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**SCHEDULED NAPPING ON THE NIGHT
SHIFT: CONSEQUENCES FOR THE
PERFORMANCE AND
NEUROPHYSIOLOGICAL ALERTNESS OF
AIR TRAFFIC CONTROLLERS**

T. Leigh Signal

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To my family; Mathew, Ailsa, Peter, Trina, and Kirsty. For always believing in me.

ABSTRACT

Rapid technological change and increasing traffic volumes worldwide are adding to the safety challenges for air traffic control. The night shift has traditionally been a period of low workload and work practices have evolved to reflect this. Thus, despite the exemplary safety record, there is a need to consider further systemic defences for maintaining performance and safety on the night shift. One possible strategy is the provision of a scheduled nap at work. In order to investigate the consequences of a scheduled nap on the night shift, 28 operational air traffic controllers were monitored across four roster cycles. Each roster cycle included one of two night shifts. Air traffic controllers were given a nap opportunity on one night shift of each type, and did not nap on the other. Information on the timing, quantity, and quality of sleep during the work week and days off was collected using actigraphy, and supported with logbook data. Sleep during the nap was measured using polysomnography, and the EEG and EOG were further utilised to determine neurophysiological alertness over the latter part of the night shift. Reaction time performance was measured three times across the night shift (beginning, middle, and end) with the psychomotor vigilance test.

Actigraphy data indicated that the backward, rapidly-rotating work schedule of air traffic controllers resulted in a progressive loss of sleep across the work week. The reduction in sleep lead to an increasing cumulative sleep debt that was at a maximum prior to the night shift. This sleep debt was not related to reaction time performance at the end of the night shift, but was found to influence neurophysiological alertness.

It was determined that the large majority of air traffic controllers were able to sleep during the scheduled 40 minute nap opportunity. However, the latency to sleep onset was long, the sleep short, and of relatively poor quality. Circadian and homeostatic factors increased the likelihood of entry into, and waking from, slow wave sleep (SWS). They were also found to influence reaction time performance and neurophysiological alertness. More variable performance and lowered alertness were seen at the end of the later starting (and finishing) night shift, possibly due to the combined influence of circadian and time-on-task factors. Homeostatic variables had less influence on performance at the end of the night shift, but greater acute sleep loss and higher cumulative sleep debts were related to increased neurophysiological sleepiness.

The short nap sleep had no measurable effect on sleep subsequent to the night shift. However, the amount of sleep obtained in the nap was related to improved reaction time

performance and greater neurophysiological alertness in a dose-dependent manner, with even small amounts of stage 1 sleep effecting a performance improvement. Performance improvement was consistent across a range of reaction time measures and consistent improvements were also evident in the neurophysiological data, with the occurrence of SEMs declining, and lower spectral power evident in all frequency bands and single frequencies.

These findings clearly demonstrate that a minimal quantity of sleep benefits the performance and alertness of air traffic controllers despite the “noise” of a field setting, thus providing a link between laboratory studies of napping and the actual work environment. The findings also fully support management endorsing a 40 minute napping opportunity for air traffic controllers working the night shift.

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CHAPTER 1

INTRODUCTION

As our 24 hour society advances technologically we are faced with a number of challenges. Automation is now ubiquitous in many workplace settings, but perhaps nowhere more so than in aviation and air traffic control (ATC) (Garland & Hopkin, 1994; Molloy & Parasuraman, 1996). The predicted growth in the number of aircraft in the skies will place an additional burden on a system already nearing its maximum capacity. Unprecedented levels of automation are seen as a way to increase the capacity of the system. Therefore air traffic controllers must be prepared for further job changes, such as more monitoring, as the degree of automated assistance increases.

Aviation is also one of the most global of industries and therefore, for reasons of financial necessity and because of societal expectations, is required to provide its services at any time of the day or night. Further, aviation, and by extension air traffic control, is an industry in which safe operations are paramount. Not only are serious accidents financially costly, with a single major accident estimated to cost an airline as much as \$500 million dollars (Lauber & Kayton, 1988), but they also often involve a large loss of human life.

There is widespread acknowledgement that 70-90% of aviation accidents are attributable to human performance errors (Lauber & Kayton, 1988; Nagel, 1988) but there is less recognition that actions or failures to act, which result in an error, fall within the range of typical human performance (Reason, 2000). From a systemic point of view the action or omission that initiates or perpetuates an unsafe event is known as an *active failure*. Given that human functioning occurs along a performance continuum, an industry or organisation must have defences in place to prevent actions at the undesirable end of this continuum from affecting operational performance. When a system feature allows, through its inadequacies or non-existence, a human action to create or exacerbate an unsafe event a *latent failure* is said to occur (Reason, 1997).

A high proportion of the aviation workforce are shiftworkers, who perform many tasks they are not ideally suited for in association with highly automated systems, becoming the unwitting cause of most industry incidents and accidents. This certainly applies in air traffic control. Although the current safety record in air traffic control is exemplary, the combined circumstances of shiftwork, the monitoring nature of the job, and changing

work demands through the worldwide growth in air traffic, suggest a proactive approach should be taken with respect to maintaining or enhancing system safety. One approach is to consider the factors that influence the likelihood of an air traffic controller committing an *active failure*. Multiple elements are likely to be important and might include factors such as; the demands of the task, the operator's level of experience, the working environment, social and domestic issues, and psychological and physiological issues.

The importance, and inter-relatedness, of all these factors is recognised, but to investigate their combined effects would be an impossible task given the infinite number of permutations of levels and combinations of various factors. Normally, in research the opposite occurs. A laboratory based study is conducted during which the conditions of other potentially confounding factors are kept well controlled while the consequence of altering a single variable of interest is studied. Such well-controlled studies are a critical part of understanding the role of a factor which influences an individual's performance and therefore their susceptibility to producing an active failure. However, it is often difficult to generalise the findings from laboratory studies to an operational environment, such as air traffic control. Field studies provide information that bridges this gap, and where interventions are being investigated field studies provide a context that tests the merit of the intervention despite the "noise" of the field setting.

The present study focuses on the physiological challenges created by shiftwork in a complex operational environment. With society's current acceptance of night work, the economic gains engendered, and the extended services provided, shiftwork and night work are a fact of modern life. Furthermore, with the recognition of the biological difficulties of shiftwork, systemic defences need to be investigated that could be used to assist people in countering the performance consequences of shiftwork. One possible defence is the provision of a nap opportunity in the workplace, thus dealing directly with the main physiological reason for shiftwork difficulties.

Although there is much laboratory based research investigating the benefits of napping, there is less focussed on napping during the night hours, and sparser still is the research that has studied this issue in the operational environment. The purpose of the present study was to investigate the efficacy of napping for performance and alertness in an operational air traffic control environment.

This chapter begins by introducing the processes of sleep, the factors that affect propensity for this state, and the physiological mechanisms that underlie and promote our diurnal functioning. This is followed by a discussion of the need for sleep, intimated largely through the behavioural consequences of reducing sleep in both an acute and cumulative manner, and the converse process, that of extending sleep. Particular consideration is given to changes in elementary functioning as well as higher order processes affected by sleep loss. To provide a comprehensive picture of the factors that determine the need for sleep, evidence is also presented on the influence of individual variability.

The subsequent section focuses on the neurophysiological changes seen with prolonged wakefulness, at sleep onset, and in association with diminished performance. This introduces the reasons why performance alters with increasing sleepiness, and proposes what might be seen, and therefore also measured, at a neurophysiological level when an individual experiences sleepiness in a working environment.

To this point, the chapter aims to provide information on when and why a shiftworker, particularly a night worker, might be more prone to an *active failure*. What follows is the consideration of a systemic defence that may provide a means of reducing the neurophysiological and behavioural signs of sleepiness and thereby maintaining operational safety. The contemporary literature addressing the consequences of napping in preparation for, and at work is presented, along with other factors that have been shown to influence the efficacy of napping.

Sleep loss, performance, and alertness are then considered in the context of air traffic control, and the current literature addressing these issues is considered. The chapter concludes with the aims of the present study.

1.1 Sleep Structure

Given that one third of our lives are spent sleeping, its importance and interest is not surprising, but until within the last 75 years, sleep was thought of as a state of inactivity, both physically and neurophysiologically (Dement, 2000). In 1937, utilising the recently developed electroencephalogram (EEG), Loomis and colleagues (1937) determined that distinct changes in the electrical activity of the brain occurred with the onset and maintenance of sleep. The process of monitoring EEG and other physiological signals during sleep is known as polysomnography. Analysis of polysomnographic data have

shown that there are two distinct states of sleep, as different from each other as sleep is from wake. These sleep states are known as rapid eye movement (REM) and non-rapid eye movement (NREM) sleep. REM sleep is known as such due to the rapid eye movements characteristic of this stage. NREM sleep is therefore sleep without the occurrence of such eye movements. NREM sleep is further divided into four arbitrarily defined stages: 1, 2, 3 and 4.

When an individual closes their eyes in an attempt to sleep, electrical activity of the brain in the range of 8-12 Hz predominates. This frequency band is known as alpha (α) and typifies relaxed wakefulness. With the onset of sleep, alpha activity disappears and is replaced by activity in a slightly slower frequency range, that of 4-8 Hz, or theta (θ). The disappearance of alpha and appearance of theta signifies stage 1 of NREM sleep, often considered a transitional stage. Another physiological sign associated with stage 1 sleep is the occurrence of slow, rolling, predominantly horizontal, eye movements (SEMs). These can be recorded by the electrooculogram (EOG) and are normally seen both prior to and during stage 1 sleep.

Stage 2 NREM sleep normally occurs several minutes after stage 1, and is considered by some sleep researchers (Carskadon & Dement, 2000) the true onset of sleep. It is determined by the presence of bursts of activity in the 12-14 Hz range, known as sleep spindles, as well as the presence of K complexes, which are characterised by a clear negative wave followed immediately by positive wave, with a combined duration of greater than 0.5 seconds.

In the normal pattern of sleep, stage 2 is followed by stage 3 and then stage 4 NREM sleep. In both these latter stages even slower electrical brain activity is seen, in the range of 0.5-4 Hz. Such activity is also known as delta (Δ). Delta waves are of large amplitude ($>75 \mu\text{V/cm}$) and the proportion of a period of the EEG containing this type of activity determines whether stage 3 or 4 has been entered, with stage 4 typified by proportionally more delta activity.

Each stage of NREM sleep is also characterised by progressively higher arousal thresholds, resulting in individuals becoming more and more difficult to wake as they move from stage 1 to stage 4 sleep. Because of the higher arousal thresholds in stages 3 and 4 they are also known as deep sleep. Waking from deep sleep has been shown to increase the likelihood of a transitory feeling of hypovigilance and drowsiness (Wilkinson

& Stretton, 1971; Naitoh & Angus, 1989), which is known as sleep inertia (Tassi et al., 1992).

In contrast to the slowing of EEG activity seen in NREM sleep, REM sleep EEG is very much like waking EEG, including low amplitude mixed frequency activity (Rechtschaffen & Kales, 1968). REM sleep is often known as paradoxical sleep, the paradox being a highly active brain coupled with total body atonia. Signals from the brain stem control the large muscle group atonia, which serves to prevent us making movements associated with dreams. Dreaming is a predominant activity of this stage of sleep and the rapid eye movements mentioned previously are thought to be associated with the dreaming process as well.

Within the sleep state ultradian rhythms exist. Each cycle is approximately 90 minutes in length and begins with NREM sleep of progressively deeper stages. After the first period of stage 4 sleep, which is approximately 70 minutes into the sleep period (Carskadon & Dement, 2000), an individual moves quickly back through the lighter stages of sleep into the first period of REM sleep of the night. Although each cycle of NREM/REM sleep is approximately 90 minutes, greater amounts of NREM stages 3 and 4 predominate in the first half of a normal nocturnal sleep. Across each successive cycle the balance between NREM and REM sleep changes with more REM sleep occurring. During a normal night of sleep approximately 25% of the total time asleep is REM sleep, 25% NREM stages 3 and 4, and the remainder stage 2 NREM sleep.

The above description is of a typical young adult. However, with age both the architectural and temporal pattern alters. Sleep in later life is characterised by more awakenings, reduced slow wave sleep (SWS), slow wave activity (SWA), and spindle activity (Landolt, Dijk, Achermann & Borbély, 1996). Such changes in sleep architecture and maintenance can create difficulties for individuals trying to sleep when their body is not physiological primed to do so.

1.2 Sleep Regulation

1.2.1 *The Circadian Pacemaker*

For an understanding of the alternation between sleep and wake, the mechanisms driving this change need to be considered. As humans, we have peaks and troughs in daily functioning across a range of physiological variables, including sleep-wake propensity

(Dijk, 1997). These changes are largely governed by internal physiological mechanisms overseen by a group of neurons located in the anterior hypothalamus and known as the suprachiasmatic nuclei (SCN) (Moore & Eichler, 1972; Stephan & Zucker, 1972). The SCN produces fluctuations with a periodicity of approximately 24 hours. Because the length of the periodic fluctuations approximates a day, the term “circadian clock” is often used when referring to the SCN. The term originates from a combination of the Latin words “circa” meaning about, and “dies” for day (Czeisler & Khalsa, 2000).

The pattern of firing of SCN cells has been shown to be self-sustaining (Klein, Moore & Reppent, 1991) and this site is believed to fill the central role of circadian pacemaker (Czeisler & Khalsa, 2000). One demonstration of this is through the transplantation of cells of the SCN from mutant hamsters to normal hamsters. A mutation in the circadian clock of the hamsters’ results in their pacemakers producing periods of around 20 hours in length, while the normal hamsters keep clock periods of the expected 24 hours. After transplanting cells from the SCN of the mutant hamster to a normal hamster the normal hamster also shows a clock period of 20 hours (Silver, Lesauter, Tresco & Lehman, 1996). These findings also indicate the endogenous nature of the circadian clock.

Under nycthemeral conditions (sleeping at night and being awake during the day) most of the human body’s systems and functions show variations influenced by the circadian pacemaker. These include body temperature, melatonin, cortisol, prolactin, growth hormone, and urine production (Dijk, 1997). Minimum temperature occurs during a normal night’s sleep at a point around 6 hours after normal sleep onset (Van Cauter & Turek, 1995). Core body temperature then rises across an individual’s subjective day, reaching its maximum a few hours before normal sleep onset. In contrast, melatonin levels peak around the time core body temperature reaches a minimum, then continues to fall across most of the normal waking day, rising again in the early evening (Rajaratnam & Arendt, 2001). Because it is not possible to directly monitor the activity of the cells within the human SCN, the change in core body temperature, or variation in plasma melatonin, are the most common markers used to track the progress of the circadian clock across its cycle.

When isolated from external time cues the circadian clock is said to “free-run”, and typically the period of the clock is longer than 24 hours. Such a change implies that under normal conditions there is a means of keeping the circadian pacemaker synchronised with the 24 hour light-dark cycle. This process is known as entrainment.

The primary mechanism of entrainment for the circadian pacemaker is exposure to light (Pittendrigh, 1981). Light information is conveyed from the retina to the SCN via the retino-hypothalamic tract (RHT). Light has a marked effect on the circadian pacemaker and is capable of advancing or delaying its phase (Jewett, Kronauer & Czeisler, 1994) depending on the intensity, duration, and timing of exposure. For an entrained individual, repeated exposure to bright light late in the day or early in the night produces a phase delay. This effectively lengthens each cycle of the circadian clock. Light exposure late at night or early in the day produces the opposite effect, of a phase advance, which has the effect of shortening the clock's period. Light exposure during the middle of the day has little effect on the circadian pacemaker.

Early studies on the circadian clock under conditions free of environmental cues indicated that the average period was about 25 hours. However, their findings are somewhat confounded by study participant's self selected exposure to room light when awake. Originally room light was thought to have no effect on the clock, although more recently this has been shown not to be the case (Boivin, Duffy, Kronauer & Czeisler, 1996). In more recent studies using protocols where individuals were isolated from all time cues and kept in conditions with constant and dim (10-15 lux) room lighting, the intrinsic circadian period is found to be closer to 24 hours, and is on average 24.2 hours (Czeisler et al., 1999).

There is also evidence that there are other external, non-photoc stimuli to which the circadian clock is somewhat sensitive (Turek & Zee, 1999). These include the sleep-wake cycle, physical activity (Van Cauter, Sturis & Byrne, 1993) and social interaction (Aschoff, Fatranská & Giedke, 1971). These additional cues are weaker than the influence of light and together with light are known as zeitgebers, from the German word meaning time givers.

If, due to travel to another time zone, the zeitgebers become displaced relative to an individual's current circadian cycle, the circadian pacemaker can re-entrain the physiological and behavioural variables that are under its control. This results in a shift of the numerous rhythms, returning them to the same phase relationship with the various time cues. Another key property of the circadian system is its ability to ensure each physiological system has a consistent phase relationship with the others (Comperatore & Krueger, 1990).

Often, after travelling to a new time zone people complain of symptoms such as daytime sleepiness, difficulty sleeping, impaired performance, and gastrointestinal complaints. These symptoms are known as jet-lag and are thought to be partly due to the body's rhythms being desynchronised from the environment. This is because the circadian pacemaker adapts to a new time zone in an exponential manner (Rajaratnam & Arendt, 2001). A further factor thought to contribute to jet-lag is the desynchronisation of the various physiological and behavioural rhythms from each other (Arendt, Stone & Skent, 2000), as each system adjusts to the new time zone at a slightly different rate from the others.

Fluctuations in a physiological variable, such as sleep propensity, are not solely the doing of the endogenous circadian pacemaker. The timing of the sleep-wake cycle and other factors such as physical activity, posture and lighting levels also have an effect. One way of teasing out the influence of the circadian clock from these other factors is to keep individuals awake, in one postural position and under constant, relatively dim lighting. This experimental protocol is called a constant routine, and under such conditions the temporal profiles of physiological variables alter, with the circadian influence becoming obvious. The variation in core body temperature differs under constant routine and normal conditions, with the drop early in the evening not being as pronounced under a constant routine, indicating that sleep itself does contribute to the drop in core body temperature seen after sleep onset (Minors, Waterhouse & Åkerstedt, 1994). There are other variables, such as melatonin, whose variation is almost independent of sleep or a constant routine and yet other physiological variables, such as hormones, that are highly sensitive to the sleep-wake cycle. For example, the release of thyroid stimulating hormone (TSH) is inhibited by sleep, otherwise it would peak in the middle of a nighttime sleep episode. Under diurnal conditions it peaks just before sleep onset and is suppressed throughout the sleep episode (Van Cauter & Turek, 1995).

If an individual is placed in isolation from all time cues they may choose to live very long subjective days, with the onset of consecutive main sleep episodes separated by up to 50 hours (Strogatz, 1986). Under such conditions the circadian clock begins to free-run, as evidenced by the body temperature rhythm, with a period close to 24 hours. However, the very long sleep-wake cycle falls outside the range of capture of the circadian pacemaker. As a consequence core body temperature becomes desynchronised from the sleep-wake cycle. This occurrence is known as spontaneous internal desynchronisation.

Alternatively, this can be forced to occur when individuals are placed on a sleep-wake schedule several hours different from a 24 hour cycle, 28 hours for example. Producing this latter effect under experimental conditions is known as the forced desynchrony protocol. Because sleep episodes occur at all circadian phases during the desynchrony protocol it is a means of allowing the investigation of the separate and interactive contribution of the circadian clock and the sleep-wake cycle to the propensity to initiate, maintain, and terminate sleep (Dijk, 1997).

Under the desynchrony protocol the circadian contribution to the drive for sleep (as determined by sleep latency and wakefulness) is evident, with the maximum occurring close to the low point in core body temperature. This circadian-influenced drive for sleep gradually diminishes across the rising limb of core body temperature, reaching a minimum approximately 240 degrees from the temperature nadir (Czeisler & Khalsa, 2000). At this point in time, sleep is rarely initiated and it has been named the evening “wake-maintenance” zone (Dijk & Czeisler, 1995). Not only is the initiation of sleep dependent on circadian phase but so is the duration of sleep episodes, with shorter episodes of sleep and more frequent terminations occurring on the rising limb of the circadian cycle in core body temperature.

1.2.2 *Sleep Homeostat*

The circadian drive for wakefulness does not rise steeply until approximately 6 hours after the nadir in core body temperature. Therefore, spontaneous awakening after a normal night's sleep is likely to occur because of factors other than a circadian initiated wakeup signal. Dijk and Czeisler (1995) suggest it is the amount of prior sleep obtained. This sleep-wake dependent aspect of sleep regulation is known as the sleep homeostat (Borbély & Achermann, 1999). It is responsible for increasing sleep propensity when sleep has been shortened or prevented, and decreasing the propensity for sleep when sufficient sleep has occurred. The homeostatic drive for sleep decreases across a sleep episode with the lowest propensity for sleep at the end of a night sleep episode. It is thought to be greatest at sleep onset and contribute to facilitating sleep over the first half of the night. As the homeostatic drive declines in the latter half of the night the circadian drive for sleep becomes greatest, thus the combined influence of both circadian and homeostatic influences maintain sleep across the entire night (Czeisler & Khalsa, 2000).

1.2.3 *Models of Circadian and Homeostatic Influences on Sleep Regulation and Structure*

Theoretical and mathematical models have been developed to further conceptualise the factors influencing sleep regulation. One of the major models is the two-process model, which was later extended and modified to the three-process model (Åkerstedt & Folkard, 1995). The two main processes are Process S, which represents the homeostatic drive for sleep, and Process C, representing the circadian influence. The third process is named Process W and represents sleep inertia on waking. This model has been used to account for phenomena such as the recovery from sleep deprivation and sleep under shiftwork conditions (Borbély & Achermann, 1999).

The temporal change in process S was initially derived from slow wave activity (0.75-4.5 Hz) in the EEG (Borbély & Achermann, 1992), and consequently the duration and quantity of SWS and SWA are seen as predictive markers of the homeostatic drive (Borbély & Achermann, 1999). The sleep homeostat declines exponentially during NREM sleep, while during waking it increases in a saturating exponential function. Although night time sleep is maintained initially through the influence of the homeostatic drive and then by the circadian process, daytime wakefulness is maintained through the circadian influence countering the increasing homeostatic drive for sleep (Czeisler & Khalsa, 2000).

Both the homeostatic and circadian processes also influence the structure, or architecture, of sleep. The duration and occurrence of SWS is determined predominantly by homeostatic factors, while circadian phase has a greater influence over the occurrence of REM sleep. Studies in which short episodes of sleep (1.5-3 hours) were allowed across the circadian cycle without variations in prior wake show that REM sleep latency, accumulation, episode duration, and propensity all varied with the phase of the core body temperature cycle (Dijk, Beersma & Daan, 1987).

The term sleep homeostasis implies that loss must be recovered by sleep of either increased duration or intensity (Dijk, 1997). During recovery from total sleep deprivation SWS (and SWA) show an immediate rebound while an increase in REM sleep is delayed (Borbély & Achermann, 1999). The amount of sleep lost is never fully regained (Åkerstedt & Gillberg, 1986) and even in studies where the opportunity for recovery sleep has been unlimited only up to 60% of the time lost is recovered (Dijk, 1997). When an individual obtains more than 4 hours sleep but less than their normal

amount, all sleep stages other than stages 3 and 4 are abridged. Once the duration of sleep is reduced to less than 4 hours, stages 3 and 4 decrease as well (Carskadon & Roth, 1991).

Recovery from sleep loss also shows predictable changes in REM sleep. If the recovery sleep is long enough, REM sleep is also lengthened (Dement & Greenberg, 1966; Webb & Agnew, 1975a; Carskadon & Dement, 1981). Because SWS is initially predominant when recovering from sleep loss, it is proposed that two full nights of unrestricted sleep are required for recovery, with the second night allowing REM sleep to also be recouped (Dijk et al., 1987).

It has been demonstrated on numerous occasions that human sleep shows a circasemidian (bimodal) rhythm, with a mid-afternoon increase in sleep propensity. This is thought to be related to an approximate 12 hour bimodal rhythm in SWS propensity (Broughton, 1989), with peaks occurring during the night and about 12 hours later. Evidence for this increase in sleep propensity has been shown by the frequency with which adults nap at this time (Evans & Orne, 1975), the occurrence of the siesta in certain cultures (Broughton, 1983), and the increase in objective sleepiness as measured by the Multiple Sleep Latency Test (MSLT)¹ (Carskadon & Dement, 1992).

This circasemidian tendency for sleep is often not seen in forced desynchrony protocols because napping is not allowed. However, Zulley and Campbell (1985) showed that under forced desynchrony protocols where individuals' disregarded the instructions not to nap, or were allowed to nap, sleep is split into two periods, with the major sleep episode occurring at the core body temperature nadir and a shorter sleep occurring 12 hours later.

Age related changes in sleep architecture and duration have been mentioned, which include an increased number of awakenings, reduced SWS, SWA, and spindle activity (Landolt et al., 1996). The cause of these changes is currently unknown, but is possibly due either to alterations in the EEG generating mechanisms or the homeostatic drive for sleep. Dijk (2000) has demonstrated that the homeostatic drive is still functioning in older individuals but possibly to a reduced extent. In forced desynchrony protocols older subjects have a core body temperature minimum earlier than their younger

¹ The MSLT is a standardised and validated test which measures the latency to polysomnographically determined sleep onset under soporific conditions.

counterparts and show lower amplitude rhythms, although the circadian period has not been found to differ. The reduced homeostatic drive is thought to interact with the lower amplitude circadian system (Dijk, Duffy, Riel, Shanahan & Czeisler, 1999) to produce changes in the sleep-wake cycle.

1.2.4 *Consequences for Shiftworkers*

Due to societal factors human beings can, and do, override signals from the circadian and sleep-wake system. The most obvious example of self-determined sleep-wake schedules is shiftwork. Shiftwork in its broadest sense is work that is scheduled outside normal daytime hours and therefore requires sleep to be displaced from its normal night time location. Because of the above mentioned characteristics of the circadian and sleep-wake system, particular problems are created for shiftworkers.

Shiftworkers often report similar symptoms to those seen with jet-lag and for similar reasons. When a shiftworker changes to a new shift schedule, such as working at night and sleeping during the day, many of the non-photic zeitgebers encourage the circadian pacemaker to shift to the new pattern of work and rest. During the process of attempted re-entrainment, the body's systems are desynchronised from these time cues and each other, like after travel to a new time zone.

For a shiftworker there is a further complication. The change in the pattern of work and rest creates conflicting cues for the circadian pacemaker, which attempts to adapt to the new pattern of activity and sleep, but is constantly drawn back to its diurnal orientation by the strength of the light zeitgeber. The result is incomplete adaptation to the new work pattern. Gander et al. (1998a) demonstrated this in overnight cargo pilots flying domestic routes. The pilots crossed no more than one time zone in 24 hours and normally worked either three or five night shifts in a row. It would be expected that if the pilots completely adapted to their night schedules then the nadir in core body temperature would shift by 12 hours. However, the study findings indicated that on average the low point in core body temperature was only delayed by 2.8 hours. In addition, frequent changes in the pattern of work and rest are thought to produce persistent desynchronisation of the circadian pacemaker from the 24 hour light-dark cycle resulting in the shiftworker constantly experiencing jet-lag like symptoms (Gander et al., 1998a).

The circadian clock also acts to shorten the sleep of the shiftworker, particularly the night worker, who would normally attempt their main sleep episode after finishing work (Åkerstedt, 1991) and on the rising limb of core body temperature. The high homeostatic drive for sleep might assist in initiating sleep but the circadian system makes maintaining sleep difficult. Irrespective of the length of prior sleep the peak in the circadian wake signal occurs approximately 6 hours after the low point in core body temperature resulting in reduced sleep duration (Gander, Rosekind & Gregory, 1998b). In the study of overnight cargo pilots (Gander et al., 1998a), it was shown that regardless of the time of sleep onset after the night shift, the pilots woke on average 6.07 hours after their nadir in core body temperature. Other disturbances such as noise (Knauth & Rutenfranz, 1972), lighting levels, social and domestic commitments may also play a role in reducing daytime sleep. The consequence is that the duration of sleep after a night shift is approximately two thirds of a normal night's sleep (Åkerstedt, 1998).

