percentage of sleep period time, for each sleep period in protocol A and protocol B. Identical symbols indicate significant differences ($p<0.05$).



Core body temperature

Due to technical problems with the equipment used in the rectal temperature measurements, we were able to obtain complete data sets from only 5 subjects in both protocols (figure 4). A circadian rhythmicity was observed in both protocols with minimum temperatures between 0400 and 0600h during the first night, reaching maximum temperatures between 1900h and 2200h, and followed again by a drop in temperatures during the nap night between 0500h and 0630h. A significant interaction (protocol by time) was found in temperature measures [$F(406,1624)=2.11$, $p<0.0001$]. Post-hoc Tukey analysis showed significant differences in temperature between protocols around the afternoon naps and the 0730h nap. In order to understand these effects more clearly a closer examination of the data around the afternoon naps and nighttime naps was conducted. A significant reduction in core temperature [$F(28,112)=5.70$, $p<0.0001$] was seen during and after the 1600h nap in comparison to the temperature of subjects awake at the same time (protocol A). A similar lowering of temperature in the 1300h nap of protocol A was found compared to awake subjects in protocol B but did not reach significance ($p<0.07$). Moreover, a similar trend ($p<0.05$) was seen in the temperature of subjects taking a nap at 0730h (protocol B) compared to awake subjects (protocol A). Thus, at the same time of day, body temperature is lower during sleep than during wake.

Complex demodulation was performed on the raw core temperature data for each subject with a filter for circadian influences (i.e., 1 cycle/24 hrs). The filtered time series for each subject were averaged (point by point), resulting in a mean curve (with standard errors) for core temperature data in each protocol (figure 5). For the circadian effect (which includes the increasing linear trend) complex demodulation accounted for 79.5% (protocol A) and 75.5% (protocol B) of the variance contained in the original core temperature data.

Figure 4: Difference between protocol A and B in the mean z-score (± s.e.) of core body temperature over the 34 hr study. (N=5) Asterisk indicate significant differences between protocol A and B ($p<0.05$). Shaded and boxed area indicate the timing of sleep periods. Arrows indicate meal or snak time.

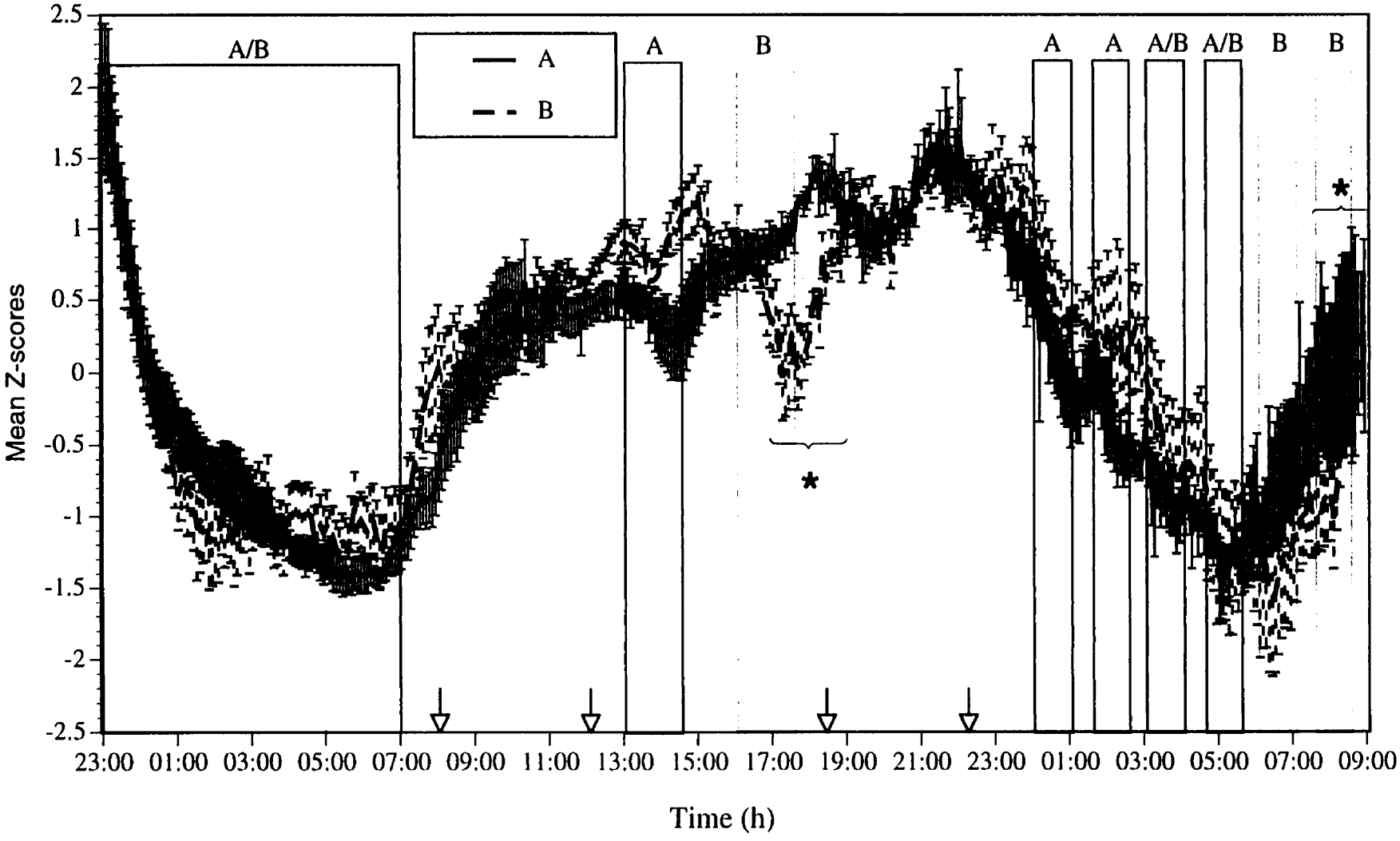
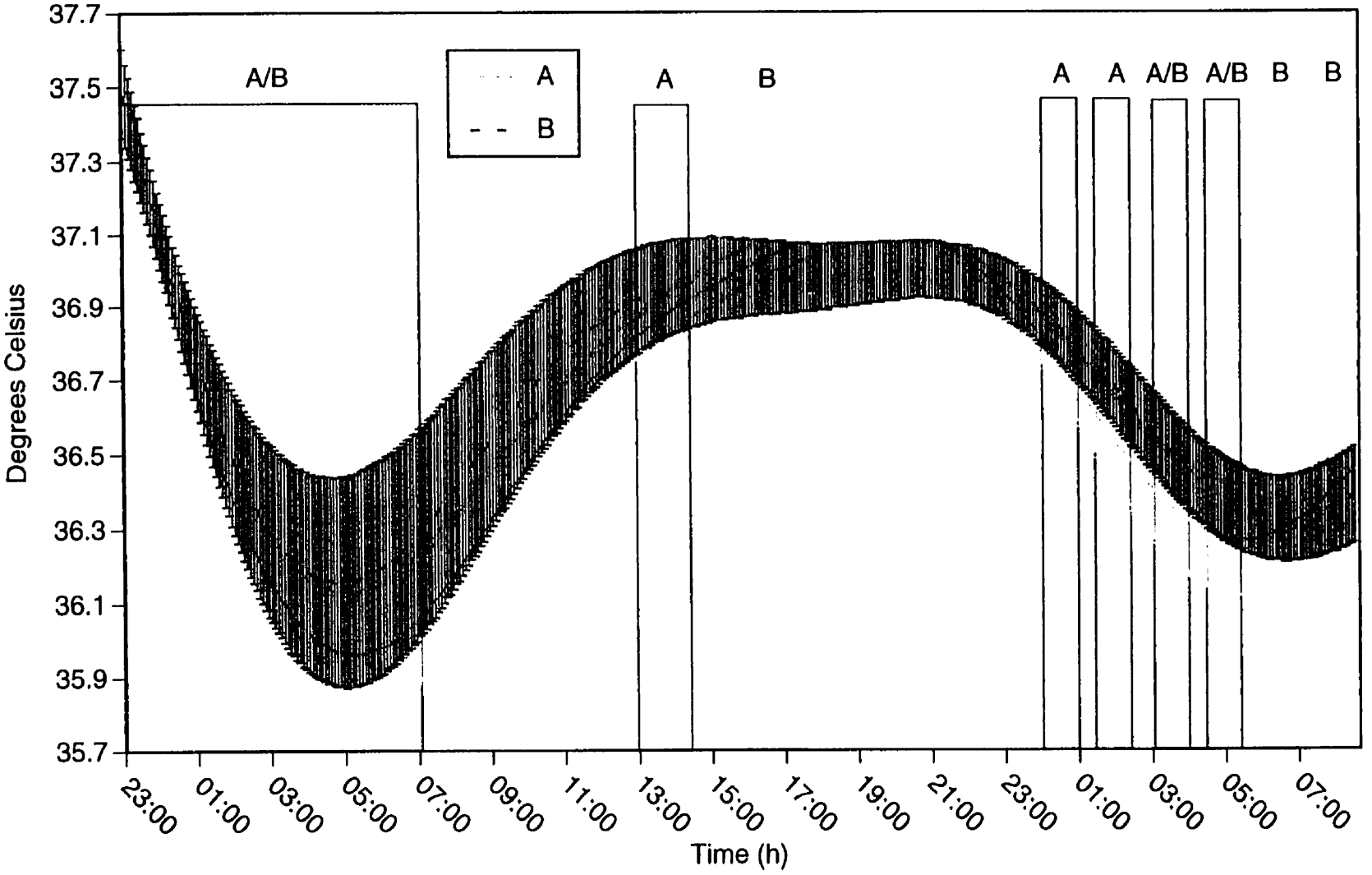


Figure 5: Means (± s.e.) of demodulated core body temperature for protocol A and B over the 34 hr study. Shaded and boxed area indicate the timing of sleep periods.



Complex demodulation confirmed the lowering effect of the late afternoon nap (protocol B) on the circadian rhythm in core temperature in comparison to protocol A.

2.0 Cognitive Performance tasks and subjective scales

Logical reasoning

Figure 6 presents the effects of napping on logical reasoning performance, measured over the last 21 hr of the study. Deterioration in the number of correct responses (top panel) and reaction time (lower panel) can be observed immediately upon awakening from night naps in both protocol A (open squares) and protocol B (filled circles).

A significant drop in the number of correct responses was found in protocol A upon awakening from the 0100 and 0230h naps compared to performance in protocol B where the subjects had not yet slept (top panel of figure 6). In protocol A, subjects performed significantly better at 0730h after completing all their naps compared to protocol B where they just had been awakened from their penultimate nap. Reaction times (RT) for correct responses (lower panel of figure 6) were longer upon awakening at 0230h compared to staying up in protocol B. On the other hand, waking up at 0830h in protocol B resulted in longer reaction time than in protocol A where subjects had been awake since 0530h.

Performance between the two protocols did not differ significantly at any other test session. Performance returned to the baseline by the end of both protocols. Although the number of incorrect responses and their corresponding RT's followed the same pattern as the correct responses (i.e., drop in the number of incorrect responses and increase in reaction time upon awakening from the

naps), no significant differences were found between the two protocols.

Sleep inertia : The effects of sleep inertia on performance were assessed by an analysis of the pre- and post-nap data only (figure 7). Control afternoon naps taken at 1300h or 1600h did not result in any significant sleep inertia effects in either performance measure. No significant differences between the two afternoon naps were found. However, significant sleep inertia effects in the number of correct responses were found, however, for all of the nocturnal naps in protocol A [$F(4,48)=4.16$, $p<0.006$] and in the first three naps (0300, 0430, 0600h) scheduled in protocol B [$F(4,48)=3.76$, $p<0.01$].

Reaction times were significantly elevated upon awakening from naps at 0000, 0130, and 0430h in protocol A [$F(4,48)=4.99$, $p<0.002$] but did not reach significance in protocol B (lower panel of figure 7). A significant recovery (represented by arrows in figure 7) in the correct number of responses and corresponding reaction times were observed between the post-nap test and the following pre-nap test taken 15 min later (pre-nap tests of subsequent naps) in protocol A. In protocol B, no significant improvements were seen after the 0300h, 0430h and 0600h naps in reaction time, and after the 0600h nap in the number of correct responses. Thus, the sleep inertia-induced decrements in performance in the naps scheduled in protocol A, and in the 0730h naps of protocol B, had recovered to pre-nap levels during the inter-nap period. In the remaining naps subjects did not regain their initial level of performance before napping again. Overall, sleep inertia effects were more pronounced in the earlier naps than in the last nap (0730h).

Figure 6: Changes in logical reasoning performance over the last 21 hr of the study. The top panel shows the mean number of correct responses and the lower panel shows the mean reaction time for correct responses over 2 min. Asterisks indicate a significant difference between protocol A and B ($p<0.02$). Shaded and boxed area indicate the timing of the naps.

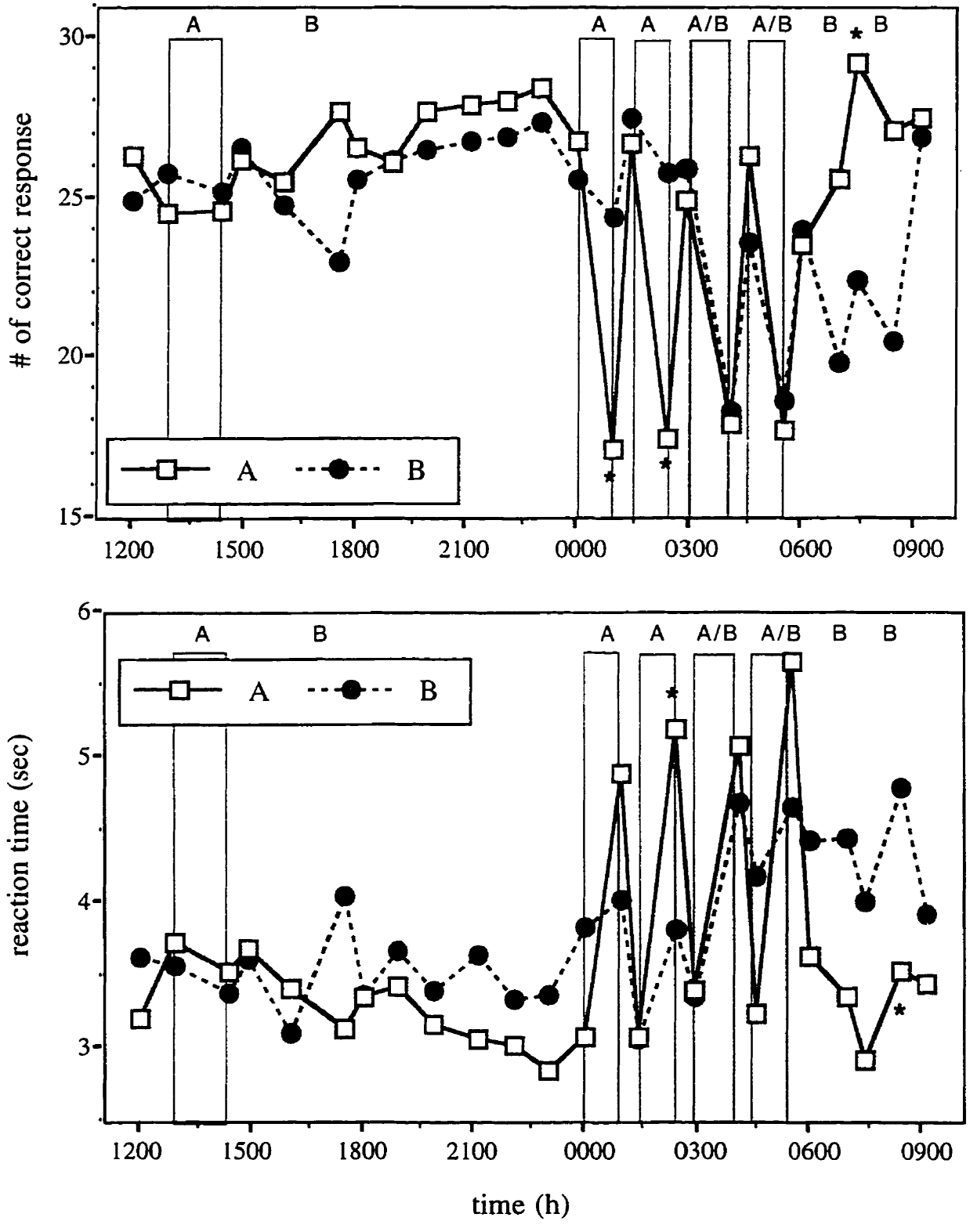
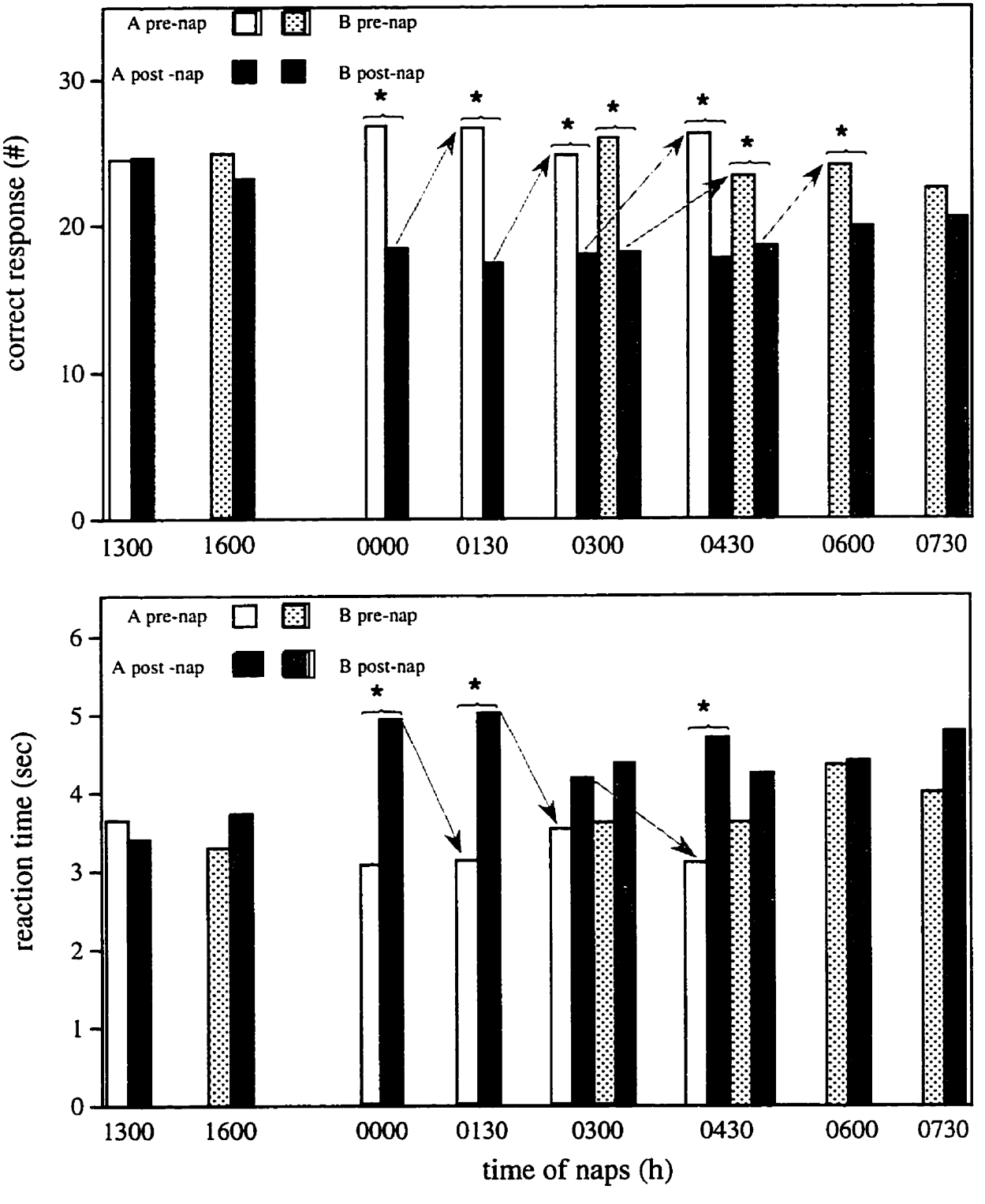


Figure 7: Post-nap sleep inertia effects and recoveries on logical reasoning performance for the afternoon and night naps in protocols A and B. Top panel shows the mean number of correct response and the lower panel shows the mean reaction time for correct response over 2 min prior to and immediately following each nap. Significant sleep inertia effects (pre- vs post-nap comparisons) are indicated with asterisks ($p<0.01$). Significant performance recoveries (post-nap vs next pre-nap comparisons) are indicated with arrows ($p<0.01$).



Serial reaction time

Figure 8 presents the effects of napping on the serial reaction time performance measured over the last 21 hr of the study. Deterioration in the number of correct responses (top panel) and reaction time (lower panel) can be observed immediately upon awakening from the naps in both protocols.

In figure 8 (top panel), a significant reduction in the number of correct responses [$F(30,346)=5.56$, $p<0.001$] can be seen upon awakening at 0100 and 0230h in protocol A compared to performance in protocol B where subjects were awake. A similar reduction in responses was observed in protocol B after completing their naps at 0700h compared to subjects just awakening in protocol A.

There was a coincidental lengthening (lower panel of figure 8) of RT's at the same times [$F(30,346)=4.59$, $p<0.001$]. All performance levels returned to daytime levels by the end of both protocols. The number of incorrect responses (top panel of figure 9) in this task showed a significant increase [$F(30,346)=3.39$, $p<0.001$] immediately after awakening at 0100 and 0230h in protocol A compared to B, and at 0700 and 0830h in protocol B compared to A. Longer reaction times (lower panel of figure 9) were observed at the same test sessions [$F(30,346)=2.29$, $p<0.001$]. Thus, the number of incorrect responses and the corresponding RT's followed the same pattern as the correct responses. Since the incorrect responses showed similar results as the correct responses, only the analyses for the correct responses will be further reported.

Figure 8: Changes in serial reaction time performance over the last 21 hr of the study. Top panel shows the mean number of correct responses and the lower panel shows the mean reaction time for correct responses over 2 min. Asterisks indicate a significant difference between protocol A and B ($p<0.001$). Shaded and boxed area indicate the timing of the naps.