1.3 Sleep Need

The term sleep "loss" intimates the existence of an optimal quantity of sleep (Carskadon & Roth, 1991). However, this logic is not universally accepted (Carskadon & Roth, 1991; Ferrara & Gennaro, 2001) and within the literature there are currently two diametrically different points of view regarding how much sleep is optimal. One school of thought is that a large portion of society is chronically sleep restricted (Webb & Agnew, 1975b; Bonnet & Arand, 1995). This is partly based on findings that young adults, when allowed to sleep *ad libitum*, obtain on average an additional 2 hours sleep per night, increasing their total sleep to 9-10 hours (Webb & Agnew, 1975b). Further, historical evidence from 1910 suggests young adults slept on average 9 hours per night compared to the 7-8 hours obtained by their present day contemporaries. Bonnet and Arand (1995) point to the range of studies that have found negative effects on performance and subjective sleepiness in association with sleep restriction.

The alternative point of view is that humans only require a core amount of sleep for normal functioning that is approximately 4.5-6 hours, or 3-4 cycles of NREM/REM sleep per night (Horne, 1991). The reasoning for this stems from findings that individuals can adjust to functioning on such quantities of sleep. In addition, when sleep duration is extended beyond the average 7-8 hours, alertness and performance do not improve (Harrison & Horne, 1995). Both Carskadon and Roth (1991) and more recently

Ferrara and De Genarro (2001) state that it is more likely that the actual amount of sleep required lies somewhere between these two extreme viewpoints.

Ferrara and De Genarro (2001) refer to this ideal amount of sleep as the “daily amount of sleep that allows a subject to be fully awake (i.e. not sleepy) and able to sustain normal levels of performance during the daytime” (pp 156). They further state that when a short or long term change in the actual amount of sleep obtained results in less sleep than an individual’s ideal daily sleep duration, a sleep debt is said to occur and accrue.

These concepts have potential implications for shiftworkers because, as discussed previously, their pattern of work and rest often prevents them getting their normal daily sleep need. At some points in a roster cycle, such as preceding the first night shift, a shiftworker may also forgo sleep completely, thereby subjecting themselves to the effects of complete sleep loss (Knauth et al., 1980). However, the degree of total sleep deprivation that they are likely to experience generally does not exceed 24 hours (Åkerstedt, 1991).

The following discussion considers the consequences of acute sleep loss, short term and long term sleep restriction. In section 1.2.3 above, the consequences of sleep loss on the architecture of sleep were discussed. However, there are also repercussions for subjective and objective sleepiness, and performance. In addition, the ramifications of sleeping longer than normal provide an insight into how much sleep an individual possibly requires.

1.3.1 Effect of Acute Sleep Loss or Restriction

Sleep Loss in Laboratory-Based Tasks

In laboratory-based studies, a number of consistent changes in performance have been identified with increasing sleep loss. One of the most profound was evident in a classic study by Williams, Lubin and Goodnow (1959) who studied 40 male participants during 72 or 98 hours of sleep loss. It was noticed that with increasing sleep loss the variability between the fastest and slowest responses on a visual reaction time task increased markedly, with the duration of the longest responses increasing while the fastest responses remained close to those seen during baseline performance. Not only did the duration of the longest responses increase but also their frequency, resulting in progressively more uneven performance. The finding of increased variability in

responding with greater sleep loss has been replicated on numerous subsequent occasions (Angus & Heslegrave, 1985; Bonnet, 1986; Dinges, Orne, Whitehouse & Orne, 1987).

In instances where individuals are able to control the speed at which they complete tasks, sleep loss results in fewer completed tasks, as each takes longer, but without a concomitant increase in error rates. This is thought to be due to general cognitive slowing and a change in strategy from speed to accuracy. Such a shift can be seen in arithmetic tasks (Dinges & Powell, 1988), logical reasoning (Angus & Heslegrave, 1985), and tracking (Mullaney, Kripke, Fleck & Johnson, 1983). A lack of appreciation of this change in strategy provides an explanation for some of the discrepant findings seen in the sleep loss and performance literature (Dinges & Kribbs, 1991).

It had been suggested that long tasks (30-60 minutes) are necessary in order to sensitively detect the effects of sleep loss on performance (Wilkinson, 1968). However, when a change in performance across the duration of a task is the focus of investigation, the effects of sleep loss can be seen as a decline in speed of responding. This time-on-task effect is noticeable in a task of 10 minutes in length after a night without sleep (Lisper & Kjellberg, 1972). Further, if the performance tasks are embedded within a high workload situation, then very short duration tasks (1 minute) can be used to sensitively detect the effects of sleep loss after only 18 hours of sleep loss (Heslegrave & Angus, 1985). If a performance task is not located within a high workload context, then the longer the task, the greater the likelihood that the effects of sleep deprivation on performance will become apparent.

Another general effect seen with sleep-deprived performance is a slowing of the fastest responses. This has been termed the “optimum response shift” (Dinges, 1992). Dinges and Powell (1989) showed that across a period of continuous sleep deprivation, the fastest 10% of responses on a visual reaction time task could be seen to decline during the first 24 hours. This effect is not as marked as the changes seen at the slower end of the responding continuum.

Changes in Cognitive and Real-World Performance with Sleep Loss

It is contentious as to how well these performance changes seen on laboratory tasks relate to “real world”, or more cognitively complex, performance. Generally the more cognitively complex a task is the greater the intrinsic level of interest. This is thought to

encourage individuals to compensate for the effects of sleep loss (Harrison & Horne, 2000) therefore it is not surprising that a current prevailing view in the literature is that sleep loss has little or no effect on high level complex tasks.

Cognitive skills are thought to be reliant on the functioning of the prefrontal region of the cerebral cortex and recent evidence has shown that this area of the brain is affected by as little as one night of sleep loss (Petiau et al., 1998; Drummond et al., 1999; Drummond et al., 2000; Thomas et al., 2000). A complicated task in the real world, such as decision-making, is likely to involve a range of cognitive skills. In a recent review, Harrison and Horne (2000) discuss evidence that suggests a raft of skills necessary for decision making are adversely affected by sleep loss. The skills that are affected include: attending to complex information while filtering out distractions, following a situation and recognising the need to apply new strategies, lateral thinking and innovation, risk assessment, maintaining interest, controlling mood and behaviour, the ability to self monitor performance, and the ability to communicate effectively (Harrison & Horne, 2000).

In a workplace context these types of skills are frequently at a premium in non-routine or emergency situations, where time pressure is also extreme. Nevertheless, some are also integral to maintaining workplace safety under normal conditions or situations where conditions are becoming less safe. For example, there is some evidence to suggest that the ability to assess risk degrades with increasing sleep loss, and that individuals become less concerned with negative consequences (Harrison & Horne, 1998). Further, motivation appears to maintain performance at pre-sleep deprived levels if wakefulness does not exceed 24 hours or sleep is not restricted to less than 50% of the normal daily amount (Dinges & Kribbs, 1991). Such findings indicate that the relationship between sleep loss, performance and motivation is not a simple one, with motivation masking the effects of sleep loss on performance but both motivation and performance gradually being diminished by increasing sleep deprivation.

It has been reported for many years that mood changes occur with sleep deprivation. The reduced ability to control mood and behaviour is reflected in the reports of increased irritability, impatience, reduced social inhibitions, inappropriate interpersonal behaviour and childlike humour (Horne, 1993). It has been shown that mood is more negatively affected when the tasks being performed are more demanding and complex (Angus, Heslegrave & Myles, 1995). Like mood, another aspect of complex performance

that is also important in inter-personal interaction is communication. Changes in communication have been recorded with sleep loss, particularly a decreased ability to clearly articulate after one night of sleep loss (Harrison & Horne, 1997). Angus and Heslegrave (1985) found participants became slower at responding to incoming messages after one night without sleep. Schein (1957) required pairs of individuals to communicate spatial information to each other, with one person verbalising the details to the other person who was then required to plot the information. After approximately 55 hours without sleep participants began to have difficulty both verbalising and interpreting the messages. Sleep deprivation appears to affect communication by reducing naturalness of speech, the decoding of word meanings, and the clarity of articulation (Harrison & Horne, 2000).

Because of the relatively autonomy under which many complex tasks are performed, an individual's ability to monitor their own performance is also often central to preserving safety margins. Discrepant findings have been reported in the ability of individuals to self-monitor their performance with increasing sleep loss. Baranski et al. (1994) found no impairment in the ability of individuals to detect changes in their cognitive performance across 3 days sleep loss. However, Harrison and Horne (2000) noticed that sleep deprivation lead to increased confidence on ambiguous tasks.

The effect of sleep loss on either simple or complex performance does not work in isolation. The circadian system also has an influence on performance across each 24 hour period, with the overall mean on a task across consecutive 24 hour periods decreasing with increasing sleep loss. These two effects combine to produce the poorest performance at the circadian nadir (Van Dongen & Dinges, 2000). For individuals working on the night shift this has important ramifications, and changes in workplace performance have been linked to these effects. For example, in a classic study by Bjerner et al. (1955) a peak in errors reading a gas meter occurred at 0300 hours on the night shift. In addition, the speed with which telephone operators connect calls (Brown, 1949) and the speed with which industrial workers joined threads (Wojtczak-Jaroszowa & Pawlowska-Skyba, 1967) reached its slowest around 0400 hours. Locomotive engineers were also found to be most likely to fail to cancel an alerting device when working the night shift, resulting in a peak in automatic brakings at 0300 hours (Hildebrandt, Rohmert & Rutenfraz, 1974).

In a workplace situation the effects of sleep loss are often not possible to distinguish from those produced by the circadian system and these combined effects are also reflected in safety related incidents and accidents (Åkerstedt, 1991). When controlling for the level of activity across a 24 hour period, a peak in single vehicle car accidents (Harris, 1977; Hamelin, 1987; Åkerstedt, Kecklund & Hörte, 2001) and military flight accidents (Ribak et al., 1983) was found to occur at the circadian nadir. Several major industrial disasters have also been linked to the combined effects of sleep loss and the circadian nadir. Examples include the nuclear power plant meltdown at Chernobyl and the launch and subsequent explosion of the NASA space shuttle Challenger.

Changes in Sleepiness and Performance with Short Term Sleep Restriction

It is not only total sleep loss that has consequences. Evidence suggests that sleep restriction also has ramifications for performance and alertness. Investigations measuring objective changes in sleepiness with greater levels of sleep restriction on a single night have found linear decreases in sleep latency on the MSLT with increasingly limited sleep (Rosenthal, Roehrs, Rosen & Roth, 1993; Gillberg & Åkerstedt, 1994; Devoto, Lucide, Volani & Bertini, 1999). Rosenthal et al. (1993) noted that curtailing one night of sleep by as little as 2 hours shortened MSLT latencies between 2.2 and 7 minutes. Harma et al. (1998) found a different relationship between sleep restriction and latency to sleep as measured by the Maintenance of Wakefulness Test (MWT). They reduced sleep to 4, 2 or 0 hours and contrasted it to 8 hours of sleep and noted an exponential decaying function between the amount of sleep obtained and sleep onset latency (SOL) in the MWT. The nature of the relationship could in part be attributed to the lack of statistical difference in SOL between the 8 and 4 hour conditions. It has however, been argued that the MWT lacks sensitivity to moderate and acute sleep loss (Porcu, Bellatreccia, Ferrara & Casagrande, 1997), and is prone to a ceiling effect when latencies reach the maximum of 20 minutes, such as in the 8 hour condition in the study by Harma et al. (1998).

Taub and Berger (1973) restricted the sleep of 10 individuals who normally slept 7-8 hours to 5 hours for one night by delaying sleep onset. They found a decrease in accuracy and speed on a vigilance task, mood to be more negative, but no change in the performance outcomes on an addition task after sleep restriction. A classic study by Wilkinson, Edwards and Haines (1966) investigated the effects of two nights of complete sleep loss or restriction to 5, 3, 2, or 1 hour of sleep, compared to a baseline amount of

7.5 hours. Their findings indicated that after one night, performance on a vigilance task was affected by sleep restriction when 3 hours or less of sleep were obtained, while an addition task was affected only when 2 hours or less of sleep were allowed. Importantly, the effects were exacerbated with two days versus one of sleep loss or restriction, indicating the cumulative nature of sleep loss.

1.3.2 *Effect of Cumulative (More Than One Night) of Sleep Restriction*

This leads to a discussion of the effects of cumulative sleep loss. For a shiftworker complete sleep loss may occur occasionally, such as in association with the first night shift, but sleep restriction across consecutive days is a more likely scenario. As the findings of Wilkinson, Edwards and Haines (1966) indicate, when sleep is restricted for more than one night the effects accumulate. This is further supported by the finding of progressively shorter latencies to sleep on the MSLT for 12 individuals whose nightly sleep was reduced to 4 hours for two successive nights (Carskadon & Dement, 1982). An additional study by Tilley and Wilkinson (1984) reduced the sleep of 8 young women to 4 hours per night for two nights by either restricting it to the 1st or 2nd half of the night. They reported a cumulative slowing of simple reaction time performance and no difference between the different sleep restriction protocols. This adds weight to the argument that the changes are due to sleep loss *per se* rather than an alteration in timing of the sleep-wake schedule.

In longer protocols, Carskadon and Dement (1981) focused on individuals who habitually slept for 9 hours and reduced their sleep to 5 hours for seven nights. Results showed a cumulative linear decline in MSLT latencies, which continued to decrease across the entire protocol. This is in contrast to subjective sleepiness ratings, as measured by the Stanford Sleepiness Scale (SSS), which plateaued after the 4th day of sleep restriction. The difference between objective and subjective sleepiness may indicate individuals are less able to judge the consequences of sleep restriction when the effects accumulate. The findings from this study must also be considered with care as they may be in part due to the selection of habitually long sleepers.

In probably the most well controlled study to date, 16 individuals had their nightly sleep reduced to approximately 5 hours per night for seven nights (Dinges et al., 1997). Cumulative, linear changes were seen in subjective sleepiness, as measured by the SSS, objective sleepiness recorded by the MSLT, and neurobehavioural performance using the

psychomotor vigilance task (PVT). Subjective sleepiness was seen to immediately increase with sleep restriction and then level off between the 2nd and 5th day of the protocol. Performance did not change significantly until the 2nd day and also plateaued, but between the 2nd and 6th days. A further continued decline in both subjective sleepiness and performance was seen in the last days of the study and was shown to continue for 8 individuals who participated in an 8th day of sleep restriction. The authors suggest this pattern of change indicates a step like increase in sleepiness with the continued decline in the latter part of the experiment indicating that an asymptote had not been reached. The change in performance across this protocol was seen to match the change in MSLT scores found in Carskadon and Dement's (1981) study.

Several other studies have reduced sleep to between 4 and 6 hours per night for up to eight nights (Webb & Agnew, 1965; Hamilton, Wilkinson, & Edwards, 1972; Herscovitch & Broughton, 1981a, 1981b; Blagrove, Alexander & Horne, 1995). These studies have generally produced inconsistent findings, but practice effects and other methodological weaknesses are confounds in many.

Sleep restriction has also been studied across prolonged periods of time (Webb & Agnew, 1974; Blagrove et al., 1995; Dinges et al., 1999). The findings from the studies by Webb and Agnew (1974), and Blagrove (1995) showed little or no decrease in performance across the protocol, although participants did report increased subjective sleepiness. Unfortunately in these studies, widely spaced measures, lack of information on the timing of sleep during the study, and practice effects create potential confounds.

Dinges et al. (1999) avoided many of these methodological problems in a study of individuals with stable sleep habits (6.6-8.6 hours of sleep per night) sleeping either 4, 6, or 8 hours per night for 14 nights. Those in the 4 and 6 hour categories showed a linear increase in the frequency and duration of lapses on the PVT, a decreased number of correct substitutions on the Digit Symbol Substitution test, and poorer performance overall on a serial addition-subtraction task. The most rapid change was seen in those sleeping 4 hours per night, with their performance by the 13th day being similar to that seen after two nights of total sleep loss. Individuals sleeping 6 hours per night showed less severe effects but by the 5th day were performing as if they had one night of total sleep loss. In a quarter of the 6 hour group and half of the 4 hour group uncontrolled periods of sleep were reported by the 6th day, with this occurrence peaking in the last 2

days. Contrary to the performance findings, ratings of subjective sleepiness and mood did not differ with reduced sleep.

This discrepancy between objective and subjective sleepiness adds to the argument that individuals cannot accurately gauge the consequences of cumulative sleep loss. On the whole this well controlled study, which frequently assessed performance, indicates the marked cumulative effects of continued sleep restriction. One concern with this study is the inclusion of individuals habitually sleeping only 6.6 hours per night. They may have been chronically sleep deprived and further sleep restriction could have much stronger effects than for someone naturally sleeping for this length of time (Ferrara & Gennaro, 2001) or alternatively they were naturally short sleepers and further sleep restriction is then likely to have less of an effect.

The results from these above mentioned studies focussing on cumulative and long term changes in sleepiness, are difficult to compare given the various experimental paradigms utilised. In addition, a large majority reported practice effects, testing sessions were often widely spaced, and many studies only had a few participants, and largely relied on the participants' honesty and reliability in maintaining the experimental protocol.

On the whole many studies report a discrepancy between subjective and objective evidence of sleepiness, with most of these noting few performance changes but a marked increase in subjective sleepiness. However, those studies with fewer methodological problems tend to report the opposite, with a lower sensitivity of the subjective ratings to sleepiness than the performance changes indicate. As for performance changes, those studies reporting few or no effects of sleep reduction are weakened by methodological problems and therefore their findings are questionable. When performance is frequently measured, using measures free of practice effects, and the amount of sleep participants are getting is well controlled, the results indicate a cumulative reduction in performance with successive days of sleep restriction (Dinges et al., 1997; Dinges et al., 1999).

This is supported by a meta-analysis of studies whose protocols included a reduction of sleep to 5 hours per night (Pilcher & Huffcutt, 1996). Their findings are somewhat surprising and point towards cumulative partial sleep loss producing more negative effects than periods of either short term (45 hours) or long term (>45 hours) total sleep loss. This goes against the linear decline in performance and sleepiness seen in other studies with increasing amounts of sleep restriction. On average, across all measures,

partial sleep loss scores were two standard deviations below those without sleep loss, while total sleep loss scores were only one standard deviation below. Their meta-analysis pointed towards motor performance being the least affected by partial sleep loss while cognitive performance and mood were more severely affected by sleep restriction than total sleep loss. These authors also point towards simple, long, cognitive tasks (10 minutes versus 6 minutes) being the most prone to sleep loss. This meta-analysis is noteworthy partly because it included studies measuring changes in actual work tasks and the inclusion of these tasks may have contributed to the unexpected differences between partial and total sleep loss.

At this point, there is sufficient evidence to indicate that a 2 hour reduction in sleep for one night impairs alertness (Carskadon & Roth, 1991) and that if sleep reduction continues, the effects on performance, and subjective and objective sleepiness accumulate (Ferrara & Gennaro, 2001). These findings seem to counter Horne's (1991) argument of the requirement for approximately 4.5-6 hours of sleep and although the findings of these studies provide an insight into the consequences of losing sleep, the idea of how much sleep is sufficient sleep is still not clear.

One way of looking at this concept is to consider how much sleep people normally get. A recent questionnaire study of 7000 New Zealand adults reported the average sleep amount to be 7.5 hours per 24 hour period (R. Harris, personal communication, December 2001). This is similar to the amounts reported in studies of other populations (Carskadon & Roth, 1991). Although it is possible that habitual sleep is not sufficient sleep, because in the study of New Zealanders 37% reported they rarely or never got enough sleep and 46% indicated that they rarely or never woke feeling refreshed (R. Harris, personal communication, December 2001). Horne (1991) provides a partial counter argument to these findings, suggesting that the process of moving from sleep to wake is a gradual one. Therefore, it is unlikely an individual would normally wake feeling immediately refreshed.

1.3.3 *Sleep Extension*

A further consideration in the search for the optimum quantity of sleep is to consider the effects of extending sleep beyond its normal duration. It has been noted that humans can extend their normal sleep amount by as much 3.5-6 hours with relative ease and without a reduction in the amount of sleep obtained on the following night (Feinberg,

Fein & Floyd, 1980; Gagnon, De Koninck & Broughton, 1985). However, sleeping for 10 hours or more has been reported to be something that not everyone can easily do (Volani, Devoto & Lucidi, 1996), even when they say they can (Webb, 1986).

Even if an individual can sleep for longer there are conflicting results regarding the consequences for daytime functioning. Detrimental effects for vigilance, complex motor performance (Taub, Globus, Phoebus & Drury, 1971), reaction time performance (Taub & Berger, 1973; Taub, 1980) and short term memory and subjective sleepiness (Taub, 1980) have been noted.

In contrast, a number of studies investigating the effect of extending sleep duration and/or time in bed (TIB) of individuals to 10 hours have found some positive effects. Increased latency to sleep onset as measured by the MSLT has been noted (Carskadon & Dement, 1982; Roehrs, Timms, Zwyghuizen-Doorenbos & Roth, 1989; Harrison & Horne, 1996b; Roehrs, Shore, Papineau, Rosenthal & Roth, 1996) but the increase in latency in all instances was relatively small. Improved reaction time performance has also been found (Roehrs et al., 1989; Harrison & Horne, 1996b) but improvement on a tracking task (Roehrs et al., 1989), mood, self-rated sleepiness or ability to detect target tones (Harrison & Horne, 1996b) were not seen.

On the whole these sleep extension studies do not tend to support the value of lengthening sleep by 2-3 hours. The only consistent change is a small increase in sleep latency as measured by the MSLT, with some positive effects for performance. The ecological validity of the small increases in SOL and performance is questionable given the greatly increased TIB required.

1.3.4 *Individual Differences*

There is one final concept that requires consideration in relation to this area of “optimal sleep”, and that is the impact of individual variability. Like many physiological characteristics, the amount of sleep people require shows a Gaussian distribution (Ferrara & Gennaro, 2001), and although the average is between 7 and 8 hours, the spread of 95% of reported values is between 5 and 10 hours (R. Harris, personal communication, December 2001). Certain individuals have been defined as naturally short, and others as naturally long, sleepers (Hartman, Baekeland, Zwillling & Hoy, 1971). On the whole women have been documented to obtain more, and have an increased need for sleep (Jean-Louis, Kripke, Ancoli-Israel, Klauber & Sepulveda, 2000). Individuals also vary in

the speed with which they fall asleep. Lavie and Zuluni (1992) identified individuals with very short daytime sleep latencies (< 6 minutes) but no complaint of daytime sleepiness. Changes in sleep quantity and architecture with age have also been mentioned previously (Landolt et al., 1996).

The response of individuals to total sleep loss has been shown to produce substantial differences between individuals. However, performance on a psychomotor vigilance task is remarkably similar within study participants on different occasions of exposure to sleep loss (Doran, Van Dongen & Dinges, 2001). Fifty eight percent of the variance in performance could be attributed to this individual specific variability.

Van Dongen and Dinges (2000) state that a tendency for either morningness or eveningness (i.e. to go to bed early and rise early or alternatively go to bed late and rise late) is the most predominant source of circadian related individual variation. Individuals at either end of this morning/evening continuum are thought to differ in the phase of their circadian clock, which is in turn reflected in neurobehavioural functioning, with some people performing consistently better in the morning and others in the evening (Van Dongen & Dinges, 2000). Morning type individuals have been shown to get less total sleep and have greater levels of alertness on waking (Carrier, Monk, Buysse & Kupfer, 1997; Taillard, Philip & Bioulac, 1999). In comparison, evening type individuals don't rate themselves as sleepier when they get less than their normal daily sleep requirement (Taillard et al., 1999) and indications are that they are more flexible in their sleep-wake cycle possibly allowing them to adapt more easily to shiftwork (Åkerstedt & Torsvall, 1981).

1.3.5 *Summary of Sleep Need*

There are consistent, negative effects of sleep loss on laboratory-based performance including increased variability in responding, cognitive slowing, time-on-task decrements and an optimum response shift. It is also reported that more complex, cognitively based skills are adversely affected, although this area is still contentious because of conflicting findings. Despite the inconsistencies in the literature on changes in complex cognitive performance with sleep loss, the robust finding of diminished vigilance and attention is critical, as there is unlikely to be any cognitive or psychomotor task that does not require this as a fundamental component (Dinges, 1992).

The consistent changes seen on simple vigilance and reaction time tasks indicate them to be sensitive probes of the functioning of the central nervous system (CNS). This is particularly so, as the findings also serve to provide a functional meaning to the physiological changes that occur (Dinges & Kribbs, 1991). Utilising well established, appropriate performance measures is also critical in order to see the effects, as well as providing a link between laboratory findings and those seen under field settings.

Although there certainly seems to be sufficient evidence for the negative, and cumulative effects of reducing sleep below the habitual daily amount, the presently available literature does not indicate the need for a greatly increased quantity of sleep. Bonnet and Arand (1995) suggest that the beneficial effects of sleep accrue in a logarithmic manner with large benefits gained from a few hours of sleep and a progressively fewer positive effects seen with increasing quantities of sleep. This position is further supported by Jewett's (1999a) study showing that the pattern of performance improvement with recovery sleep follows a saturating exponential function, and further explains why sleep extension has few positive effects.

On this basis it is likely that for the average individual 7-8 hours of sleep per night is sufficient for normal daytime functioning. However, because of the variability seen between individuals, it is not possible to determine an overall sleep need that can be applied to everyone. Instead, Ferrara and De Genarro (2001) suggest that each person's baseline sleep need should be individually determined and that this can best be done by allowing someone to go to bed when tired and rise in the morning when feeling refreshed and without being woken by an alarm. A reduction in sleep below this individual baseline quantity, either acutely or in a cumulative manner, produces negative consequences for performance and alertness.

1.4 Physiological Changes with Increasing Sleepiness

Possibly the most marked effect of either acute sleep loss or a cumulative sleep debt is increasing sleepiness (Bonnet, 2000). The term sleepiness is widely used, often without discussing what is meant by it. In the present context its use is limited to describing the subjective feeling of, and objective evidence for, the pressure for sleep (Åkerstedt, Torsvall & Gillberg, 1987). When an individual is experiencing sleepiness they are somewhere on the continuum between fully awake and asleep (Pivik, 1991). Another term often used interchangeably with that of sleepiness, is drowsiness. It is also deemed

in this context to refer to the subjective or objective experience of transitioning between the sleeping and waking state. The expression *alertness* is sometimes used interchangeably with that of drowsiness and sleepiness, again without qualification. In this context it is presumed to represent one end of the sleepiness continuum, that of fully awake and being able to remain so without assistance from external stimuli (Dinges & Kribbs, 1991).

The preceding section discussed *what* happens to performance and alertness with increasing sleep loss or restriction but not *why* these changes occur. One hypothesis that explains the increasing variability in responding on externally paced reaction time tasks or the trade of speed for accuracy on self-paced tasks, is that of state instability (Doran et al., 2001). It posits that with greater levels of sleep deprivation, performance becomes more variable or delayed due to the build of the homeostatic drive for sleep combined with the endogenous circadian influence on alertness and attention, which when not masked by greater effort from the performing individual, becomes apparent through transient periods of sleep. These short sleep episodes have been termed microsleeps (Murray, 1965). When a microsleep occurs at the same time that a response is also required, the likely result is a prolonged reaction response, also known as a lapse. The state instability hypothesis builds upon the earlier “lapse hypothesis” which was based on findings that prolonged reaction times were coupled with EEG activity that indicated a shift from wakefulness to sleep (Williams et al., 1959).