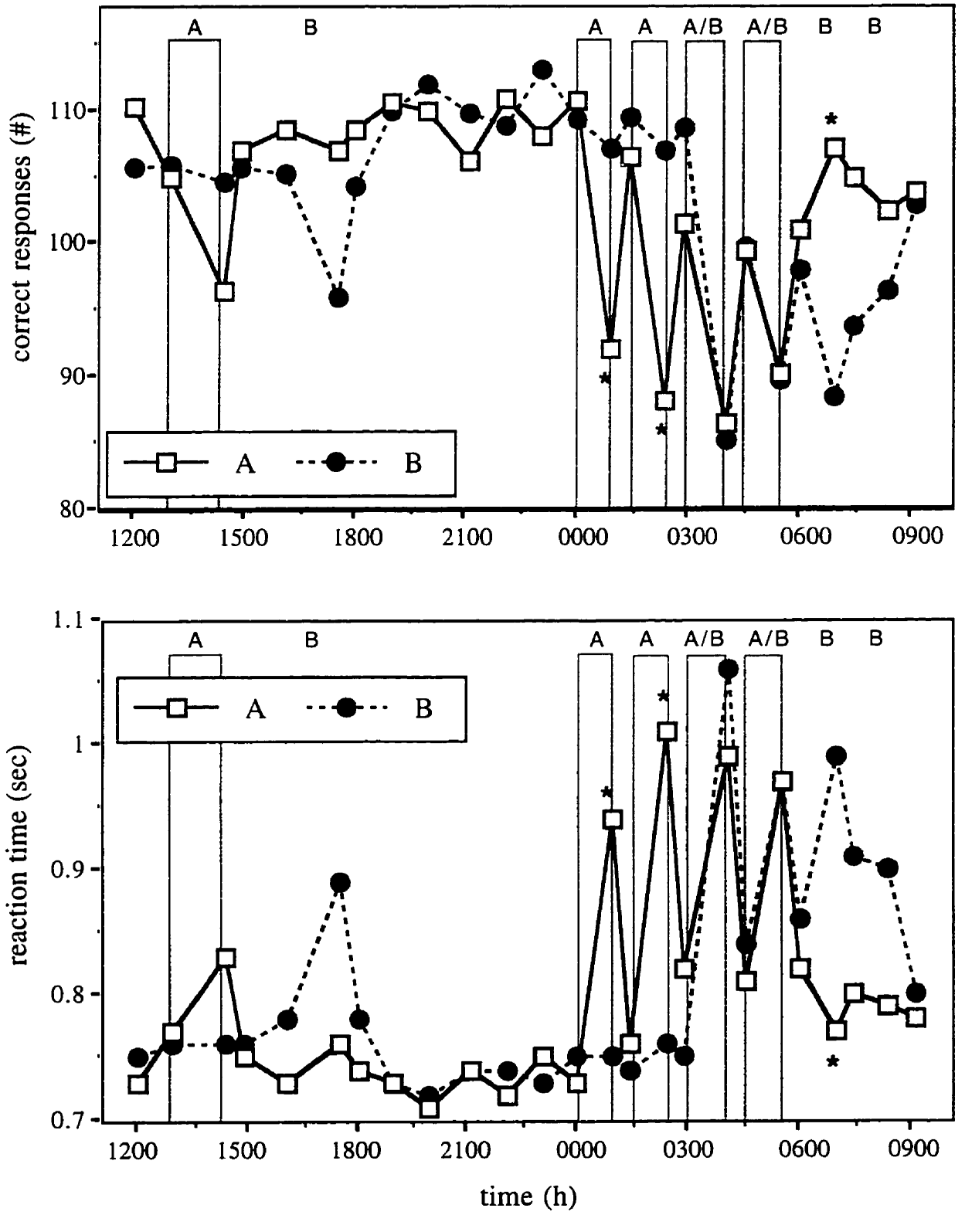
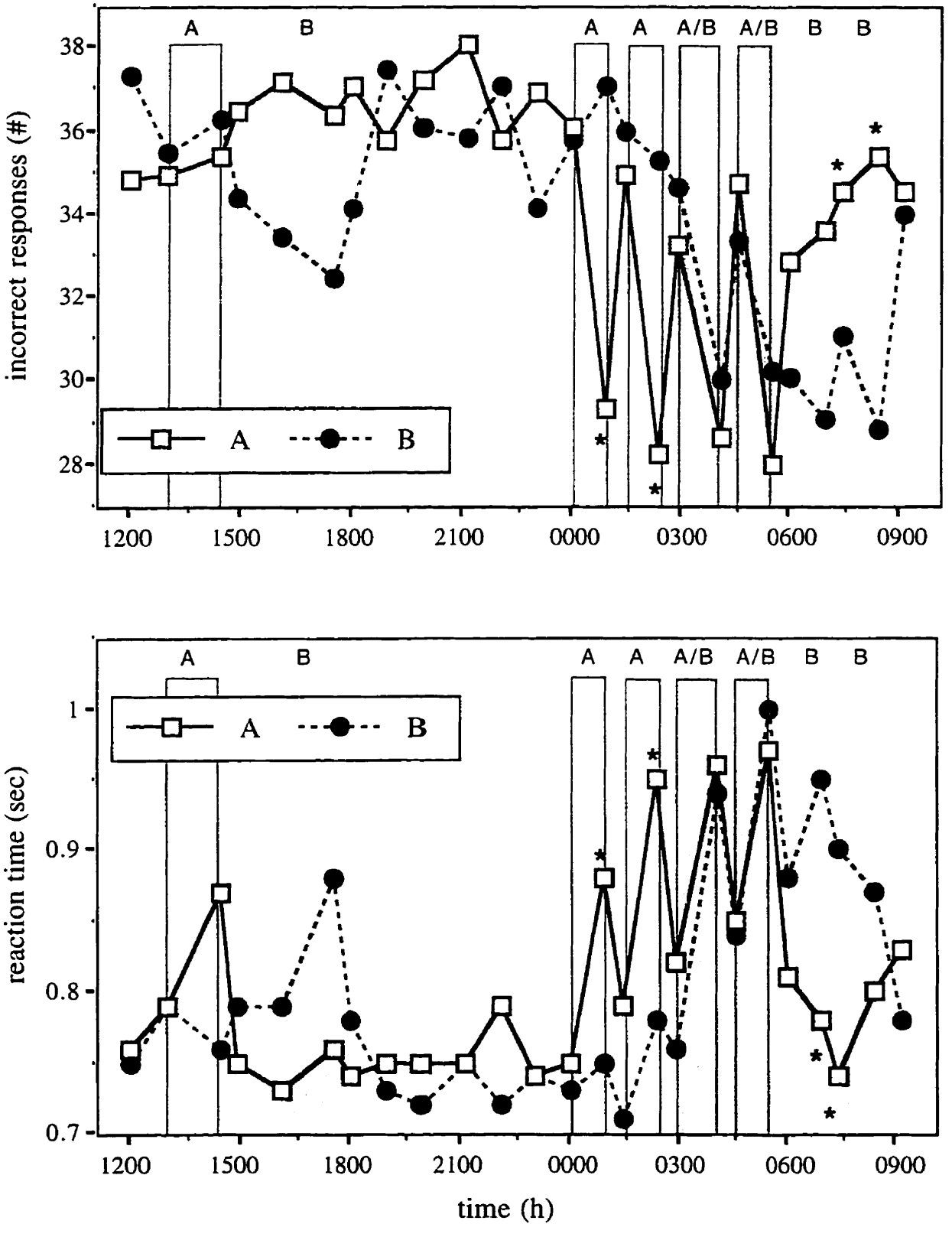


Fig. 9: Changes in serial reaction time performance over the last 21 hr of the study.Top panel shows the mean number of incorrect responses and the lower panel shows the mean reaction time for incorrect responses over 2 min. Asterisks indicate the significant difference between protocol A and B ($p<0.001$). Shaded and boxed area indicate the timing of the naps.



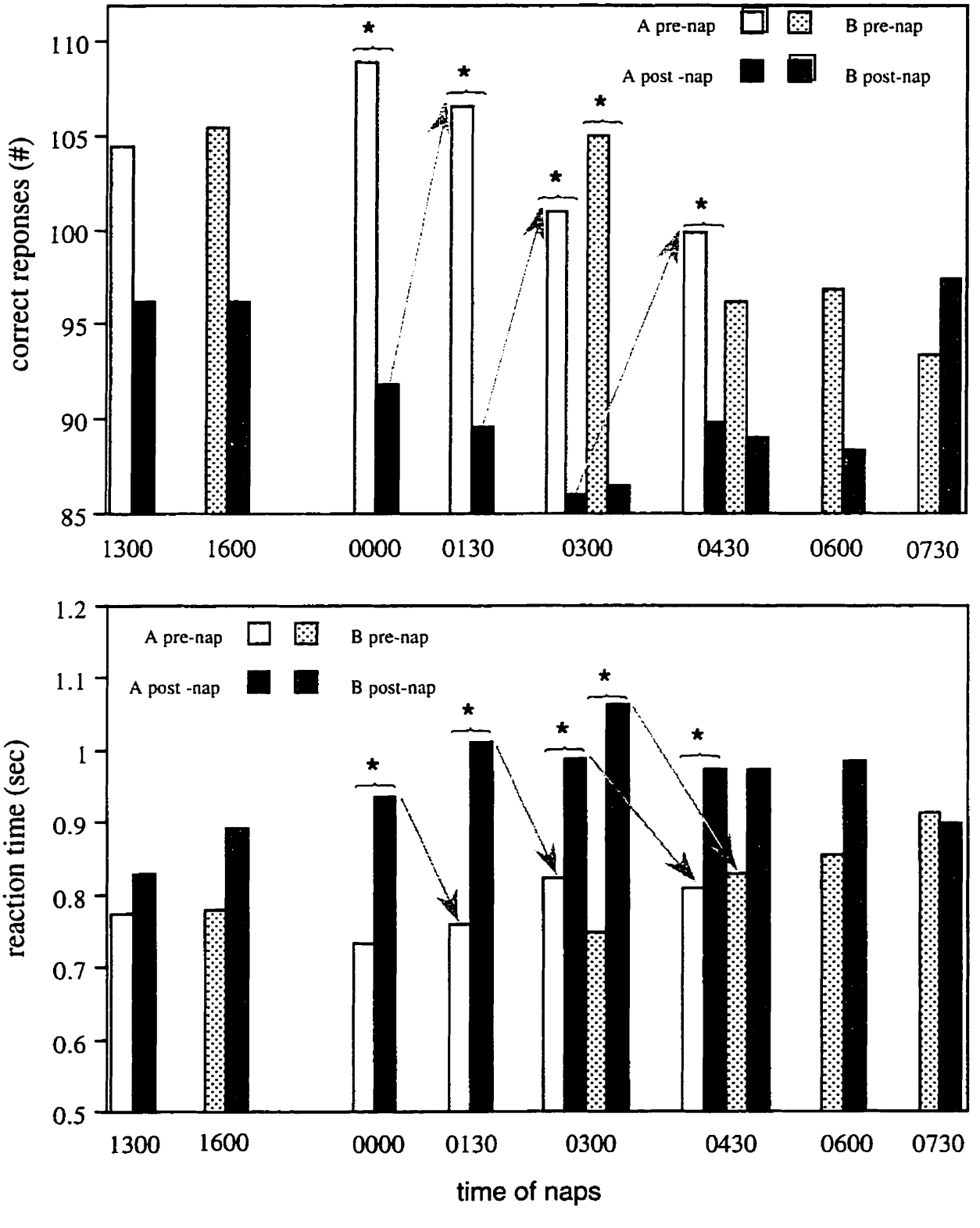
Sleep inertia : Pre- and post-nap analyses of serial reaction time performance are expressed in figure 10. Even though performance dropped upon awakening from the afternoon naps (1300 and 1600h) in comparison to pre-nap performance levels, no significant sleep inertia effects were found in either performance measure. There were also no significant differences in pre- and post-nap performances between the two naps. However, significant sleep inertia effects in the number of correct responses (top panel of figure 10) for all of the night naps were seen in protocol A [$F(4,48)=2.15$, $p<0.05$], but only the first nap of protocol B reached significance [$F(4,48)=6.76$, $p<0.001$].

Reaction times were significantly elevated upon awakening from all the night naps in protocol A [$F(4,48)=2.63$, $p<0.05$] and after the 0300h nap in protocol B [$F(4,48)=2.95$, $p<0.03$]. Again subjects' performances (number of correct responses and reaction times) in protocol A had recovered significantly after 15 min of being awake. On the other hand, the number of correct responses after

the 0300h, 0430h and 0600h naps and reaction time after the 0430h and 0600h naps did not recover significantly in protocol B. Overall sleep inertia effects were more pronounced in the earlier naps than in the later one (0730h) where performance was better upon awakening than prior to the nap.

In summary, both tasks showed consistent sleep inertia effects following awakenings from naps scheduled at 0000, and 0130, and not with naps during the afternoon or at 0730h. Comparable levels of sleep inertia effects were seen in naps taken at 0300 in both protocols. After 15 min of being awake, deteriorated performance measured immediately upon awakening from the naps always recovered in protocol A but not in protocol B.

Figure 10: Post-nap sleep inertia effects and recoveries on serial reaction time performance for the afternoon and night naps in protocols A and B. Top panel shows the mean number of correct response and the lower panel shows the mean reaction time for correct response over 2 min prior to and immediately following each nap. Significant sleep inertia effects (pre- vs post-nap comparisons) are indicated with asterisks ($p<0.05$). Significant performance recoveries (post-nap vs next pre-nap comparisons) are indicated with arrows ($p<0.05$).



Self-report scales

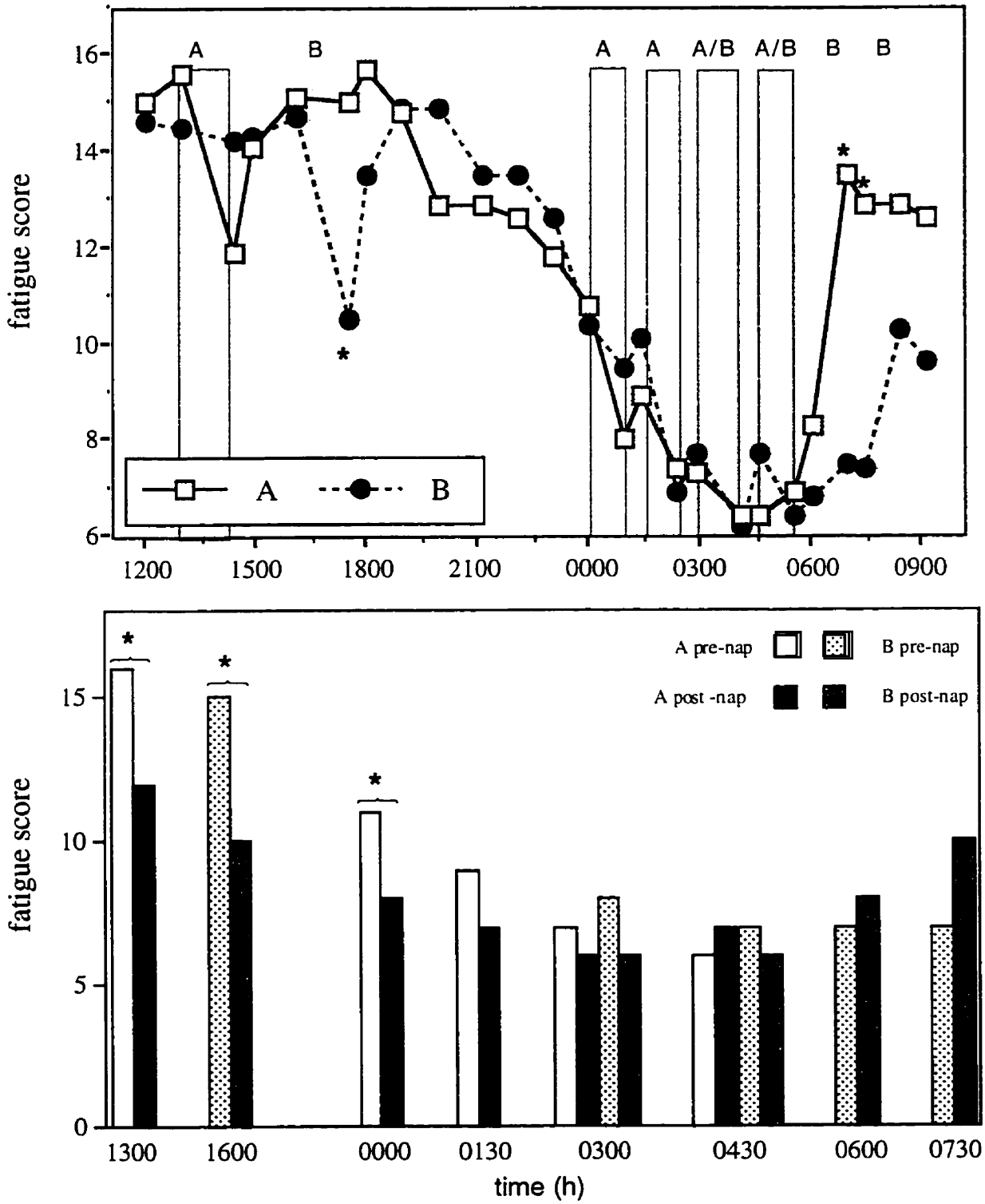
Figure 11, 12, 13, and 14 presents the effects of napping on the self-report ratings of fatigue, sleepiness, mood, and drowsiness over the last 21 hr of the study. Irrespective of the protocol, a progressive deterioration in the subjective levels of fatigue, sleepiness, drowsiness, and mood could be seen from 1900h up to 0600h. The lower panels in these figures show the pre- and post-nap ratings expressed as histograms to facilitate the comparison between the two protocols.

Fatigue

Subjective levels of fatigue followed a circadian rhythm in both protocols with a nadir in the fatigue score between 0400-0500h (top panel of figure 11). However, figure 11 (top panel) indicates that subjects were significantly more fatigued in protocol B, as shown by lower scores after the naps at 1730, 0700 and 0730h, than at the same times in protocol A [$F(30,360)=4.15$, $p<0.001$]. Ratings between the two protocols did not differ significantly at any other test sessions. The most fatigue occurred at 0400h but subjective ratings started to improve between 0700h and 0900h in both protocols. Subjects who napped early in the night (protocol A) recovered from fatigue at a significantly greater rate in the morning hours than subjects napping late at night (protocol B).

Sleep inertia : A significant sleep inertia was observed after the afternoon naps in both protocols (lower panel of figure 11) and after the nap taken at 0000h in protocol A ($p<0.001$). In both protocols, the naps taken during the nights did not affect the recovery from fatigue in the inter-nap period. No sleep inertia was evident with fatigue scores after late night naps (protocol B).

Figure 11: Changes in subjective levels of fatigue over the last 21 hr of the study (top panel). Significant differences between protocol A and B are indicated with asteriks ($p<0.001$). Shaded and boxed area indicate the timing of the naps. The lower panel presents the mean score prior to and immediately following each nap. Significant sleep inertia effects (pre- vs post-nap comparisons) are indicated with asterisks ($p<0.01$). The larger numbers on the ordinate represents less subjective fatigue.



Sleepiness

The pattern of change in the sleepiness ratings was similar to the fatigue ratings in figure 11. Figure 12 (top panel) shows a significant post-nap increases in sleepiness after 1730, 0700 and 0730h naps in protocol B compared to protocol A ($p<0.001$). The highest rating of sleepiness peaked between 0230h and 0530h in both protocols.

Sleep inertia : Significant sleep inertias in sleepiness scores (lower panel of figure 12) were observed upon arousal from the afternoon and first night naps in protocol A ($p<0.01$), and from the afternoon nap in protocol B ($p<0.005$). In both protocols, napping did not improve sleepiness scores either immediately upon awakening from a nap or when rated 15 min later prior to the next nap.

Mood

Circadian variations in the positive (top panel) and negative (lower panel) components of the NHRC mood scale were found (figure 13) mirroring the changes seen in fatigue and sleepiness (figure 11 and 12). Similar to these latter scales, a significant decline was seen in positive mood at 1730, 0700 and 0730h ($p<0.001$)], and a significant increase in negative mood at 0700 and 0730h ($p<0.001$) in protocol B, compared to the same times in protocol A. The same tendency towards an increase in the negative mood score was seen at 1730h but it did not reach significance. The more negative moods and lower positive moods were obtained between 0300h and 0530h in both protocols.

Sleep inertia : Figure 14 presents histograms of the positive (top panel) and negative (lower panel) NHRC mood scores before and after each nap.

Figure 12: Changes in subjective levels of sleepiness over the last 21 hr of the study (top panel). Significant differences between protocol A and B are indicated with asteriks ($p<0.001$). Shaded and boxed area indicate the timing of the naps. The lower panel presents the mean score prior to and immediately following each nap. Significant sleep inertia effects (pre- vs post-nap comparisons) are indicated with asterisks ($p<0.01$). The larger numbers on the ordinate represents greater subjective sleepiness.

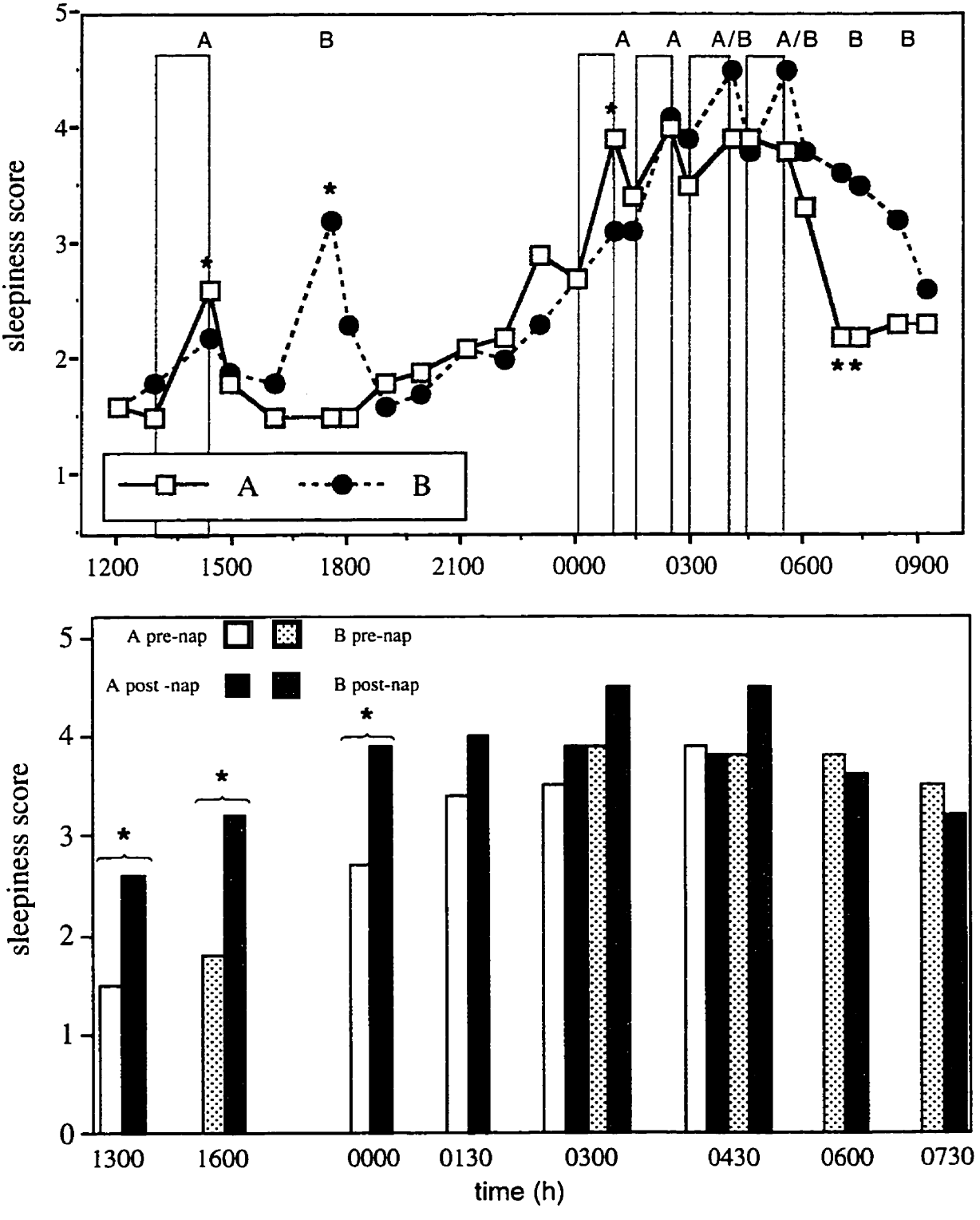


Figure 13: Changes in subjective levels of positive (top panel) and negative (lower panel) mood over the last 21 hr of the study (top panel). Significant differences between protocol A and B are indicated with asteriks ($p<0.001$). Shaded and boxed area indicate the timing of the naps. The larger numbers on the ordinate represents a more positive mood and a more negative mood.

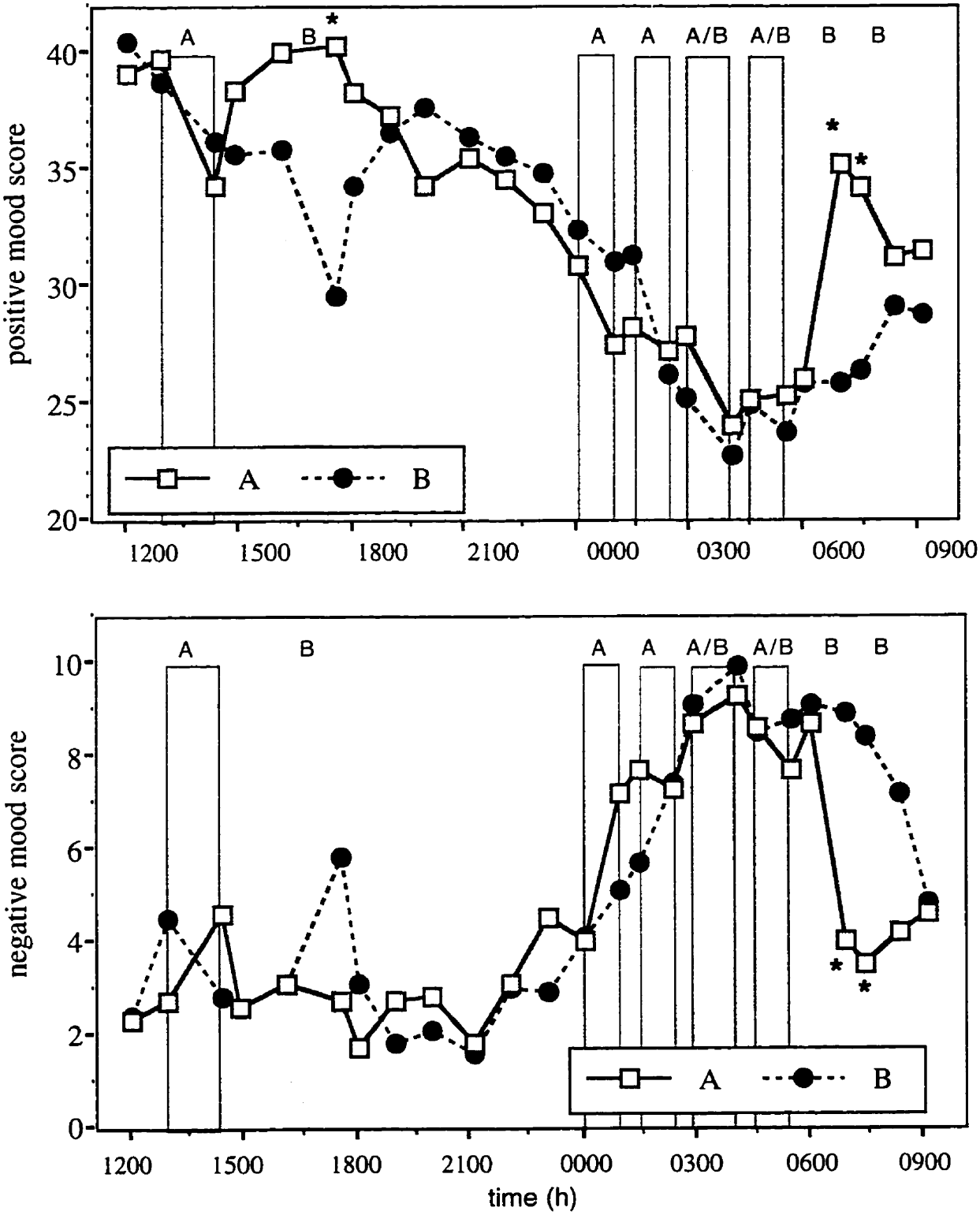
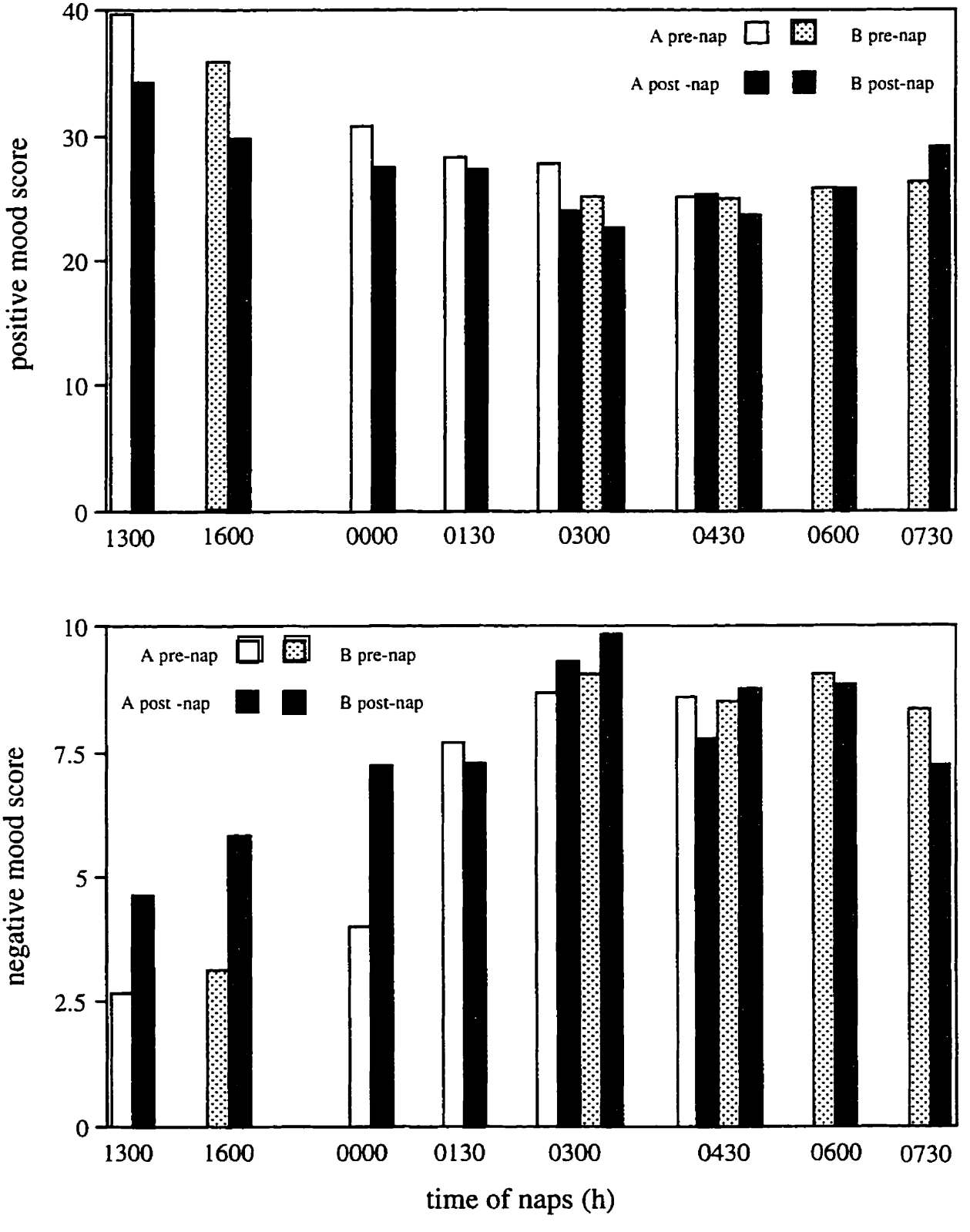


Figure 14: Mean score of subjective levels of positive (top panel) and negative mood (lower panel) prior to and immediately following each nap in protocols A and B.



Although analyses of positive and negative scores showed no significant adverse effect of sleep inertia for any of the naps in both protocols, sleep inertia trends were observed in negative mood after the afternoon naps and the 0000h nap. In both protocols, subjects did not report greater positive mood and lower negative mood during the inter-nap period. Overall abrupt awakening did not significantly affect mood ratings.

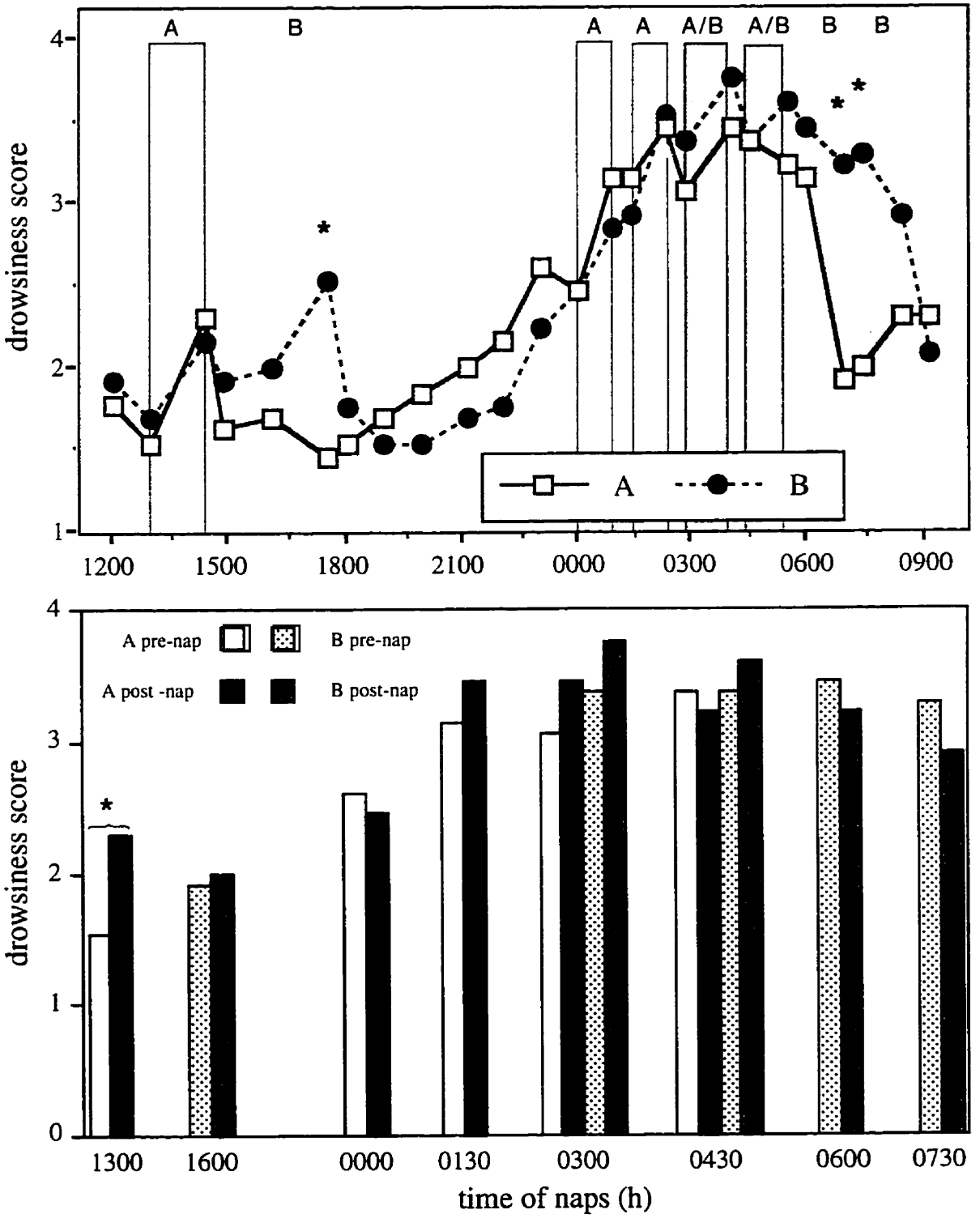
Drowsiness

Drowsiness scores (figure 15) reflected similar changes to those observed in sleepiness (figure 12). A significant increase in drowsiness was seen at 1730, 0700 and 0730h in protocol B ($p<0.001$) compared to the same times in protocol A. The highest ratings of drowsiness occurred between 0230h and 0530h in both protocols.

Sleep inertia : Sleep inertia effects were not as evident as for sleepiness, subjects reported feeling significantly drowsier upon awakening from the afternoon nap in protocol A ($p<0.001$) than prior to the nap (lower panel of figure 15). No sleep inertia effect were found after the naps in protocol B. At night, no significant recovery in the drowsiness levels were observed between the post-nap tests and the pre-nap tests (15 min later) for the following naps in both protocols.

Overall, subjects reported feeling more fatigued, sleepy, and drowsy upon awakening from the afternoon naps compared to pre-nap levels. All self-report scales showed a circadian rhythmicity with peaks in the afternoon or early evening and nadirs in the early morning.

Figure 15: Changes in subjective levels of drowsiness over the last 21 hr of the study (top panel). Significant differences between protocol A and B are indicated with asteriks ($p<0.001$). Shaded and boxed area indicate the timing of the naps. The lower panel presents the mean score prior to and immediately following each nap. Significant sleep inertia effects (pre- vs post-nap comparisons) are indicated with asterisks ($p<0.01$). The larger numbers on the ordinate represents greater subjective drowsiness.



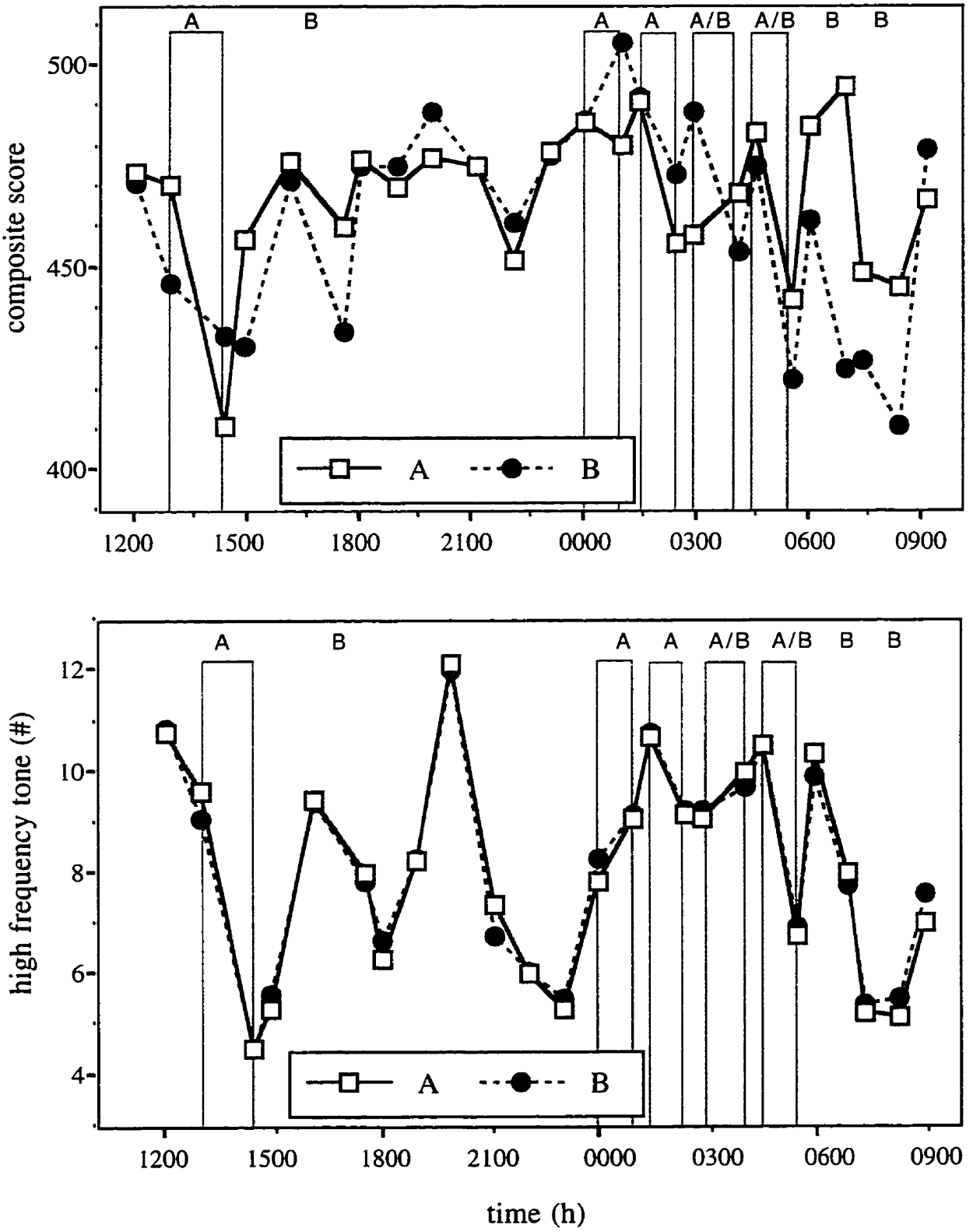
Synthetic work environment (Synwork)

Figure 16 presents the effects of napping on the composite score measurements for the synthetic work task (top panel) and the number of high frequency tones sounded in the auditory sub-task (lower panel) over the last 21 hr of the study.

The composite score in figure 16 (top panel) is the total performance score the subjects received during 4 min on all of the sub-tasks in Synwork. No significant differences in the scores between protocol A and B were found throughout the entire experiment (top panel of figure 16). Overall, the lowest values were seen at 1430, 0530, 0800, and 0830h, and the highest scores occurred at 2100, 0100 and 0130h in both protocols ($p<0.001$). Note the similarities in the composite score curves (top panel of figure 16) and the high frequency tone curves sounded in the auditory sub-task (lower panel of figure 16).

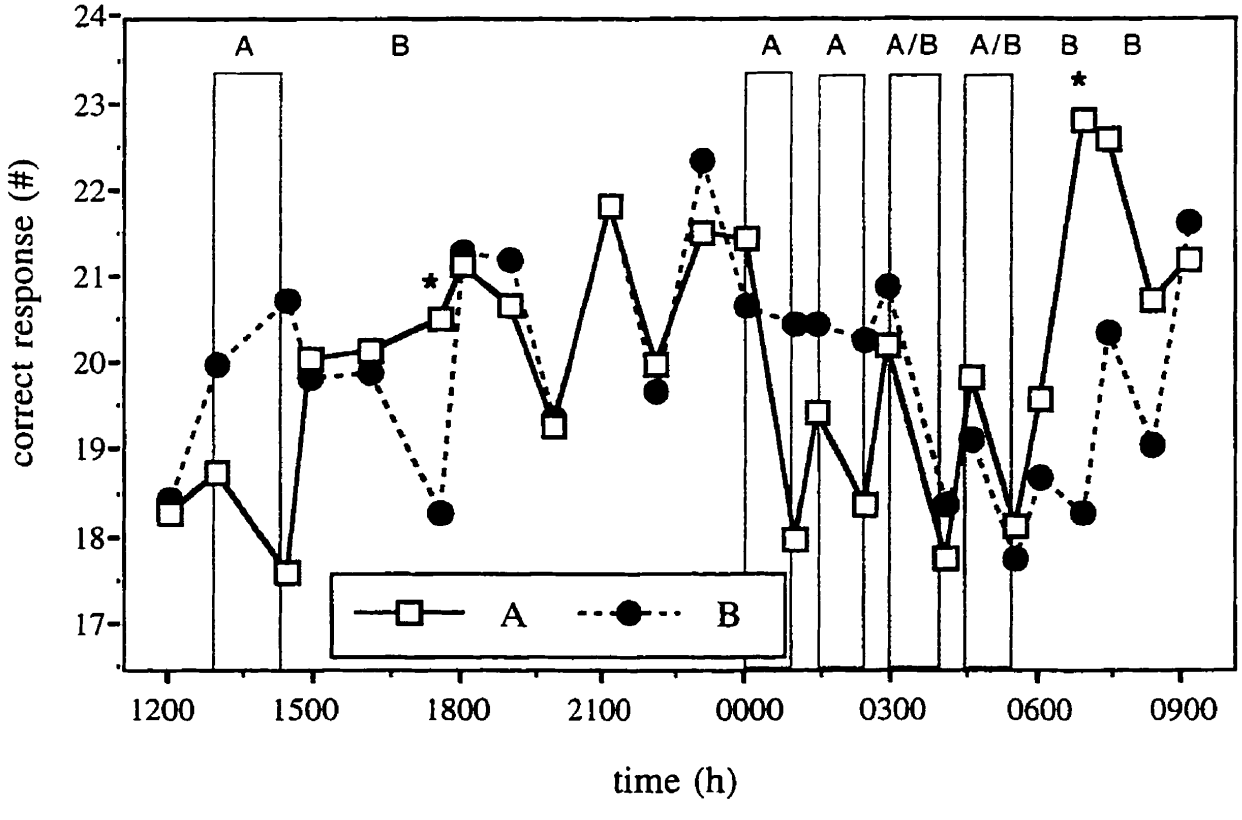
The number of high frequency tones sounded in the auditory sub-task varied significantly from one test session to the next one [$F(30,330)=87.83$, $p<0.0001$] but did not differ between the two protocols (lower panel of figure 16). The high tone probability varied between 0.08 and 0.27 from session to session even though it had been set to be 0.20. Since the correct detection of a high tone was awarded 10 points, the variability in the number of high tones sounded affected considerably the maximum number of points the subjects were able to achieve and the time allocated to respond to the tones in each session. Moreover, it is likely that the allocation of resources by the subjects to the three remaining sub-tasks had been affected by the variability of high frequency tones sounded in the auditory sub-task.