Other performance changes such as general slowing on cognitive tasks, time-on-task changes, and the optimum response shift are either only partly due to lapsing or not at all. Dinges and Powell (1988) removed the influence of lapses on reaction time performance and analysed the data in a minute-by-minute manner. A decrement in responding was still evident, indicating that despite the number and duration of lapses rising with increasing time-on-task there was an independent overall decline in responding. Mackworth (1968; 1969) believed that such changes were due to the soporific effects of an uninteresting task. Dinges (1989) proposes that with increasing sleep loss, an individual becomes more reliant on environmental and contextual factors to sustain attention and, where these are removed, an increased susceptibility to habituation becomes obvious. The changes in optimum responding have been suggested to be due to a lack of ability to continually attend to a task as well as produce the necessary rapid motor response.

Although habituation contributes to declining performance on some tasks, the neurophysiological changes which are responsible for performance lapses and increased variability in responding are able to be objectively measured and are relatively well understood. Furthermore, they are believed to be the cause of the diminished performance rather than a downstream effect of it. Thus, to measure or detect increased sleepiness it would seem logical to do so at the most basic level possible.

1.4.1 *What is the Electroencephalogram (EEG)*

In order to understand the measurement and detection of microsleeps, it is first important to clarify what it is that is being recorded. The EEG records the electrical activity of neurons, which normally fire across a range of frequencies broadly including 0.1-100 Hz. In the waking EEG, frequencies between 8-30 Hz predominate with very little activity above or below this range (Niedermeyer, 1999). As was discussed briefly at the beginning of this chapter, with the onset of sleep, frequencies slower than 8 Hz become more predominant. Traditionally EEG frequencies have been divided in broad bands and named. Those of theta, alpha and delta have already been discussed but other bands commonly referred to are beta (14-30 Hz, β), and gamma (γ), which normally includes frequencies above 30 Hz. Depending on the circumstances, bands may also be broken down into upper and lower parts. The EEG is normally plotted as voltage against time, with voltage indicating the size, or amplitude, of the signal. The normal amplitude of EEG signals ranges between 10 and 100 μ V, with most between 10 and 50 μ V.

During waking, low amplitude, mixed frequency, asynchronous activity, generally in the beta range predominates. With increasing sleepiness populations of neurons begin to fire in synchrony (Steriade, 2000). The synchronous activity continues and generally slows with deeper NREM sleep. Control of this synchronisation is thought to be through one or more neural pacemakers, which have been identified at the cellular level for synchronous frequencies associated with sleep spindles and in the delta range. Current knowledge regarding the mechanisms controlling the occurrence of activity in other bands, such as alpha, is limited. The psychophysiological significance of many rhythms is also still unknown.

Wakefulness is largely controlled by the reticular formation in the brain stem, and signifies a readiness of neuronal networks to receive information from external sources

(Jones, 2000). This allows individuals to be conscious of themselves and the world around them. The transmission of information from external sources to the cerebral cortex is through the thalamus and is enhanced during waking. Signals received by the thalamo-cortical relay are output one for one during this phase. Steriade, McCormick and Sejnowski (1993) were the first to establish that at the first signs of drowsiness, the input of sensory information to the thalamus remains unchanged but the output from the thalamo-cortical relay decreases and continues to decrease with increasing NREM sleep depth, thereby also resulting in a decreased level of consciousness.

One of the most predominant frequency bands seen with early changes from wakefulness to sleep is that of alpha, which characteristically waxes and wanes. Between individuals the amplitude of alpha varies, as does its prominence, with this frequency being dominant in 20% of people, and 25% showing little or no alpha activity. With eye-closure, and under conditions of relaxed wakefulness, alpha is predominant in posterior regions of the brain, particularly the occipital area (Niedermeyer, 1999). With attention, especially visual stimuli and mental effort, alpha activity is attenuated. As alertness increases, alpha is replaced by low voltage mixed frequency activity, most obvious when the eyes are opened.

In the normal waking EEG, a small amount of unorganised theta can occasionally be seen, which is thought to be generated by a different mechanism from alpha (Niedermeyer, 1999). Both theta and the slower frequencies of delta, have been associated with cholinergic activities and central cholinergic pathways (Steriade, Gloor, Llinas & Lopes da Silva, 1990).

1.4.2 EEG/EOG Changes With Increased Sleepiness

It has already been discussed that with increasing sleep loss, restriction, or disruption, the homeostatic drive for sleep builds, and eventually becomes so powerful that the brain begins to show short periods of sleep (microsleeps) (Kribbs & Dinges, 1994). These microsleeps occur uncontrollably and with increasing frequency as the homeostatic drive escalates. The following section reviews the evidence of homeostatic changes in the waking EEG with increasing sleep loss, as well as the abundance of literature regarding the EEG and EOG related changes that occur with the onset of sleep. Such electrophysiological changes have implications for individuals in situations where they are

required to be fully alert in order to perform competently (Santamaria & Chiappa, 1987; Harrison & Horne, 1996a).

In traditional sleep scoring, the EEG is visually inspected for changes and certain events, but there is often much information contained in the EEG that the human eye cannot distinguish (Campbell, 2000). By transforming data from the time domain to the frequency domain (for example by Fast Fourier Transform) it is possible to describe the constituent frequency components of a complex waveform. Often the resulting spectral data are log transformed prior to comparisons being made, in order to reduce the effect of differences in amplitude of the EEG in different frequency bands. Compared to performance or subjective measures, quantitative EEG (QEEG) changes seen during wakefulness have the advantage of being relative quick to calculate, producing a continuous scale of EEG activity (Åkerstedt, 1991), and being independent of an individual's level of motivation, their ability to fall asleep, or their perception of the situation.

Homeostatic Changes in the EEG

Early studies have shown that theta and alpha power increase during prolonged tasks and with increased subjective sleepiness in individuals with their eyes-open (Fruhstorfer, Langanke, Meinzer, Peter & Pfaff, 1977; O'Hanlon & Kelley, 1977). Using constant routine protocols, more recent studies have reported that, with prolonged wakefulness, increased spectral power is seen in the eyes open EEG in the 6.25-9 Hz range (Cajochen, Brunner, Kräuchi, Graw & Wirz-Justice, 1995), between 1 and 7 Hz when recording from frontal derivations (Cajochen, Knoblauch, Kräuchi, Renz & Wirz-Justice, 2001), and for frequencies between 0.75-9 Hz, and 12.25 and 25 Hz (Aeschbach et al., 1997).

In some instances a circadian modulation in spectral power has also been demonstrated (Aeschbach et al., 1997; Lafrance & Dumont, 2000; Cajochen et al., 2001). Theta power shows the greatest circadian amplitude, with the trough for this frequency, delta and lower alpha occurring around the time of the evening "wake-maintenance" zone (Aeschbach et al., 1997). During normal night hours the increase in power in delta, theta, and lower alpha (7.25-9 Hz) was most pronounced (Aeschbach et al., 1997), with a peak at 1-7 Hz occurring just after the nadir of core body temperature (Cajochen et al., 2001).

It has been suggested that the increased power in these frequencies seen in the waking EEG, reflects an increasing homeostatic drive for sleep, similar to the increased power in

lower frequencies manifested in NREM sleep (Cajochen et al., 1995; Aeschbach et al., 1997; Cajochen et al., 2001). This is supported by the attenuation of spectral power with intermittent naps (Cajochen et al., 2001). Further, the finding of changes in EEG power across the day indicates the sensitivity of the EEG to the processes that occur with extended wakefulness (Aeschbach et al., 1997; Lafrance & Dumont, 2000).

Across a shorter period (12.5 hours) of prolonged wakefulness at night, similar changes to those mentioned above have been recorded (Åkerstedt & Gillberg, 1990).

Furthermore, when there were large increases in alpha and theta power, maximal subjective sleepiness ratings were made and SEMs were noted. However, the changes in the EEG were only pronounced when extreme subjective sleepiness was recorded (above 7 on Karalinska Sleepiness Scale). The authors made several methodological suggestions including analysing data in 1 Hz increments, as this identified spectral peaks between 5 and 9 Hz. They also suggest that under these conditions, EEG recorded when the eyes are closed is less sensitive to sleepiness due to the almost immediate sleep onset seen at eye closure, and although the number of blinks and rapid eye movements made scoring difficult, they felt that the occurrence of SEMs was the most sensitive indicator of sleepiness. Finally, they also noted large inter-individual differences on all indices.

Physiological changes during prolonged wakefulness have also been recorded in an industrial workplace setting across both an afternoon and night shift (Åkerstedt, Kecklund & Knutsson, 1991). Increased power in both the theta and alpha bands was noted, but alpha power only increased significantly across the night shift. Five individuals were also seen to fall asleep, for an average of 43 minutes, during the night shift. Prior to, during and after these sleep periods an increase in alpha power to 200% above baseline was seen (baseline levels being the mean power in the first hour of an afternoon shift). Increases in theta to this extent were only noted during the sleep periods.

The findings from this study were weaker than those in previous studies of objective sleepiness by these authors. They thought this was due to the removal of the sleep episodes from the data subjected to power spectral analysis, which was done because of the conceptual difference between sleepiness (fighting sleep) and sleep (succumbing to the fight). It was hypothesised that higher power in the alpha and theta bands may have been seen if the individuals' work environment prohibited them from falling asleep, as the study participants would then have to fight sleep. In addition, the level of physical

activity required in their tasks may have assisted in masking sleepiness. It was therefore felt that the EEG would provide information on manifest sleepiness, or sleepiness that was expressed given the current level of stimulation. It was also established that increases in alpha and theta were seen in “bursts”, which therefore calls into question the desirability of averaging the power in short epochs over longer periods of time. It may be better to consider the frequency or percentage of epochs that exceed a certain criterion.

EEG and EOG Associated With Sleep Onset

The study of sleep onset has a long history, originating with the work of Loomis et al. (1937). This group made the connection between a change in consciousness and a parallel set of changes in the EEG. The three general EEG changes first identified by Loomis and colleagues (1937) as signalling sleep onset have since been greatly expanded upon. Santamaria and Chiappa (1987) have published extremely detailed findings regarding the neurophysiological changes seen in the EEG and EOG with increasing drowsiness and sleep onset.

A main point from the study by Santamaria and Chiappa (1987) was that the electrophysiology of the wake-sleep transition is complex and not only do variations exist between individuals but a given person may vary in how they transition between wake and sleep on separate occasions. The variability of the drowsy EEG has also been expressed by others investigating the wake-sleep transition (Liberson & Liberson, 1965; Mulsby et al., 1968; Broughton & Hasan, 1995).

Santamaria and Chiappa (1987) organised the transition from wake to sleep into four groups depending on the changes seen in the EEG. The first general area of change noted with drowsiness was an alteration in the distribution of alpha. They theorised that for an alert individual, eye closure acts to passively prohibit any desynchronising input from occipital locations in the cortex resulting in the appearance of alpha in this location. Subsequent removal of non-visual inputs from other cortical locations, such as the central and anterior regions gradually allows alpha to appear there also. Changes in the distribution of alpha have been reported elsewhere (Fischgold, Schwartz & Dreyfuss-Brisac, 1959) and also more recently (Broughton & Hasan, 1995). Broughton and Hasan (1995) proposed that the anterior spread of alpha was in fact a separate rhythm and often the first *reliable* sign of drowsiness.

The next category of change identified by Santamaria and Chiappa (1987) was a decline in amplitude of the EEG, seen particularly in posterior alpha. Loomis et al. (1937) determined this change to be the second of the three stages seen during sleep onset. A decrease in the amplitude of alpha has also been identified by other researchers (Davis, Davis, Loomis, Harvey & Hobart, 1938; Liberson & Liberson, 1965; Maullsby et al., 1968; Badia, Wright & Wauquier, 1994; Wright, Badia & Wauquier, 1995). However, the timing of the change varies between studies.

A decline in alpha amplitude is often followed by the appearance of various lower frequencies (Santamaria & Chiappa, 1987). Various topographical patterns are seen, and the general trend is for the intervals between episodes of alpha activity to lengthen. This gradual interposition of frequencies slower than alpha has been reported in the very early studies of sleep onset (Davis et al., 1938; Fischgold et al., 1959; Kuhlo & Lehmann, 1964; Liberson & Liberson, 1965), and also more recently (Badia et al., 1994; Broughton & Hasan, 1995; Wright et al., 1995), but often the temporal sequence of these changes is reported differently.

The final category of changes seen is a combination of those above and depends on a number of factors, such as sleep pressure, individual differences, and age (Santamaria & Chiappa, 1987). Under high sleep pressure, alpha disappearance is followed closely by the appearance of slower frequencies. If sleep pressure is not high, a decrease in amplitude occurs first and slowing occurs later, or the two changes occur together. The different proportions of the patterns discussed above are probably due to the moment-by-moment balance between wake and sleep generating systems and it is likely that individual differences are generated through unique mechanisms for the interaction between these two systems.

Despite the complex changes in the EEG during sleep onset, the currently accepted criteria for sleep onset stipulates the presence of EEG activity of low voltage, mixed frequency, with predominantly 2-7 Hz activity (Rechtschaffen & Kales, 1968). The standard definition of the transition from wake to stage 1 is said to be a general slowing of the EEG, with a decrease in the frequency and amplitude of alpha.

Behavioural Categorisation of Sleep Onset EEG Changes

All the studies mentioned so far in this section have defined the sleep onset period solely by reference to the EEG. Several studies have used behavioural measures in conjunction

with EEG recordings to investigate the changes during the transition from wake to sleep (Ogilvie, Simons, Kuderain, MacDonald & Rustenburg, 1991; Ogilvie & Simmons, 1992; Hori, Hayashi & Morikawa, 1994).

Ogilvie et al. (1991; 1992) operationally defined sleep as failure to respond to an auditory tone, and proposed latency to responding as an indicator of where on the wake-sleep continuum an individual was. Spectral data from 5 seconds of EEG preceding the auditory tone were sorted into 5 categories according to response times. During wakefulness (category 1) there was little total power in the EEG, which gradually increased across the categories, particularly in the lower frequencies. Across the categories, delta power showed no increase until sleep onset (category 5), while theta power showed a relatively constant increase across all 5 categories. Alpha power was seen to decrease until category 5, where a sharp increase occurred. Sigma power showed a similar change between category 4 and 5, but preceding that was relatively stable. Beta power showed the same pattern as alpha. Ogilvie and Simmons (1992) noted that the process of transitioning into sleep was oscillatory, and that this behavioural method of looking at the sleep onset process allowed for transient fluctuations, as did the short scoring epochs.

Hori et al. (1994) also used 5 second epochs to categorise the EEG during the wake-sleep transition. They determined 9 categories that covered from eyes-closed wakefulness through to stage 2 sleep. Responses to a tone, if made, were significantly longer in stages 8-9, which were in turn longer than those in stages 5-7, and the fastest responses were in stages 1-4.

SEMs and Sleep Onset

SEMs have been shown to be a reliable indicator of drowsiness (Santamaria & Chiappa, 1987; Ogilvie, Wilkinson & Allison, 1989; Hori et al., 1994), and said to occur in all individuals (Maulsby et al., 1968; Santamaria & Chiappa, 1987). It has also been suggested that for 50% of individuals they are the first signs of drowsiness (Maulsby et al., 1968).

SEMs are linked with EEG changes indicating drowsiness (Aserinsky & Kleitman, 1953; Kuhlo & Lehmann, 1964; Liberson & Liberson, 1965; De Gennaro, Ferrara, Ferlazzo & Bertini, 2000; Wright & McGown, 2001). In the standardised criteria for sleep scoring, slow eye movements of several seconds duration are said to be predominant during stage

1 sleep, particularly during the early portion of this stage, but they do not persist into stage 2 sleep (Rechtschaffen & Kales, 1968). SEMs also precede sleep onset, although their exact timing in relation to this event is questionable. Carskadon (2000) reports that SEMs are normally seen 1-2 minutes prior to the onset of sleep, although in daytime sleep recordings they have been seen up to 15 minutes prior to sleep onset.

1.4.3 *Declining Performance and Concomitant EEG/EOG Changes*

The relationship between EEG/EOG indicators of sleepiness and performance decline is of key importance in a workplace setting. Early studies in this area noted that slowed responses, made when an individual's eyes were closed, were associated with a reduction in alpha activity and the occurrence of slower, 2-5 Hz (Bjerner, 1949), and theta, activity, (Mirsky & Cardon, 1962; Williams, Granda, Jones, Lubin & Armington, 1962). Further early studies also associated increased alpha (Daniel, 1967; O'Hanlon & Kelley, 1977) and theta power (Horvath, Frantik, Kopriva & Meissner, 1976; Fruhstorfer et al., 1977), and decreased beta power (O'Hanlon & Beatty, 1977) in the eyes-open EEG, with reduced performance.

More recent studies have focussed on the relationship between EEG and performance across periods of extended wakefulness. Using data collected during a vigilance and letter discrimination task across 15 hours of wakefulness, Belyavin and Wright (1987) calculated canonical variates to investigate changes in EEG power. The main canonical variate explained 94% of the variance between the number of missed responses and was associated with increasing delta and theta power and decreased beta₁ (14-21 Hz).

Increases in alpha power were only significant when relatively few misses occurred (25-50%) on the visual vigilance task. For a greater number of misses, the direction of change in alpha power became more variable. Increases in theta and decreases in beta₁ were consistent between individuals, with decreases in beta₁ being the most useful discriminator for both tasks. Despite these findings, the changes in EEG could only be used to discriminate the highest error rates from the best performance. This indicates, as the authors suggest, a need to understand the variability that exists between individuals.

Torsvall and Åkerstedt (1988) studied individuals performing a 45 minute monotonous vigilance task across the night under laboratory conditions. For the 1 minute leading up to a miss, all EEG and EOG variables except eye blinks showed an increase compared to the hit. This was the same for the single 7.5 second epoch prior to the event for all

variables except alpha power. The authors noted that the state of dozing off was reached gradually and that once it occurred an oscillation between alertness and sleepiness continued. From these findings it was estimated that sleepiness equated to a 300% increase in alpha power above baseline (first 3 hits), 200% increase in theta, and SEMs seen one fifth of the time. Large individual differences were seen but the patterns were generally homogenous, with all participants showing an increased number of SEMs, an increase in alpha power even if during baseline they showed little alpha rhythm. Increases in theta and delta were seen in all but one individual.

Unlike Belyavin and Wright (1987) and Torsvall and Åkerstedt (1988), Corsi-Cabrera (1996) found spectral power in all frequencies from 4-20 Hz to increase in the one second of EEG recorded prior to each visual stimuli with increasing wakefulness.

Many of these above mentioned studies have ignored the influence of circadian phase on the EEG/EOG-performance relationship. Higuchi (2001) investigated changes in diurnal EEG power while participants completed a discriminatory auditory vigilance task with their eyes open. Power in the theta, alpha, and beta bands varied over time, with greater power seen in the afternoon and evening compared to the morning. The largest increase in alpha power was seen at 1400 hours, possibly indicating a circadian influenced increase in sleepiness at this time. Cajochen et al. (1999) undertook a constant routine experiment across ~32 hours of wakefulness to determine whether the association between EEG/EOG and performance persisted independently of circadian phase. The authors noted two major findings, first the demonstration that both circadian and homeostatic influences can be seen in EEG spectral power, and although this protocol did not allow the separation of these effects, the findings suggest that frontal EEG power below 7 Hz is primarily homeostatically determined due to its near linear time course. Second, although different measures were used, similar findings on the relationship between EEG spectral power and performance were seen to those reported in the studies mentioned below, by Jung et al. (1997). This is despite the time interval between performance measurement and EEG recording being approximately 10-15 minutes.

Using more sophisticated analytical techniques, Makeig and Inlow (1993) investigated the variability in eyes-closed EEG across time, and within individuals, as they performed a 28 minute simulated sonar task. To investigate the existence of a linear relationship between performance and the EEG spectrum a local error rate was determined. With increasing

local error rate, a decrease in alpha power and increase in low theta and delta power were seen. As the error rate became high a reduction in power at 10 Hz and a rise at 4 Hz was noted. In a later study Makeig and Jung (1995) showed that these above changes were consistent for eyes-open EEG at all frequencies other than at 10 Hz. In a further recent study by this group, Jung and Makeig (1997) studied individuals during a simulated auditory and visual sonar task and showed that there was a monotonic relationship between EEG power at Cz in the narrow frequency bands of 3.7 and 14.7 Hz and performance, with EEG power increasing as error rates also increased. No obvious relationship was seen between error rates and frequencies near 10 Hz, indicating that the alpha band provides little useful information on performance when an individual's eyes are open. The findings from these three studies demonstrate the need to look at changes in specific frequencies rather than across frequency bands, and the authors noted that such patterns were stable within, but variable between, individuals.

In a study utilising a combined auditory and visual detection task, Makeig and Jung (1996) demonstrated an 18 second cycle in detection performance. This was accompanied by decreased spectral power near 4 Hz and 40 Hz in the seconds preceding undetected targets, while before detected targets the reverse was seen. A later study confirmed a cyclic pattern of performance near 18 seconds, with poor performance accompanied by an increase in spectral power at 3-4 Hz (Makeig, Jung & Sejnowski, 2000)

A series of studies investigating the changes in the EEG and EOG with sleepiness have been conducted in workplace settings. The EEG and EOG of locomotive engineers were recorded during both a day and night trip, and the engineers were divided into a high and low sleepiness group depending on their self-reports (Torsvall & Åkerstedt, 1987). During the day trip spectral power in the alpha band did not differ between the two groups, while at night alpha power was significantly greater for all study participants and in particular for those who were in the high sleepiness group. Similar findings were seen for theta power and the number of SEMs.

Kecklund and Åkerstedt (1993) recorded the midline EEG of 18 truck drivers during either an evening or night drive. Power spectral data from the last 6 hours of the drive were analysed in 7.5 second epochs and was either averaged in 1 hour blocks or as the number of "bursts" above a certain criterion (power in the traditional frequency bands either 100%, 150%, or 200% above baseline values recorded during the beginning of the

drive). The frequency of bursts of theta and alpha power were found to increase across the duration of the drive, with this effect being most pronounced for alpha power in the last 2 hours of those in the night group. Only average power in the theta band increased over time when analysed in 1 hour blocks. From these findings the authors recommended analysing data in bursts in order to allow transient increases in power to be captured and that the criterion for a burst be relatively low, such as 100-150% increase above baseline.

In contrast, in a later study of 30 minute periods of night driving (Gillberg, Kecklund & Åkerstedt, 1996) no increase in EEG/EOG signs of sleepiness were seen across the duration of the drive. The EEG was scored by determining the percentage of a 30 second epoch which showed increases in alpha, theta or SEMs. Only a 5 minute period of eyes closed EEG recorded prior to and after the drive showed increases in sleepiness (eyes-open did not significantly differ). The authors suggested that in this instance the experienced drivers could have been less vulnerable to the effects of sleepiness or alternatively the task was short and alerting. Unfortunately, no prior sleep history of participants was provided, making the interpretation of these findings difficult.

1.4.4 Summary of Physiological Changes With Increasing Sleepiness

Studies that have focussed on changes in waking EEG power over extended time frames have produced somewhat disparate findings, partly due to the variability in time frames looked at, and the frequency bands utilised. Nonetheless, homeostatic influences are generally evident through increasing power in frequencies below 25 Hz, with the most prominent increases seen in the lower frequencies (approximately 1-9 Hz). Across the night hours, increases in delta, theta and alpha power below 9 Hz, are most prominent, while theta and alpha show much smaller increases during the daytime. In conjunction with this homeostatic rise there are also circadian variations in spectral power, with the daytime maximum occurring towards the late afternoon or evening, followed by a drop around the time of the evening wake-maintenance zone, then a continued rise across the night hours if wakefulness continues.

The changes seen with the onset of sleep provide an insight into the complex and variable EEG patterns that might be seen in a workplace situation with increasing sleepiness. Without large EEG montages these often fleeting, and location specific changes, would not be able to be detected. However, although the changes are complex

and variable, grossly it can still be said that under eyes-closed conditions, power in alpha and beta band decreases with increasing drowsiness while theta and delta power increases (Pivik, 1991).

It must be kept in mind that the changes seen with sleep onset were generally seen under conditions where individuals were relaxed, had their eyes-closed and were being encouraged to fall asleep. This is in contrast to what would normally be occurring in a working environment. Åkerstedt (1991) states that the EEG parameters recorded under relaxed eyes-closed conditions are essentially irrelevant when the interest is on the workplace monitoring of sleepiness. This view is probably slightly extreme, as sleep onset is likely to be very dependent on the working environment, and although most employers would not encourage an individual to relax enough to fall asleep while working, it is not to say that under soporific conditions an individual might lose the battle to keep their eyes open and would eventually relax enough to fall asleep. In fact, Åkerstedt himself (1991) recorded instances of individuals falling asleep at work. What has not been determined is whether the changes seen under normal sleep onset are the same as those seen when an individual has been attempting to keep their eyes open and remain awake. Despite this, it is likely that the changes seen during normal sleep onset have some similarity to, or provide some insight into, what might be seen when an individual loses the battle to remain awake at work.

One striking neurophysiological difference between eyes-open and eyes-closed conditions under different levels of sleepiness is the prominence of alpha. As has been mentioned, there is strong evidence indicating that under eyes-closed conditions a decline in alpha power indicates increased sleepiness, while in eyes-open conditions it is the appearance of alpha which is the marker of sleepiness (Åkerstedt & Gillberg, 1990). Unless it can be definitively determined whether an individual has their eyes open or closed, focussing on changes in the alpha band becomes problematic.

It is not definite whether the changes seen in conjunction with increasing wakefulness and preceding, or during, performance lapses are the precursors to sleep or not. Nevertheless, such changes are definitely associated with increasing sleepiness, they are similar to sleep, and most importantly are coupled with a reduced behavioural response. In addition, both the oscillatory nature of the sleep onset process and the ephemerality of decreased alertness when performing a task, highlight the importance of looking for

short “burst” like events in the EEG data, in addition to focussing on any global change in spectral power in association with increasing sleepiness.

It has been argued that the variations seen in the EEG and EOG are only seen when alertness is below that normally seen at the circadian nadir (Van Dongen & Dinges, 2000) and therefore sleepiness has to be severe to be detected through such neurophysiological measures, which in turn may limit their usefulness. Despite this point of view, there are still many reports of the EEG and EOG being sensitive enough to measure decreased alertness during the night shift and in working environments e.g. (Aeschbach et al., 1997; Lafrance & Dumont, 2000). It may be that in these studies, individuals were particularly sleepy. On the other hand, it may be that this level of sleepiness is not unusual in night workers, thereby making such neurophysiological measures entirely pertinent.

From all the studies of EEG related changes with increasing wakefulness, during sleep onset, and in conjunction with performance changes, the findings indicate that, although changes can possibly be detected when frequencies are grouped into bands, more specific changes are likely to be seen when individual frequencies or narrow bands are also examined. This supports the idea that analysis of EEG data should probably involve at least two stages, the first a broader overview using traditional bands, and the second looking more closely at frequency specific variations. A further complication with the analysis of EEG data is the enormous variability seen between individuals. When looking at sleep onset, which has been investigated most thoroughly in this regard, there is also evidence that a single individual varies in the process through which they transition from wake to sleep. This suggests that individual data should be looked at closely before group analyses are undertaken, and it may be necessary to individualise peak frequencies for use in subsequent investigations.

1.5 Countermeasures to Sleepiness

The increased sleepiness and decreased performance seen with sleep loss are issues of operational concern for shiftworkers, as are any factors that increase an individual's susceptibility to error, or what Reason (2000) has labelled an *active failure*. Night workers face the biggest challenge, as the effects of any prior sleep loss are coupled with the greatest circadian propensity for sleep and the nadir of performance. Further, for a diurnal individual, the homeostatic drive for sleep continues to build across the night

shift. Given all the factors weighted against the shiftworker it is necessary to consider if there are measures that can be taken to improve their ability to remain awake and function during times of greatest vulnerability.