Figure 16: Napping effects on the synthetic work task composite score measurements (top panel) and the number of high frequency tones sounded in the auditory sub-task (lower panel) over the last 21 hr of the study. Shaded and boxed area indicate the timing of the naps.



Consequently, it was not possible to compare performance between pre- and post-nap sessions and to determine the specific effects of sleep inertia on the auditory sub-task or on the other sub-tasks. Individual sub-task analyses comparing the two protocols did not show any consistent effect other than a high variability from test session to test session. However, the correct number of responses given in the arithmetic sub-task (figure 17) showed that subjects in protocol B completed more additions correctly at 1430h compared to subjects in protocol A, who in turn completed more correct additions at 0700h after completing their nap schedule ($p<0.001$). Figure 17 also illustrates the high session to session variability.

Figure 17: Napping effects on the arithmetic sub-task over the last 21hr of the study. Asterisks show the significant difference between protocol A and B in the mean number of correct response over 4 min ($p<0.001$). Shaded and boxed area indicate the timing of the naps.



3.0 PERFORMANCE IN RELATION TO PRIOR SLEEP COMPOSITION

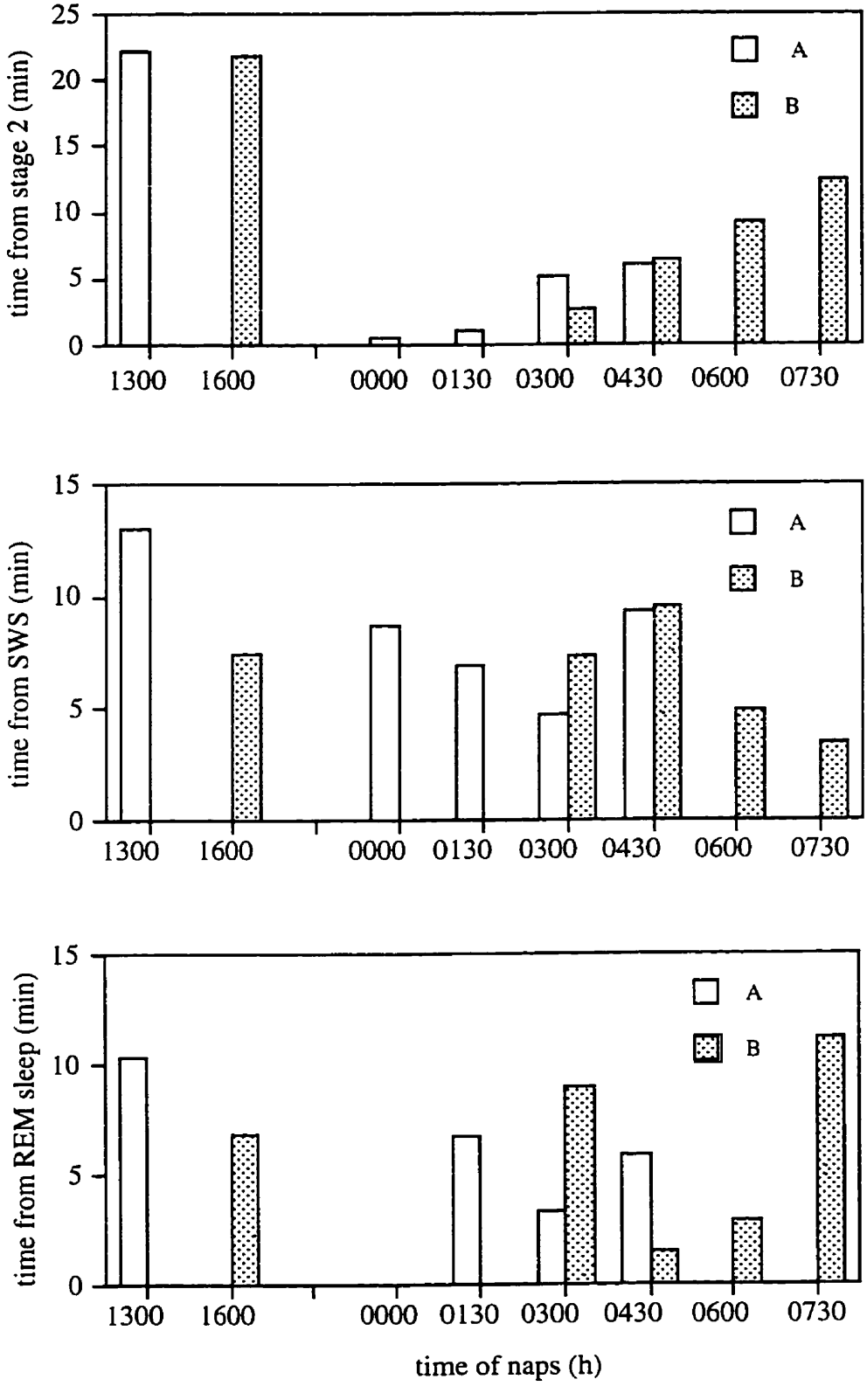
Table 4 shows the sleep stage from which subjects were awakened in each protocol and nap expressed as a percentage of the total number of subjects awakened per nap. On average, subjects were awakened 37% of the time from stage 2, 31% from SWS and 25% from REM sleep. Awakenings from SWS occurred mostly after the 0000h, 0130h and 0430h nap of protocol A, and the 0300h nap of protocol B. REM awakenings predominated after the 1300h and 1600h afternoon naps, and the 0300h and 0430h naps of protocol A and the 0600h and 0730h nap of protocol B.

In the following section, analyses of variance were used to determine the effects of stage at awakening (SWS and REM) on post-nap performance for the logical reasoning and serial reaction time tasks. Moreover, correlations between performance measures immediately upon awakening and sleep variables (such as SWS, REM, and stage 2 in min; SWS%, REM%, and stage 2%; SWS, REM, and stage 2 sleep latencies) were assessed to investigate whether they may have influenced performance upon awakening. The possible influence of the amount of time between the end of a specific sleep stage (stage 2, SWS, and REM sleep) and forced awakening at the end of a nap on post-nap performance level was also assessed. The amount of time between the end of stage 2, SWS, and REM sleep and forced awakening is represented in figure 18 for the afternoon and nocturnal naps in protocols A and B.

Table 4: Sleep stages from which subjects were awakened from the afternoon and night naps in in protocols A and B and expressed as a percentage of the total number of subjects.
Shaded area represent the stage from which subjects were awakened more often within each nap.

Sleep Period	Protocol	Sleep stage at awakening				
		Wake	Stage 1	Stage 2	SWS	REM
1300h -1430h	A	16.67%	0.00%	25.00%	16.67%	**41.67%**
1600h -1730h	B	15.38%	15.38%	23.08%	15.38%	**30.77%**
0000h - 0100h	A	0.00%	7.69%	7.69%	**84.62%**	0.00%
0130h - 0230h	A	0.00%	0.00%	**46.15%**	**46.16%**	7.69%
0300h - 0400h	A	0.00%	0.00%	**46.15%**	23.07%	30.77%
	B	0.00%	0.00%	30.77%	**53.84%**	15.38%
0430h - 0530h	A	0.00%	0.00%	15.38%	**53.85%**	30.77%
	B	7.69%	0.00%	**46.15%**	15.38%	30.77%
0600h - 0700h	B	0.00%	7.69%	**53.85%**	7.69%	30.77%
0730h - 0830h	B	0.00%	7.69%	**53.85%**	7.69%	30.77%

Figure 18: Time between the end of the last episode of stage 2, SWS and REM sleep forced awakening from the afternoon and nocturnal naps in protocols A and B.



Logical reasoning

No significant difference between the two protocols were found in the specific effect of SWS and REM awakenings on post-nap performance for correct responses and reaction times. No significant effects of stage upon awakening were found on the post-nap number of correct responses. As illustrated in figure 18 (right panel), only a trend towards increased post-nap reaction times ($p< 0.09$) can be seen when subjects are awakened from SWS.

In protocol A, a significant correlation was found between SWS and post-nap reaction times after the nap scheduled at 0130h. A positive correlation ($r= 0.67$, $p < 0.01$) indicated that greater amount of SWS (> 15 min) resulted in longer reaction times. No correlations between any sleep variables and post-nap performances reached significance in protocol B. The amount of time between the end of stage 2, SWS and REM sleep and forced awakening were not significantly correlated with post-nap performances in both protocols.

Serial reaction time

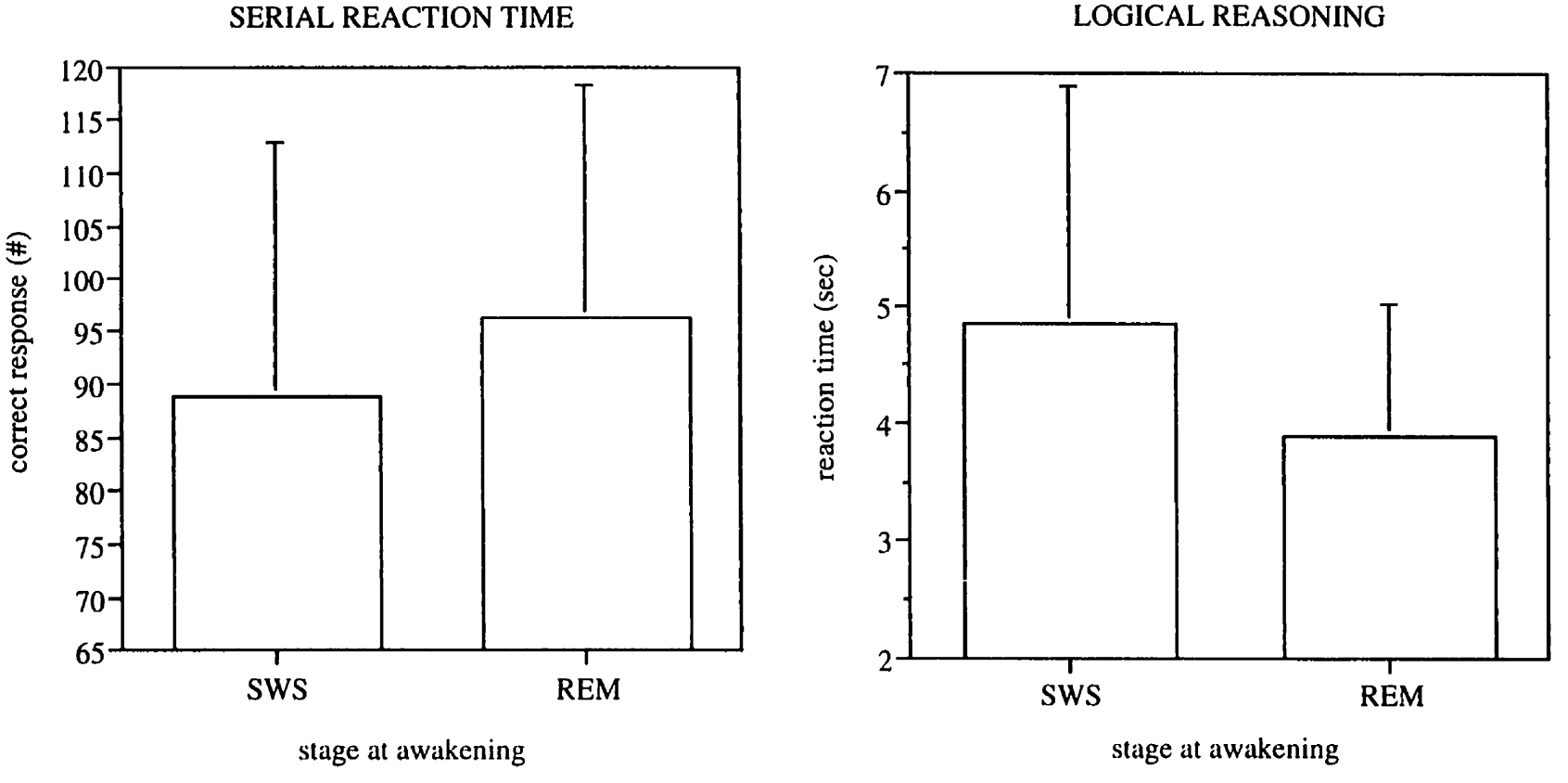
No significant difference between the two protocols were found in the specific effect of SWS and REM awakenings on post-nap performance for correct responses and reaction times. Only a trend towards a reduced number of correct responses ($p< 0.08$) can be seen when subjects are awakened from SWS compared to REM awakenings (left panel of figure 18). No significant effects of stage upon awakening were found on post-nap reaction times.

In protocol A, SWS durations in the 0130h nap were positively correlated with reaction times ($r= 0.60$, $p < 0.02$). The more time subjects spent in SWS, the slower they performed. No correlations between any sleep variables and post-nap performances reached significance in protocol B. The amount of time

between the end of stage 2, SWS and REM sleep and forced awakening were not significantly correlated with post-nap performances in both protocols.

In summary, no significant differences were seen in the effect of sleep stage at awakening and sleep stage duration on post-nap performances between the two protocols. Overall, SWS durations and awakenings showed a tendency to be associated with greater post-nap performance impairments.

Figure 19: Effects of stage upon awakening (SWS and REM) on the post-nap number of correct responses for the serial reaction time task (left panel), and on the post-nap reaction times for the logical reasoning task (right panel). Vertical bars show standard deviations.



CHAPTER V

DISCUSSION

Previous studies that have evaluated the circadian variations in sleep inertia have not been not conclusive. Sleep inertia effects had been reported to be more intense if awakenings occurred during the circadian trough of core temperature, or no circadian effects were found. However, these studies evaluated sleep inertia in a context where sleep deprivation varied concomitantly with time of day. The present study has been designed to investigate nocturnal variations in sleep inertia by varying the placement of a series of naps during the normal sleep period in a context with no prior sleep loss. Sleep inertia, which is usually defined as a deterioration of post-sleep performance in comparison to pre-sleep status, was measured during two series of four 1 hr naps by comparing changes in mental performance prior and immediately after each nap. In the first series (protocol A), naps were scheduled between 0000h and 0430h. In the second series (protocol B), naps were scheduled between 0300h and 0830h. Particular to this design was the introduction of afternoon naps in order to control for the possible confounding effect of prior wake time and sleep loss that may have resulted from the delayed sleep time in protocol B compared to protocol A. To minimize this variable, 90-min naps were scheduled in the afternoon 9.5 hr prior to the first nocturnal nap of both protocols. In addition, two of the naps in each protocol were purposely scheduled to occur at the same clock time (0300h and 0430h) to evaluate any possible interaction between time of night effect and the order of presentation of the naps. Finally, performance tasks were chosen either because of their known sensitivity to sleep inertia (SRT and LRT), or to assess the sensitivity of

the task (Synwork task) to sleep inertia.

Previous studies (Angus and Heslegrave, 1983; 1985) have reported decreases in cognitive performance of 30 % in comparison to baseline values after only 18 hr of wakefulness. However, Dinges *et al.* (1987) showed that naps taken at 1500h and 0300h prior to sleep loss (prophylactic naps) and after 6 or 18 hr of wakefulness had a positive effect on cognitive performance for more than 24 hr after the naps. The scheduling of afternoon prophylactic naps in the current study, was chosen to coincide with the highly reported tendency to spontaneously nap at this time of the day (Tune, 1969; Taub, 1971; Dinges, 1989b; Campbell, 1984; Lavie, 1989; 1992). Afternoon naps were expected to prevent performance decrements prior to the first nocturnal nap in both protocols. Sleep inertia effects were expected after nocturnal naps but no circadian effects on sleep inertia were anticipated. Moreover, since deterioration in cognitive performance found immediately upon awakening return to normal performance levels in approximately 15 min under situations with no sleep loss (Wilkinson and Stretton, 1971; Dinges *et al.*, 1980; 1985), post-nap performances were expected to return to normal levels when measured in a subsequent test session scheduled 15 min later. Based on the results of Dinges *et al.* (1988) which did not show any dissociation between subjective ratings and performance measures upon awakening if the naps were taken prior to sleep deprivation, subjective ratings of fatigue, sleepiness, mood and drowsiness in the present study were expected to demonstrate sleep inertia effects in concordance with sleep inertia effects on cognitive performance. Sleep infrastructure during the nocturnal naps should be dependent on the circadian placement of the naps and the order of presentation of the naps. Core body temperature was expected to show the well known circadian rhythm in both protocols and a lowering of temperature was anticipated during the naps in

comparison with body temperature prior to the nap.

The discussion chapter of this thesis will address the following:

1. Section 1.0: The sleep composition of the naps and control sleep periods.

2. Section 2.0: The variations in core body temperature throughout the 24 hr of each protocol.

3. Section 3.0: The general effects of the two protocols on cognitive performance and subjective measures.

4. Section 4.0: The effects of the circadian placement of the naps on sleep inertia.

5. Section 5.0: The duration of sleep inertia and how it can affect sleep inertia measurements.

6. Section 6.0: The effects of sleep inertia on subjective reports of fatigue, sleepiness, mood and drowsiness with performance measures.

1.0 SLEEP COMPOSITION

Sleep composition of the various sleep periods in the two protocols was calculated to determine the relationships between sleep composition and the sleep stage upon awakening from naps with subsequent sleep inertia effects measured by cognitive performance and subjective ratings. The sleep composition of the various sleep periods was expressed as a percentage of sleep period time to facilitate comparisons with published data for normal adult men between 20 and 30 yrs old (Williams *et al.*, 1974). In addition, the first control night (either protocol A or B) and second control night (remaining protocol) for each subject were compared to each other to assess the effect of

blood sampling procedures on sleep composition since each subject slept the second control night (either in protocol A or B) with an I.V. catheter.

The composition of normal nocturnal sleep in healthy subjects has been well documented (Williams *et al.*, 1974; Carskadon and Dement, 1989). Sleep stages usually measured in minutes can be expressed as percentage of total sleep time. The normal sleep period in young adults (20 to 30 years old) consists of 2 to 3 % wakefulness, 45 to 60% stage 1+2, 15 to 30% stage 3+4 (SWS), and 20 to 35% REM sleep (Williams *et al.*, 1974). We found no differences between the two control sleep periods in both protocols, but some significant differences from the values reported in the literature. Sleep efficiencies greater than 90% were found in both protocols. More specifically, the mean time of wakefulness of 4.8% in protocol A and 6.8% in protocol B during the nocturnal sleep period was significantly greater than the 2 to 3% found by Williams *et al.* (1974). SWS was at the lower limit 15 to 30% range (15.5% in protocol A, 15.6% in protocol B). Our subjects exhibited significantly less REM sleep (16.9%) than the normal 20 to 35% reported by Williams *et al.* (1974). As in most sleep studies, the differences in sleep composition between this study and Williams *et al.* (1974) may be attributed to the alteration of the normal sleep architecture when subjects are exposed to a new environment and recording conditions (often called the first night effect). As in the present study, this phenomena may include less REM sleep and more wakefulness but also more stage 1 sleep, longer sleep latencies, and less SWS (Agnew *et al., 1966*; Mendels and Hawkins, 1967; Schmidt and Kaelbling, 1971). The addition of one or two adaptation nights prior to the first experimental night in the laboratory may have avoided the first night effects and allowed a better estimation of subjects baseline sleep patterns. On the other hand these additional nights may have jeopardized the subject's compliance in

participating in a study already scheduled over two separate weekends. However, even though REM in our group was depressed on the first night, it was not expected to affect sleep for the following night in any quantifiable way (Williams *et al.*, 1974). Moreover, the overall percentage of time spent in wakefulness, SWS, and REM sleep during the control sleep period were comparable between the two protocols.

The presence of an I.V. catheter did not adversely affect the subjects' sleep. This is in agreement with other studies who found that the presence or absence of blood sampling during sleep did not affect sleep composition in normal subjects (Kerkhofs *et al.*, 1989), or patients suffering either from sleeping sickness (African trypaniosomasis) (Buguet *et al.*, 1993) or depression (Kerkhofs *et al.*, 1989). In contrast, a study of healthy older men and women (Vitiello *et al.*, 1996) showed that catheterization of a vein prior to sleep resulted in a decrease in total sleep time, sleep efficiency, REM, and SWS (in women only). However, these catheterization effects were not uniform across all sleep variables or subjects. A second study of older subjects (Adam, 1982) also showed significant reductions in total sleep time, sleep efficiency, REM sleep, and significantly more wakefulness within sleep on the catheter nights. Results from both studies were obtained with an older population. Although in the present study some subjects fell asleep quicker and spent less time in REM sleep when they slept with the I.V. catheter, we can conclude that sleep in normal young male subjects was minimally affected by the presence of an I.V. catheter.

Previous studies have shown that under a normal sleep-wake schedule there is a strong tendency for naps to occur naturally in the afternoon (Tune, 1969; Taub, 1971; Dinges, 1989b; Campbell, 1984; Lavie, 1989; 1992). Moreover, a second peak of SWS has been observed during these afternoon naps (Karacan

et al., 1970; Weitzman *et al.*, 1980; Gagnon and De Koninck, 1984; Gagnon *et al.*, 1985). Consequently, the duration of the afternoon naps (90 min) in this study, and their circadian placement were selected to correspond with the afternoon increase in sleep propensity (Tune, 1969; Taub, 1971; Dinges, 1989; Campbell, 1984; Lavie, 1989; 1992) and to allow time for no more than one NREM-REM cycle (Carskadon and Dement , 1989). We expected that the afternoon naps would resemble the first NREM-REM cycle of nocturnal sleep composed of predominantly SWS and a limited amount of REM sleep. Actually, all subjects fell asleep easily and both afternoon naps were comprised of all sleep stages, with short latencies to sleep onset (4.1 min) and sleep efficiencies of approximately 88%. A significantly larger amount of SWS was present in the 1600h nap in protocol B compared to the 1300h nap in protocol A. Two factors may have contributed to this specific difference. First, the 1600h nap was preceded by a longer time spent awake which has been found to be one of the major factors influencing positively the amount of SWS (Webb and Agnew, 1971; Borbely *et al.*, 1981). Second, the circadian placement of the 1600h nap was closer to the subject's maximum body temperature which has been previously reported to be closely related to an increase in SWS (Broughton, 1975; Lavie and Scherson, 1981; Gagnon and De Koninck, 1984; Campbell and Zulley, 1989). Another possibility is the additive effect of both factors which would also result in a greater amount of SWS in the 1600h afternoon naps.

As the naps were only 60 min in length, the night naps should show fewer sleep stage changes and vary in composition according to their circadian placement. The first naps of each protocol should contain more SWS than the naps taken later in the night. Indeed, both first naps had significantly more SWS than the last naps of each protocol. No significant difference was found in the amount of SWS between the first night naps of protocol A (0000h) and B (0300h). The

introduction of the 90 min afternoon nap and the equalization of the amount of prior wakefulness between the afternoon nap and the first night nap (9.5 hr) appears to have created a comparable pressure to sleep in both protocols. In fact, no significant differences in any sleep variables were observed between naps of protocol A and B when naps were compared in the order of presentation (nap 1-A versus 1-B, nap 2-A versus 2-B, ...).

To estimate how successful the naps were at providing sleep, the cumulative amounts of sleep from all naps were calculated and compared between protocols. When the comparisons were done without including the afternoon naps, subjects in protocol B fell asleep faster and had better sleep efficiencies than in protocol A. Furthermore, subjects in protocol B accumulated significantly more REM sleep during all of the nocturnal naps. This is consistent with previous findings showing increased REM sleep propensity in the early morning hours (Czeisler et *al.*, 1980; Zulley, 1980; Gagnon and De Koninck, 1984). This significant difference with protocol A disappeared if the afternoon nap was included in the cumulative sleep calculations. As mentioned earlier, although the 1600h afternoon naps were comprised of more SWS than the 1300h ones, this did not significantly affect the amount of SWS in the subsequent nocturnal naps. There was no difference in the accumulated amount of SWS between the two protocols.

This lack of any difference in the cumulative amount of SWS between the two protocols may be the result of the comparable durations of prior wakefulness (9.5 hr) between the termination of the afternoon nap and the beginning of nocturnal naps in both protocols. In situations where the subjects have slept the night before (no prior sleep loss), the amount of SWS in a subsequent sleep period has been found to be primarily dependent upon the duration of prior wakefulness (Webb and Agnew, 1971; Borbely *et al.*, 1981). However, our

SWS results in protocol B are in contrast to the observations of Karacan *et al.* (1970) that afternoon naps with more SWS are followed by a reduction in nocturnal SWS. In the present study, only the sleep latencies and consequently sleep efficiency in all naps were found to differ between protocols. In protocol A, sleep latencies in the nocturnal naps were similar to those of the overnight sleep, but this produced lower sleep efficiencies in the naps as a result of the shorter sleep durations of those naps (60 min). In the present study, shorter nocturnal sleep latencies were observed towards the early morning (protocol B) in comparison to the beginning of the night (protocol A). One possible explanation is that subjects were more sleep deprived in protocol B than protocol A even though they had SWS in the afternoon nap of protocol B. However, this may not be the only explanation since sleep latencies and efficiencies within each protocol showed significant improvement from the first to the last nocturnal nap. In other words, subjects fell asleep easier towards the early morning in both protocols. Consistent with the present findings, Richardson *et al.* (1982) showed that nocturnal sleep latencies in young subjects with no prior sleep loss declined steadily during the night with a minimum around 0730h and rose sharply around 0930h. They also reported a second minimum around 1530h. Balkin and Badia (1988) also observed shorter sleep latencies towards the early morning (0700) which they attributed to the accumulation of sleep loss throughout their four nights of disrupted sleep. However, their data showed a similar trend of shorter sleep latencies in the morning during the first night of sleep disruption prior to any sleep loss. This variation of sleep latency with time of night does not explain the significant differences found in the sleep latencies in protocol A compared to protocol B when the naps were taken at the same clock time (0430h). One important difference between the two protocols at this time of the night was the amount of prior sleep. Subjects in protocol A had already the opportunity to sleep in the

preceding 3 naps (0000h, 0130h, and 0300h) whereas the subjects in protocol B had only one nap (0300h) prior to the 0430h nap. This difference in the accumulated sleep between the two protocols prior to the 0430h nap may have created a higher need for sleep in protocol B than protocol A, resulting in a shorter sleep latency.

In summary, the composition of the control sleep in both protocols was typical of a normal night's sleep. As hypothesized, greater amounts of SWS were found in the 1600h afternoon nap (protocol B) than in the 1300h nap (protocol A). This finding may be attributed to longer prior wakefulness (3 hr) and/or to the circadian placement of the nap in comparison to the 1300h nap in protocol A. However, the nocturnal naps in protocol A did not demonstrate significantly higher levels of SWS compared to later naps (protocol B) as previously expected. As predicted, the naps in the second half of the night in protocol B demonstrated higher amounts of REM compared to the earlier naps taken in protocol A.

2.0 CORE TEMPERATURE

It is well-known that sleep is accompanied by a decrease in core temperature occurring immediately after sleep onset and being most prominent during the first two hours of sleep (Gillberg and Akerstedt, 1982). Gillberg and Akerstedt (1982) indicated a connection between the initial temperature change (falling) and the rapid progression of sleep towards SWS at all bedtimes. Moreover, Mills *et al.* (1978) have shown that, at the same time of day, core temperature is lower during sleep than during wakefulness. The well-known circadian rhythmicity of core body temperature was observed in both of our protocols. In addition, a lowering of body temperature during and shortly after the afternoon naps occurred. However, only the 1600h afternoon nap showed a significant

lowering of core temperature compared with temperature prior to the nap and to corresponding times of wakefulness in protocol A. In addition, significantly lower core temperatures were observed in subjects napping at 0730h in protocol B compared to those in awake subjects in protocol A. Interestingly, no other naps showed a significant drop in core temperature.

Several factors may explain these differences in the effect of sleep (and SWS) on core temperature. First, the duration of the nap will have an influence on the lowering effect of sleep on core temperature. Gillberg and Akerstedt (1982) found that the temperature fall was greater immediately after sleep onset and that this effect lasted for about 70 to 80 min. Moreover, they reported a connection between the initial temperature fall and the presence of SWS, with sleep at all bedtimes progressing rapidly towards SWS. Also, they found higher temperature levels during the first SWS periods than during the first REM periods indicating that SWS is related to a rapid fall in temperature rather than to low absolute levels. In agreement with Gillberg and Akerstedt findings, the significantly greater amounts of SWS observed in the 1600h afternoon nap (90 min) may have contributed to the larger decrease in core temperature compared to the 1300h nap. In the nocturnal nap situation of the present study, no significant difference in the amount of SWS were observed between the two protocols in the naps taken in the same order of presentation or at the same clock time. Even when temperatures were compared at the same clock time (0000h and 0130h) when the subjects napped (protocol A) and when they didn't (protocol B), no significant difference were found between core temperatures. The timing of the nap within the circadian rhythm of core temperature will establish the initial core temperature prior to the nap which can vary substantially depending if the nap is scheduled during the rising or descending portion of the rhythm. The lowering effect of sleep (SWS) on core

temperature may vary according to the initial core temperature. Gillberg and Akerstedt (1982) computed the difference in temperature between the first and sixth 10-min periods and compared these differences for the 7 bedtime conditions (2300, 0300, 0700, 1100, 1500, 1900, and 2300h). They found no significant variations in the rate of initial temperature changes (decrease) with time of day. However, the computed differences differed from zero (no change) for all bedtimes except for the 0700h and the 1900h conditions which showed a tendency towards a lesser fall in core temperature. Gillberg and Akerstedt (1982) suggested that the initial effect of sleep (SWS) on temperature was not strong enough in the 0700h condition to counteract the rising tendency of the circadian temperature rhythm in the morning hours. No explanations were given for the 1900h condition. Nevertheless, they concluded that the lowering influence of sleep on temperature was less during the rising phase of the core temperature rhythm.

In the present study, although no significant differences in temperature were found because of the small number of subjects, the timing of the afternoon naps does not explain a significantly greater fall observed in core temperature in the 1600h nap compared to the 1300h one since both naps were scheduled during the rising phase of the circadian temperature rhythm. Nocturnal naps were scheduled in the falling and rising phase of the circadian temperature rhythm. As in Gillberg and Akerstedt's study, sleep in the 0730h nap was not strong enough to counteract the rising tendency of core temperature observed at this time in the control nights. No significant drop in core temperature were observed between before and after the 0730 nap.

The circadian placement of the nap will also affect the corresponding awake temperature according to the direction of the slope. Such effects resulting from the circadian placement of the nap may further explain the significantly lower

temperature observed in the late afternoon nap taken at a time when if awake core temperature continues to rise steeply. In the nocturnal nap situation, subjects napped (protocol A) at a time when core temperature was falling even when the subjects were kept awake (protocol B). As a result, the lowering effect of sleep on temperature was masked by the falling phase of the circadian temperature rhythm and no significant difference were observed in core temperature between sleep and wakefulness, with the exception of the 0730h nap. A significantly lower core temperature was observed in subjects napping at 0730h in protocol B compared to the core temperature (which had already started to rise) of awake subjects in protocol A. This differs from Gillberg and Akerstedt study where temperature during sleep was lower than during wakefulness only during the low or falling phase of the circadian temperature rhythm. However, the authors suggested an alternative interpretation of their data since their waking temperatures were obtained from the condition where subjects spent the longest period awake (24 hr of sleep loss) resulting in a possible delay of core temperature through sleep deprivation.

Prior to the first nocturnal nap of protocol A, a possible tendency for a phase delay in core temperature data was observed in protocol B (figure 4). However, complex demodulation performed on the raw temperature data (figure 5) did not show any significant phase delay in protocol B compared to protocol A. On the other hand, complex demodulation confirmed the lowering effect of the 1600h nap on the circadian rhythm of core temperature in protocol B. A small increasing linear trend present in both protocols suggest a tendency towards a lesser fall in core temperature during the nap nights. The slowing of the decrease in core temperature during its descending phase may be explained by the intermittent internap awakenings. Although this effect was not found to be significant on a nap to nap basis it resulted in an overall increasing linear

trend in core temperature. This effect of wakefulness on core temperature has previously been observed by Aschoff et *al.* (1974).

In conclusion, sleep was accompanied by a decrease in core temperature, the decrease being more prominent in naps of longer durations and large amounts of SWS as in the 1600h afternoon naps. In the case of naps scheduled between 0000h and 0700h, the lowering effect of sleep on temperature was masked by the falling phase of the circadian rhythm. Finally, sleep was not strong enough to counteract the rising tendency of core temperature in the 0730h nap. However, core temperature during the 0730 nap did not rise as much as when the subjects were awake at the same clock time (protocol A) since core temperature were significantly lower during the nap. Unfortunately the small number of subjects with complete core temperature data limited the power of the evaluation of a possible phase delay in protocol B, the lowering effects of naps on core temperature as well as the possible correlation between core temperature and sleep inertia effects on performance discussed in the following sections.

3.0 GENERAL EFFECTS OF THE SCHEDULE ON PERFORMANCE AND SUBJECTIVE MEASUREMENTS

Logical reasoning and serial reaction time tasks

The hourly measurement of performance during the experiment permitted the evaluation of overall levels of performance throughout the study with the exception of performance measured upon awakening from a sleep period. Up to the first night naps (0000h and 0300h), performance measured with the logical reasoning and serial reaction time tasks showed no significant difference between protocols. Previous studies with no afternoon naps had shown substantial decrements (30% or more) in logical reasoning and serial

reaction time performance after 18 hr of wakefulness (Mullaney *et al.*, 1983; Naitoh and Angus, 1989). The expected deterioration of performance at the beginning of the night in protocol B after 18 hr of wakefulness was apparently prevented by the recuperative effect of the afternoon nap. Since there was no comparable protocol without an afternoon nap, it would be premature at this point to conclude that only the afternoon nap permitted performance to be maintained. However, these performance results are in agreement with previous studies (Gillberg, 1984; Godbout and Montplaisir, 1986; Dinges *et al.*, 1987; 1988) showing the positive effects of prophylactic naps on cognitive performance 1.5 to 10 hr after the naps in comparison with subjects without any naps. Finally, cognitive performance measured after the completion of each series of naps returned to similar levels than prior to the beginning of nocturnal naps in both protocols. These findings are consistent with previous studies (Hartley, 1974; Lubin *et al.*, 1976; Mullaney *et al.*, 1983; Haslam, 1985) which showed that moderate levels of sleep reduction (between 4 and 6 hr per day) and fragmentation (60 to 80 min naps) do not result in any significant differences in cognitive performance in comparison to baseline values.

To estimate how well the subjects performed in this study, we compared the present results with the results of Heslegrave and Angus (1985) who also used a shorter version of the logical reasoning and serial reaction time tasks (2 min). In both studies, performance tasks were self-paced and subjects were given instructions to work as quickly and accurately as possible. For the logical reasoning task, Heslegrave and Angus (1985) reported 12 to 14 correct responses per minute and an error rate lower than 1 per min during the daytime prior to any sleep deprivation. In the present study, subjects also scored between 12 and 14 correct responses per minute with an error rate of 3.5. Subjects accurately answered 55 times per minute in the serial reaction time

task, whereas the subjects of Heslegrave and Angus responded correctly 70 times per minute. In both studies, the total number of responses for the SRT were approximately 73 per min.

Interestingly, the number of errors per minute reported in this study were three (LRT) or five times (SRT) greater than the error rate reported in the Heslegrave and Angus study. The difference between the two studies may be related to some differences in the testing conditions. Some of these differences included the use in the present study of only male volunteers, a low workload instead of a high one, a shorter practice time, a 2 min task instead of 1 min, and finally the serial collection of blood samples.

None of the previously mentioned differences between the studies could explain the higher number of errors found in the present study. Gender effects have previously been examined and did not show performance differences (Davies and Parasuraman, 1982). Lower workloads and short task durations are not expected to result in higher error rates (Angus and Heslegrave, 1985; Dinges, 1992a). Shorter practice time and collection of blood samples are unlikely responsible for the larger number of errors per minute since the error rates were maintained throughout the study and the total number of responses were similar to Heslegrave and Angus' study. Finally, as expected when performing short duration self-paced tasks such as the logical reasoning and serial reaction time, "sleepy" subjects preferred to reduce their work rate immediately upon awakening in order to maintain their accuracy (cognitive slowing).

Subjective scales

Previous studies have reported minimum subjective ratings of sleepiness in the early afternoon followed by an increase thereafter (Heslegrave and Angus,

1985; Monk *et al.*, 1985; Monk, 1987). However, minimal ratings of fatigue and sleepiness in the present study were reached prior to the afternoon naps and maintained until 1800h in both protocols. It seems reasonable to think that fatigue and sleepiness ratings did not increase because of the beneficial effects of sleep taken in the afternoon naps. Beneficial effects of afternoon naps on mood parameters have been previously documented in subjects prior to any sleep loss (Dinges *et al.*, 1988). Significant improvement of mood variables following naps were found in comparison to bed rest and control wake periods. The present results showed that fatigue and sleepiness ratings began to increase and positive mood ratings to decrease shortly after 1800h, while the negative mood scores started to increase after 2100h. These self-reports of fatigue, sleepiness and mood during the evening were consistent with those reported by previous studies (Heslegrave and Angus, 1985; Babkoff *et al.*, 1991).

As with the performance measures, subjects' self-reports were not different at the beginning of the night between the two protocols. Within one and a half hour after the last awakening of protocol A (0700h), subjects started to report feeling significantly less fatigued, sleepy and drowsy than in protocol B. At this time (0700h) in protocol B, subjects were still following the alternating schedule of 60 min asleep and 30 min awake. No significant difference was observed between the two protocols 30 min after the last nap of protocol B (0900h). Nocturnal subjective ratings will be further discussed in section 5.

Synthetic Work Environment task

In the present study, an attempt was made to evaluate the effect of time of day and sleep inertia on the newly designed Synthetic Work Environment task (Ellsmore, 1991, Ellsmore *et al.*, 1991) and to compare it with more typical

performance tasks (LRT and SRT). As in previous studies (Ellsmore, 1991, Ellsmore *et al.*, 1991; Savu, 1991, Ellsmore *et al.*, 1995), subjects in the present experiment had a positive attitude towards the task and preferred it to conventional tasks. The synwork task was designed to simulate the complexity of a situation requiring simultaneous attention to several tasks.

The strategy to perform this task, as reported by the subjects at the end of the study, was to start by memorizing the Sternberg set of letters when shown at the beginning of the task. They then performed the arithmetic task while monitoring the other two tasks "from the corner of their eyes". When necessary, they would interrupt the arithmetic task to respond to either the auditory or the Sternberg tasks. After giving a certain number of arithmetic responses, the subjects reported that it was time to reset the cursor from the reaction time task. They repeated this pattern until the end of the 4 min task. No other strategies were reported by the subjects but it is not known how this strategy can vary with increasing sleepiness.

In the present study, when overall measures (session response rate and composite score) were plotted and visually compared with plots from logical reasoning and serial reaction time tasks, considerable variability could be seen in the synwork variables. A careful examination of each sub-task revealed that the auditory sub-task was responsible for this variability (figure 15). In the auditory sub-task, two tones at different frequencies (low, 5000 Hz; high, 7000 Hz) were randomly presented to the subjects every 5 sec. Subjects were required to identify and respond only to high tones. The probability for a high frequency tone which was originally set at 20% was found to actually vary between 8% and 27% from one test session to the other. Since every correct detection of high tone was awarded 10 points, the variability in the number of high tones could impact directly on the maximum number of points for this sub-

task, and the time allocated to the three remaining sub-tasks. Consequently the composite score and session response rate would be affected. As Savu (1991) has shown in his experiment, there are significant interactions among the four sub-tasks, and when task difficulty is manipulated in one of the tasks (i.e., increasing the pointer speed), it will result in the deterioration in the performance of another sub-task (i.e., addition sub-task). Such variability in the high tone probability of the auditory sub-task was not expected since there was no indication of any problem in previous studies (Ellsmore, 1991, Ellsmore *et al.*, 1991; Savu, 1991, Ellsmore *et al.*, 1995). A closer examination of Savu's experimental data (1991) showed a similar but somewhat smaller variability (18% to 29%) in the probability of high tones sounded from session to session. The larger variability observed in the present study may be due to the shorter duration of each test session (4 min) in comparison with 15 min test sessions in Savu's experiment. This task had not been used previously for such a short time, all previous studies having used test session lasting a minimum of 15 min. Two options can be considered in order to eliminate the impact of the variable high frequency probability in shorter test sessions. One possibility is to add a "low sound report" box. Subjects would then need to respond to every high and low frequency tone sounded in order to get points. Since the total number of tones (high and low) was kept constant from one session to the other, this additional report box would mean an identical maximum number of points for every test session, with the subjects still being required to correctly distinguish a high from a low sound. Alternately, default setting of sound rate might need to be smaller than the actual every 5 sec to avoid overload. Another possibility is to modify the section in the program responsible for the high tone probability in a way that would reduce considerably the variability.

Despite its variability from one session to the other, the high tone probability

was kept constant between the two protocols (A and B) for any given test session allowing a comparison between the two protocols. Only the arithmetic sub-task showed significantly more additions correctly completed at 1430h in protocol B than A, whereas more correct additions were completed at 0700h in protocol A than B. In both circumstances, sleep inertia effects may have been responsible for the reduction in the number of correct additions considering that the 1430h test session in protocol A and the 0700h test session in protocol B were scheduled immediately upon awakening from a nap. Further analyses to determine the effects of sleep inertia on the synwork task by comparing pre- and post-nap test sessions were not possible because the number of high frequency tones sounded occasionally differed significantly before and after the nap (figure 15, lower panel). Finally, in the present study the number of correct additions completed by the subjects improved continuously throughout the day (until 0000h) but were similar between the two protocols (fig. 16). Performance improvements on all aspects of the task were also observed by Ellsmore (1991) during their 4 test sessions conducted per day over a period of 4 to 12-days. Thus, in the case of the arithmetic task, more research is needed in order to distinguish between time of day effects and/or practice effects.

In summary, the afternoon naps prevented the expected deterioration in performance at the beginning of the night in protocol B, and the usual increase in subjective sleepiness and fatigue during the late afternoon until 1800h in both protocols. Subjects' work rates on the LRT and SRT were comparable to other studies even though their error rates were higher without prior sleep loss. Performance measures returned to their initial levels once the nocturnal naps were over. Both subjective and performance measures were not significantly different between the two protocols with the exception of test sessions scheduled after the naps. Finally, the use of shorter duration test session in

synwork may have revealed some limitations of this new complex task.

4.0 EFFECTS OF THE CIRCADIAN PLACEMENT OF NAPS ON SLEEP INERTIA

If given the opportunity to nap prior to any accumulation of sleep loss, is there a best time to sleep in order to minimize the unwanted effect of sleep inertia upon awakening? In other words, as Naitoh *et al.* (1993) questioned "is there a best time to wake up?".