The notion exists that physical and or mental toughness, or prior experience with sleep loss provides an antidote to the effects of sleep loss. However, there is almost no evidence that these factors prevent the neurophysiological or behavioural outcomes of sleepiness (Dinges, Whitehouse, Orne & Orne, 1988). Instead such factors may increase an individual's level of motivation and in turn mask the effects of sleep loss for a short period of time. Other countermeasures, such as breaks, have been shown to increase alertness as measured by the EEG (Landström & Lindholm Häggqvist, 1988; Horne & Reyner, 1996), but with a gradual return to sleepiness beginning immediately. Nevertheless, they may be important in providing relief from time-on-task fatigue, and in combination with activity and food, they have been shown to be helpful during driving (Englund, Ryman, Naitoh & Hodgdon, 1985). Studies of the effects of food on its own are limited and findings unclear (Åkerstedt & Landström, 1998). Similarly the relationship between physical activity and alertness is uncertain (Englund et al., 1985). Increased sound, using different tones, has been shown to improve subjective sleepiness (Landström, Englund, Nordström & Åström, 1994), but this countermeasure as well as others, such as lowered workplace temperature, exposure to bright light, and the long term use of melatonin are unlikely to be practical, even if shown definitively to be effective in improving performance and alertness (Åkerstedt & Landström, 1998).

Caffeine is probably the most widely used countermeasure to sleepiness during the night hours. In some instances caffeine has been found to be just as (Schweitzer, Muehlback & Walsh, 1992; Horne & Reyner, 1996), if not more, effective than a short sleep in improving performance and alertness (Rogers, Spencer, Stone & Nicholson, 1989), but other studies have shown the opposite effect (Bonnet et al., 1995). However, when taken in combination with a short sleep it is more effective than sleep alone (Bonnet & Arand, 1994; Reyner & Horne, 1997). Unfortunately, caffeine interferes with the architecture of subsequent sleep, blocking NREM sleep (Landolt, Dijk, Gaus & Borbély, 1995), and there are many people who do not, or cannot, consume it (Åkerstedt & Landström, 1998).

Despite what is known about the structure of sleep, the strong influence of circadian and homeostatic factors on the propensity and maintenance of sleep, and the

neurophysiological and behavioural changes that occur with sleep loss, the primary function of sleep is still not clear (Bennington, 2000). Nevertheless, it seems that when sleep is lost, the only enduring solution is to fulfil the body and brain's requirement for sleep. As indicated earlier, the benefits of sleep are suggested to accrue in a logarithmic manner (Bonnet & Arand, 1995) which is further supported by the findings of Naitoh (1982) and Haslam (1981) that indicate small amounts of sleep are disproportionately effective in maintaining performance compared to complete sleep loss. Thus, obtaining some sleep, even if markedly less than a normal night's sleep, seems intuitively to be a countermeasure to workplace sleepiness.

In the literature, a single episode of sleep that has a maximum duration of half the length of a normal nocturnal sleep is defined as a nap (Naitoh & Angus, 1989). The following section considers what is currently known about the prevalence of napping and the consequences, both positive and negative. Due to the focus of the present thesis on shiftworkers, studies which have investigated the usefulness of short episodes of sleep at work, and prior to work, are discussed as well as other relevant literature that provides information of the factors that influence the outcomes associated with napping.

1.5.1 *Who Naps?*

Shiftworkers have been identified as a population of individuals who frequently nap (Dinges, 1989), probably due to sleep loss associated with their work schedule (Dinges, Orne, Evans & Orne, 1981). Åkerstedt and Torsvall (1985) reported that around 1700 hours, prior to their shift, 18% of night workers napped for an average of 2 hours. The same percentage were reported to nap in association with working a morning shift, although the duration was shorter (90 minutes), and the nap was taken after the shift at approximately 1430 hours, rather than preceding it. Similar findings have also been reported in earlier surveys of shiftworkers (Andersen, 1970; Knauth et al., 1980).

Reports of napping at work are less common, although Andersen (1970) reported that 50% of police were able to obtain a short nap when working the night shift and Kogi (1981) noted that 40-50% of Japanese shiftworkers regularly napped during the night shift.

Other than shiftworkers, the practice of napping is probably most widespread in cultures where a siesta is part of the normal daily routine (Taub et al., 1971). In non-siesta cultures, habitual nappers have been identified and are most commonly young college

students (Evans & Orne, 1975) or older individuals whose nocturnal sleep is no longer sufficient in quantity or quality (Dinges, 1989).

1.5.2 *Why Nap? Evidence for the Beneficial Effects of Napping*

Napping Strategically at Work and/or During the Night Shift.

It has been theorised that, along with offsetting the effects of sleep loss, the circadian nadir, and the homeostatic drive for sleep, napping on the night shift may serve the further purpose of anchoring an individual's circadian rhythms (Matsumoto & Harada, 1994). As discussed earlier, the change from a diurnal schedule to a nocturnal one provides cues to the circadian pacemaker to adjust to the new pattern of work and rest, but strong cues from the light-dark cycle counter this message, possibly resulting in constant circadian desynchronisation for the night worker. A short sleep during the night shift may assist in keeping the night workers' circadian rhythms on a diurnal schedule, reducing the degree of circadian readjustment that occurs. However, currently there is little evidence to support this belief.

If napping is to be conducted in the work place, operationally it is necessary to consider several issues. These include whether individuals can nap in the workplace, how much sleep they must obtain in order for the beneficial effects to become apparent, whether the placement of the nap in relation to circadian phase is important for effectiveness, how operationally relevant any improvements are, and how long they last for. Negative consequences must also be considered, as well as other factors that might alter the potential usefulness of napping.

As mentioned previously, a nap is defined as a single episode of sleep, with a maximum of half the duration of the normal nocturnal sleep. Based on this definition, a nap could be relatively long, and in most working environments it would be completely untenable to consider that an individual could complete their work tasks and also be able to get, for example, 4 hours of sleep. Therefore, in reviewing the literature in this area, consideration was only given to studies where naps of up to 2 hours were investigated. Although there may be working environments where longer episodes of sleep are feasible, in a work period of approximately 8 hours, a maximum of 2 hours (or a quarter of the work period) away from the workplace would logically seem to be around the most an organisation could tolerate financially and logistically. There are only a handful

of studies which have investigated the usefulness of napping during the night shift, with the large majority of those taking place in a laboratory setting.

Angiboust and Gouars (1972) determined the effects of a 2 hour nap at 2400 hours after 17 hours of wakefulness. Visual vigilance was tested between 0300 and 0500 hours and compared to a no-nap group, and the performance of a group tested between 1000 and 1200 hours after a normal night of sleep. Findings indicated that performance for those who napped was better than for the non-nappers and was close to that seen in the daytime group.

Gillberg (1984) looked at the effectiveness of napping after a night of reduced sleep. Twelve male subjects were required to take a 1 hour nap during a “night shift” in the laboratory when the preceding night’s sleep had been limited to 4 hours. Each participant completed three study conditions a week apart, one included an early nap at 2100 hours, the second involved a late nap at 0430 hours, and the third was a non-napping control condition. Reaction time performance was tested at 1900 hours and again at 0700 hours the next morning. A sleep latency test was also completed, with the first occurring at the same time as the early nap. Participants did not know whether they would be awakened at sleep onset (first 20 seconds of stage 1 sleep) or after the 60 minute nap. A second sleep latency test was repeated after the late nap.

Polysomnography data showed the late nap to be longer and have less stage 1 sleep. Pair-wise comparisons showed the late nap to produce faster mean reaction times and faster means for the 5 slowest reaction events, than the control or early nap conditions, but the overall effects between these conditions was not significant. For sleep latencies, no overall differences between conditions were found but pair-wise comparisons showed that, in the sleep latency test at the end of the night, the late nap resulted in longer latencies than both the early nap, and no nap condition, with no difference between the early nap and control. Subjective sleepiness did not differ between conditions. The authors suggest their findings provide evidence that a nap can counter the performance decline seen during the circadian nadir, and that the late nap is particularly effective in doing this. The closer proximity of the later nap to the nadir and its slightly longer length were the explanations offered for this.

Rogers et al. (1989) investigated the effect of a 1 hour nap at 0200 hours compared to 200 mg of caffeine taken just prior to 2315 hours. Six individuals completed each of these conditions with a third control condition of no nap or caffeine. No prior sleep loss

preceded any of the experimental conditions. The 60 minutes of nap sleep was timed from the first consecutive 5 minutes of stage 2 sleep and average total sleep time was reported to be 62.5 minutes, with a SOL of 20.8 minutes. If an individual was still asleep at 0400 hours they were woken, followed by a battery of tests starting at 0415, 0630 and 0845 hours. Out of the eight performance measures used, the nap only improved auditory vigilance at 0415 hours and 0630 hours, and digit symbol substitution at 0630 hours, while caffeine showed a greater number of positive effects. There is a concern that sleep inertia may have affected performance during the first testing session after the nap, but this is difficult to confirm. No details were provided on the duration of time between waking and testing, other than at least half an hour had elapsed. Based on these findings the authors suggest that naps would provide little benefit during a work period, particularly when the overnight deterioration in performance has already begun, but they do consider that naps may serve to prevent further sleepiness. The small number of participants in this study does need to be considered when giving weight to these findings.

In order to investigate whether the timing and duration of a nap of 30 or 50 minutes interacted, and the subsequent consequences for early morning alertness and performance, Sallinen et al. (1998) required 14 experienced shiftworkers to spend five nights in the laboratory, with each night separated by at least 16 days. The testing situation was simulating a night shift commencing at 2300 hours, and on four of the five “night shifts” a 30 minute or 50 minute nap was allowed at either 0150 or 0440 hours. Findings indicated that the later nap resulted in more total sleep, more stage 1, stage 2, and SWS, faster latencies, and fewer awakenings. Reaction time performance and subjective sleepiness were not significantly affected by any single nap, but overall, napping resulted in improved mean reaction time performance and slightly lower ratings of sleepiness in comparison to control values. Napping also reduced performance lapses. On a repeated test of sustained wakefulness (RTSW), latencies were longer for early naps when compared to controls. There was no statistically significant difference between the short and long nap on any measure, except that the longer nap reduced the amount of SWS in the subsequent daytime sleep. Although naps were somewhat beneficial, they did not reverse the general decline in performance across the night shift, but instead limited its extent. The authors note the weaker effects of the nap in improving subjective sleepiness, which follow the findings of Gillberg (1984) and Dinges et al. (1988). However, in contrast to Gillberg’s (1984) findings, Sallinen et al. (1998) also noted the

beneficial effects of the earlier nap. The authors also reinforce the operational significance of a nap reducing the occurrence of performance lapses, which would be important in any monitoring task.

Other studies have investigated the usefulness of napping on the night shift but they have used only subjective reports. Matsumoto and Harada (1994) noted that control room workers in a chemical plant, who were allowed to nap for approximately 2 hours at either 0100 or 0300 hours, reported increased fatigue across the night shift, but the increase was not as great compared to individuals who were not able to nap. Similar findings were reported by Saito and Sasaki (1996), who compared people who did not nap to those who had either a 1 or 2 hour nap at 0300 hours. Both naps decreased subjective ratings of fatigue, with no difference seen between the different naps despite the increased length and amount of SWS in the 2 hour nap. Bonneford et al. (2001) followed a group of night workers at an industrial plant, who were allowed a 1 hour nap at work, for a year. They noted the feasibility and acceptance of napping at work, the worker's improved satisfaction with night work, reports of higher vigilance post the nap, and a general improvement in quality of life.

In one of the few field-based studies, 21 line pilots operating in a three crew 747 cockpit on trans-pacific flights were monitored during four middle legs (a leg being a single flight) of an eight leg trip (Rosekind et al., 1994). The legs were between different locations but all were of a similar duration, crossed a similar number of time zones, and included two daytime westward legs and two night time eastward legs. The aim of the study was to determine whether a 40 minute nap opportunity, taken during a low workload cruise portion of flight, could improve the performance and alertness of flight crew.

Throughout the flight EEG, EOG, and EMG were recorded from each individual for two purposes; first to determine the quantity and structure of sleep obtained during the napping opportunity, and second to measure neurophysiological alertness during the last 90 minutes of the flight. Alpha or theta activity in the EEG, and SEMs in the EOG, were visually identified and binned according to the duration of the event (5-10 seconds, 11-15 seconds, and greater than 15 seconds). Performance was assessed via the PVT, which was completed pre-flight, at top of climb (TOC), immediately prior to the nap, soon after the nap, at top of descent (TOD), and post-flight. Prior to, during, and after

the eight leg trip, sleep quantity and quality were ascertained using actigraphy and logbook data.

Nine crew members were assigned to a non-rest group and 12 to a rest group. During the nap opportunities 93% of individuals were able to sleep. After excluding data from the non-sleepers, the average total sleep time was 26 minutes, sleep efficiency was 64%, latency to the first 1.5 minutes of sleep was 5.6 minutes, and 62% of the nap was spent in stage 2 sleep. Only the amount of stage 1 and SWS sleep differed between day and night flights. During the day, 37% of the nap was stage 1 sleep, while at night this declined to 25%. In contrast, SWS comprised 4% of the total sleep on a day flight and 8% on a night flight. No REM sleep was seen in any nap. The data from the non-nappers during their 40 minute non-nap opportunity indicated that four individuals slept on five occasions, with two episodes of sleep exceeding 10 minutes. It was not mentioned whether the EEG and EOG data, recorded at times other than during the napping opportunity and the 90 minutes prior to landing, were analysed for sleep. Therefore it is possible that other episodes of sleep occurred, and if so they could have contributed to the observed changes in performance and neurophysiological alertness. The decision by the authors to exclude individuals who did not sleep from the summary sleep statistics may also be somewhat misleading.

Performance was compared between nappers (whether or not they slept). Median performance on the PVT was slower for those who did not have the opportunity to nap. Importantly, their pre-nap performance was also significantly worse, suggesting motivational factors influenced the performance of those who were provided with the opportunity to nap. On the other hand, pre-nap differences between groups were not seen for the number of performance lapses, but immediately post the nap and at TOD, those who did not nap had significantly more lapses. Regression lines fitted to the 10 minutes of PVT data were compared for individuals whose initial performance did not differ between day and night flights. A significantly greater deterioration in reaction time performance was noted for individuals who did not nap during night flights, compared to all other conditions.

Across all the neurophysiological data from the last 90 minutes of the flight, 154 microevents were identified, with 120 of those occurring in seven out of nine individuals who did not nap and 84% of those attributable to four of those seven. Only 34 microevents were seen in the last 90 minutes of the flight from the group of individuals

who did nap, and these were from six individuals, with two individuals producing 59% of these events. Most events (54%) were between 5 and 10 seconds in duration, with only 15% being longer than 15 seconds. Statistical analyses indicated the difference in the number of microevents between the nap and non-nap group was significant, with more SEMs and microevents seen during the night rather than the day flights. Subjective sleepiness ratings did not differ significantly between groups.

A more recent field study of flight deck napping studied three person crews during outbound day and inbound night flights on North-Atlantic routes (Simons & Valk, 1997). The subjective alertness and performance of both the napping and non-napping pilot was measured using the Stanford Sleepiness Scale and a combined dual vigilance and tracking task. Fifty nine pilots (mean age 38.2 years) were allowed a 40 minute nap opportunity during the cruise portion of the flight. The amount of sleep obtained prior to the flight and during the stopover was determined via actigraphy. Forty eight percent of pilots stated they did not sleep on the outbound trip and 41% did not sleep on the inbound leg. Of those who slept, the mean self-reported total sleep time was 19 minutes, with a latency of 12-13 minutes.

There was no statistically significant difference between those who napped and those who did not for performance on the post-nap vigilance task. Although, tracking performance was improved on both the outbound and inbound legs. Subjective sleepiness was also lower at TOD after napping, but only on the inbound flight. The pilot remaining awake reported higher ratings of sleepiness while his/her colleague napped compared to pre- and post-nap ratings, with 6 out of 20 reporting moderate to high levels of sleepiness. Higher ratings of sleepiness were correlated with poorer pre-trip and stopover sleep quality and efficiency.

Naps that Precede Work

Numerous studies have indicated a short sleep prior to a night of wakefulness will improve subjective alertness (Harma, Knauth & Ilmarinen, 1989; Sugerman & Walsh, 1989; Bonnet, 1991; Schweitzer et al., 1992) and performance (Nicholson et al., 1985; Dinges et al., 1987; Bonnet, 1991; Schweitzer et al., 1992; Caldwell & Caldwell, 1998). In contrast, a single study found that individuals who napped at home prior to the first night shift reported greater subjective sleepiness and showed poorer performance than those who did not nap (Rosa, 1993). With the findings generally supporting napping

prior to a night of wakefulness, a shiftworker should be encouraged to sleep prior to work, in an attempt to reduce any prior sleep debt, and to ensure preparedness for work. Despite a shiftworker's good intentions, social or domestic commitments in the afternoon or evening may preclude an individual from obtaining such sleep, thus making napping on the night shift a countermeasure to consider.

1.5.3 *Potential Negative Effects of Napping*

Among the possible negative effects of workplace napping are the perception employers have of those who nap. An employer who has not been educated on the consequences of sleepiness and the benefits of napping may interpret an employee's desire and ability to nap, as laziness and a lack of professional ethics (Muzet, Nicolas, Tassi, Dewasmes & Bonneau, 1995). Japan is probably the most forward thinking country with regard to napping. In this country many industries and workplaces actively promote napping on the night shift (Matsumoto & Morita, 1987).

Employees may be reluctant to nap at work if, during their initial effort, they found it difficult to fall, or stay, asleep. Such difficulties may occur for a variety of reasons, but can result in an individual being disinclined to attempt to nap on subsequent occasions. Difficulties sleeping at work may be in part countered by having an area adapted for napping that is close to the working area, and is well sound attenuated, kept at a comfortable temperature, and ventilated. The implementation of napping in a working environment should be well planned and set up formally rather than on an *ad hoc* basis, since once employer's and employee's negative attitudes to napping are entrenched, they may be extremely difficult to change (Muzet et al., 1995).

There is also the concern, and some evidence to support this concern (Tepas & Carvalhais, 1990), that following napping the day sleep will be of reduced duration and quality. This might be a particular problem for older shiftworkers who are already attempting their day sleep under a diminished homeostatic sleep drive. In their study of short naps, Sallinen et al. (1998) noted that a 50 minute napping opportunity reduced the amount of SWS in the subsequent daytime sleep, but no change was seen for a 30 minute napping opportunity. Further, the amount of SWS in the subsequent daytime sleep for the non-nappers was the same as that seen in the nap group, if their nap SWS sleep was summed with their daytime SWS sleep. Similarly Matsumoto and Harada (1994) noted that night workers who napped reported less subsequent day sleep, but the day sleep plus

nap sleep was not significantly different from the amount of day sleep obtained by non-nappers. In contrast, Rogers et al. (1989), found a 1 hour nap at 0200 hours had no effect on post night shift sleep quantity or structure, while caffeine reduced the amount of SWS obtained in daytime sleep.

Probably the greatest apprehension surrounding implementing napping in a workplace is the feeling, and effect of, sleep inertia on waking. Sleep inertia has been described as the transient confusion, sleep drunkenness and reduced performance seen on sudden awakening (Dinges, Orne & Orne, 1985b), a transient period of hypovigilance (Jewett et al., 1999b), a state of increased sleepiness and disorientation (Bruck & Pisani, 1999) and the state of being physically awake while cognitively still asleep (Naitoh, Kelly & Babkoff, 1993). Its significance is such that it has been incorporated into different theoretical models (Achermann & Borbély, 1992; Folkard & Åkerstedt, 1992) and is noted as a possible cause of air accidents, which have been reported as of increased incidence the closer the pilot is to waking from a normal nights sleep (Ribak et al., 1983).

Waking from SWS (Dinges et al., 1981) and spending longer in SWS, are thought to produce more severe sleep inertia on awakening (Dinges, Orne & Orne, 1985a). Salame (1995) investigated the sleep inertia effect on spatial memory and logical reasoning after a 1 hour nap at either 0000 hours or 0300 hours. They found that after an average of 43 minutes of sleep, 4-12% of participants woke spontaneously, 22-35% woke from stage 2 sleep, 61% from SWS and a very small percentage from REM sleep. In contrast to the findings of Dinges et al. (1985a) there was no observed correlation between these various sleep stages and performance on waking.

There is still some debate about the duration of sleep inertia effects, with some studies reporting no measurable effects after a minute (Webb & Agnew, 1964) while others have seen effects up to 3 hours after waking. Jewett (1999b) studied 15 individuals from the 1st minute through to 4 hours after waking from a night of 8 hours sleep. Subjective sleepiness ratings, and a 2 minute addition task, were completed regularly and both showed an asymptotic improvement with the most rapid rise seen in the first hour, and the asymptote reached around 2-4 hours, after waking. A saturating exponential function provided a good fit to the data, which is similar to the pattern of findings of Folkard and Åkerstedt (1992) and Achermann et al. (1995). After shorter periods of sleep, such as those studied by Tassi et al. (1992), Salame (1995), and Sallinen (1998), sleep inertia effects were seen for at least 15 minutes but no longer than 30 minutes after waking.

Sleep inertia is reported to be most severe with greater prior sleep loss, and to last for longer, such that 50 hours of sleep loss results in several hours of sleep inertia after a 2 hour nap (Naitoh et al., 1993). Under conditions of no sleep loss, the effect is reported to last up to 20 minutes (Wilkinson & Stretton, 1971; Dinges et al., 1981; Dinges, 1990). This is possibly through the increased intensity of the homeostatic drive for sleep, which in turn increases the depth of sleep.

It has been indicated that sleep inertia is worst near the nadir of core body temperature (Naitoh, 1981; Dinges et al., 1985a), but is seen at all times of the day. Naitoh (1981) demonstrated sleep inertia effects to be more severe, and last longer, on waking from a nap of 2 hours taken after 45 hours of waking than after the same length nap after 53 hours of waking. The earlier nap was taken at the circadian nadir. In contrast, Naitoh (1993) investigated changes in sleep inertia using a logical reasoning task across 64 hours of continuous work, during which naps of 20 minutes were allowed every 6 hours, and found a clear circadian rhythm in performance but no variation in sleep inertia. The findings from this study are somewhat questionable given the small number of subjects. However, the study by Salame et al. (1995) also failed to find a time of night effect for sleep inertia.

Efforts have also been made to investigate whether the effects of sleep inertia can be minimised or eliminated by noise (Tassi et al., 1992), the napping environment (Dinges et al., 1981), or caffeine (Van Dongen et al., 2001). Tassi et al. (1992) demonstrated that noise eliminated sleep inertia after a 1 hour nap at midnight, but not for a nap of the same length at 0300 hours. In a study of 2 hour naps every 12 hours across an 88 hour sleep deprivation protocol, Van Dongen et al. (2001) showed that caffeine, in an amount equivalent to a quarter of a cup of coffee every hour, was effective in removing the effects of sleep inertia compared to a placebo. Caffeine also increased the latency to sleep onset, and was seen to decrease the amount of REM sleep and SWA. The alerting effects of the naps on psychomotor performance were not seen to differ between the two groups. The authors believe that caffeine was effective in reducing sleep inertia through its action as an adenosine antagonist.²

² Adenosine is a neurotransmitter thought to be released during extended wakefulness due to glycogen depletion. Sleep allows glycogen levels to be restored but adenosine probably continues to be released and persists on waking, resulting in sleep inertia. It is thought that normally, reuptake or metabolic processes eventually remove adenosine on waking, hence the relative duration of sleep inertia and the rapid reduction by caffeine.

Additional Factors Influencing the Effectiveness of Naps on the Night Shift

The relationship between prior sleep loss and napping also needs to be considered. Studies that have investigated napping during periods of continuous wakefulness provide some insight into this issue, although not all their findings can be directly applied to shiftworkers, particularly when the periods of continuous wakefulness exceed 24 hours.

It is suggested that the longer the period of prior wake, the greater the amount of sleep required to effect a performance improvement. Both Naitoh (1982) and Haslam (1982) showed a nap of 3-4 hours after a prolonged period of wake (20 or 90 hours) improved performance, but not back to baselines levels. It has also been demonstrated that a 2 hour nap taken early in a period of sleep deprivation is more effective than the same duration nap taken later in the protocol (Dinges et al., 1987).

Naitoh (1981) demonstrated that after 45 hours of wakefulness, a 2 hour nap at 0400 hours was of almost no benefit in the 6 following hours, whereas a nap of the same length after 53 hours of wakefulness at 1200 hours did have positive effects for performance. The lack of an effect for the nap at 0400 hours was thought to be related to the combined effect of circadian phase and prior sleep loss, which may have produced prolonged sleep inertia, thus producing the null finding. These findings raise the issue of a possible interaction of circadian phase with prior sleep loss. However, a further study reported contrasting findings (Dinges et al., 1987; Dinges et al., 1988). Two hour naps, taken either at the circadian acrophase or nadir, across a 56 hour sleep deprivation protocol, did not differ in their effects on PVT performance, even after 30 or 42 hours of prior wake.

An additional issue raised by the study of Dinges et al. (1988) is the time taken for the effects of the nap to be evident, and the duration of any benefit. A nap taken early in the sleep loss protocol, prior to real sleep loss occurring (after 6 or 18 hours of prior wake), did not show its beneficial effects immediately, but once apparent, the improvement lasted for most of the remainder of the 56 hour protocol. On the other hand, naps taken after 30 hours of sleep loss resulted in an immediate performance improvement, but the effects were not maintained for more than 12 hours.

The napping environment is also likely to be influential in determining the quality of sleep obtained and therefore also the benefits of the nap. In a study of possible measures to reduce the effects of sleep inertia on waking, Dinges et al. (1981) compared

a 1 hour nap opportunity in the afternoon under sleep-conducive conditions (in bed, dark, temperature controlled, sound attenuated room), to the same duration nap opportunity under less ideal conditions (sitting in a lounge chair, lit room, and with noise). Sleep latency and total sleep time did not differ between the two conditions but there was less stage 1 and more stage 4 sleep under the sleep-conducive condition. Despite the effect on sleep structure, there were no significant differences in performance between these two conditions.

1.5.4 Summary of Napping as a Countermeasure

On the whole, these studies support the efficacy of napping on the night shift for improving performance and alertness. Even though the findings are not strong, they are reasonably consistent. Probably the most reliable finding is that napping limits, rather than reverses, any performance decline or increase in subjective sleepiness across the night shift. Given that the laboratory findings were not overwhelmingly convincing, the clear benefits reported by Rosekind et al. (1994) in the flight crew field study are somewhat surprising, especially given the range of factors not able to be controlled in a field setting that could have masked or confounded the results. The clear findings may largely be the result of the measures utilised, as the neurophysiological correlates of alertness showed the effects of napping most strongly.

A number of factors need to be considered when assessing the potential benefits of napping. One of the most important issues is whether individuals can sleep in the workplace during a relatively short opportunity. Naitoh (1992) reported that the efficiency of short sleeps was approximately 50%, and Åkerstedt et al. (1993) noted efficiencies for 1 hour naps ranged between 46% and 97%. However, both these studies included sleep taken at all times in the circadian cycle and after varying periods of prior wakefulness. In laboratory based studies of naps during a simulated night shift, the efficiencies reported were generally higher, ranging between 69% and 94% (Gillberg, 1984; Rogers et al., 1989; Sallinen et al., 1998), and efficiencies of approximately 64% were seen in the NASA flight deck napping study (Rosekind et al., 1994). In contrast, Simons et al. (1997) reported low sleep efficiencies, of around 50%. Without further evidence regarding the efficiency of sleep obtained in the workplace, it is difficult to know how applicable the findings of laboratory studies are to workplace situations. Such information is certainly still needed but is likely to be specific to particular conditions,

such as the amount of prior sleep loss generated by a work schedule, and the environment in which the nap can be taken.