Three methods have been commonly used to measure sleep inertia. One method compares post-nap performance with "baseline" performance taken sometime prior to the nap and any accumulation of sleep loss (Seminara and Shavelson, 1969; Wilkinson and Stretton, 1971). By comparing performance measures at different times in the study but within the same subjects, this method avoids the possibility of finding differences between groups that would simply be the result of individual differences. However, comparison of performance to a "baseline" performance taken sometime at a different time of day and/or prior to any accumulation of sleep loss poses problems as any significant difference could be the result of either a time of day effect, a sleep loss effect, or a sleep inertia effect.

A second method has compared performance upon awakening from the nap with performance measured at the same time in a group of subjects who did not nap (Tassi *et al.*, 1992; Naitoh *et al.*, 1993). This method has the advantages of comparing performance measured at the same time of day in both conditions and providing a good comparison with what performance would be at this time of the day without the "benefit" of a nap. However, consideration should be given to the use of a different group of subjects in the two conditions and the possibility of a significant difference resulting from individual differences. In the

present study, the possibility of having "control protocols" without nocturnal naps was considered. However, one important disadvantage of having the same subjects in the "control protocols" is that they would be required to spend two more weekends in the laboratory in order to complete a control protocol for each afternoon nap (1300h and 1600h). This would have considerably reduced the compliance of the subjects to participate in such a study while doubling the time needed for its completion.

Finally, the most common method used to measure sleep inertia is to compare performance immediately upon arousal from sleep with performance measured prior to sleep (Langdon and Hartman, 1961; Dinges *et al.*, 1981; 1985; Glovinsky *et al.*, 1990; Stampi, 1992; Mullington and Broughton; 1994). This method has the advantages of comparing performance measures within the same subjects and evaluating the immediate "benefit" of a nap by comparing performance levels of the subjects just prior to the nap with that immediately after the nap. However, comparisons of performances measured at different times of the day (pre- and post-nap) should be carefully considered because of the possible confounding effects of time of day on performance tasks especially in the case of longer sleep periods (several hours).

In the present study, comparison between pre- and post-nap performance was the method chosen to measure sleep inertia effects. This method was chosen because naps were of short durations (60 and 90 min), and comparisons of performance were done within the same subjects.

Afternoon naps

The present results for the logical reasoning and serial reaction time tasks demonstrated no significant sleep inertia effects after the 90 min afternoon naps scheduled at 1300h and 1600h. Moreover, after both afternoon naps, post-nap

performances (30 min after awakening) were back to pre-nap levels and did not differ from performance at the same time in the two protocols in subjects who had either been awake for 3 hr (protocol A) or had not slept yet (protocol B). Other studies found that 2 hr midday naps produced minimal sleep inertia in both healthy sleep deprived and non sleep deprived narcoleptics adults (Mullington and Broughton, 1994; Lavie and Weler, 1989). On the other hand, Dinges *et al.* (1981) found substantial sleep inertia on the descending subtraction and simple reaction time tasks after 60 min afternoon naps in non-sleep deprived adults. Stampi (1992) semipolyphasic sleep study reported greater daytime sleep inertia following 50 min naps than 20 min naps, with intermediate levels after 80 min naps. However, when only the afternoon naps were examined, both the 80 min and 20 min naps resulted in comparable but smaller sleep inertia effects (70% of pre-nap) than the 50 min naps (50% of pre-nap) on the DST performance task.

These results illustrate the importance of the duration of the nap on sleep inertia. Nap durations of approximately an hour (50 or 60 min) produced the strongest sleep inertia effects while longer naps (80, 90 and 120 min) produced less sleep inertia. In naps shorter than 2 hr and taken prior to sleep loss, minor changes in nap duration may directly affect sleep inertia by modulating the sleep stage probability from which the subjects might be awakened. Greatest decrements have been observed upon awakening from SWS while REM arousals produced the least decrements (Dinges, 1990; Stampi, 1992). In addition, several authors (Dinges *et al.*, 1981, 1985; Lavie and Weler, 1989; Stampi, 1992) had previously reported that the greater the amount of SWS, the more severe the performance decrements upon awakening. Despite the fact that subjects in the 1600h nap (protocol B) had significantly greater amounts of stage 4 sleep and total SWS than in the 1300h nap (protocol A), it was not

associated with significantly greater performance decrements upon awakening. A closer examination of the proximity of SWS periods to sleep termination showed that SWS ended on average more than 20 min prior to awakening in both protocols. Therefore, most of the afternoon nap awakenings for both protocols occurred from REM (36%), wake (16%) or stage 1 (7%), comprising 59 % of the total awakenings. Consistent with previous findings, sleep stage upon arousal was one of the key factors responsible for the smaller performance impairments upon arousal.

Nocturnal naps

A reduction in the number of cognitive responses (cognitive slowing) on self-paced short duration tasks has been reported to typically occur with sleep deprivation (Wilkinson, 1961; Lubin, 1967; Mullaney *et al.*, 1983; Angus and Heslegrave, 1985; Dinges *et al.*, 1988). Dinges (1992a) suggested that the sleepy subjects chose "willingly" and "strategically" to "trade speed for accuracy". Thus, sleepiness occurring upon abrupt awakenings (sleep inertia effects) was expected to result in a similar type of performance deficit (cognitive slowing) on self-paced tasks than sleepiness caused by extended wakefulness (sleep deprivation effects). Indeed, the logical reasoning and serial reaction time tasks measured in the present study showed significant reductions in the number of cognitive responses immediately upon awakening from the nocturnal naps. Interestingly, the subjects chose to reduce their work rate in order to maintain the same level of accuracy as prior to the naps. In a study designed to determine whether sleep inertia effects were qualitatively different from sleep deprivation effects, Balkin and Badia (1988) looked at performance and subjective sleepiness during four night of sleep disruption/restriction. In contrast to previous studies (Tassi et *al.*, 1992; Naitoh et *al.*, 1993; Mullington and Broughton, 1994) and the present findings, sleep inertia effects produced

both an increased error rate and a reduced work rate on an addition task. Although Balkin and Badia's study revealed that both speed and accuracy of calculation were affected by sleep inertia, they also found an equally deleterious effect of sleep loss across the night on both the error and work rates.

The primary purpose of this study was to determine if performance measures of sleep inertia were influenced by time of night. A comparison of pre- and post-nap performance showed that sleep inertia effects on awakenings from a series of naps taken early in the morning (protocol B) affected performance less than awakenings from a series of naps taken at the beginning of the night (protocol A). In other words, post-nap performance measures in protocol B were not significantly different from pre-nap performance measures whereas post-nap performance measures in protocol A were always significantly different from pre-nap performance measures.

In fact, if sleep inertia is sensitive to a time-of-day effect, one would expect to find a more severe sleep inertia around the circadian trough in body temperature (around 0400h) than earlier at night as in the studies of Dinges *et al.* (1985), and Rosa *et al.* (1983). Naps taken during the nadir of the circadian rhythm in core temperature produced stronger sleep inertia effects upon awakening than naps taken at the peak even when they were preceded by up to 12 more hours of sleep loss (Dinges *et al.*, 1985), or when subjects were awakened from baseline sleep period with no prior sleep loss (Rosa *et al., 1983*). Whereas, if sleep inertia effect is primarily a function of prior sleep deprivation, one would expect stronger sleep inertia effects by the end of the night and/or study (Dinges *et al.*, 1985; Rosa and Bonnet, 1985). However these suggestions could not explain the absence of time of day effects on sleep inertia in Balkin and Badia (1988) and Naitoh et *al* (1993) studies. Balkin and Badia (1988) found stable sleep inertia effects (measured by the mean number

of addition problems correctly completed) across the night. Even the cumulative sleep loss effect of 4 nights of consecutive sleep disruptions did not reveal any time of night effects on sleep inertia. Naitoh *et al* (1993) also failed to find any circadian rhythmicity in sleep inertia (measured by the number of problems attempted in the logical reasoning task) upon awakening from 20 min naps taken every 6-hr during a 64-hr of continuous work period.

Our findings did not show the expected time of day effect on sleep inertia since all of the naps in protocol A (between 0000h and 0430h) showed strong sleep inertia effects in comparison to the last naps of protocol B (0600h and 0730h). No other pre-nap versus post-nap comparisons showed any significant sleep inertia effects. Moreover, these smaller sleep inertia effects measured in the early morning naps of protocol B are in contrast to the stronger sleep inertia effects expected with the additional 3 hr of wakefulness in protocol B in comparison to protocol A. On the other hand, these results further support the previous findings of Dinges *et al.* (1987; 1988) suggesting the usefulness of prophylactic afternoon naps in preventing performance deterioration following longer period of wakefulness. Thus, neither sleep deprivation (stronger sleep inertia by the end of the study) nor time of day (stronger sleep inertia during the trough of the core temperature rhythm) adequately explain the smaller sleep inertia effects found in the early morning.

The sleep composition of the nap and/or stage upon awakening which are also assumed to be related to the severity of sleep inertia (Webb and Agnew, 1964; Feltin and Broughton, 1968; Tassi *et al.*, 1992; Dinges at al., 1981; 1985) may be important factors to consider. In the present study, a trend towards an increase in the severity of sleep inertia effect on post-nap performance was only seen in relation to the preawakening sleep stage. In both protocols, awakenings from SWS produced worse LRT reaction times and SRT correct

number of responses in comparison to REM awakenings which resulted in performance levels much closer to pre-nap levels. These results are generally consistent with those of Dinges (1990), Tassi *et al.* 1992, and Naitoh *et al.* (1993) which showed greater performance decrements after awakening from SWS while arousal's from REM sleep produced the least performance impairments. In agreement with Dinges (1990), performance immediately upon awakening was also positively correlated with depth of sleep (related to the amount of SWS). In protocol A, the greater the amount of SWS accumulated during the 0130h nap, the slower subjects performed the LRT and SRT tasks. In contrast, Davidson (1987) found no relationship between the correct number of responses per min in the logical reasoning task and the amount of SWS, whereas the correct number of responses per min in the serial reaction time task was positively related to the amount of SWS. In Davidson's study, however, the logical reasoning task was performed after the serial reaction time task which started 8 min after awakening.

The weak relationship observed in the present study between post-nap performance and sleep depth (SWS) and stage upon awakening are nevertheless consistent with the performance results which show less sleep inertia effects immediately upon awakening from early morning naps (protocol B). This relation points towards a deterioration in the subject's performance in association with the presence of SWS especially when closer to awakening. Although the amount of SWS did not differ significantly between protocol A and B when the naps were compared in the order of presentation, awakenings from SWS occurred mostly after all of the naps of protocol A and the first nap (0300h) of protocol B. As Dinges (1990) pointed out, it is difficult to separate sleep composition from stage upon awakening in shorter sleep periods. On the other hand, sleep depth may not be only linked to the presence of SWS since Bonnet

(1985) observed some sleep inertia effects upon arousal in subjects who were denied most of their SWS and REM sleep. Sleep deprivation or extreme sleep reduction and disruption, may create stronger sleep pressure (strong sleepiness or sleep tendency) resulting in a stronger subsequent sleep inertia irregardless of the stage subjects are awaken from. Alternately, naps taken prior to sleep deprivation or in addition to nocturnal sleep (as in the present study) might not be influenced by any strong sleep pressure and result in sleep inertia effects which vary in relation with stage upon awakening. Perhaps sleep depth and/or stage upon awakening only modulate an already present sleep inertia effect.

One other possible mechanism suggested by Dinges (1990) which is related to the sleep inertia process, concerns the decline in cerebral metabolism resulting from a drop in core temperature (thermal down-regulation) which is evoked by sleep (especially SWS). Dinges explains that a low cerebral metabolic activity which would covary with sleep pressure (therefore sleep depth) would impair cognitive performance if a subject was abruptly awaken unless a rise in the metabolic activity had already occurred. He further suggests that the metabolic activity would increase "through the passage of time (e.g., circadian variation), a change in sleep stage (e.g., accumulation of REM sleep), or increased physical activity at awakening (e.g., getting out of bed)". Essentially, core body temperature would covary with sleep inertia effects on cognitive performance because of its relation to cerebral metabolic activity. In the present study, smaller sleep inertia effects were found during the early morning naps when body temperature was either at its lowest or was rising. These results support Dinges' hypothesis since the three last naps of protocol B would be the ones to most likely have higher metabolic activity if indeed metabolic activity is correlated to body temperature. However, it is more difficult to explain the lack

of significant sleep inertia effects on performance upon awakening from the afternoon naps especially in protocol B (1600h). The 1600h nap resulted in a significant drop in body temperature attributed to greater amounts of SWS which in turn should be associated with lower cerebral metabolic activity and result in greater sleep inertia impairments. It is possible that the drop in temperature was insufficient to produce a substantial brain metabolic decline and as a result produce significant sleep inertia effects. Further evaluation of body temperature as a covariant of sleep inertia effects on cognitive performance is necessary.

In summary, when sleep inertia was examined by comparing pre- and post-nap performance, awakenings from a series of early morning naps (protocol B) resulted in less sleep inertia than naps taken at the beginning of the night (protocol A). The absence of any significant sleep inertia after the afternoon naps may be explained by the longer duration (90 min) of the naps and by awakenings occurring mainly from wakefulness, stage 1 and REM sleep. On the other hand, the smaller sleep inertia effects upon awakening from the early morning naps could not be adequately explained by the accumulation of sleep loss which had been expected to produce stronger sleep inertia effects. However, the severity of sleep inertia effects was related to the hypothesized lower brain metabolic activity associated with the drop in core temperature at the beginning of the nigh (protocol A) and marginally associated to the sleep composition of the nap and/or stage upon awakening.

5.0 DURATION OF SLEEP INERTIA

Sleep inertia has been characterized in most studies by poor performance immediately upon awakening compared to pre-sleep status (Naitoh and Angus, 1989), which eventually comes back to normal performance in approximately

15 min under normal circumstances (Wilkinson and Stretton, 1971; Dinges *et al.*, 1980; 1985) and can be more prolonged under sleep deprivation (Naitoh, 1981; Dinges, 1990). In the present study, each nocturnal nap was followed by two 15 min cognitive test sessions, one upon awakening followed immediately by a second one. The second, third, and fourth naps of protocol A and B were started as soon as the second test sessions were completed. Since performance was expected to be back to normal levels after 15 min of wakefulness (Dinges *et al.*, 1985), results from the second test session were used as a measure of pre-nap performance for the following nap. Therefore, it is possible that the present findings of smaller inertia effects in the morning (0600h and 0730h) were a product of how sleep inertia was measured. As a matter of fact, when post-nap performances in both tasks were compared between naps and protocols, no significant differences in sleep inertia effects on performance were found. These results suggest that sleep inertia persisted beyond the 15 min allotted between the first performance measurement (upon awakening) and the second one just prior to the following nap. Performance on the LRT and SRT showed a significant recovery during the 15 min spent awake between each naps of protocol A but not after naps taken at 0430h and 0600h of protocol B. This delayed recovery of performance after 15 min of wakefulness was even more evident after the serial reaction time task than the logical reasoning task despite the fact that the SRT task was performed after the LRT. Duration of sleep inertia may affect differentially different types of performance tasks. Selective effects of sleep inertia on different performance measures was also observed by Mullington and Broughton (1994) between a subtraction test and a serial reaction time task. Again the serial reaction time task showed more sensitivity to sleep inertia effect. Since post-nap performances did not always return to normal levels in protocol B and they were sometimes slightly but not significantly better than post-nap performance of

protocol A, the difference between pre- and post-nap performance in protocol B became smaller resulting in an apparent significantly smaller sleep inertia effect. In fact, no significant differences in sleep inertia effects were found between early and late nocturnal naps.

While the present study was not designed to analyze any progressive improvement of sleep inertia effects over time on task, the repetition of each task 15 min later did allow us to compare subject's performance between the two test sessions. The use of a longer task duration would have permitted us to examine the duration of sleep inertia but not different performance tasks. Furthermore, longer duration tasks need to be carefully chosen so they are not strongly affected by boredom or create a lack of interest, since performance impairments caused by sleep inertia would be difficult to distinguish from performance impairments resulting from boredom or lack of interest.

Very few studies have monitored the duration of sleep inertia by measuring performance over the course of several minutes. Feltin and Broughton (1968) observed that "arousal from SWS was progressive, whereas following REM sleep subjects were initially more alert, then tended to lack interest and fall asleep (non-motivated test)". As mentioned earlier, the present results showed only a trend to stronger impairments of performance upon arousal from SWS in comparison to REM sleep awakenings in both protocols. The observations of Feltin and Broughton suggested that the prolonged sleep inertia effects found after the 0430h and 0600h naps in protocol B may be related to a larger number of subjects being awakened from REM sleep in protocol B than in protocol A. However, table 3 shows that in both protocols the same number of subjects were awakened from REM sleep in the three last naps and prolonged sleep inertia effects were observed only in protocol B but not in protocol A. Thus, REM awakenings alone can not explain the difference in sleep inertia duration

between naps and protocols. Balkin and Badia (1988) found a successive improvement in the number of correct additions completed (averaged across 4 nights) with each test at 1.5, 7.5, and 13.5 min after awakening but no effect of time of night. This reduction in sleep inertia across the three post-awakening tests was due to a decline in the mean error rate (25% to 10%) and an increase in the mean number of problems attempted. In Balkin and Badia's study, it is possible that any difference in sleep inertia duration across the night could have been masked by the accumulation of sleep loss from night 1 to 4. Moreover, Tassi *et al.* (1992) reported a sleep inertia duration of 15 min after an early night nap (0000h to 0100h) and 9 min after a 0400h to 0500h nap on a spatial memory test. They did not mention, however, if the difference in sleep inertia duration between the two nap timings was significant. In the present study, shorter sleep latencies observed in protocol B, combined with time of day effects are possible explanations for the apparent longer duration of performance impairments caused by sleep inertia in the 0430h and 0600h nap in protocol B.

Another explanation could refer to the hypothesized link between strong sleep inertia effects on performance and a decline in brain metabolic activity resulting from a drop in body temperature. The rise in core temperature observed during and after the 0600h nap (figure 4) may be associated with a rise in brain metabolic activity. Thus, higher metabolic activity would result in better performances immediately upon awakening (smaller sleep inertia effects) and shorter duration of impairments in protocol B. Although performances upon awakening in protocol B were slightly better (but not significantly) than post-nap performances in protocol A, they did not recover significantly after 15 min of wakefulness. The expected rise in brain metabolic in protocol B may not have been sufficient to result in significant performance improvements. Moreover, the

slowing of core temperature decrease and possible corresponding rise in metabolic activity resulting from the internap wakefulness in protocol A would not seem sufficient to explain the significant performance improvements found in protocol A.

In summary, in the present study the intensity of sleep inertia upon awakening was not significantly influenced by time of day but the duration of impairments was longer following naps taken at 0430h and 0600h in protocol B. In other words, post-nap performances measured immediately upon awakening in protocol A were not significantly more impaired than post-nap performances in protocol B. The apparent smaller sleep inertia effect in protocol B was a product of a reduced difference between pre-nap performances, which had not returned to the subjects' initial performance levels, and post-nap performances. The present results demonstrate the importance of how one characterizes (defines and measures) sleep inertia effects when evaluating the effects of different circadian placements of a nap. A recent study by Mullington and Broughton (1994) comparing the efficacy and time of day effects of sleep inertia upon awakening from daytime naps in narcoleptic patients illustrate the importance of how sleep inertia is measured. The authors concluded that single midday long naps (2 hrs at 1540h) produced minimal sleep inertia compared to shorter afternoon naps (20 min) especially the ones scheduled around 1540h. Comparisons of sleep inertia between naps were done by transforming post-nap performances into a percentage of pre-nap levels (post-nap/ pre-nap X 100). However, as observed on the graphs, performance levels prior to the long nap were below pre-nap levels of the short nap resulting in an apparent smaller sleep inertia effect upon awakening from the longer naps. In fact, both naps appeared to produce similar post-nap performance impairments.

6.0 PERFORMANCE VERSUS SUBJECTIVE MEASURES OF SLEEP INERTIA

During each test session, subjective questionnaires were administered after the logical reasoning and serial reaction time tasks, allowing the subjects to use, if they wanted, their perception of how well they had performed in the previous tasks to evaluate their fatigue, sleepiness, mood, and drowsiness. Subjects' ratings indicated a detrimental effect of sleep inertia on fatigue and sleepiness immediately upon awakening from afternoon naps. Similar results of fatigue and sleepiness were found following the midnight naps but not with any other nocturnal naps.

Although performance scores did not deteriorate significantly after the afternoon naps in the present study, the subjects felt more fatigued and sleepy than prior to the nap. On the other hand, a consistent sleep inertia effect was observed following nocturnal naps while the corresponding subjective ratings did not show any awareness of a decreased post-nap performance compared to pre-nap level except for the midnight naps. Self-ratings indicated increasing fatigue and sleepiness, and decreasing mood across the night (circadian variation). These results can be contrasted with Dinges' (1990) conclusions indicating that subjects self-report of sleepiness immediately upon awakening will reflect their level of performance if they nap prior to 18 hr of sleep loss. Otherwise they will misjudge (usually underestimate) how sleepy they feel in comparison to their performance level. In other words, the dissociation between subjective ratings and performance measures upon awakening does not occur if the nap is taken prior to sleep deprivation. However, Dinges *et al.* (1985) reported previously a higher sleepiness rating immediately following an afternoon nap (1500h) taken only after 6 hr of wakefulness in comparison with a lower sleepiness rating given after a 0300h nap taken after 18 hr of wakefulness. Furthermore, performance measured upon awakening was significantly better in the 1500h

nap compared to the 0300h nap. As in the present study, Dinges *et al.* showed that although performance did not deteriorate as much in the afternoon than at night, subjects reported feeling sleepier in the afternoon. A similar discrepancy between self-ratings and performance measures were found at night by Balkin and Badia (1988). Stable sleep inertia effects were measured across the night with a 5 min addition task whereas self ratings indicated increasing sleepiness across the night especially between the beginning of the night (0040h and 0140h) and the early morning (0440h and 0540h). A potential explanation for the differences between subjective ratings at different times of day and performance impairments may be that subjects compared their mood at the time of measurement with how they expected it to be at this time of day (circadian variation) without considering how "well" they performed on the tasks. In other words, if the subjects perceived their mood to be (e.g. sleepy) different from what they would expect at this time of day (e.g. not sleepy in the afternoon), they may give a higher rating than if they felt sleepy at night when they expected to feel sleepy.