Given that individuals who nap in a workplace environment may not get much sleep, it is necessary to consider whether there is a minimum amount of sleep required before beneficial effects are seen. Due to the different methods and measures utilised in studies of napping at night, the findings are difficult to compare. To date, as far as the author is aware, no one study has systematically compared a range of short periods of sleep (for example between 10 minutes and 2 hours) during a night shift. Sallinen (1998), Simons (1997) and Rosekind (1994) showed that naps of less than 30 minutes during a night shift have beneficial effects, while Naitoh, (1992) reported that in continuous operations, the minimum effective nap duration was 4-10 minutes. Together, these findings suggest that even a short sleep at work is likely to be of benefit. Because of the asymptotic relationship between sleep duration and performance, the more sleep an individual could get before reaching the asymptote, the better their performance.

Nonetheless, due to the normal architecture of sleep, there is an increased likelihood of waking from, or having more SWS, when a sleep episode exceeds 40-50 minutes (Carskadon & Dement, 2000). This in turn increases the likelihood and severity of sleep inertia. Further, longer sleep episodes are more likely to reduce the quantity and quality of daytime sleep following the night shift. Thus, a balance between maximising the positive effects of a nap, while also minimising the negative effects, must be obtained when napping is to occur in a working environment.

The timing of the nap in relation to circadian phase is a further point to consider, particularly as evidence suggests that a nap taken closer to the circadian nadir is longer, and possibly contains more SWS. However, there is little evidence to suggest such naps are markedly more effective in improving performance.

A possible interaction of circadian phase with prior sleep loss has been seen when sleep loss is around 45 hours (Naitoh, 1981), but this finding has not been supported in other studies (Dinges et al., 1987). These findings suggest that night time naps may be most beneficial if the amount of prior sleep loss is not excessive (i.e. less than 2 days), since with increasing sleep loss the negative outcomes of napping may become more prevalent and persistent. The findings of Hartley (1974) and Gillberg (1984) suggest that once

sleep inertia is overcome, performance closest to the nap will be maximally improved, followed by a subsequent decline.

Overall, these studies suggest that a nap taken earlier in the night shift may provide the advantages of a short period of sleep (as long as it is not too far in advance of critical operational times) while also minimising the negative consequences that occur with deeper sleep at the circadian nadir, and with greater prior wake.

The final issue to consider is the ability of a nap to improve operational performance. Currently, there do not appear to be any studies that have measured operational performance pre and post a workplace nap. Even so, there is evidence of faster responding and decreased lapsing subsequent to napping, which is likely to be of operational relevance. The decreased occurrence of neurophysiological signs of sleepiness would also be likely to influence workplace performance. Nevertheless, these proposed relationships have not yet been demonstrated.

1.6 Air Traffic Control

The following section of this chapter provides an overview of the job of an air traffic controller. To place the present study in context, the research that has addressed issues of sleep loss, the associated changes in performance and alertness, and countermeasures in air traffic control are reviewed and discussed.

1.6.1 What is Air Traffic Control?

The fundamental task of controlling is to ensure the safe and efficient passage of aircraft, both on the ground and in the air. To achieve this there are various controlling positions, which due to the specific airspace they control and the systems they work with, often require the application of different controlling methods in order to separate aircraft. Generally either a minimum vertical distance or a minimum horizontal distance must be applied between two aircraft. In New Zealand there are four main controlling jobs. These are aerodrome control, terminal approach control, area control, and oceanic control. Other countries have similar positions, that would perform similar tasks, but different job titles are sometimes employed.

Depending on the number of aircraft movements, and the type of operations occurring, an airport might have an aerodrome controller to ensure the safe separation of aircraft from each other. At small aerodromes with low traffic levels, and/or few commercial

operators, aircraft are often responsible for separating themselves from each other. When an airport warrants an aerodrome controller, the controller would direct aircraft taxiing on the ground, as well as those taking off and landing via VHF radio. Maintaining the required separation between aircraft is achieved visually, with the controller looking out the control tower window to confirm the locations of aircraft.

Once an aircraft has taken off, and is leaving the surrounding airspace (known as a Control Zone or CTR), it is likely to be passed to a terminal approach controller. This normally only occurs for aircraft operating under instrument flight rules (flying with reference to instruments), or aircraft operating under visual flight rules (flying with reference to the ground or water) that choose to remain in controlled airspace. Terminal approach controllers ensure that traffic descending down into, and climbing out from, a CTR are separated from each other. The airspace they control (Terminal Control Area or TMA) generally sits above the CTR (see Figure 1-1). To keep traffic separated, terminal approach controllers use a combination of radar and standard procedures that ensure separation of aircraft from other flights and terrain. Terminal approach controllers communicate with aircraft via VHF radio and rely heavily on information from the radar screen to ensure minimum separation standards are maintained.

There are two types of radar. Both primary and secondary radar are used in New Zealand. The primary radar emits signals that bounce off objects in their path. The radar system then detects the echoes of the original signal, and uses this information to measure the distance of the object from the radar. The secondary radar sends signals that automatically trigger a return signal from a device in the aircraft called a transponder. The returned data includes the height of the aircraft and a special code that can be electronically matched to flight plan details. Secondary radar information allows controllers to determine the location of aircraft and also its height, speed, and other aircraft details.

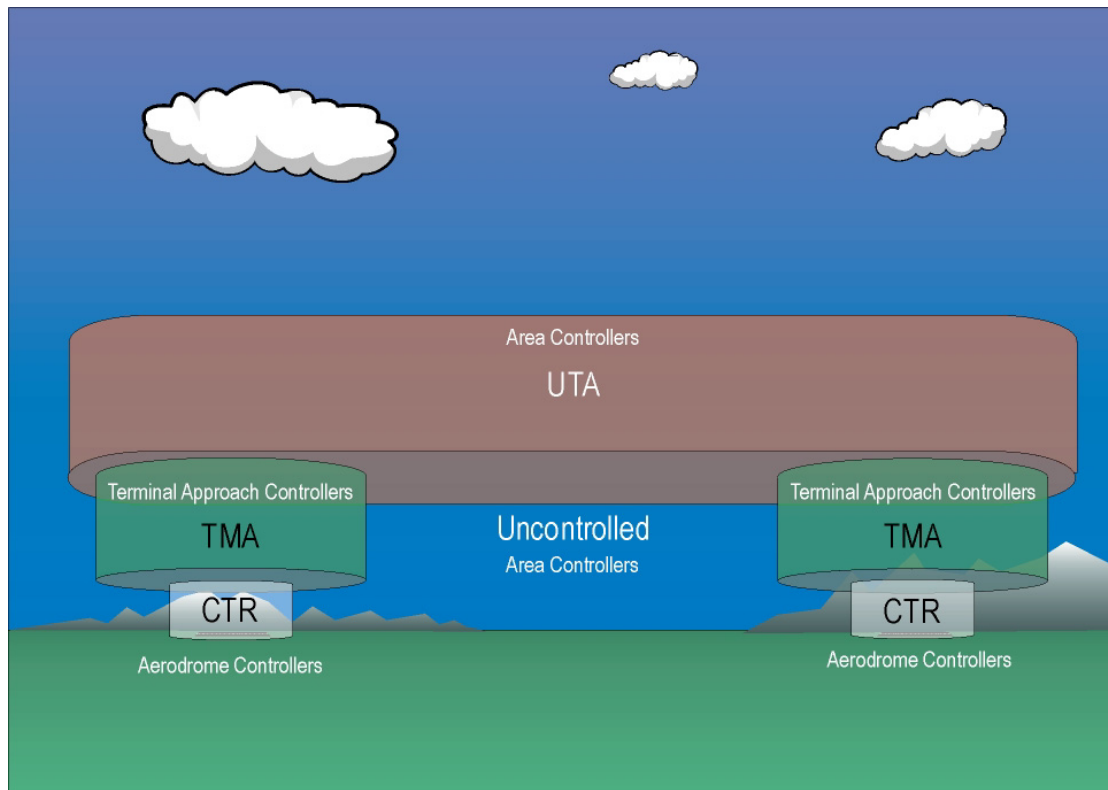


Figure 1-1: Division of Airspace by Controlling Position and Location

Area controllers are responsible for separating aircraft en-route between terminal control zones, while in upper terminal airspace (UTA). They also provide traffic and weather information to certain aircraft in some types of uncontrolled airspace (see Figure 1-1). They too use radar as their primary means of separating aircraft, but the minimum horizontal and vertical distance that must be maintained between aircraft are normally greater than those in a TMA. The physical amount of airspace they control is likely to be much greater than that of terminal controllers, but the density of the traffic is less. Normally aircraft follow standard flight routes between locations, therefore assisting the controller to separate aircraft from each other. Depending on the time of day and traffic levels, area airspace may be broken up into sectors, with one area controller responsible for a single sector. When traffic levels decrease, such as during the night hours, the sectors are often grouped together as one and a single controller becomes responsible for a much greater portion of airspace. Part of the task of any controlling position is to update the flight plans of aircraft. When traffic levels are high, often the controlling tasks are split, with one controller acting as a planner and assisting the controller responsible for providing aircraft separation.

As an aircraft approaches its destination, it is passed from the area controller to the destination terminal approach controller then eventually, to the aerodrome controller. Flights are handed from one controller to another using electronic and manual methods. When a flight passes outside the domestic airspace of a country, it is handed to an oceanic controller. Due to the limited distance over which radar can be effective oceanic controllers use advanced satellite technology to communicate with and separate aircraft. The minimum distances that must be maintained between aircraft in oceanic airspace are greater than those applied under radar control. Until satellite technology was employed, communication between the oceanic controller and the aircraft was entirely by HF radio which is still used as a backup. The data received from the satellite system are plotted onto a display providing the oceanic controller with a visual picture of the traffic situation.

Despite the diverse types of airspace and methods of separation applied by the various controlling positions, all controllers utilise a broad range of cognitive skills to maintain the efficient and safe movement of aircraft. Individuals seeking to be air traffic controllers are assessed prior to acceptance into a training programme to determine if they have the attributes necessary to acquire these cognitive skills. In New Zealand there is a single air traffic control service provider (Airways Corporation of New Zealand Ltd.), which also trains new air traffic controllers. In addition to passing a series of screening tests, potential air traffic controllers must meet certain medical requirements and then undergo a 12 month period of training. The training involves learning about the air traffic control systems, rules and regulations, and basic visual and radar controlling techniques. Newly trained controllers are normally employed in aerodrome control positions and receive on the job training to complete an aerodrome control rating and a licence to operate. As positions become available, those wishing to work in the radar control environment are initially employed in an area control position. Further training is required on radar control procedures, and the local operating procedures. Each controlling position requires further training and the issue of the appropriate rating. The New Zealand Civil Aviation Authority licenses air traffic controllers in a similar manner to pilots. Like pilots, controllers must complete regular competency based checks and undergo annual medicals to ensure they are fit to work.

1.6.2 Is Fatigue a Known Problem in Air Traffic Control?

It has been previously mentioned that air traffic control, due to the services that are provided, inherently involves shiftwork and therefore also the associated problems. A recent study by Della-Rocco (1999) attempted to determine the extent of fatigue in ATC, through the analysis of two separate databases. A review of voluntary incident reports made to the Aviation Safety Reporting System (ASRS) between 1988 and 1996 identified 153 reports (2.7% of the total reports) as mentioning fatigue. This is similar to an earlier study (Roske-Hofstand, 1995), which stated that 3% of all air traffic control ASRS reports submitted between 1986 and 1994, listed fatigue as at least one of the causal factors. The second database examined by Della-Rocco (1999) was the FAA's Operational Error/Deviation System (OEDS) database, which contains mandatory reports on operational air traffic control errors. The relationship between shift related variables and operational errors was investigated. Only the length of time on position was found to have a very small (.04), but significant, correlation with the severity of the error. No other relationships reached significance but many analyses were hampered by incomplete or poor data.

In 1990 a review of the UK's Confidential Human Factors Incident Reporting Programme (CHIRP) was conducted by the Committee on Regulation of Air Traffic Controllers' Hours (CRATCOH) (1990). It focussed on the 215 voluntary air traffic control reports received by the database since its establishment in 1986. Of those, 28 (13%) referred directly to fatigue and fatigue-induced problems.

Due to methodological issues associated with voluntary reporting, and data quality issues, it is likely these reported levels are an underestimation of the scale of the problem. Whether they are or not, as Heslegrave et al. (1996) states "in few other occupations do performance changes as a result of fatigue have the potential public concern and consequences as they do in air traffic control" (pg. 183).

1.6.3 Work Schedules and Associated Sleep Loss in Air Traffic Control

In the United States, Canada, Australia, and New Zealand a counter-clockwise (or backward), rapid rotation of shifts is commonly employed. This pattern of work involves subsequent shifts starting earlier than the previous one, reducing the amount of time between periods of work and effectively compacting the working week. Normally, 4-5 days are worked followed by 2-3 days off before the cycle repeats. A typical pattern

seen in New Zealand would be an evening shift, afternoon shift, early morning shift, followed by a night shift, with two days off. The night shift commences late in the evening on the day that the early morning shift finishes. This short break between shifts is known as a “quick turn around”.

To some extent a rapidly-rotating pattern of work is considered advantageous because it minimises the chronic desynchronisation associated with schedules that rotate more slowly (Luna, French, Mitcha & Neville, 1992), largely because an individual's circadian clock remains diurnally orientated (Dahlgren, 1981; Comperatore & Krueger, 1990; Luna, 1997). Further, permanent night shifts, or longer periods of night shifts, are considered undesirable, not only because of the physiological and social consequences but also because the low workload at night is argued to adversely affect the proficiency of air traffic controllers (Melton & Bartanowicz, 1986; Hopkin, 1988; Luna et al., 1992; Luna, 1997). Probably the main reason for the rotating schedule, particularly the backward rotation, is support for it from controllers (Schroeder, Rosa & Witt, 1998). The compressed work week on a backward, rapidly-rotating schedule allows a longer period of time between working weeks, and the rapid rotation of shifts during the work week provides some time off at all phases of the 24 hour day. Social isolation is thus minimised (Luna, 1997).

Despite support from controllers for these types of work schedules, there are concerns that they may lead to less sleep, greater disruption to an individual's circadian rhythms, and result in more complaints of fatigue compared to a clockwise rotating schedule.

In the United States, the pattern of two evening shifts, two day shifts and a night shift (2-2-1) is common. A comparison of the 2-2-1 shift to five consecutive day, evening, or night shifts across a seven day period showed there was no significant difference in the amount of sleep obtained (Saldivar, Hoffman & Melton, 1977), although across the five working days controllers on the rotating shifts averaged 18 minutes less sleep per 24 hours. Cruz and Della-Rocco (1995a; 1995b) noted similar findings. The total amount of sleep obtained across the week did not differ between the five day and 2-2-1 schedules. Although, controllers obtained significantly more sleep on a 2-1-2 pattern (two afternoons, midday shift, two evenings). Nevertheless, the amount of sleep obtained in each 24 hour period decreased across the week for both the 2-2-1 and 2-1-2 schedule. Schroeder et al. (1998) also found a decrease in the quantity of reported sleep across both the backward rotating schedule and four 10 hour days.

Rhodes et al. (1996) determined that across a number of different backward rotating shifts, and a schedule of five consecutive night shifts, controllers got 25-30% less sleep than they did on their days off. Polysomnographic data indicated early onset of SWS, REM sleep, and increased levels of SWS, suggesting individuals experienced an increased homeostatic sleep drive because of their sleep debt. Much of the reduced total sleep across a week of the backward rotating schedule can be attributed to the quick turn around between a day (or morning) shift and the night shift, during which controllers report the shortest sleep of the working week (2.3-2.7 hours, Rhodes et al. (1996); 2.4 hours, Cruz and Della-Rocco (1995a; 1995b); 3.5 hours, Saldivar et al. (1977); 3.75 hours, Schroeder et al. (1998)). Early starting shifts are also responsible for reduced sleep quantity and quality in air traffic controllers (Costa, Schallenberg, Ferracin & Gaffuri, 1995). With day shifts that start at approximately 0700 hours, the average total sleep length is between 5.6 and 6.3 hours, with the shorter durations reported prior to the earliest starting shifts (Cruz & Della-Rocco, 1995a; 1995b; Della-Rocco & Cruz, 1995). Mollard et al. (1997) noted that early shifts resulted in earlier awakenings but no change in bed times, thus producing the shortened sleep episodes. Despite the sleep loss, approximately 50% of controllers working a backward rotating schedule reported that they were satisfied with the schedule they worked (Saldivar et al., 1977).

In the study comparing the 2-2-1, 2-1-2, and straight day shifts, subjective ratings of fatigue were made on the SSS at the beginning of each work day and prior to the drive home (Cruz & Della-Rocco, 1995a; 1995b). Ratings of sleepiness were consistently higher prior to driving home than at the beginning of the shift, and sleepiness ratings were higher on the last day of the work week compared to the first three days of the schedule. Across the entire work week there were no significant differences between the three different schedules.

Rhodes et al. (1996) measured the performance of controllers on tests from the Walter Reed Performance assessment battery at the beginning, middle, and end of shifts across the working week. Across backward rotating schedules, and those including a quick turn around, performance generally worsened across the week. However, whether this change was statistically significant is not clear. Schroeder et al. (1998) found similar results to those above, with a steady increase in reaction time performance and less positive and more negative mood across the days of a 2-2-1 schedule. While these studies suggest reduced sleep and a decline in functioning across the work week, they do

not provide strong support for the notion that rapidly, backward rotating shift schedules are necessarily worse than other roster patterns.

Air traffic controllers working a rapidly-rotating schedule face a particular challenge on the night shift, as they must function through the nadir of their diurnally orientated circadian rhythms (Luna, 1997). The consequences of working at night for alertness and performance in non-air traffic control environments have already been discussed. In studies involving air traffic controllers, higher subjective ratings of fatigue (Grandjean, Wotzka, Schaad & Gilgen, 1971; CRATCOH, 1990; Luna, French & Mitcha, 1997) and confusion, and less vigour are reported on the night shift compared to the day shift (Luna et al., 1997). Smith et al. (1971) note greater subjective fatigue and sleepiness at the end of the night shift than the beginning, and a greater difference pre- and post-shift compared to day or evening shifts. Lower activity levels and possibly sleep have been recorded on the night shift (Luna et al., 1997), as well as an increased incidence of napping at work for those who work night shifts, compared to those who do not (Cruz, Della-Rocco & Hackworth, 2000). Mollard et al. (1997) also found ratings of sleepiness to be highest after the night shift and when getting up early, but controllers reported an early starting day shift, rather than the night shift, to be the hardest shift. This may be explained by the quick turn around that preceded the day shift.

Folkard et al. (1984) report the occurrence of night shift paralysis (a momentary, incapacitating paralysis associated with a high pressure to sleep) is four times higher for controllers whose shift schedule enforces a quick turn around between the morning and night shift, compared to those who schedules do not include this pattern of work.

Few studies have objectively investigated the performance of controllers on the night shift. Heslegrave et al. (1996) note that controllers reported impairment in performance at the end of an 8 hour night shift equivalent to performance at the end of a 12 hour day or evening shift. If the night shift was extended, performance was rated as severely impaired by the end of the shift. Findings from the same study suggest that the cognitive performance of controllers was more impaired in the middle of the night shift, at the time of the circadian nadir, than at the beginning or end of the shift (Rhodes et al., 1996), and over consecutive night shifts, performance declined. Schroeder et al. (1998) found the slowest reaction times and greatest number of errors to occur when controllers worked the night shift, but because the night shift was always the last shift of the week, it was not possible to attribute this solely to shift timing.

An additional concern is the safety of individuals getting to and from work. Nearly twice as many controllers (27%) reported falling asleep driving to or from work after working 1-4 consecutive night shifts compared to those who hadn't worked a night shift in the last year (Heslegrave et al., 1996). Cruz et al. (2000) report a higher incidence (44%) of controllers falling asleep while driving to or from work if their schedule included night shifts. Both studies reported a two fold increase in the likelihood of having fallen asleep when driving for controllers who work night shifts compared to those who do not.

In the study by Rhodes et al. (1996) EEG data were collected from 8 controllers in 30 minute segments across consecutive night shifts. Due to a large amount of lost data, statistical comparisons could not be made, but trends suggested a general decrease in power across the night shift, higher relative delta power at the beginning and end of the night shift, and an increase in relative theta power across the night shift. The decrease in total spectral power across the night is somewhat surprising, but included frequencies from 1.5-60 Hz, so a reduction in power in the higher frequencies may have masked a general increase in power at the lower end of the spectrum. The increase in power in the delta and theta band at the end of the night shift suggests increased sleepiness, and possibly microsleeps at this point in time. Lille and Chéliout (1982) also recorded physiological variables, including EEG and ECG, from air traffic controllers across a day and a night shift. After midnight on the night shift, heart rate was higher and delta activity was more prominent, compared to the day shift. The authors explained these differences as being due to the greater physiological cost of maintaining efficient performance during periods of lower workload.

In a diary study of British air traffic controllers, Spencer et al. (2000) demonstrated that the subjective level of workload was significant in predicting self-rated fatigue at the end of a period on duty. There were also interactions of the level of workload with the continuous time awake and the time spent continuously controlling. At low levels of workload there was no effect of time awake on ratings of fatigue, whereas at medium levels of workload fatigue ratings increased once time awake exceeded 10 hours. Under high workload conditions, fatigue ratings increased after only 6 hours since the last sleep. The effect of time spent continuously controlling only influenced fatigue ratings under conditions of high workload and periods of controlling greater than 2 hours. Other factors found to predict fatigue levels included the time of day and the time on duty (length of total work time). Because the time of day and time on duty did not interact,

they can be presumed to be additive, such that the highest levels of fatigue are seen at the end of a long duty that finishes around 0600 hours. This highlights the need to consider the maximum length of the night shift. Further, although the level of workload seems to be important in predicting ratings of fatigue, the night shift is normally a time of low workload. Air traffic controller errors have been found to be more prevalent under conditions of moderate to low workload (Schroeder & Nye, 1993). Moreover, most of these errors were due to inattention, failure to maintain situational awareness, misuse or misinterpretation of radar data, or inadequate vigilance (Redding, 1992).

In addition to workload, there are other factors that potentially interact with the arrangement of work to influence the occurrence of sleepiness, such as age. Older controllers are reported to find their work schedules more difficult to cope with (Rhodes, Szlaptis, Hahn, Heslegrave & Ujimoto, 1994; Heslegrave et al., 1996), which may be related to changes in the circadian system that lead to a reduction in the amount of sleep older shiftworkers are able to obtain (Gander, Nguyen, Rosekind & Connell, 1993). It is also important to note that older individuals are poorer at maintaining their performance on attention, and monitoring tasks (Thackery & Touchstone, 1981), and generic psychomotor, perceptual, and cognitive tasks (Rhodes et al., 1996) associated with controlling. More recently, age has been strongly related to performance on simulated controlling tasks (Becker & Milke, 1998). However, it is also suggested that older controllers compensate in other ways, such as using their experience, so that operational performance is not necessarily affected.

1.6.4 Countermeasures in Air Traffic Control

Roske-Hofstrand (1995) stated that, particularly in the US air traffic control system, fatigue is often viewed as an individual experience rather than a professional hazard. This provides a possible reason why countermeasures to fatigue have not been more urgently investigated in this environment. Given the above findings indicating sleep loss across the backward, rapidly-rotating shift schedule and increased subjective and objective sleepiness, and decreased performance on the night shift, it is not surprising that several researchers have recommended napping as a fatigue countermeasure in the air traffic control environment (Rhodes et al., 1996; Luna, 1997; Cruz et al., 2000).

Findings supporting napping as a possible countermeasure to fatigue have been previously discussed, but these studies were conducted in a laboratory setting or with a

different occupational group. There appears to be only one study that has investigated the usefulness of this countermeasure for air traffic controllers (Della-Rocco, Comperatore, Caldwell & Cruz, 2000). In a simulated working environment, 59 air traffic controllers completed three early morning shifts, and then a night shift after an 8 hour break (during which no sleep was allowed). Using a between-subjects design, naps of either 120 or 45 minutes, both ending at 0345 hours, were allowed on the night shift. Performance after these naps on a simulated air traffic control task and vigilance task were compared to the performance of air traffic controllers who did not nap. Napping opportunities and the break for non-napping controllers was analysed for sleep. These findings are not reported, other than the exclusion of two non-napping controllers due to sleep in excess of 30 minutes.

Measures from the simulated air traffic control task showed no effect of the nap conditions or circadian influences. Correct responses on the vigilance task increased after the long nap compared to the no-nap condition at tests conducted both immediately after the nap, and at the end of the shift. Differences between the long nap and the short nap were evident at the end of the shift. For the number of false responses on the vigilance task, the long nap again improved performance at both time points compared to the non-napping condition, while the short nap resulted in fewer false responses compared to no nap at the end of the night shift.

1.7 Chapter Summary and Study Aims

Air traffic controlling involves working within an automated system that places the human operator in a monitoring role. In addition, controllers must perform a diverse range of cognitive tasks, all of which require attention and vigilance as a core component (Dinges & Kribbs, 1991). Therefore to perform these job-related skills and tasks effectively a controller must be able to remain alert, irrespective of the time of day or night (Saldivar et al., 1977; Melton & Bartanowicz, 1986; Price & Holley, 1990; Koenig, 1995; Schroeder, Rosa & Witt, 1995; Heslegrave et al., 1996).

As this chapter has shown, shiftwork-related sleep loss can degrade performance and neurophysiological alertness. Napping is a potential countermeasure to sleep loss, but only one previous study has investigated napping as a countermeasure in air traffic control. However, this study was not conducted in the operational environment.

The present study therefore aims to determine the efficacy of napping on the night shift for maintaining the alertness and performance of operational air traffic controllers.

Using a within-subject design, controllers are monitored over four shift cycles. Each shift cycle is differentiated by the timing of the rostered night shift (early starting or late starting), and whether a workplace nap is allowed or not. Chapter 2 discusses the design and methodology of this study in detail.

Each of the subsequent chapters then addresses a series of related questions. Chapter 3 examines the sleep consequences of backward, rapidly-rotating work schedules, including whether the length, timing, and quality of sleep episodes change across the shift cycle, and whether air traffic controllers are cumulatively and/or acutely sleep deprived when working the night shift. It also investigates whether the pre-planned workplace naps affect the length, timing, and quality of post night shift sleep. Chapter 4 describes the polysomnographic analysis of the napping opportunities, including how much sleep controllers were able to obtain during the nap, and whether the amount and quality of nap sleep are related to the level of acute sleep loss, length of prior wake, and the timing of the nap. This chapter also examines whether there is any evidence for a “first night effect”, i.e. whether sleep obtained during the first study night differs from sleep obtained on subsequent study nights. Controller’s assessments of their nap sleep are compared with the polysomnographic measures, and the occurrence of sleep outside the napping opportunity is evaluated. Chapter 5 examines the performance changes across the night shift and the effects of napping. It begins by addressing whether sleep and circadian factors are reliably associated with performance levels and subjective sleepiness at the start of the night shift. The influence of architectural aspects of nap sleep, the amount of sleep obtained, and the opportunity to nap, on performance and subjective sleepiness are then considered. The final question in this chapter is whether additional sleep related factors have a predictive relationship with performance and subjective sleepiness subsequent to the nap. Similar questions are then posed in Chapter 6, but the focus is on the outcomes for neurophysiological alertness at the end of the night shift. Initially, the amount of analysable EEG data are discussed. It is then examined whether the amount of nap sleep, the architectural aspects of this sleep, or the opportunity to nap can improve, or limit a decline, in neurophysiological alertness. The last question of this chapter deals with the influence of additional sleep related factors on neurophysiological alertness.

The final chapter brings together the findings of chapters 3-6, and discusses these in detail, particularly with regard to their collective meaning and their relationship to previous research. Because this study was conducted in an operational environment, recommendations are then made for controllers, the employing organisation, and the regulator. Finally, future research issues that were highlighted by the present research are considered.

CHAPTER 2

METHOD

2.1 Participants

All air traffic controllers working in Christchurch Area control were invited to participate in the study. Thirty five controllers were approached and 28 returned positive responses, resulting in a response rate of 80%. Seven controllers declined to participate in the study for a variety of reasons. These included home and personal commitments, current difficulties coping with night shifts, not being currently rostered on night shifts, and having other training responsibilities.

2.2 Measures

This section describes the measures chosen for use in the present study along with the reasons for their choice. Initial data processing is outlined, but detailed information on variables and analyses are presented in the relevant subsequent chapters.

2.2.1 *Actigraphy*

To determine the impact of the backward, rapidly-rotating shift schedule on the sleep of controllers, and in order to investigate whether individuals commencing the night shift were sleep deprived, controllers wore an activity monitor (*Mini-Mitter™*) across an eight day study period (see Figure 2-1 for further detail). The *Actimatch™* is a small, lightweight device approximately the size of a wristwatch that utilises an accelerometer to measure the occurrence and degree of motion. Such devices have been proven highly sensitive to sleep, with correlations of approximately 0.96 to polysomnographically scored sleep in both normal and sleep disordered populations (Sadeh, Hauri, Kripke & Lavie, 1995; Kushida et al., 2001). However, unlike polysomnography, actigraphy cannot discriminate sleep from still wakefulness, resulting in an overestimation of total sleep time and sleep efficiency (Kushida et al., 2001). Despite these limitations actigraphy is a widely accepted, recognised means of monitoring sleep objectively over extended periods of time in a non-invasive, low-cost manner (Ancoli-Israel, 2000).

Using digital integration, the sensor in the watch measures all accelerations and integrates frequency of movement with intensity to produce a voltage. Increased speed and frequency of movement produces greater voltages. These voltages are sampled at 32 Hz

are stored as activity counts. In the present study, data were accumulated for a period of one minute before being stored, and then the counter was reset to zero.

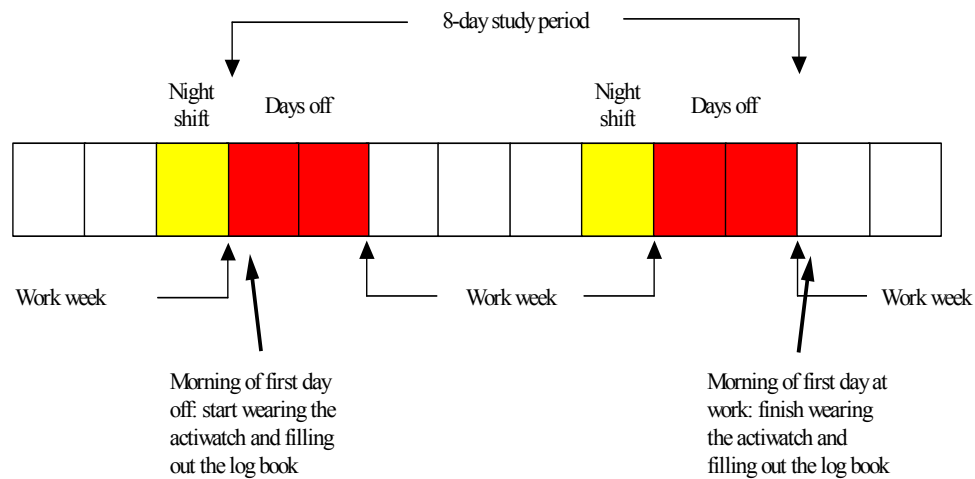


Figure 2-1: An Eight Day Study Period: Indicating Times of Wearing an *Actiwatch*TM and Completing the Logbook

The accelerometer in the *Actiwatch*TM is omnidirectional for sensitivity to movement. However, the shape of the sensor makes it more sensitive to movement in certain directions (vertical movement of the watch when located in a normal position on the wrist). Because of this sensitivity in one direction, standardised placement of the watch is important. In addition, no two accelerometers give identical readings for the same motion. To minimise this problem the *Actiwatch*TM has a calibration coefficient to normalise data between watches. This removes most variation between units. However, relative levels of activity are thought to be more reliable measures between watches when making inferences than absolute values. This is also largely due to variations in positioning the watch. Because watch location and positioning are so important in reproducibility of the data, study participants were shown how to place the watch on their wrists by the experimenter. This was in conjunction with verbal and written instructions (see Appendix A).

While wearing the *Actiwatch*TM, participants completed a sleep diary to provide reference information for the actigraphy output. This sleep diary was developed from log books used in studies by Gander (1998a; 1998b; 1998c; 1998d) and modified in consultation with other New Zealand shift workers and air traffic controllers (see Appendix B).

Participants were asked to record, using the 24 hour clock, the times they started trying to sleep, when they think they fell asleep, when they think they woke up, and when they finished trying to sleep. In addition, they were asked to record the times at which they started and finished work and to mark whenever they took the watch off.

At the end of a study period, *Activatches*TM and logbooks were collected from the controllers and data downloaded via a serial port connection to a PC. Data were analysed using the *Sleepwatch*TM software in conjunction with the logbook data. The software algorithm scores each one minute epoch between bed-time and get up time as either sleep or wake. To be identified as a wake epoch the activity count for that particular epoch, as well as the epochs immediately prior to, and after it are compared to a pre-set sensitivity value. If the activity count exceeds the threshold value the epoch is scored as wake. For each epoch the total sum of all activity counts is calculated for a period of approximately five minutes, centred on the scored epoch. The contributions of epochs adjacent to the scored epoch are one fifth of their activity count. Epochs two minutes from the scored epoch contribute one twenty fifth of their counts.

The sensitivity threshold in *Sleepwatch*TM could be set at a high (80), medium (40) or low value (20). A high sensitivity, or low activity count, requires relatively little movement before wake is scored, while a low sensitivity, or high activity count, has the opposite effect of only scoring wake when much larger amounts of movement occur. The population involved in the present study were sleeping both during the day and at night, and the expectation was that day sleep would be lighter and more fragmented, compared to the deeper and less broken night sleep. Choosing a low sensitivity threshold could therefore result in an underestimation of the amount of sleep occurring in daytime sleep episodes, which are expected to be more restless. On the other hand, utilising a high sensitivity setting would potentially overestimate the amount of sleep occurring during night time sleep periods. On this basis a medium sensitivity threshold was deemed most appropriate as it would not bias the output towards either day or night sleep.

Using this sensitivity threshold with one minute epochs, a single epoch with a total activity count of 40 or more, would be scored as wake. If the activity count was less than 40 the two immediately adjacent epochs would contribute one fifth of their total activity counts and the two epochs further out would contribute one twenty fifth of their activity counts. If this total exceeded 40 the central epoch would be scored as wake.

After setting the medium sensitivity threshold the raw actigraphy data were viewed in *Sleepwatch*TM alongside the logbook data and a range of variables were either set or generated.

The variables were then copied directly from *Sleepwatch*TM to an Excel file, which represented each individual's eight day period of actigraphy. These files were then imported to *SPSS*TM and *SAS*TM databases for statistical analysis.

2.2.2 Psychomotor Vigilance Test

The performance of controllers was assessed by the use of a validated, reliable and sensitive test of vigilance and reaction time (Dinges, Mallis, Maislin & Powell, 1998). The Psychomotor Vigilance Test (PVT-192), is based on a reaction time test developed by Wilkinson and Houghton (1982) and the analyses are designed by David Dinges and John Powell. The testing device is now produced by CWE Inc., and is distributed by Ambulatory Monitoring, Inc. It is widely used in studies of fatigue, sleep deprivation, and performance and has been shown to be sensitive to changes in alertness associated with circadian phase (Dinges, 1991; Wyatt et al., 1997), levels of acute sleep deprivation (Dinges et al., 1994), increasing cumulative sleep deprivation (Dinges et al., 1997; Rowland et al., 1997) and the combined effects of shiftwork with time zone changes (Rosekind et al., 1994). The advantages of using the test in the present study include its known psychometric properties, ease of use in an operational context, and lack of practice effects. The PVT was designed for evaluating an individual's ability to sustain attention and respond rapidly to events (Dinges & Powell, 1985), making it a suitable test for this occupational group.

The test requires individuals to respond as rapidly as possible to the presentation of numerals on a 4 digit LED numeric display. It is a "simple" reaction time test, in that it does not involve a choice between responses but does provide feedback on performance via the numerals on the LED display, which are the time in milliseconds taken to react.

Using software provided with the PVT-192 hardware, the test was initialised prior to each use, with test length set at 10 minutes, inter-stimulus periods able to range between a minimum of 2000 milliseconds and a maximum of 10000 milliseconds, and a lapse categorised as any response longer than 500 ms (which is twice that of the mean response time). These values are the standard test parameters used in the validated version of the PVT (Powell, 1999).

Two buttons for responding were available on the device and the test was initialised so that only the button associated with the individuals dominant hand was activated for responding. Instructions to subjects were developed from the standard test instructions and were presented to all subjects on a printed card to standardise the information (see Appendix C)

Each subject completed a 1 minute trial of the PVT prior to the study. The PVT shows no practice effects (Dinges, 1991), therefore a brief trial of the test was sufficient to familiarise subjects with the structure of the test before data collection began. Prior to beginning the test a Visual Analog Scale (VAS) was presented on a LCD display that is located below the numeric LED display. The stimulus “Sleepy?” was presented along with a 10 point scale anchored “Yes” and “No”. Participants used the left button on the PVT to move the cursor to the desired location on the scale, and then pushed the right button to register their choice. This scale was presented again at the completion of the test.

Along with the reaction events being recorded by the PVT device, the voltage created by the commencement of a stimulus was output to a channel recorded on-line by the ambulatory neurophysiological *Embla*TM recorder. This was followed by a second signal that occurred when the participant responded to the stimulus. The result was a square wave that corresponded to the reaction time for each stimulus.

Figure 2-2 below shows a participant completing the PVT while combined neurophysiological and reaction time data are recorded online. All tests were completed in a room without distractions. The test box could be held in both hands or placed on a desk. Subjects could choose to use either their index finger or thumb to push the response key, but were required to use the same method of responding for all tests.



Figure 2-2: Individual Completing the PVT While Neurophysiological Data are Simultaneously Recorded Online

After each study night, data from the PVT were downloaded to a PC in ASCII format. Each night's data was then separated into individual tests and stored on the hard drive of a laptop. In October 2000, Ambulatory Monitoring, Inc., issued a notice to all PVT-192 users that a minor error had been identified in the firmware of the PVT. The error resulted in the recording of an incorrect reaction time for the first event of every trial. The erroneous value was always within the expected range of values, so had remained undetected for a number of years. To rectify the problem, PVT analysis software had been altered to discard the first value of each trial. This updated *REACT*TM software was employed to discard the first value of each trial in the present study and to then output a report containing a number of summary variables. Data from each report were copied to an Excel spreadsheet, and then joined with all other outputs from each trial. The Excel spreadsheet was then imported into *SPSS*TM and *SAS*TM databases for statistical analysis.

2.2.3 *Neurophysiological Data*

The *Embla*TM ambulatory recorder (*Flaga hf*TM) collected nine channels of physiological data that was used for three purposes: the Alpha Attenuation Test, to determine neurophysiological alertness, and to score the sleep of participants who were napping.

Gold *Grass*TM electrodes were placed according to the 10-20 system at the EEG sites, C4, P4, O2, Oz, and A1. The recording montage created in *Somnologica*TM included the following five channels: C4-A1, O2-A1, O2-Oz, O2-P4, and Oz-P4. These channels were chosen for a variety of reasons but included; the best detection of the slower EEG frequencies associated with decreased alertness, to allow the scoring of sleep, to minimise known causes of artefact, and to include both widely spaced referential and closely placed bipolar channels. Ideally, a full 10-20 montage would have been used but this was not feasible or sensible given the time constraints associated with connecting such a montage and the additional requirements this would place on participants. Five channels were used as a compromise.

The C4-A1 channel was used to score sleep according to standard criteria (Rechtschaffen & Kales, 1968) and therefore allow comparisons to be made with other studies. The O2-A1 channel is recommended as an adjunct to C4-A1 for sleep scoring. This is to provide redundancy in the event the C4 electrode loses contact as well as the fact that its occipital location provides a good view of changing alpha activity, which is important in determining sleep onset (Harris, 1991). It is also a referential arrangement and widely spaced in contrast to the remaining three channels. The choice of location of the three bipolar channels was largely based on the need to detect the slower frequencies of alpha and theta seen with decreasing alertness. Alpha is known to be prominent in the occipital area and theta is also generally seen posteriorly (Niedermeyer, 1999). These rear, closely placed electrodes are also less likely to record eye movement artefact (Reilly, 1999) and their use provides a combination of referential and bipolar channels within the montage. Because inter-hemispheric differences were not of interest, and because comparisons were to be made within subjects, electrodes were all placed on the right side of the head. Handedness was recorded in the event of outliers.

Each electrode site was gently abraded with *Omniprep*TM to reduce impedance to less than 5000 ohms. Conducting electrode cream, which once dry, also helped hold the electrode

in place, was applied to the cup of the electrode (*EC2™*). For further stability the electrode was covered with a square of adhesive *tegaderm™* and strong *hypafix™* tape.

Left and right Electroculogram was recorded from the left and right outer canthus, with the left electrode positioned 1 cm up from the horizontal plane and the right 1 cm down. Both electrodes were identical to, and applied in the same manner, as the EEG electrodes. They were referenced to the same auricular reference (A1). These electrode sites allow the recording of vertical and horizontal eye movements as well as the detection of electrode artefact (Carskadon & Rechtschaffen, 2000). EEG activity reflected in the EOG is seen as in phase movements on both channels and electrode artefact will also register as in phase or only in one channel. In contrast eye movements produce out of phase deflections. These electrode positions are also recommended for standardized sleep scoring. The disadvantage of this electrode arrangement is the inability to differentiate between horizontal and vertical eye movements, and that oblique eye movements produce relatively flat tracings (Rechtschaffen & Kales, 1968). In addition, vertical eye movements are not as easily discriminated with this electrode arrangement (Harris, 1991). However, in this study slow rolling eye movements are of the greatest interest. Such movements are recorded more consistently in the horizontal axis (Santamaria & Chiappa, 1987), and are therefore easily discernable with these placements.

Electromyogram (EMG) was recorded bipolarly from two electrodes positioned on the mentalis/submentalis muscles. Again gold *Grass™* electrodes were used and applied in the same manner as electrodes at other sites. However, if the participant was male, due to facial hair, collodion was used in addition to the other methods to help secure the electrodes. EMG is considered important to record when scoring sleep because of its importance in determining REM sleep stages (Rechtschaffen & Kales, 1968), but because of the short periods of sleep being recorded REM sleep was unlikely to be seen. It was principally recorded to assist in the identification of movement and therefore in screening the other physiological channels for movement artefact.

Electrocardiogram (ECG) was recorded for the purposes of identifying any ECG artifact that appeared in other recorded channels. Disposable *3M Red Dot™* electrodes were placed in the left and right clavicular space and connected to snap on leads.

All electrode wires were grouped together at the back of the head then the wires were held in a bundle with plastic cable binding. This was done to reduce movement artefact caused by wires moving or being pulled and to improve common mode rejection. The top of the cable was taped to the participant's clothing to hold it secure. The bottom of the cable fed into a pouch positioned on the participant's waist. From here each electrode wire was connected to a multi-purpose cable, with electrode inter-connectors employed for channels using the same site. The leads of the multi-purpose cables fed into the *Embla*TM recorder. The recorder was held in a separate bag along with the battery pack, which was also positioned on the participant's waist on the hip opposite to the bag holding the connectors and multi-purpose cables.

The analogue inputs to the *Embla*TM were sampled simultaneously at 2000 Hz and digitised to 16 bit resolution by a Sigma-Delta AD converter. Data were then down sampled to 200 Hz. At this time the input signal is filtered sharply so that it does not contain frequencies greater than the Nyquist frequency. The signal was then digitally pass-band filtered at 0.5-90 Hz and stored either on the hard drive of a laptop or to a PCMCIA card in the *Embla*TM recorder.

Alpha Attenuation Test

The Alpha Attenuation Test (AAT) was developed to meet the need for a simple objective test to assess sleepiness in non-laboratory settings. Other commonly used objective tests for assessing sleepiness are the Multiple Sleep Latency Test (MSLT) and the Maintenance of Wakefulness Test (MWT). Both the MSLT and MWT require participants to be lying down in a soporific situation, with their eyes closed. This situation is often not feasible in field settings where lengthy, resource-intensive processes are not an option. The much simpler, less intrusive AAT, has been shown to correlate well with both the MSLT and the MWT (Stampi, Stone & Michimori, 1993; Virkkala, Pihl, Harma & Muller, 2000).

The AAT has the added advantage of not being dependent on a subject's motivation (or not) to fall asleep. Further it is not reliant on the subjective scoring of the onset of sleep, or the premise that the ability to fall asleep quickly is related to neurophysiological alertness.

The test requires participants to be seated facing a mark or cross on the wall approximately 2 metres ahead at eye level. The participant is then asked to alternately

open and close their eyes, for a period of 2 minutes at a time, and for a total of 8 minutes. The test begins with a 2 minute period of eyes open focussed on the mark on the wall, then 2 minutes of eyes closed remaining awake. This is followed by a further 2 minutes of both eyes open and eyes closed periods. Full details of the test format and instructions can be found in Appendix D.

The test is based on the principle that when an individual's eyes are open their alpha activity increases as a function of increased sleepiness (Daniel, 1967). In contrast with increased sleepiness, alpha attenuates in the 'eyes-closed' subject (Santamaria & Chiappa, 1987). A ratio of the alpha power in the eyes closed EEG to alpha power in the eyes open EEG is calculated and is known as the Alpha Attenuation Coefficient (AAC) (Heitmann, Stampi & Anandan, 1995). Larger ratios indicate neurophysiological alertness due to the relatively high levels of alpha power seen in an alert eyes-closed individual compared to the low power seen when their eyes are open. Smaller ratios therefore indicate lowered neurophysiological alertness because of the attenuating alpha power when a sleepy individual closes their eyes, compared to the increasing alpha power seen when their eyes are open.

These data were collected during every testing session, and preceded each psychomotor vigilance task, but was not analysed for the purposes of this thesis.

Neurophysiological Correlates of Alertness

Neurophysiological alertness was assessed during the night shift through the continuous recording of EEG and EOG.

Measures From the Electroencephalogram

For this thesis only the EEG from the last hour of each night shift was utilised. The focus was limited to potential EEG changes post the napping opportunity, and a further reduction in data was based on the premise that the end of the night shift was of greatest operational interest. It was the furthest point in time from the napping opportunity, and if changes in EEG indices of alertness could be seen as a result of the napping opportunity, then it could be reasonably expected that they also existed closer to the napping opportunity.

An overarching concern in relation to the use of this type of data, was the quantity and quality of data that would be obtained through recording in an ambulatory environment.

To this researcher's knowledge, no other studies that have conducted continuous ambulatory EEG/EOG recordings in the workplace, have discussed the amount of data contaminated by artefact. For this reason, and to try and limit the number of channels of EEG looked at, a separate study was conducted (J. Gale, personal communication, January 20, 2002). The study aimed to determine if, when conducting an ambulatory recording, there was a best EEG channel or channels that should be used to maximise the amount of data available for analysis. Further aims were to examine whether there was a pattern to the artefact and the reliability of subjective artefact detection and rejection. It was considered that there might be a pattern in the occurrence of artefact if, for example, as individuals became sleepier they blinked more or moved more in an attempt to stay awake. Therefore more data would be removed due to artefact resulting in an under representation of sleepy data. The opposite could also occur, with individuals moving less as they became sleepier, resulting in an overestimation of sleepy data.

The findings of the study showed that significantly more data were retained from the Oz-O2 channel compared to all other single channels. Furthermore, when comparing the amount of data retained in this single channel to the combination of Oz-O2 with another channel, there was no significant increase in the amount of data that were artefact free. There was no overall pattern to the occurrence of artefact, but within individual recordings trends for both increasing and decreasing artefact as time progressed were seen.

In an attempt to more objectively distinguish clean EEG from that contaminated with artefact, a series of criteria were developed. The scoring criteria are listed in Appendix E. Three trained scorers (JG, LS and SG) visually screened the entire night's recording from 6 individuals. A kappa value was calculated in order to assess inter-rater reliability (a value between 0.4 and 0.75 is regarded as 'fair to good agreement'). These results are all taken from the findings of the study by J. Gale et al. (personal communication, January 20, 2002) and can be seen in Table 2-1. Eleven further recordings were then screened for artefact by JG, and the remaining recordings were screened by LS.

Table 2-1: Kappa Statistics for Inter-Rater Reliability

Rater 1	Rater 2	Kappa (κ)
JG	LS	0.601
JG	AG	0.546
LS	AG	0.576
Grand Mean		0.574

Automated artefact detection and rejection methods do exist, but they have not yet proven to be more sensitive and specific than the human scorer and no one method is without shortfalls. There is a reasonable amount of support in the literature for the use of algorithms to “subtract” eye movement artefact from the EEG. However, EEG can also be recorded in the EOG, so that removing the EOG from the EEG channel will also result in the loss of EEG data. Because of difficulties such as these it was decided to use only visual screening, despite the time consuming nature of the task. Other complex computational methods, such as Independent Components Analysis (ICA), show promise but they are only slowly becoming accessible and are not yet practical for non-engineering personnel.

A further consideration when viewing and analysing the EEG data was the length of the data epoch. A 5.12 second epoch was chosen for use, as this resulted in a base 2 number of data points being analysed, and therefore meeting the assumptions of the Fast Fourier Transform (FFT). It was also long enough to be operationally significant, in that decreased levels of alertness for periods of approximately 5 seconds could result in critical operational information being missed. Although longer epochs would be faster to score and analyse, they would also be more prone to being rejected due to short bursts of artefact. Relatively short epochs also provided greater sensitivity through ability to detect transient changes in alertness.

When scoring the EEG, the time at the end of the recording was first determined. A point in time exactly 1 hour prior to this was then the start point for viewing and scoring the data. A purpose designed LabVIEW programme was used for this and more detail on the structure and functions of the programme can be found in Appendix F.

A contextual epoch of 10 seconds was displayed on the screen in a large, central, waveform graph. The middle portion of this graph was the 5.12 second epoch to be scored and analysed. This central epoch was clearly delineated and was also expanded and displayed in an additional smaller waveform graph. The spectral power within the 5.12 second epoch was also plotted. See Appendix F, Figure F-1 for detail.

Each epoch was viewed and scored as being free from artefact, or not, using the criteria outlined in Appendix E. No filters were applied to the data, and it was viewed at an amplitude of 40 $\mu\text{V}/\text{cm}$. It was increased to this size because the amplitude of the EEG was low due to the small inter-electrode distance. Once scored, the power spectral information for the epoch, its scored status, and an index value representing its location in the recording, were written to a file by the LabVIEW programme. The data from the subsequent epochs were appended to the same file.

Epochs overlapped each other by 50%, and a Hanning window was applied prior to the FFT to reduce spectral leakage. With a sample rate of 200 Hz, the spectral output was calculated for 0.195 Hz increments (sample rate divided by total number of data points in epoch = $200/1024$).

The resulting file was in text format, which was then imported into a *SPSS*TM or *SAS*TM database for statistical analysis. Files could also be read by a further purpose built LabVIEW programme which plotted the spectral information as a 3-dimensional array.

Measures From the Electrooculogram

EOG channels were scored using *Somnologica*TM for the presence of SEMs. This was done at the same time as the entire recording (other than the napping opportunity) was scored for sleep. The following section outlines the procedure used for the scoring of sleep, including the filters, gain, and epoch length applied to the EOG channels.

The criteria for an SEM is presented in Appendix G. The duration of each SEM was marked on the EOG traces as an event. At the cessation of scoring these events were summarised in a report that could be exported to an Excel spreadsheet. The spreadsheets were joined into one file and imported into *SPSS*TM and *SAS*TM databases. The various variables generated and utilised are detailed in Chapter 6.

Sleep During the Napping Opportunity

Of the nine channels of neurophysiological data recorded, six were utilised in the scoring of sleep. These were C4-A1, O2-A1, EOG-L, EOG-R, EMG, and ECG. Experienced sleep scorers, using *Somnologica*TM software, viewed the data collected during each pre-planned napping opportunity. On a separate occasion the ambulatory data from the remainder of night shift were also viewed. The EEG channels of C4-A1 and O2-A1, plus EOG-L, EOG-R, EMG, and ECG were presented in that order (see Figure 2-3). All these channels, except that of ECG, are considered advisable for sleep scoring according to the criteria of Rechtschaffen and Kales (1968).

All EEG data were filtered with a low pass filter of 30 Hz and viewed at an amplitude of 75 $\mu\text{V}/\text{cm}$, in order that waves equalling 1 cm or greater met the criteria for the delta waves of stage 3 and 4 sleep. The EOG channels were viewed at 100 $\mu\text{V}/\text{cm}$ with a low pass filter of 30 Hz applied. No high pass filter was applied to either the EEG and EOG data because of the application of a broad pass-band filter of 0.5-90 Hz when data were recorded. The low pass filter of 30 Hz was selected to remove high frequencies which were not of interest and to allow viewing of slow waves, characteristic of delta activity in the EEG and SEMs in the EOG, through to sleep spindle activity in the EEG and REM in the EOG. Such a filter setting also minimises high frequency interference from, for example, the EMG (Carskadon & Rechtschaffen, 2000).

The EMG channel was viewed at the amplitude necessary to detect changes in the signal (normally approximately 20 $\mu\text{V}/\text{cm}$), as absolute amplitude is not relevant to polysomnography, rather only the relative changes are important (Carskadon & Rechtschaffen, 2000). The EMG signal had a high pass filter of 10 Hz applied, a low pass of 70 Hz and a notch filter set at 50 Hz. These filter settings are standard (Carskadon & Rechtschaffen, 2000) and allow the removal of alternating current interference as well as preventing slow signals from interfering with the EMG tracing.

The ECG signal viewing amplitude was not altered and no filters were applied, as this signal was only present in order to assist in the possible identification of ECG artefact in other channels.