It is also interesting to note that even after 15 min of wakefulness, when most of the sleep inertia effects on performance had dissipated, subjective ratings did not improve significantly between each night nap. In contrast, Balkin and Badia (1988) showed that self-rated sleepiness decreased with time since awakening when all measurements were averaged across the four nights. They also noted that the three post-nap sleepiness ratings tended to diverge by the morning, which meant a larger decrease in sleepiness in the morning. They hypothesized that this probably reflected "circadian variations in preparedness for awakening". Instead of reflecting preparedness for awakening, the divergence may also reflect a relative sensitivity by the subjects to judge their sleepiness improvement. A small change in how sleepy they feel when they

score high (early morning) may be better detected than the same change when they score low (beginning of the night) on the sleepiness scale.

Another possible explanation for the difference between subjective ratings and performance levels immediately upon awakening may be related to a differential sensitivity of subjective ratings to sleep loss. However, Baranski *et al.*, (1994) found that sleep deprivation had no apparent effect on the ability of the subjects to self monitor their performance on a sequential addition task with no feedback. When subjects were asked to evaluate on a scale from 1 to 6 how confident they were about their previous response on the addition task, it was found that the validity of their judgment was not affected by sleep deprivation. Moreover, subjective ratings (fatigue, sleepiness, and mood) from the same studies showed, over the course of the experiment, similar declines and plateaus to performance tasks (Angus and Heslegrave, 1985; Heslegrave and Angus, 1985). Even when the subjects were given a 2 hr nap opportunity, performance and mood showed similar sleep inertia effects that gave way to an attenuated circadian-modulated deterioration of performance and mood (Angus *et al.*, 1992). Two field investigations (military personnel) have also found nap benefits for both performance and mood (Haslam, 1985; Opstad *et al., 1978*). However, mood parameters in Dinges *et al.* (1988) did not benefit from 2 hr nap opportunities and showed a significant variation over time as a function of sleep loss and circadian timing whereas circadian-modulated performance decline was attenuated or prevented by the naps. In the present study, irrespective of the naps (i.e. in both protocols), a progressive circadian-modulated deterioration in mood variables was observed from 1900h to 0600h. A significant difference was observed between the two protocols in ratings given at 0700 and 0730. Subjects who had napped early in the night (protocol A) recovered at a greater rate than subjects still napping (protocol B). However, as

soon as the subjects in protocol B finished napping, subjective ratings did not differ between the two protocols. It seems that, in our experimental conditions, the expected circadian-modulated recovery of mood variables in the morning was delayed by the presence of naps.

Thus, in the present study, it remains unclear why subjective ratings do not reflect subjects' performance decrements immediately upon awakening and their following performance improvements as time awake passed. Subjective measurements of fatigue, sleepiness, mood and drowsiness are fundamentally different from the ability to perform; they are more sensitive to circadian variations; or more influenced by time spent asleep or a combination of both. If subjective ratings are more sensitive to circadian variation, they may be related to some physiological process such as core body temperature or cerebral metabolic rate as hypothesized for sleep inertia effects on cognitive performance in the previous section. Interestingly, subjects' ratings indicated a detrimental effect of sleep inertia on fatigue and sleepiness upon awakening from the afternoon naps, whereas a significant drop in body temperature was observed during and after the 1600h nap in comparison to temperature prior to the nap. A similar trend in temperature lowering was observed in the 1300h nap. A detrimental effect of sleep inertia on subjective ratings was also found following the midnight nap even though the temperature drop during the midnight nap did not reached significance. A shorter nap duration and a circadian-related fall in temperature that started prior to the nap may have been responsible for the lack of any significant difference. No other significant deterioration in subjective ratings could be attributed to sleep inertia effects but subjects reported feeling more fatigued and sleepy at 0700 and 0730h compared to the group who had already finished napping (protocol A). At the same time, significantly higher temperatures were observed in the non-napping

group (protocol A) compared to the ones who were still napping (protocol B). Unfortunately, our incomplete data set of core temperatures did not permit any further evaluation of a possible relation between body temperature and subjective ratings. However, this may suggest a need to further investigate possible relations between physiological variables and subjective ratings, and it may help to understand some discrepancies in the effects of sleep inertia on subjective reports and cognitive performance measures.

In conclusion, subjective ratings seem to be more affected by circadian rhythm and sleep loss than by the presence of short naps, and in this case by the severity of sleep inertia. These specific self-reports of fatigue, sleepiness, mood and drowsiness can not be used to evaluate the severity of sleep inertia on performance tasks. More appropriate subjective scales are needed to reflect performance decrements upon awakenings.

CHAPTER VI

SUMMARY AND POSSIBLE APPLICATIONS FOR THE PRESENT FINDINGS

The main purpose of this study was to evaluate time of night effects of sleep inertia on performance and subjective measures immediately upon awakening from nocturnal naps. Confirming previous findings by Balkin and Badia (1988), consistent performance impairments measured upon awakening from nocturnal naps implied that there was no particular time of night (prior to 0730h) when individuals could be suddenly awakened and their performance would not be significantly impaired by sleep inertia. Only the 0730h nap showed less performance impairments upon awakening in comparison to the other naps. Nevertheless, it can be said that in general the intensity of sleep inertia does not vary with time of night in a situation with no prior sleep loss and controlled prior wakefulness. In contrast with previous findings (Webb and Agnew, 1964; Feltin and Broughton, 1968; Dinges et *al.*, 1981, 1985; Tassi et *al.*, 1992; Naitoh et *al.*, 1993), the intensity of sleep inertia was only marginally associated with prior sleep architecture or stage upon awakening. This may suggest that under lower sleep pressure (no prior sleep loss) sleep architecture and stage upon awakening only modulate already present sleep inertia effects.

Interestingly, naps taken late in the night (protocol B) resulted in longer performance decrements in comparison to earlier naps (protocol A). In contrast, Balkin and Badia (1988) found no time of day effect on performance recovery and Tassi et *al.* (1992) reported shorter duration of impairments after a late nocturnal nap (0400h). Contrary to Tassi et *al.* (1992), stage upon awakening

could not explain delayed recoveries in the late naps in protocol B. A circadian effect is a possible explanation. In addition, delayed recoveries of performance to initial levels were even more evident in the serial reaction time task. Perceptual motor tasks (e.g. SRT) may be more sensitive to time of night effects than semantic memory tasks such as the LRT.

The fact that subjective ratings of fatigue, sleepiness, mood and drowsiness could detect small deteriorations in performance upon awakening from the afternoon naps but not larger impairments and recoveries at night, suggest that time of day may have a stronger influence on these measures than sleep inertia effects. This lack of correspondence between subjective ratings of sleepiness and measures of performance have also been found in other studies with partial or no prior sleep loss (Hescovitch and Broughton, 1981; Johnson et *al.*, 1990, 1991). Strong correlations between the two types of measures are usually found under sleep loss experiments (Glenville and Broughton, 1979; Gillberg et *al.*, 1994; Akerstedt and Kecklund, 1994). The present findings may be important since these type of scales are often the only available method for assessing sleepiness and fatigue in the workplace. In addition, some work situations do not provide the workers with continuous feedback on their performance (e.g. control room, driving) and subjective signals of sleepiness are sometimes the only information on which individuals can base their decisions (e.g. rest breaks, nap) in order to avoid mishaps.

The evaluation of a possible covariation between sleep inertia effects and core temperature (because of its hypothesized link to brain metabolic activity) was not conclusive. The steep rise in core temperature and corresponding increase in brain metabolic activity observed during and after the 0600h nap could not explain the presence of significant sleep inertia effects on performance upon

awakening and the absence of significant performance improvements in the internap period. However, significant performance recoveries were found during the internap periods in protocol A despite smaller increases observed in core temperature. On the other hand, a closer relationship between core temperature and subjective ratings was observed in the present study.

In view of the present findings, napping during work hours may be a useful "alertness" strategy in the workplace as long as the negative effects of sleep inertia are considered. Naps can be used in both a preventive and operational manner to maintain performance. The present findings have shown that prophylactic afternoon naps can be used to maintain performance and reduce the hours of continuous wakefulness before a nocturnal work period. In terms of short nocturnal naps (1 hr), early night naps should be favored over later ones especially under circumstances where it is important that the negative effects of sleep inertia on performance dissipate after 15 min of continuous wakefulness. Moreover, subjective ratings of fatigue, sleepiness, mood, and drowsiness should not be used in order to evaluate subjects' post-nap performance levels or to evaluate if their performance levels are back to acceptable levels. Before implementing naps in the workplace it is essential to delimitate what constitutes a significant performance decrement in each specific work setting. The potential negative effects of sleep inertia after a nap should be considered and weighted against potential benefits on performance especially if a nap can be interrupted by an emergency situation requiring immediate rapid and high level of performance.

CHAPTER VII

CONCLUSIONS

This study provides some insights into the nature and relationship between time of night and sleep inertia effects on performance tasks and subjective ratings. In the context of naps taken with no prior sleep loss and controlled prior wakefulness, we can conclude that:

1. When pre- versus post-nap performance measures are compared to evaluate sleep inertia effects:

Performance measures upon awakening from the early nocturnal naps are more affected by sleep inertia effects than after late nocturnal naps.

2. When only post-nap performance measures of all the naps are compared with each other:

No significant differences in sleep inertia effects on performance were found between early and late nocturnal naps.

3. When the duration of post-nap impairments is considered:

A delayed performance recovery to initial levels occurred only with late nocturnal naps.

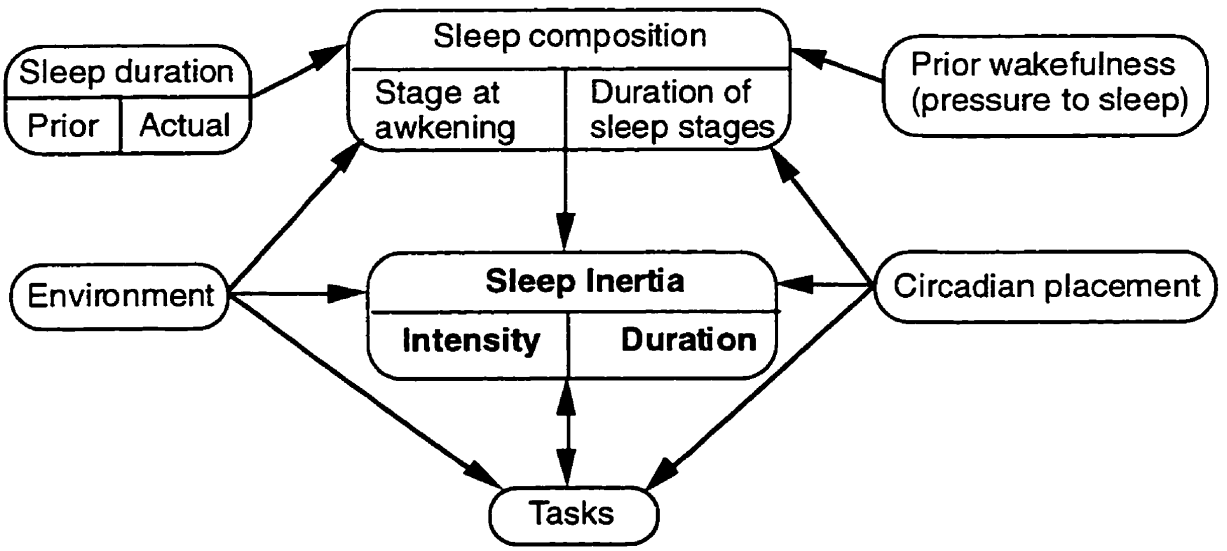
4. Subjective measures versus cognitive performance measures:

Subjective ratings of sleepiness, fatigue, mood and drowsiness did not reflect sleep inertia effects as defined by performance tasks. Moreover, subjective measures did not show any improvements after 15 min of wakefulness as observed in performance measures.

The present results demonstrate the importance of how one characterizes (defines and measures) sleep inertia effects and how this can change the conclusions of the study. Consequently, sleep inertia should not only be defined and measured by comparing pre- versus post-nap performances but it should also consider the recovery rate from sleep inertia and its relation to initial performance levels.

Finally, sleep inertia was indeed affected by the circadian placement of the nap but it may in fact be the result of the combined effects of the environment, type of task, prior wakefulness, sleep duration, sleep composition, and circadian placement. Furthermore, it is possible that at any one time each factor does not have an equal influence on sleep inertia. A schematic view of the interactions between these factors is illustrated in figure 20.

Figure 20: Interactions between factors influencing sleep inertia.



This schematic view shows that sleep duration and prior wakefulness can impact on sleep inertia by their effects on sleep composition. Environment and

the circadian placement of the naps can affect sleep inertia directly as well as indirectly. All of these factors must be considered when attempting to examine sleep inertia.

Recommendations for future studies:

More research is necessary to define the duration of sleep inertia under different circumstances (e.g. shift work, night work) and time of day (e.g. between 0900h and 0000h). The effects of sleep inertia on performance tasks might be further evaluated by using tasks involving other characteristics (e.g. multitasking, feedback) in order to determine which type of tasks would be more likely affected by sleep inertia. Future research is necessary to evaluate or design subjective scales which are sensitive to sleep inertia effects as defined by performance impairments. There is a need to further investigate the use of techniques such as displaying noise in the room, bright light exposure or washing the face with cold water immediately upon awakening in order to dispel sleep inertia more quickly. From a practical point, there is a need to conduct fields studies simulating emergency awakenings in situ with real workers and tasks and evaluate nap effectiveness and possible sleep inertia effects. Finally, more research is necessary to investigate possible changes in the brain at the biochemical level during wakefulness, sleep and sleep inertia.

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APPENDIX A: Statement of informed consent

SLEEP INERTIA STUDY

I, _____, hereby give consent to participate as a test subject in the above titled experiment under the supervision of Dr. Harvey Moldofsky and Valérie Gil (Ph.D. student).

I understand that the study involves two protocols (A & B), each of which will require me to spend approximately 36 hours in the sleep laboratory (starting Friday night and ending Sunday morning) on two occasion about 4 weeks apart. On each occasion, after having one full night's sleep, I shall remain awake and sleep only when permitted for the remaining 26 hours.

I understand that standard measurements of my sleep will be made in the sleep laboratory throughout the two occasions. At the beginning of the study, wires will be applied to my head and body to permit measurement of my sleep. I understand that my body temperature will be continuously monitored with a thin flexible wire inserted in my rectum (rectal thermistor), and that the slight initial discomfort associated with its insertion will quickly subside. I understand that these monitoring measures will be effective for the duration of the experiment (36 hours), on both occasions.

I understand that a thin tube (catheter) will be inserted into a vein in my arm, so blood samples can be withdrawn. In protocol A, the tube will be inserted prior to baseline sleep and removed 36 hours later. In protocol B the catheter will be inserted after the baseline at noon and removed 21 hours later. Before, during and after sleep 8 ml (about 1 1/2 tablespoons) blood samples will be removed. I understand that the amount of blood to be withdrawn during protocol A will be about 264 ml and during protocol B about 152 ml. I understand that over the course of the study (one month) the maximum total blood withdrawn will be 416 ml, less than one Red Cross blood donation. I understand that protocol B may precede protocol A. I understand that, aside from the possibility of some minor

bruising and discomfort from the catheter in my arm, no untoward effects are anticipated from the procedures.

I agree to perform cognitive and performance tasks at various times throughout the study. I also agree to refrain from smoking, using any medications and drugs, including alcohol, pain relievers, caffeine, and tranquilizers on the days of the study, unless my doctors feel that such medication is important to my health.

I understand that the complete schedule require 1 1/2 days on 2 occasions. I shall receive, for my participation in the study, an honorarium of $225 after each completed occasion for a total of $450. I have been informed that I may, at any time, revoke my consent and withdraw from the experiment and that the investigators may terminate my involvement in the experiment at any time, regardless of my wishes.

I understand that there may not be any immediate or direct personal benefit from the analysis of my sleep and blood samples, but the information might be helpful in the understanding of sleep inertia.

I hereby permit any data obtained as a result of this study to be presented at scientific meetings and published in scientific literature provided my anonymity is protected. This consent is voluntary and has been given under circumstances in which I can exercise free power of choice.

Date: _____

_____ _____

Subject Signature Witness Signature

APPENDIX B: Volunteers profile

Subjects	Age (years)	Weight (Kg)	Height (cm)	Average night sleep (hours)	Education (years)	Morningness/Eveningness (years)
S1	35	76	174	8	16	morning
S2	28	61	169	7	19	morning
S3	20	84	196	7	14	neither
S4	23	104	185	8	18	morning
S5	30	82	185	7.5	19	neither
S6	20	68	182	8	14	neither
S7	21	84	180	7.5	18	neither
S8	31	62	182	8.5	13	morning
S9	24	87	185	8.5	16	neither
S10	27	113	191	6.5	18	neither
S11	34	84	185	6.5	22	morning
S12	24	78	183	8	18	neither
S13	22	71	175	7	16	neither
Mean	26	81	182	8	17	
(s.d.)	5	15	7.1	0.7	2	

APPENDIX C: Performance Measures and Subjective Scales

LOGICAL REASONING

PROVIDE ANSWERS TO THE FOLLOWING GRAMMATICAL REASONING PROBLEMS. ANSWER EACH QUESTION WITH EITHER A "T" FOR TRUE OR "F" FOR FALSE.

e.g., A PRECEDES B

A B [T/F]:

THE 16 POSSIBLE STATEMENTS DESCRIBING THE ORDER OF A AND B WERE:
A PRECEDES B
B PRECEDES A
A IS PRECEDED BY B
B IS PRECEDED BY A
A DOES NOT PRECEDE B
B DOES NOT PRECEDE A
A IS NOT PRECEDED BY B
B IS NOT PRECEDED BY A
A FOLLOWS B
B FOLLOWS A
A IS FOLLOWED BY B
B IS FOLLOWED BY A
A DOES NOT FOLLOW B
B DOES NOT FOLLOW A
A IS NOT FOLLOWED BY B
B IS NOT FOLLOWED BY A

EACH OF THE ABOVE STATEMENTS WAS FOLLOWED BY EITHER:

A B OR B A

SERIAL REACTION TIME

THE MESSAGE THAT HAVE BEEN SENT OVER THE COMMUNICATION SYSTEM DURING THE PRECEDING PERIOD HAVE TO BE FILED NOW TO CONSERVE COMPUTER STORAGE SPACE.

THE ADDRESSES OF EACH MESSAGE WILL BE PRESENTED TO YOU AND YOU MUST HIT THE CORRESPONDING KEY TO FILE THAT MESSAGE. THE KEYS ARE MARKED AS:

11 FOR 11 CMBG

12 FOR 12 CMBG

13 FOR 13 CMBG

14 FOR 14 CMBG

YOU ONLY HAVE TWO MINUTES TO FILE ALL THE MESSAGES, SO PLEASE WORK AS QUICKLY AS YOU CAN, BUT ACCURATELY.

e.g., MESSAGE 021

14 CMBG

SAM FATIGUE CHECKLIST

FOR EACH OF THE FOLLOWING ITEMS, PLEASE ENTER THE NUMBER CORRESPONDING TO HOW YOU FEEL RIGHT NOW:

1. WORSE THAN
2. SAME AS
3. BETTER THAN

ITEMS: VERY LIVELY

EXTREMELY TIRED

QUITE FRESH

SLIGHTLY POOPED

EXTREMELY PEPPY

SOMEWHAT FRESH

PETERED OUT

VERY REFRESHED

FAIRLY WELL POOPED

READY TO DROP

NHRC MOOD SCALE

FOR EACH OF THE FOLLOWING ITEMS, PLEASE ENTER THE NUMBER CORRESPONDING TO HOW YOU FEEL RIGHT NOW:

1. NOT AT ALL
2. A LITTLE
3. QUITE A BIT
4. EXTREMELY

ITEMS:

Active	Grouchy
Alert	Happy
Annoyed	Jittery
Carefree	Kind
Cheerful	Lively
Able to concentrate	Pleasant
Considerate	Relaxed
Defiant	Satisfied
Dependable	Sleepy
Drowsy	Sluggish
Dull	Tense
Efficient	Able to think clearly
Friendly	Tired
Full of pep	Able to work hard
Good-natured	

Scoring: The sum of the scores on the following 10 items is the negative score: annoyed, defiant, drowsy, dull, grouchy, jittery, sleepy, sluggish, tense, and tired. The sum of the scores on the remaining items is the positive score.

STANFORD SLEEPINESS SCALE

PLEASE ENTER THE NUMBER CORRESPONDING TO HOW YOU FEEL RIGHT NOW:

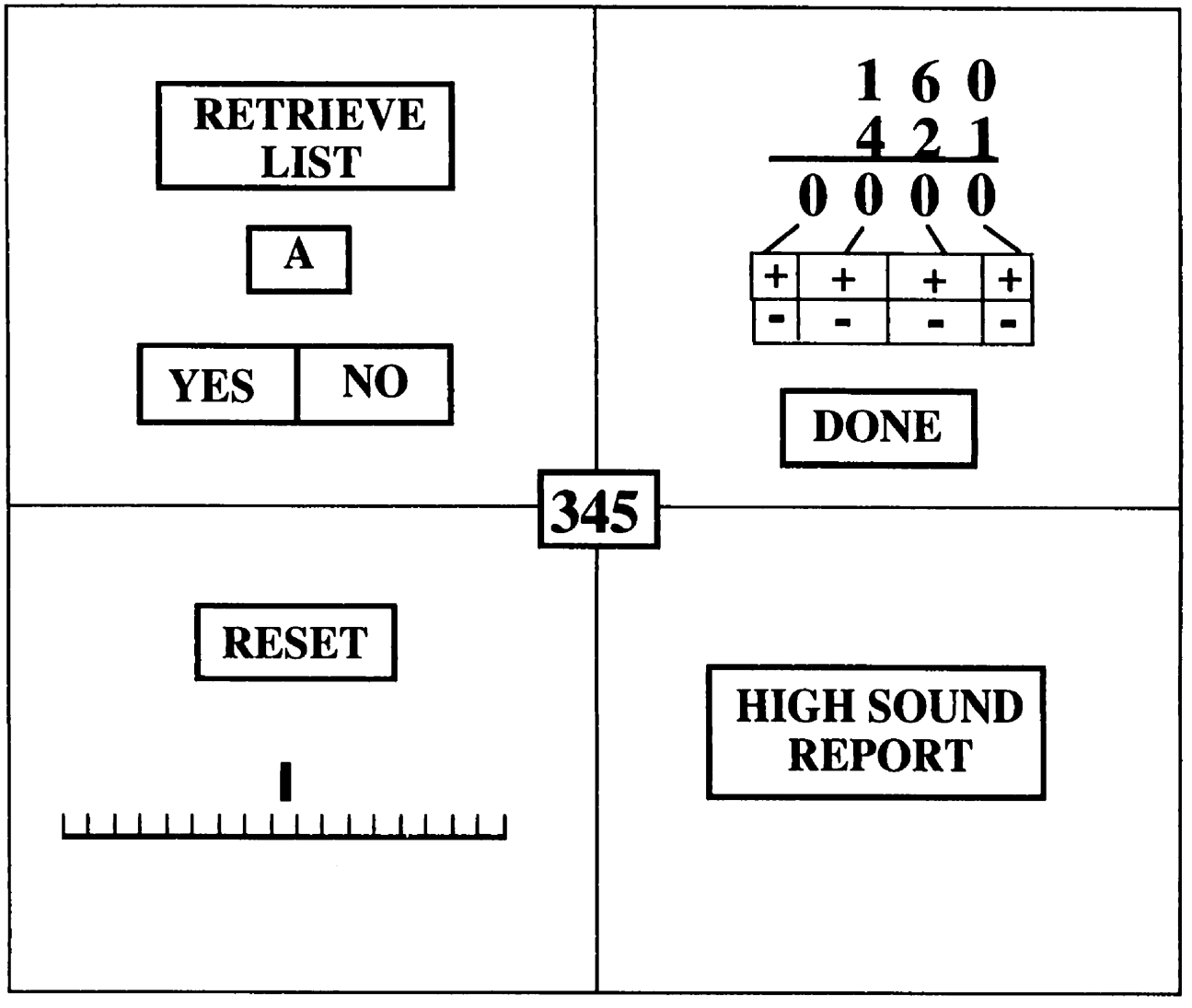
1. Feeling active and vital; alert; wide awake.
2. Functioning at high level, but not at peak; able to concentrate.
3. Relaxed; awake; responsive; but not at full alertness.
4. A little foggy; not at peak; let down.
5. Foggy; beginning to lose interest in remaining awake.
6. Sleepy; woozy; prefer to be lying down; fighting sleep.
7. Almost in reverie; sleep onset soon; losing struggle to remain awake.