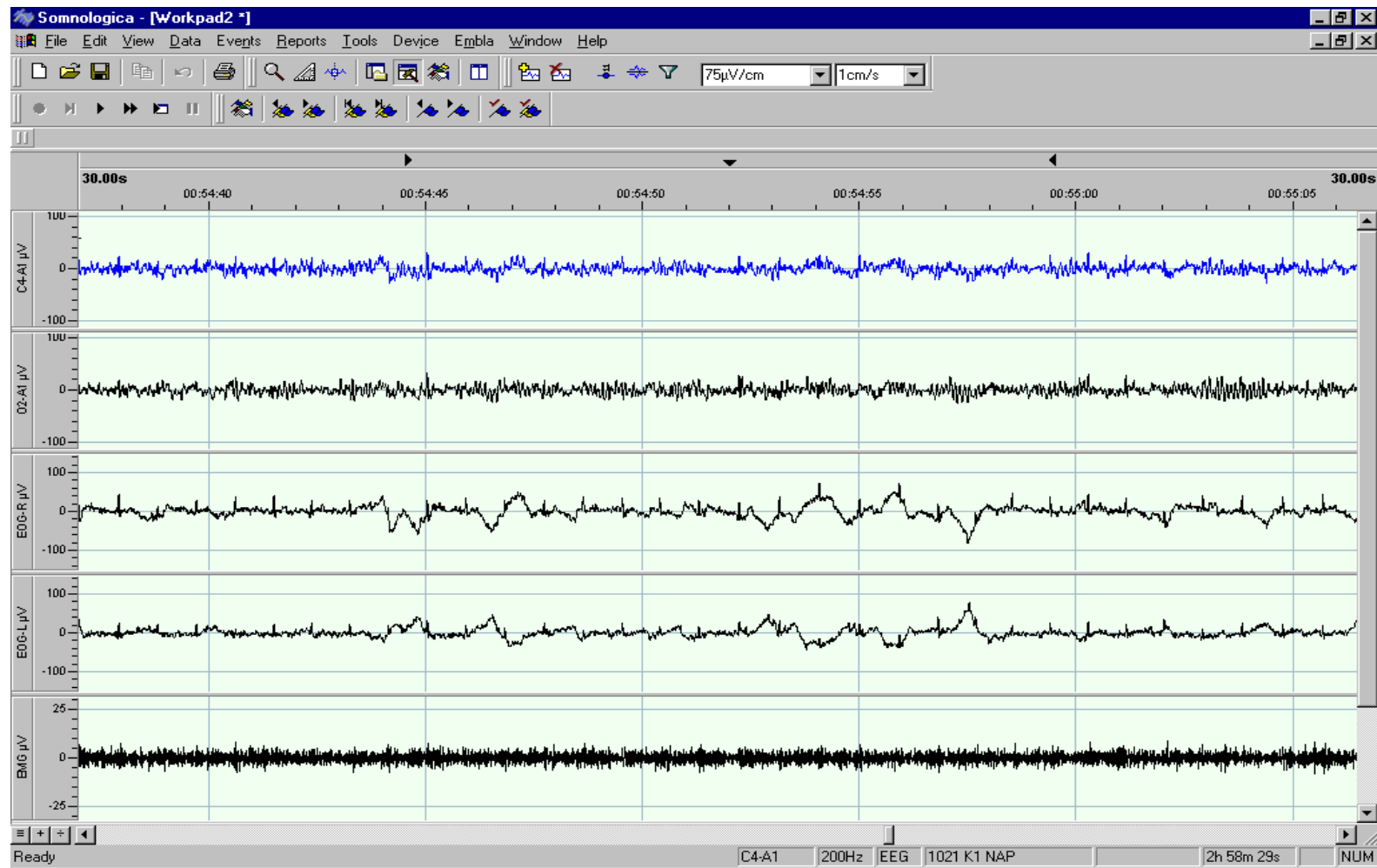


Figure 2-3: Template Used For Sleep Scoring All Data

An event marker was connected to the recorder, which the study participants pressed when they began trying to sleep and again when they had finished trying to sleep during their napping opportunity. This resulted in a mark being placed on a trace and the first of these indicated the commencement point of sleep scoring for the napping opportunity. If the marker was not available then information from the *Actiwatch*TM and logbook were utilised to determine the start of the napping opportunity, along with an assessment of the recording by the sleep scorers.

Epochs of 30 seconds were chosen for scoring, as this is the most widely utilised length (Carskadon & Rechtschaffen, 2000) and allows comparison of the sleep data to that from other studies.

Two experienced sleep scorers viewed and marked the sleep stages in each nap independently, according to the criteria of Rechtschaffen and Kales (1968). EEG arousals were also scored according to the criteria of the American Sleep Disorders Association (1992). These arousals, occurring after at least 10 seconds of NREM sleep, are characterised by an abrupt shift in EEG frequency and are required to be a minimum of 3 seconds duration, with a minimum of 10 seconds of sleep between arousals. Because the standard sleep scoring process is not designed to detect transient events such as arousals, they are often overlooked. However, it was considered important to score arousals, as it has been shown that fragmentation of sleep, like shortened sleep, leads to increased sleepiness (American Sleep Disorders Association, 1992).

Completed scoring sessions were then compared for reliability. When the inter-rater reliability was below 70% the recordings were re-scored on a consensus basis. This occurred for 11 of the 54 scoring sessions. Those recordings that were re-scored did not contribute to final inter-rater reliability scores.

Mean inter-rater reliability was 92.86% ($SD = 4.69$, $Range = 83.30-100$), which was calculated by averaging a series of weighted correlations. The correlations were weighted by the total number of epochs in each sleep stage derived from tables comparing the scoring of one individual to another (see Table 2-2 as an example). The mean value of the non-parametric rank correlation, Kendal's Tau was 0.91 ($SD = 0.086$, $Range = 0.55-$

1.0) and Cramer's V, a measure of association based on chi-square, had a mean value of 0.97 ($SD = 0.051$, $Range = 0.71-1.0$).³

Table 2-2: Example of Correlation/Sensitivity Table from *Somnologica*™ for Comparing Scorings

	Wake	REM	S1	S2	S3	S4	MT	Correlation
Wake	22	0	2	0	0	0	0	91.7
REM	0	0	0	0	0	0	0	100.0
S1	0	0	15	0	0	0	0	100.0
S2	0	0	1	38	0	0	0	97.4
S3	0	0	0	4	1	0	0	20.0
S4	0	0	0	0	0	0	0	100.0
MT	0	0	0	0	0	0	0	100.0
Sensitivity	100.0	100.0	83.3	90.5	100.0	100.0	100.0	91.6

Once inter-rater reliability was determined the summary statistics generated by *Somnologica*™ were output to an Excel spreadsheet. Calculating the average of the values from both scorers generated final values for each variable. These final variables were then imported into *SPSS*™ and *SAS*™ databases and are described in further detail in Chapter 4.

Sleep Scoring of the Remainder of the Night Shift

Separately from the scoring of the napping opportunity, the remainder of the night shift was scored for possible sleep. Using an identical process to that described above, a single trained sleep scorer viewed all the ambulatory recordings excluding the napping opportunity and scored each 30 second epoch for sleep according to the criteria of Rechtschaffen and Kales (1968), as well as scoring SEMs. A second trained sleep scorer independently scored 20% of the recordings for both sleep and SEMs. For the sleep scoring an inter-rater agreement of 100% was found.

³ The possible values of Kendall's tau range between -1 and +1, with larger absolute values indicating stronger relationships, while Cramer's V ranges between 0 and 1, with values close to 1 indicating a high degree of association between the scoring variables.

Inter-rater reliability was more difficult to calculate for the scoring of SEMs due to limitations with the *Somnologica*TM software. However, in 47% of recordings there was complete agreement in the scoring of SEMs. In the remaining instances the second sleep scorer marked 1-2 more SEMs per recording than the sleep scorer who viewed the entire set of recordings. A conservative approach was taken and the quantity of SEMs used in analyses were derived from the individual who scored all the recordings.

2.2.4 *Questionnaire Data*

Pre-Study Questionnaire

On the first meeting with participants, they were asked to complete a pre-study questionnaire, which included questions on age, gender, and experience as an air traffic controller. Questions were also asked on normal pre-shift preparation, whether a nap was normally taken at work, and the quantity of caffeine and food consumed on the night shift. The final questions included whether they had ever been diagnosed with a sleep disorder and their subjective estimate of baseline sleep need. Appendix H is a copy of this questionnaire.

Post-Study Questionnaire

At the end of each night shift, participants completed a short questionnaire (see Appendix I). Questions were asked on pre-shift preparation, the normality of the night shift, workload, consumption of caffeine and food, as well as the length and quality of sleep obtained during the nap (if relevant). If relevant, they also recorded the location at which they took their nap.

The questions on the quality of sleep were derived from the Karolinska Sleep Diary, a subjective scale that has been validated against polysomnography (Åkerstedt, Hume, Minors & Waterhouse, 1994a; Åkerstedt, Hume, Minors & Waterhouse, 1994b; Åkerstedt, Hume, Minors & Waterhouse, 1997; Kecklund, Åkerstedt & Lowden, 1997). They utilised a 10 cm VAS, marked with adjectives such as “very good” to “very poor”. The question on workload also utilised a 10 cm VAS, anchored “very low” to “very high”. All other questions were yes/no, opened ended or required an amount or time to be recorded.

2.3 Procedure

Through consultation with air traffic controllers, union representatives, and management the study design was finalised. Ethical approval for the study was received from the Wellington Ethics Committee, which also granted approval on behalf of the Auckland and Canterbury Ethics Committees.

It was originally proposed that this study would be run at both Auckland and Christchurch air traffic control centres, as both these locations included rosters where two air traffic controllers worked the night shift. This arrangement would safely allow one individual to obtain a nap, while the other individual covered the radar position. However, in the early stages of the study, due to industrial issues occurring at Auckland centre, it was decided to only ask for participation from Christchurch based controllers.

In July 1999 all controllers working in Christchurch Area Control were invited to participate in the study. They were initially informed of the study by a memo from the centre manager, which was followed by a handout prepared by the Sleep/Wake Research Centre detailing the study method and the requirements of participants (Appendix J). Forms stating whether each individual controller was willing to be approached about the study or not, were returned to a Union representative, who in turn followed up on controllers who did not reply to this request within a month. On the first meeting with each air traffic controller that had chosen to participate in the study, the study method and requirement of participants was discussed and then an informed consent form was signed (Appendix K).

At the beginning of the data collection phase, a number of trial nights were completed in order to allow any minor problems in the study design to be identified and rectified prior to starting the main period of data collection. Three controllers were approached and participated in one study night each over the month of August 1999. These study nights went smoothly and the main period of data collection commenced in early October 1999 and continued through until the end of April 2000, by which time the very large majority of data were collected. Three final periods of study data were collected in the months of May and June 2000.

The requirement for the relatively long data collection period was in large part due to the sheer amount of data collected and the fact that each controller who participated in the study was involved in four, eight day periods of data collection. Each of these eight day

periods commenced at the end of a four day work period and included the two days off, the next cycle of four shifts, and the subsequent two days off (see Figure 2-1). On all occasions the night shift occurred as the last shift of the four in the working week. Two different timings of the night shift were included in the study. One shift started at 2230 (known as a K1 shift) and the second began at 2330 (known as a K2 shift). Ensuring that each individual involved in the study completed four study periods, preceded and followed by days off, and that the shifts worked included at the very minimum an early morning shift followed by a night shift, extended the period during which data were collected. This was because each individual needed to be at an appropriate point in the roster to capture the times of interest.

The order in which each individual completed the four study conditions and the length of time between each condition depended entirely on the order in which they occurred within the roster. A strictly counter-balanced design was not feasible, as the duration over which data were collected would have been greatly extended in order for the four study conditions to be completed in a particular order for each participant.

For each shift type, one study night involved a 40 minute pre-planned napping opportunity, and on the other night shift the controller was asked to remain awake, thus allowing the effectiveness of a nap to be assessed on both types of night shifts worked.

Throughout each eight day study period controllers wore an *Actiwatch*TM and completed the logbooks, as detailed in section 2.2.1. On the night shift controllers were asked to come into work 30 minutes early in order to have the electrodes for recording sleep and neurophysiological data connected. They then completed the AAT and the PVT prior to beginning work. Neurophysiological data collected during this time were recorded straight on to the hard drive of a laptop.

Once the first AAT and PVT were completed, the *Embla*TM recorder was unplugged from the laptop, and attached to the individual's waist so that they were completely mobile. They then commenced their first period of work on the night shift. Controllers wearing the *Embla*TM at work can be seen in Figure 2-4.



Figure 2-4: Controllers Wearing the Embla™ While at Work.

All subjects in the study completed the AAT and PVT three times on each study night shift. The second testing session occurred after the napping opportunity (or an equivalent time on the non-napping night shift) just prior to returning to work, and the third session was completed at the end of the night shift, once an individual had finished work. A schematic of the timing of events on each type of night shift is presented in Figure 2-5.

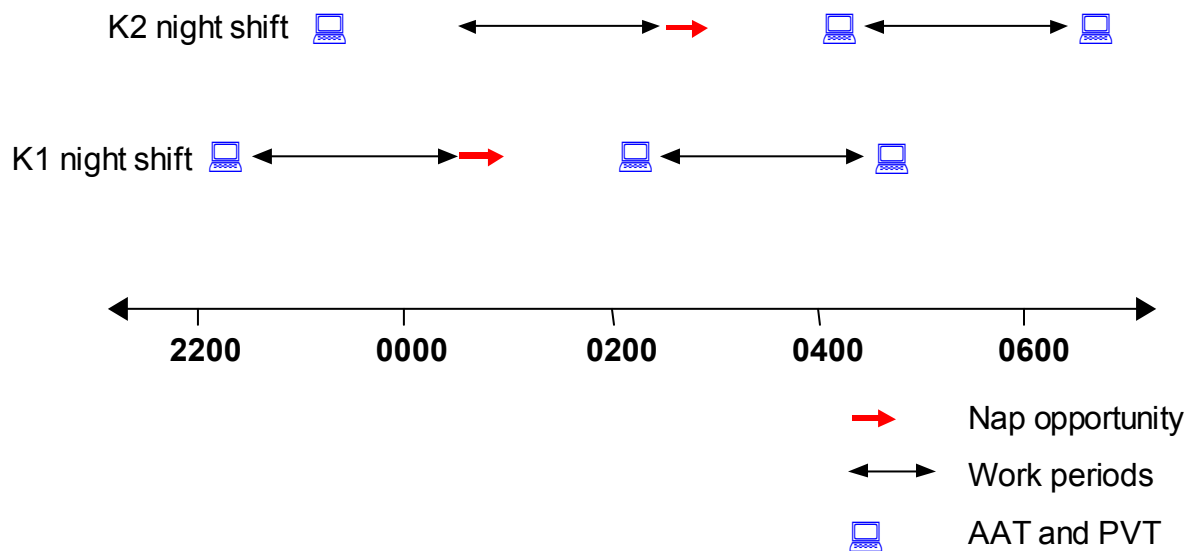


Figure 2-5: Timing of Events on the Night Shifts

On each type of night shift the 40 minute nap opportunity was planned for approximately 2 hours (for a K1 night shift) or 3 hours (for a K2 night shift) after the commencement of the shift, which was at 0030 hours or 0230 hours respectively. At this point in time, those individuals who were assigned to a napping study night were asked to commence their nap. If they normally napped at work, they utilised the same facilities. If not they chose a quiet place such as the sickbay or an office. In all situations, mattresses, blankets, and pillows were available if they had not brought their own.

Once organised for their nap, the time allocated for sleeping began. Participants also pressed the event marker on the *Embla*TM to indicate the commencement of the napping opportunity. The researcher returned to wake participants 40 minutes after the napping opportunity had commenced. Each participant was woken by a knock on the door and once the individual had responded to the researcher that they were awake, they were left to organise themselves. At this point in time they were also asked to press the event marker on the *Embla*TM once more to indicate the end of the napping opportunity.

After the third and final AAT and PVT the electrodes were removed and controllers went home. Data from the PCMCIA card in the *Embla*TM recorder were then downloaded to the laptop in 2 separate files. One was for the first half of the night, which included the nap opportunity. It finished prior to the second set of tests of the night. The second file was for the remainder of the night shift.

2.3.1 *Data Management*

All databases that were generated from the various measures were checked for accuracy and outliers. Every fourth set of actigraphy data (which equated to one individual's data across one shift cycle) were cross-checked with the raw excel file to ensure values had been transferred accurately. The same procedure of cross-checking with the original Excel files was applied to the polysomnography and PVT data. Descriptive statistics were generated (mean, standard deviation, range) for each variable in each database and checked for plausible values. Univariate outliers were identified by the calculation of z scores, with those having values in excess of 3.29 ($p < .001$), considered to be potential outliers (Tabachnick & Fidell, 1996). Histograms, box plots, normal probability plots and detrended normal probability plots were also generated and viewed, where necessary, to assist in the detection of univariate outliers. Identified values were checked against the original data for accuracy and if valid, were retained in the database.

The amount and pattern of missing data in each database was evaluated. Each individual had actigraphy and/or logbook data for each experimental condition and out of 1172 entries 85.5% had actigraphy data or were days on which no sleep period began. For 12.9% of the entries only logbook data were available and 1.6% of entries were missing. Because the amount of missing data was minimal it was not considered problematic and in subsequent analyses cases with missing data were excluded from the specific analysis. It was noted however, that out of the 19 missing entries, 11 occurred on the last day of the study period and 4 on the first day. Participants taking the watch off a day early or forgetting to put the watch on at the commencement of a study period respectively explain this.

With regard to the PVT data, of the 321 tests completed (107 study nights multiplied by 3), 308 complete sets of PVT summary statistics were available. At the completion of a study night involving 2 participants, all PVT data were lost. This occurred on one other occasion with a single study participant resulting in the total loss of 9 trials of data. A further 3 trials of data from a single individual were removed from the PVT database as they had only completed one of the four study nights before ceasing their involvement in the study. Finally, a single trial at the end of a night shift was not completed due to failure of the equipment. Because the amount of missing data was negligible and spread across three separate study conditions, the 13 cases with missing PVT data were excluded from the performance related analyses.

For each of the 54 naps, and the 107 night shifts, no neurophysiological data were lost. However, in one instance the file for a night's recording was corrupt and unable to be used. Details of data removed due to artefact are discussed in detail in Chapter 6.

Data were screened for normality according to the grouping required for each analysis. Both statistical and graphical methods were utilised. Significance tests for both skewness and kurtosis values were calculated, using conventional but conservative alpha levels (.01-.001), which are considered appropriate for moderate sample sizes (Tabachnick & Fidell, 1996). The Shapiro-Wilk and Kolmogorov-Smirnov statistics were also calculated. Where additional information was considered helpful in determining normality, histograms, box plots, normal probability plots and detrended normal probability plots were produced and assessed. In the present study, departures from normality were dealt with in ungrouped data and where it was necessary to remove outliers or apply transformations such procedures are identified in relation to specific analyses. The medians are reported in descriptive statistics for variables that displayed non-normal distributions.

In general, the statistical procedure utilised for data analysis was the mixed linear model and was performed in the *SAS*TM system for Windows (version 8.0). The mixed linear model is an extension of the general linear model and is so called because it allows the modelling of both fixed and random effects. Fixed effects are the “traditional” predictor variables, while random effects are additional variables also assumed to impact on the variability of data. The strength of the mixed model is that data are permitted to exhibit correlation and non-constant variability, therefore allowing modelling of the variances and covariances as well as the means of data.

The assumptions of the mixed model are that data are normally distributed, the expected relationships are linear, and the variances and covariances exhibit a structure matching one of those available in the modelling procedure. The assumptions of normality, linearity, and homoscedasticity can be checked by analysing the residuals (Jones & Kenward, 1989; Tabachnick & Fidell, 1996) and with each model a histogram, normal probability plot and the Shapiro-Wilk or Kolmogorov-Smirnov statistic was generated to assess the structure of the residuals. Where outlying residual values were identified the value was assessed and the model was re-run without the value(s). Residual plots were then re-checked to evaluate the effect of removing the outlier(s). In all instances the process used was to determine if removing the outlying value(s) altered the residuals and

the findings of the model. If the residuals changed slightly but the outcome of the model remained the same, then the reported results include the outlying values. However, if the outcome of the model did alter then the results reported are those minus the outlying values. In all analyses it is clearly stated whether outlying values were identified, the effect of their removal, and if the final results exclude the outlier(s) or not.

In specifying the covariance structure between measurements the procedure applied was to run a general covariance structure, known as “unstructured”, which makes no assumptions regarding equal variances or covariances. However, because of the computational capacity required in calculating such a structure this is not always possible to achieve (Littell, Henry & Ammerman, 1998). If, in the present study, the unstructured option would run, the correlation matrix was assessed to see if there was a pattern that fitted the available covariance structures. This structure was then applied and compared to alternative structures to confirm it was the best fit. In the instance that the unstructured option could not be calculated, then theoretically suitable covariance structures were applied and compared.

Other covariance structures that were considered appropriate to apply to data include the compound symmetric structure, which assumes multiple measures have the same variance and that all pairs of measures for an individual have the same covariance. Such a structure implies that the only aspect of covariance between repeated measures is due to the contribution of the individual, irrespective of the relationship of measures to each other over time. A second covariance structure utilised was the autoregressive structure. The pattern of covariance specified by this structure is that of increasing covariance with increasing spacing between measures within an individual (Littell, Pendergast & Natarajan, 2000). This was coupled with an inter-individual random effect.

Schwarz Bayesian Criteria (SBC) was used to objectively assess the fit of a covariance structure, with the highest value indicating the most suitable structure. This approach is considered the most conservative and adjusts the REML log likelihood value by imposing a penalty according to the number of parameters estimated (Littell et al., 1998).

When main and interaction effects were statistically significant, post hoc *t* tests were used to investigate comparisons of interest. To avoid Type I errors, Holm’s sequentially rejective procedure was employed to adjust the level of significance given the number of

comparisons being made. This is considered a conservative approach, but slightly less restrictive relative to the Bonferroni test (Aikin & Gensler, 1996).

CHAPTER 3

THE SLEEP CONSEQUENCES OF BACKWARD, RAPIDLY-ROTATING WORK SCHEDULES

3.1 Introduction

Previous occupationally based research, as well as the current body of knowledge on the functioning of the circadian system, suggests that the shift cycle worked by air traffic controllers in the present study prevents people from obtaining the normal amount of sleep they require. Progressively earlier start times are thought to limit night time sleep, resulting in less sleep than required and over a period of days the building of a cumulative sleep debt. Attempting to get to sleep earlier in anticipation of the earlier rising time is physiologically difficult. Coupled with this, the short turn around between the morning and night shift further limits sleep opportunities and this sleep is attempted at a less than optimal time in the circadian phase. The first part of this chapter seeks to determine whether the specific shift cycles of this group of New Zealand air traffic controllers have the expected effects on sleep length and timing.

The reasons for providing a napping opportunity to individuals working at night have been previously mentioned. An individual's ability to use this sleep opportunity, and in turn the potential benefits, may be mitigated or conversely enhanced by their sleep status prior to commencing the night shift (Dinges, 1992). Thus it is important to determine whether air traffic controllers are normally sleep deprived when working the night shift. This issue is the focus of the second section of this chapter.

Any effects of a nap on the night shift are thought to be entirely beneficial. However, with the amount of acute, and by extension cumulative, sleep loss possibly diminished by napping episodes, there is the potential that the short sleep obtained at work may impinge on the quantity and quality of post night shift sleep. Addressing this issue forms the basis for the third part of this chapter.

Specifically then, the relevant research questions and hypotheses for this chapter are as follows:

- Q1. Does the length, timing, and quality of sleep episodes change across the shift cycle worked by air traffic controllers?

- H1A. It is hypothesised that due to the compressed, backward rapidly-rotating shift cycle worked by air traffic controllers, the end of sleep episodes across the working week will become progressively earlier while sleep start times remain unaltered.
- H1B. It is hypothesised that this will lead to progressively shorter sleep episodes across the working week, but these will not differ in quality.
- Q2. Are air traffic controllers cumulatively and/or acutely sleep deprived when working the night shift?
- H2. It is hypothesised that because of the expected decrease in sleep across the working week, air traffic controllers will be both acutely and cumulatively sleep deprived when working the night shift.
- Q3. Do workplace naps affect the length, timing, and quality of post night shift sleep?
- H3. Because of the short opportunity for sleep during the workplace naps, it is hypothesised that this sleep will have no significant effect on the quality and quantity of post night shift sleep.

3.2 Method

3.2.1 Measures

To determine the impact of the backward, rapidly-rotating shift schedule on the sleep needs of controllers, and in order to investigate whether individuals commencing the night shift were sleep deprived, variables generated from the *Actimatch*TM, logbook, and pre-study questionnaire, as described in Chapter 2, were analysed.

The following variables were utilised:

Sleep timing variables

- **Bedtime:** A time based on an event marker in the actigraphy data (cross-checked against the log book entry). Participants were asked to press the event marker on the *Actiwatch*TM when they began trying to sleep. This was to ensure time spent in bed, but not trying to sleep (such as reading a book) was not included in the actigraphy calculations. In the *Sleepwatch*TM software this was set manually. For actigraphy outputs that had no event markers, consistent guidelines were followed in the interpretation of data to determine bedtime (see Appendix L). Bedtime was the time at which the *Sleepwatch*TM programme began to sample epochs of data to determine if the participant was asleep or awake.
- **Get up time:** Also a time based on an event marker in the actigraphy data (cross-checked against the log book entry). Participants were asked to press the event marker again on the *Actiwatch*TM when they finished trying to sleep, for identical reasons as above. In the *Sleepwatch*TM software it was set manually, and when no event markers were available the guidelines in Appendix L were followed. Get up time marked the last epoch used by the *Sleepwatch*TM algorithm to determine if the participant was asleep or awake.
- **Sleep start:** This parameter may either be set manually or derived automatically via the software algorithm and represents time of sleep onset. It has been demonstrated that subjective reports of sleep are discrepant from physiological measures of sleep (Carskadon & Dement, 1977; Gander et al., 1998), with the trend being an over-estimation of time taken to fall asleep (Rosekind & Schwartz, 1988a, 1988b). Therefore, it was decided to use the automated option to calculate sleep start. The *Sleepwatch*TM software searches for the first period of 20 consecutive epochs from the bedtime mark, in which a maximum of one epoch contains a non-zero value. This procedure is carried out in a step-wise manner. The first epoch in the group of 20 meeting the criterion is set as sleep start time. On this basis, it is possible for sleep start to be the same as bedtime. In the instances where actigraphy data were not available but a subjective estimate of sleep start was recorded in the logbook, then this time was entered into the main database as sleep start.

- **Sleep end:** This parameter represents the time of sleep termination and like sleep start can either be set manually or derived automatically. As with sleep onset, individuals may be poor at judging exactly when they woke up and how long they have been awake for (Carskadon & Dement, 1977; Rosekind & Schwartz, 1988a, 1988b), so to ensure accuracy and consistency this value was also calculated by the software algorithm. The algorithm examines the ten minutes immediately prior to get up time and scores the last epoch with no movement as sleep end. Because very little movement is required to set sleep end, the logbook entry was cross-checked for accuracy. Sleep end can be identical to get up time. As with sleep start, when actigraphy data were unavailable but a subjective estimate of sleep end was recorded in the logbook, this time was entered in the database as sleep end.

Sleep length variables

- **Time in bed (TIB):** Is the length of time in hours and minutes between bedtime and get up time and represents the amount of time spent attempting to sleep. This variable was of interest because it indicates the length of time participants had, or made, available for sleeping.
- **Assumed sleep:** Is the difference between sleep start and sleep end times in hours and minutes, and represents the period of time individuals are assumed to be sleeping.
- **Actual sleep time:** This is the amount of time activity levels are below the threshold sensitivity value, and is calculated by subtracting actigraphically determined wake time from assumed sleep time. If actigraphy data are not available, this variable cannot be calculated. Actual sleep time was analysed because it estimated more closely the true quantity of sleep obtained.
- **Self-reported sleep need:** The amount of sleep reported by participants as normally needed on an undisturbed night to feel fully rested. This value was reported in the pre-study questionnaire.

Sleep quality variables

- **Actual sleep time percentage:** This variable is the percentage of assumed sleep that was actigraphically scored as sleep, and is calculated by dividing actual sleep time by assumed sleep time and multiplying by 100.
- **Sleep efficiency:** Represents the amount of time in bed spent sleeping and is determined by dividing “actual sleep time” by “TIB” and multiplying by one hundred.
- **Movement and fragmentation index:** This variable was considered an index of the restfulness of sleep and therefore provides an insight into the quality rather than the quantity of sleep obtained. It was comprised of the “number of minutes spent moving percentage” added to the “immobility phases of one minute percentage”.

The first component of the movement and fragmentation index; “number of minutes spent moving percentage” was determined by dividing the “number of minutes moving” during assumed sleep time by “assumed sleep” and expressing this as a percentage. “Immobility phases of one minute percentage” was calculated by dividing the number of phases during which an individual was immobile for exactly a minute (no more or no less) by the total number of immobile phases. This is expressed this as a percentage.

As the resulting index value increases, the quality of the sleep is considered poorer, due to greater fragmentation and more movement.