THE DROWSINESS SCALE

BASED ON THE FOLLOWING SCALE, PLEASE INDICATE THE NUMBER WHICH BEST DESCRIBES HOW YOU FELT DURING THE LAST EEG TRIAL

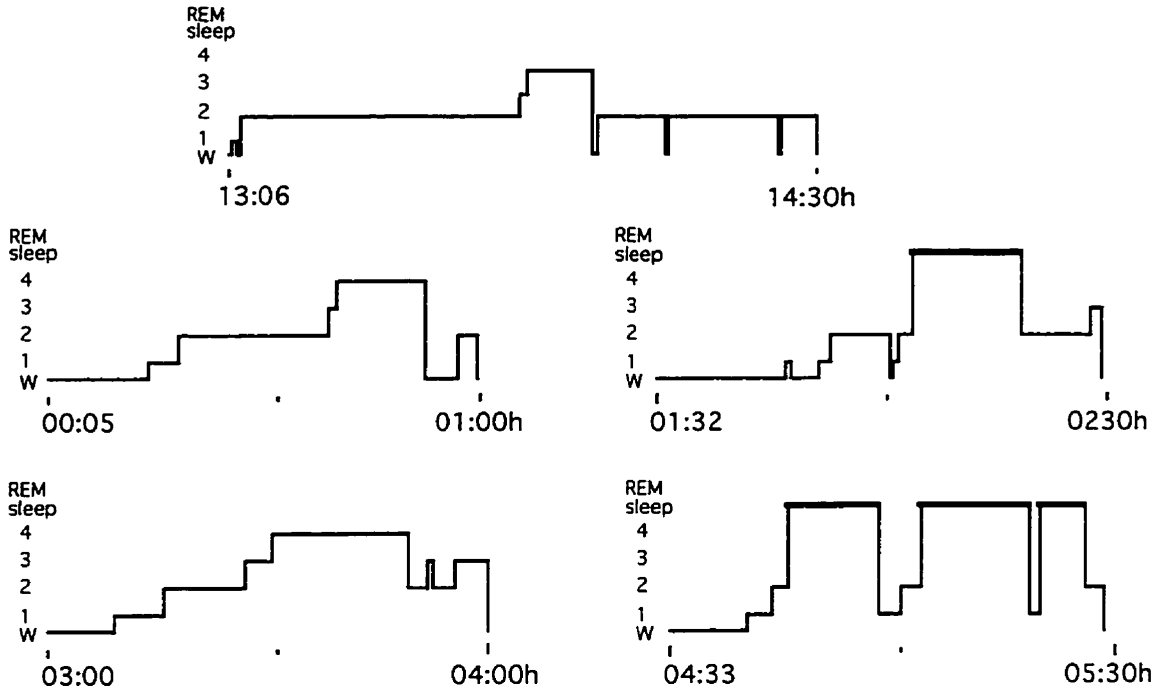
1. I WAS ALERT AND WIDE AWAKE.
2. I WAS ALERT, SOMEWHAT RELAXED BUT NOT AT MY PEAK.
3. I WAS VERY RELAXED AND EVEN A BIT DROWSY.
4. I WAS VERY DROWSY AND ALMOST FELL ASLEEP.
5. I WAS EXTREMELY DROWSY AND I THINK I MIGHT HAVE FALLEN ASLEEP BRIEFLY.
6. I FELL ASLEEP A NUMBER OF TIMES AND IT WAS DIFFICULT TO STAY AWAKE FOR EVEN SHORT PERIODS.
7. I FELL ASLEEP AND STAYED ASLEEP UNTIL THE TASK WAS OVER AND I HAD TO BE AWAKENED BY THE EXPERIMENTER.

SYNTHETIC WORK TASK SCREEN



APPENDIX D: Hypnograms for subject 3:

Protocol A



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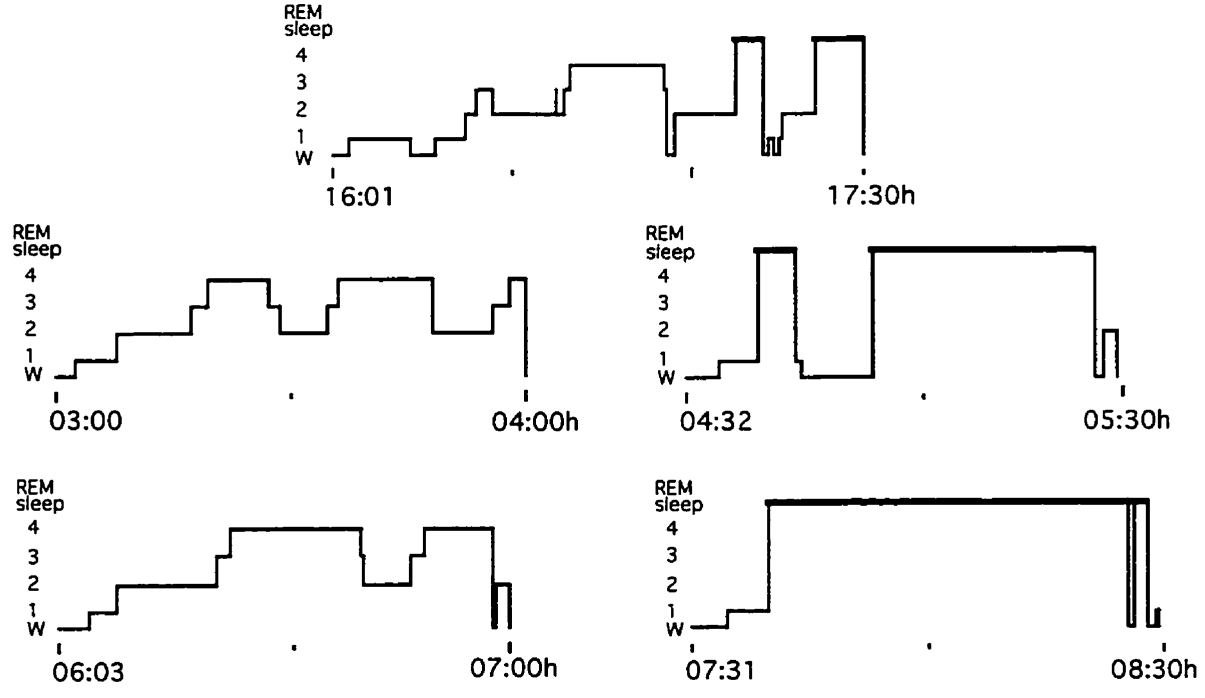
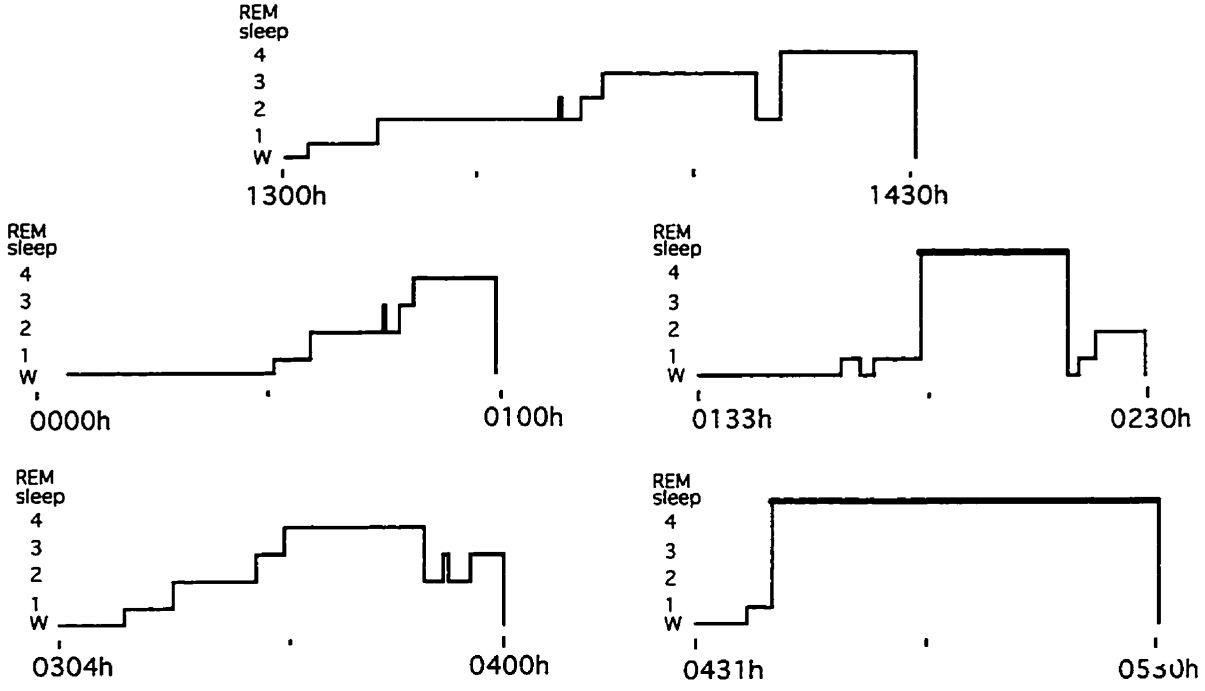


Hypnograms for subject 7:

Protocol A



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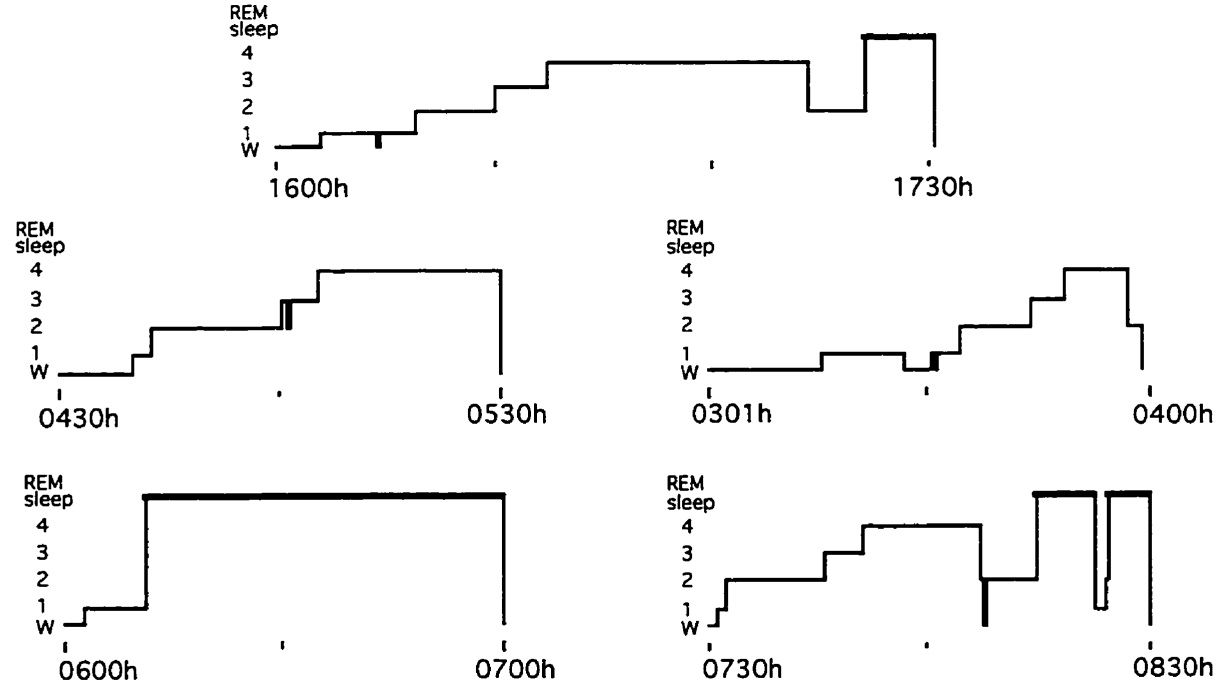


APPENDIX E: Sleep measures for control nights slept with and without an IV catheter in protocols A and B (mean ± s.d

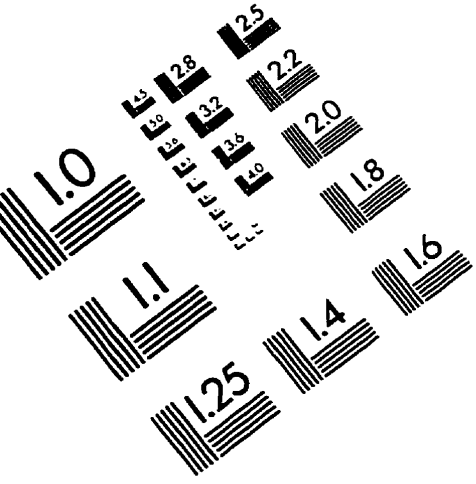
Sleep measures	group of 7 subjects				group of 6 subjects			
	Protocol B	(no IV)	Protocol A	(IV)	Protocol A	(no IV)	Protocol B	(IV)
Time in bed	454.1	(27.8)	451.7	(5.9)	463.6	(19.1)	442.1	(17.1)
Stage changes	69.2	(19.9)	71.3	(18.2)	72.6	(26.1)	68	(15.7)
Sleep period time (min)	435.6	(30.2)	449.2	(5.1)	461.9	(8.5)	430.1	(25.8)
Total sleep time (min)	417	(28.7)	444.17	(7.5)	453.4	(9.2)	416.4	(26.2)
Lat. to sleep onset (min)	18.6	(18.2)	5.03 *	(5.3)	8.5	(8.7)	13.71	(11.2)
Lat. consolidated sl. (min)	29.8	(18.9)	7.36	(8.0)	14.33	(10.6)	22.14	(8.3)
Sleep efficiency (%)	91.34	(3.8)	95.6	(3.6)	91.86	(6.4)	90.6	(6.7)
Wakefulness (min)	20.7	(10.9)	22.2	(9.0)	33.75	(19.9)	27.21	(27.2)
Stage 1 (min)	13.54	(8.7)	12.25	(12.6)	14.9	(5.1)	16.21	(11.3)
Stage 2 (min)	249.35	(24.5)	276	(20.7)	271.75	(38.8)	238.35	(43.8)
Stage 3 (min)	37.8	(19.7)	32.45	(16.8)	34	(29.0)	21	(8.7)
Stage 4 (min)	34.75	(28.6)	32.08	(22.7)	34.5	(26.4)	50.2	(32.5)
SWS (stage 3+4, min)	72.62	(36.3)	64.5	(22.5)	68.5	(16.3)	71.2	(29.3)
REM sleep (min)	78.21	(11.9)	70.5 *	(42.2)	72.4	(29.6)	76.9	(26.8)
Wakefulness/SPT (%)	4.78	(2.6)	4.95	(2.0)	7.35	(4.4)	6.37	(6.6)
Stage 2/TST (%)	57.65	(6.2)	61.45	(4.6)	58.75	(7.8)	55.45	(10.2)
SWS/TST (%)	16.4	(7.6)	14.31	(5.0)	14.8	(3.4)	16.6	(7.1)
REM sleep/TST (%)	17.92	(2.0)	15.67	(9.4)	15.7	(6.4)	17.8	(5.7)
Number of SWS episodes	3.71	(1.4)	4.7	(1.9)	3.8	(2.3)	3.8	(0.9)
Duration of SWS episodes	21.7	(7.5)	15.9	(6.7)	22.8	(10.3)	21.1	(7.8)
SWS latency (min)	41.8	(19.8)	25	(12.6)	25.9	(8.6)	29.4	(7.8)
SWS stability index	79.7	(10.9)	76.3	(11.1)	81.4	(12.3)	83.2	(13.3)
Number of REM sleep episodes	3.85	(0.7)	3.2	(1.5)	4	(1.4)	4	(1.1)
Duration of REM sleep episodes	23	(8.5)	24.1	(15.9)	25.2	(22.7)	32.05	(26.1)
REM latency (min)	87.77	(55.5)	159.1	(93.5)	180.2	(55.9)	144.3	(66.5)
REM sleep stability index	80.1	(9.8)	81.5	(11.2)	78.5	(10.2)	79.6	(12.2)

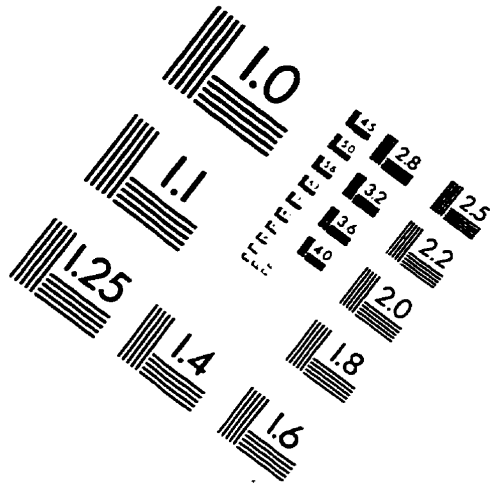
Asterisks represent significant differences ($p < 0.05$) within a group

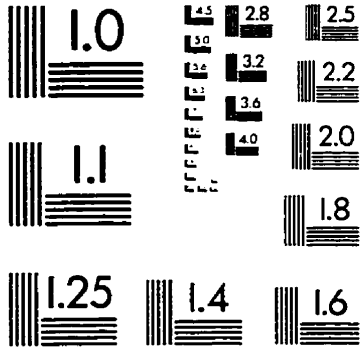
APPENDIX F: Glossary of Abbreviations

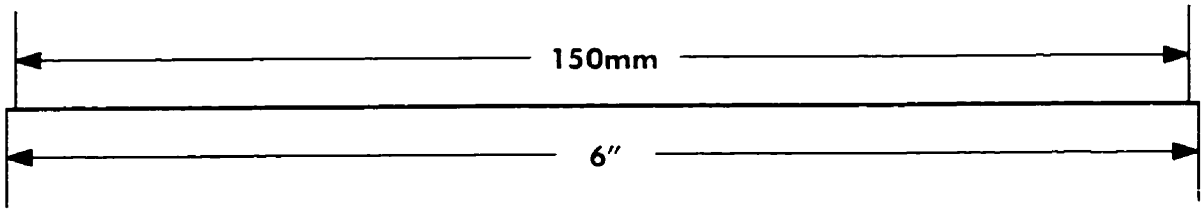
CBS	complex behavior simulator
CPS	cycle per second
DST	descending subtraction task
EEG	electroencephalogram
EMG	electromyogram
EOG	electroocculogram
IV	intravenous line
LRT	logical reasoning task
MAST	memory and search test
MDP	multi-dimensional pursuit
MSLT	multiple sleep latency test
NREM	non-REM
REM	rapid eye movement
SRT	serial reaction time task
SWS	slow wave sleep
TST	total sleep time

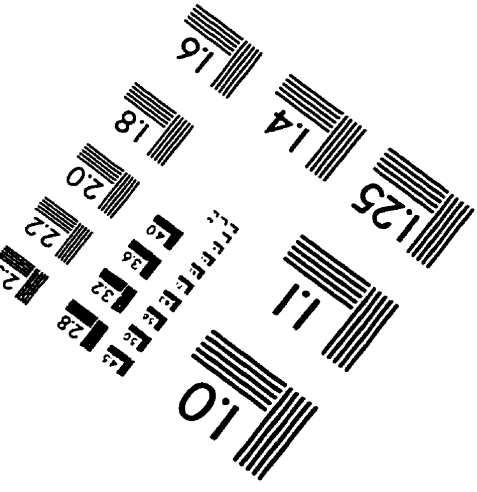
IMAGE EVALUATION
TEST TARGET (QA-3)

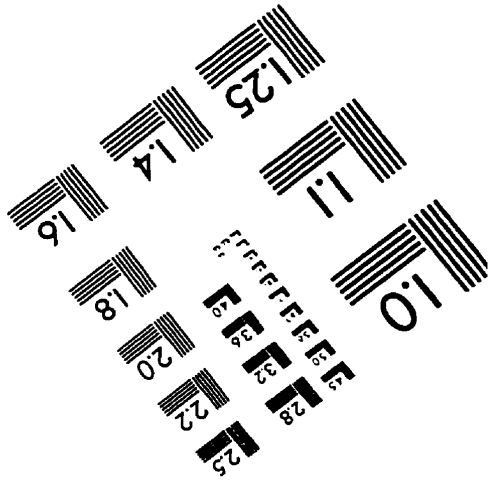












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