- **Mean activity score:** This is calculated by dividing the sum of all activity counts between sleep start and sleep end times by the number of epochs during the assumed sleep period, indicating the magnitude of activity on a per epoch basis during sleep.

The above variables were copied directly from Sleepwatch to an Excel file, which represented each individual’s eight day period of actigraphy. These files were then joined and imported to a *SPSS*TM and *SAS*TM database for statistical analysis.

Work duration variable

- **Shift length:** The actual length of time worked. Calculated by taking the difference between shift start and shift end times reported in the study logbooks.

3.2.2 *Statistical Analyses*

Descriptive statistics are presented in order to gain an understanding of the overall pattern of data for different variables. Where normal distributions exist, the mean, standard deviation, and range are given, while if departures from normality were identified the median and range is reported instead.

Because of the need to account for random variation due to individual variability and to allow for any pattern of variance between repeated observations, mixed model ANOVAs and ANCOVAs were employed as the standard method of analysis.

A wide range of mixed models have been utilised in order to answer each of the questions addressed in this chapter. Variations in the structure of the models are detailed in the relevant results sections.

3.3 **Details of Study Participants**

Participants had a mean age of 35.5 years ($SD = 6.9$, $Range = 26-56$, $N = 28$) and mean experience controlling of 11.3 years ($SD = 8.5$, $Range = 2-34$, $N = 28$). Nineteen participants were male and 9 were female. There were no reports of individuals suffering from a diagnosed sleep disorder.

3.4 **Sleep and Work Patterns in the Study Period**

This section provides an overview of the pattern of work and sleep associated with each of the experimental conditions. Details regarding the way sleep episodes were categorised are discussed, as well as a comparison between rostered and reported hours of work.

In order to see the change in the pattern of sleep associated with the work schedule it was necessary to focus on main night sleep episodes. This was done because there were individuals who occasionally napped in addition to their night sleep, and inclusion of these sporadic shorter sleeps with the night sleep data would produce outlying values that obscured the pattern of the majority of sleep episodes. The various sleep episodes seen across the study week were therefore categorised as follows:

- A main night sleep episode was defined as any sleep that started after 2000 hours but before 0400 hours, and finished before 1200 hours. These criteria were applied to each night, except for workday 3, where a sleep was considered a “main night sleep”

if it ended prior to the morning shift start time rather than 1200 hours. These main night sleep episodes account for 66% of the total sleep episodes recorded in the study and 81% of the total sum of TIB, assumed, and actual sleep.

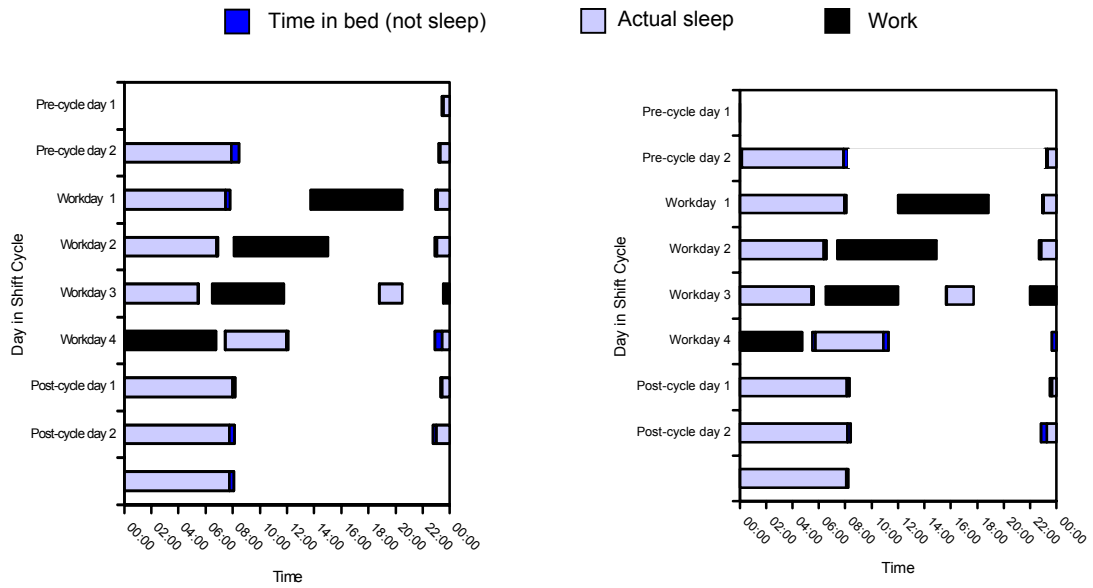
- Daytime sleep episodes pre- and post- the night shift. Sleep parameters that were deemed as pre-night shift, were calculated from values that fell between the earliest recorded end time of the morning shift and the rostered start of the night shift. Sleep post the night shift was considered as such when it occurred after the earliest finishing time of the night shift but before 2000 hours that day.
- Scheduled sleep on the night shift was defined as any sleep taken during the workplace naps.
- Short additional sleep episodes were any sleep that did not fit into the above categories. These important, infrequent shorter sleeps represent 10% of the total sleep episodes in the entire actigraphy database (9% of all summed TIB and assumed sleep episodes). They are considered in section 3.6 where baseline sleep episodes and sleep debts were calculated.

3.4.1 *Sleep Episodes*

In order to see the pattern of work and sleep across the study period, main night sleep episodes, sleep pre- and post- the night shift, and the naps obtained on the night shift, as well as all recorded work periods were plotted in Figure 3-1. Ninety percent of all sleep episodes in the study period were plotted in these figures, which include 91% of the sum of all recorded TIB and assumed sleep values in the database. For the purpose of the plots in Figure 3-1, nap episodes on the night shift were calculated from logbook data rather than polysomnographic information.

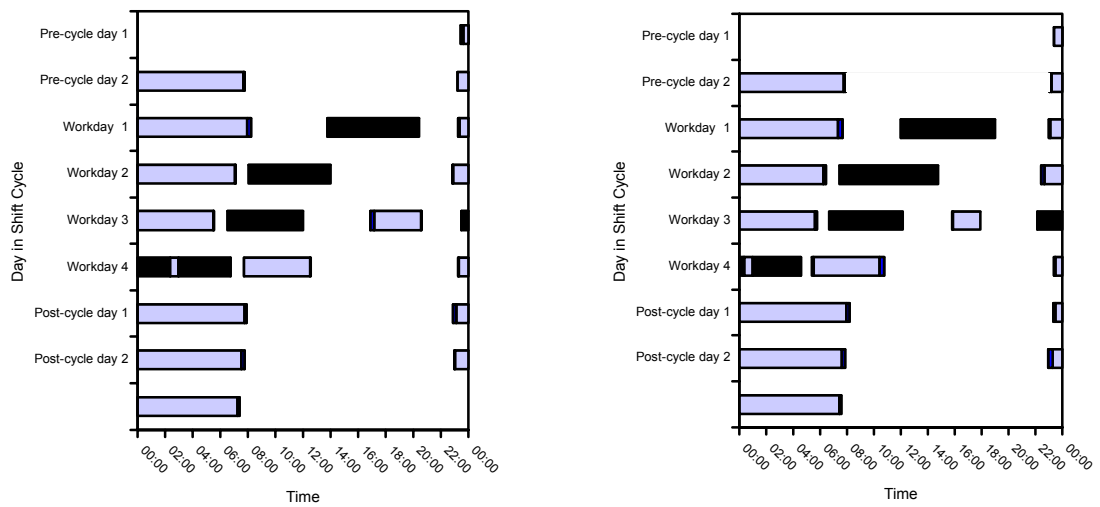
Actigraphy and logbook data were plotted from the beginning of the data collection period, which was the morning of a first day off, through the shift cycle that involved a night shift, until the beginning of work on the first day of the next shift cycle. Because not all values were normally distributed, median bedtimes, sleep start, sleep end, get up, work start, and work end times for each day in the shift cycle are plotted. Non-normal distributions of sleep times occurred when sleep had been truncated by work or, for example, after the night shift, when bedtimes commence from the end of the shift onwards.

Figure 3-1 shows four graphs, each associated with one of the study conditions. The study day is listed down the y-axis and the 24 hour day from midnight to midnight across the x-axis. The graphs clearly show the backward rotating shift pattern, with work periods starting earlier each day in the cycle. The timing of the napping opportunity can also be seen.



K2 Shift With No Nap Opportunity

K1 Shift With No Nap Opportunity



K2 Shift With a Nap Opportunity

K1 Shift With a Nap Opportunity

Figure 3-1: Sleep and Work Patterns During the Four Study Conditions.

3.4.2 *Work Hours*

As outlined in Chapter 2, controllers who are rostered on a four day shift cycle that has a night shift as the last shift, all work a relatively standard pattern. The work patterns differ slightly between cycles that include the K1 night shift or the K2 night shift. The typical rostered shift cycles are detailed in Table 3-1. Both these rosters are examples that do not include weekend shifts, when the timing of shifts alters slightly due to different traffic demands.

Table 3-1: Rostered Timing of Shifts in The Two Shift Schedules Included in the Study

	Schedule Including K1 Night Shift	Schedule Including K2 Night Shift
Workday 1	12:00-19:05	13:45-20:55
Workday 2	07:25-14:55	06:50-14:00
Workday 3	06:30-12:15	06:30-10:55
Workday 4 (K1 or K2)	22:30-06:00	23:30-06:45

A shift cycle that included a K1 night shift would typically include about 28 rostered hours of work per cycle, while a shift cycle that included a K2 shift would normally involve about 26 hours of rostered work. However, because the opportunities for sleep depend largely on the amount of time available outside of work, it is necessary to determine what hours are actually worked as opposed to what is rostered.

In this particular work context, rostered shift commencement times are adhered to, as the arriving controller is often required to relieve another individual at their radar position. However, shift end times often vary due to staffing levels or changing traffic demands. Because of this, the timing of shifts was not considered as critical as the length of the work periods.

The analyses reported in this section investigate shift type or napping condition differences for the total amount of time spent working across both the entire eight day study period and across the four rostered work days. Both periods of time were looked at because controllers could accept a shift on their rostered days off. The dependent and independent variables utilised in each model are detailed in Table 3-2.

Table 3-2: Dependent and Independent Variables for Analyses Related to Work Duration.

Dependent Variables	Independent Variables ⁴
<i>Work hours</i>	
Total hours worked in eight day study period	Shift cycle Nap Shift cycle * Nap
Total hours worked in four day work week	
Shift length	Shift cycle Nap Work day Shift cycle x Nap Work day x Shift cycle Work day x Nap Shift cycle x Nap x Work day

No significant differences were found for shift type and napping condition in either the mixed model ANOVA of total working hours over the eight day study period, or the four rostered working days. On this basis, total work durations were combined across both the eight day study period and the four rostered working days.

Controllers report working an average of 27.2 hours ($SD = 4.55$, $N = 107$) over the entire eight day study period, including rostered days off, with total working hours ranging widely from a minimum of 13 hours to a maximum of 39.9 hours (Figure 3-2). Across the four rostered workdays, controllers reported a median total work length of 25.25 hours ($N = 107$), which ranged from 13 hours to 28.5 hours, as can be seen in Figure 3-3.

⁴ Shift cycle = shift cycle worked (includes either a K1 or K2 night shift)

Nap = napping condition on night shift (nap opportunity provided or not)

Work day = day within work week (days 3 to 6 within the 8 day study period)

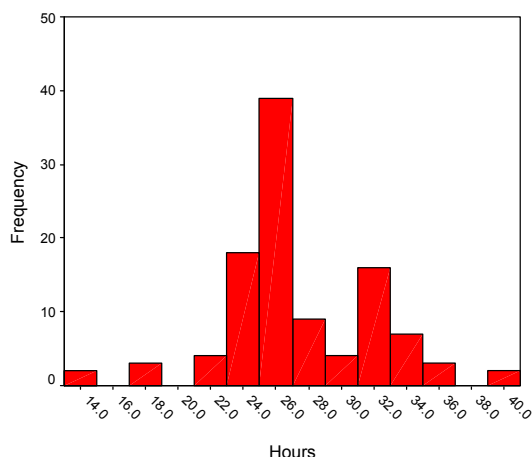


Figure 3-2: Total Hours Worked Over the Eight Day Study Period

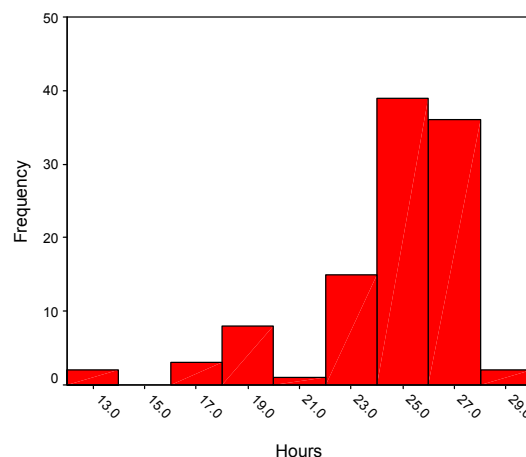


Figure 3-3: Total Hours Worked Over Workdays 1-4

Table 3-3 displays the mean, standard deviation and range for reported shift length on each rostered workday. Most shifts were between 6-7.5 hours in length, with the exception of the early morning shifts on workday 3, which were approximately 5 hours long. Like total working hours, the reported length of individual shifts also ranged widely.


Contrary to what was rostered, the total number of hours worked across either a shift cycle including a K1 or K2 night shift did not differ. It was therefore decided to investigate whether particular shifts on individual days differed between the two schedules. A mixed model ANCOVA was conducted in order to determine if there were shift type or napping condition differences for the length of each shift over the four working . The results indicated that the interaction of nap by study day was not significant. However, the main effect of study day ($F_{(3,290)} = 244.36, p < .001$) and the interaction of shift by study day ($F_{(3,290)} = 14.21, p < .001$) were found to be significant. Therefore, using the appropriate data from the coefficient matrix for least-square means, separate t tests were calculated for each shift by study day interaction.

Table 3-3: Descriptive Statistics, Post Hoc Tests, and Estimates of Differences for Shift Type on Each Rostered Work Day

Day	Type of Night Shift in Cycle	Mean Shift Length (hours)	<i>SD</i>	<i>N</i>	<i>Range</i>	Significant Post Hoc Tests for Shift Type by Day in Cycle	Estimated Difference (hours) Between Shifts and Confidence Intervals
Work day 1	K1	6.70	0.65	53	4.50-7.50		
	K2	6.57	0.65	54	4.50-7.92		
Work day 2	K1	7.12	0.58	52	5.50-8.58	$t_{(372)} = 3.40, p < .001$	0.46 (0.18-0.73)
	K2	6.68	0.78	54	4.50-8.42		
Work day 3 (morning shift)	K1	4.68	0.83	53	2.50-6.25	$t_{(369)} = -2.96, p = .003$	0.38 (0.11-0.64)
	K2	5.07	0.78	54	3.50-7.50		
Work day 4 (night shift)	K1	6.68	0.62	53	5.50-8.92	$t_{(369)} = -5.00, p < .001$	0.64 (0.37-0.91)
	K2	7.33	0.45	54	6.67-8.50		

The results of these tests are reported in Table 3-3, and show that the length of the shift on workday 1 did not differ significantly between the two different shift cycles, and both shifts were shorter than the rostered shift. On workday 2, individuals reported working shorter hours in the K2 shift cycle than the K1 shift cycle, which follows the rostered pattern. However, on both these working days the mean shift length recorded was less than the rostered duration. In the subsequent two shifts, the early morning shift of workday 3 and the night shift, those working the K2 shift cycle had significantly longer shifts than those rostered on a K1 shift cycle. This is contrary to what was rostered. In addition, both the morning shift and night shift in the K2 shift cycle are, on average, longer than was rostered.

Table 3-4 details the mean length, standard deviation, and range of shifts worked on rostered days off. Forty five of the 424 rostered non-work days in the entire database were worked (11%) and these shifts were reasonably evenly distributed across the four non-work days. Of these additional shifts, 22% were worked by two individuals, and 45% were worked by five individuals. Conversely, five individuals did not work any additional shifts on rostered days off.

Table 3-4 : Mean Length, Standard Deviation, Range, and Percentage of Shifts Worked on Rostered Days Off

Day	Mean Shift Length (hours)	SD	N	Range	Percentage of Days on Which a Shift Was Worked
Pre-duty day 1	6.82	0.67	12	5.75-7.50	12.1%
Pre-duty day 2	6.45	0.80	10	5.17-7.50	9.3%
Post-duty day 1	6.13	1.10	9	4.42-7.50	8.6%
Post-duty day 2	6.48	0.80	13	5.17-7.67	12.4%

3.5 Question 1: Length, Timing, and Quality of Sleep Episodes Across the Shift Cycle

3.5.1 Analyses and Data Management

In order to see if the length, timing, and quality of sleep varied across the working week, analyses focussed on changes associated with main night sleep episodes, as defined in section 3.3.

In addition, the recorded hours of work of study participants were assessed because of the potential effect long hours of work would be expected to have on the opportunity for sleep. Table 3-5 lists all the dependent variables which were the focus of the analyses associated with the first question addressed in this chapter. The independent factors and interactions included in each mixed model in this section are also detailed in Table 3-5. In all analyses involving these variables, the covariance of values between days in the study period was modelled most appropriately by an autoregressive structure.

Table 3-5: Dependent and Independent Variables for Analyses Related to the Length, Timing, and Quality of Sleep Across the Shift Cycle

Dependent Variables	Independent Variables ⁵
<i>Sleep length</i>	
TIB	
Assumed sleep	
Actual sleep	
<i>Sleep timing</i>	
Bedtime	Shift cycle
Sleep start	Nap
Sleep end	Day
Get up time	Shift cycle x Nap
	Day x Shift cycle
	Day x Nap
	Shift cycle x Nap x Day
<i>Sleep quality</i>	
Actual sleep time percentage	
Sleep efficiency	
Movement and fragmentation index	
Mean activity score	

3.5.2 Results: Length of Main Night Sleep Episodes

Table 3-6 below details the mean length, standard deviation and range of the main night sleep episodes across the eight day study period. To determine if the length of main night sleep episodes altered significantly across the eight day study period, a series of

⁵ Shift cycle = shift cycle worked (includes either a K1 or K2 night shift)

Nap = napping condition on night shift (nap opportunity provided or not)

Day = day within the study period (1 to 8)

mixed model ANCOVAs were performed. Details and results of the models are specified in Table 3-7.

The sleep length parameters were found to differ across the days of the study period. Figure 3-4 displays the mean length of TIB, assumed sleep, and actual sleep for main night sleep episodes over the study period. The night of workday 3 is the night shift, therefore no main night sleep episode occurs and this day was excluded from analysis. The pattern of changing sleep lengths did not differ between shift cycles with K1 or K2 shifts, or between shift cycles with and without naps. Nor were there significant interactions between these variables and the day of the shift cycle.

Post hoc tests were calculated to assess which main night sleep episodes differed from each other. The arrows in Figure 3-4 indicate the days for which significant post hoc tests were present, the results of which can be found at the end of this chapter in Table 3-21. All sleep length parameters for workday 1 and workday 2 differed from each other and all other days, while sleep length on pre-cycle days 1 and 2, workday 4, and post-cycle days 1 and 2 did not differ significantly from each other.

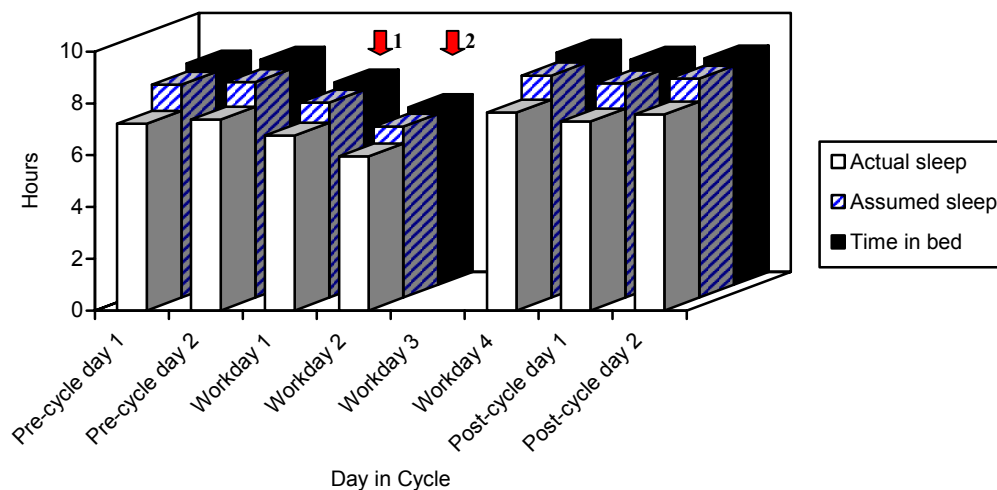


Figure 3-4: Length of TIB, Assumed Sleep, and Actual Sleep for Main Night Sleep Episodes Across the Eight Day Study Period.

Table 3-6: Mean, Standard Deviation, and Range for TIB, Assumed Sleep, and Actual Sleep of Main Night Sleep Episodes for Each Day in the Study Period

Day	TIB (h)				Assumed Sleep Length (h)				Actual Sleep Length (h)			
	<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>
Pre-cycle day 1	8.55	1.25	4.07-12.70	99	8.23	1.49	3.97-12.10	99	7.22	1.05	3.92-9.63	90
Pre-cycle day 2	8.70	1.35	5.42-12.43	106	8.33	1.43	5.17-12.38	106	7.38	1.23	4.20-10.43	99
Workday 1	7.82	1.22	5.40-10.83	104	7.53	1.23	5.05-10.13	104	6.76	1.09	4.43-9.13	99
Workday 2	6.86	0.92	5.00-9.05	106	6.61	0.96	4.58-8.95	106	5.95	1.79	4.28-8.12	99
Workday 3												
Workday 4	8.97	1.45	4.78-13.02	105	8.58	1.40	3.78-12.28	105	7.65	1.18	4.43-11.42	97
Post-cycle day 1	8.70	1.37	3.27-12.48	106	8.28	1.31	3.15-12.38	106	7.30	1.12	3.03-10.42	100
Post-cycle day 2	8.74	1.72	0.43-12.43	70	8.46	1.73	0.43-12.38	70	7.57	1.24	4.47-10.23	66

Table 3-7: Details and Results of Mixed Model ANCOVAs for the Sleep Length Parameters of Main Night Sleep Episodes Over the Study Period

Dependent Variable	Fixed and Interaction Effects in Mixed Model	Significant Main Effects	Significant Interaction Effects
TIB	shift, nap, day, shift x nap, day x shift, day x nap, day x shift x nap ⁶	Day in cycle $F_{(6, 521)} = 39.98, p < .001$	
Assumed sleep	As above	Day in cycle $F_{(6, 520)} = 34.49, p < .001$	
Actual sleep	As above	Day in cycle $F_{(6, 481)} = 28.58, p < .001$	

⁶ Shift = shift cycle worked (includes either a K1 or K2 night shift)

Nap = napping condition on night shift (nap opportunity provided or not)

Day = day within the study period (1 to 8)

3.5.3 Results: Timing of Main Night Sleep Episodes

Figure 3-5 shows the distribution of sleep start and end times for main night sleep episodes across the study period. Each time value on the plot in Figure 3-5 is related to the day on which the sleep commenced, therefore the sleep end times actually occur on the following day. For example, the sleep end time plotted for workday 2 is the sleep that ends on the morning of workday 3, which is prior to the early morning shift.

In Figure 3-5 the median sleep start and sleep end time is represented by the black horizontal bar in the centre of each box, while the inter-quartile range containing 50% of the values centred about the median is shown by the box itself, and the highest and lowest values are the whiskers extending from the box.

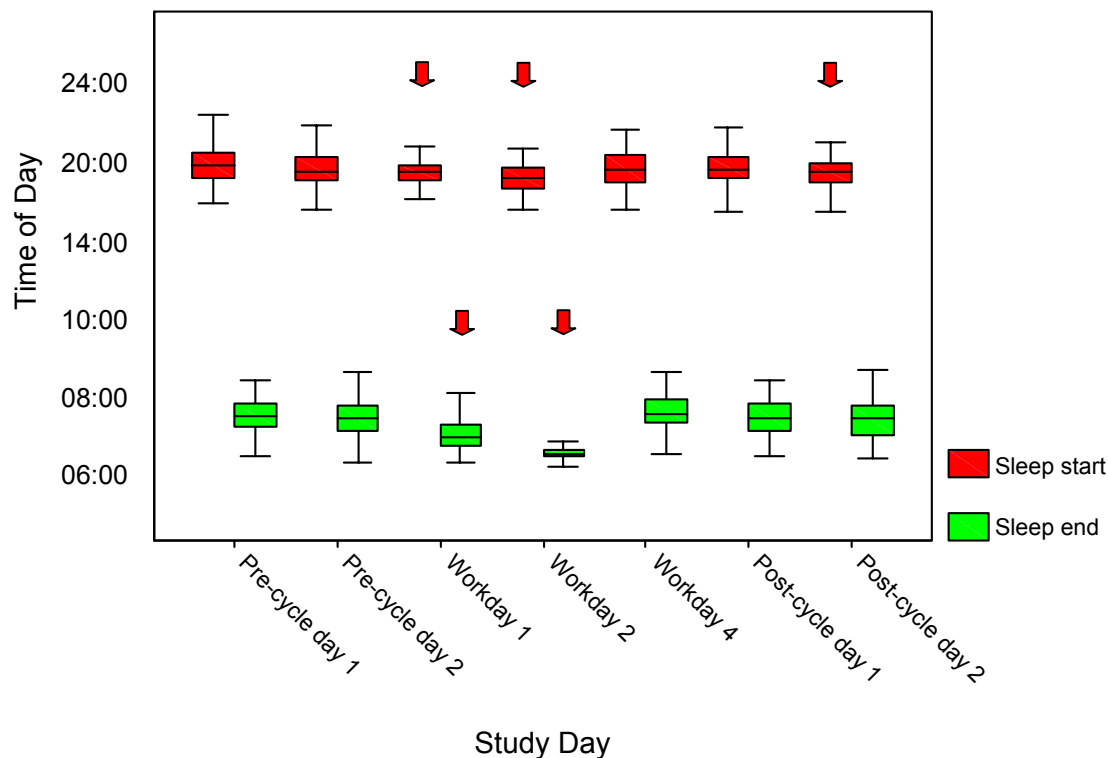


Figure 3-5: Median, Inter-Quartile Range, and Range of Sleep Start and Sleep End Times for Main Night Sleep Episodes Over the Study Period.



To investigate if rising times altered significantly across the study period and while bedtimes remained relatively unchanged, mixed model ANCOVAs were run. As detailed in Table 3-8 the results of the ANCOVA indicate that the bedtimes and sleep start times of the main night sleep episodes across the study period differed significantly across the

working week. Post hoc tests identified the days on which bedtimes and sleep start times differed. The numbered arrows in Figure 3-5 indicate these differences, which are further detailed in Table 3-22 at the end of this chapter.

The results in Table 3-8 also indicate that sleep end and get up times changed significantly across the study period. The results of the post hoc tests detailed Table 3-22 show that the sleep end and get up times of the main night sleep episode starting on workday 1 (and finishing the morning of workday 2) are significantly earlier than all other days, apart from workday 2. The sleep end and get up time of the main night sleep episode commencing on workday 2 (and finishing on the morning of workday 3) is the earliest sleep end and get up time of any main night sleep episode across the study period. This is the sleep that occurs prior to the start of the early morning shift, which also had the shortest main night sleep length parameters.

Table 3-8: Details and Results of Mixed Model ANCOVAs for the Sleep Timing Parameters of Main Night Sleep Episodes Over the Study Period

Dependent Variable	Fixed and Interaction Effects in Mixed Model	Significant Main Effects	Significant Interaction Effects
Bedtime	shift, nap, day, shift x nap, day x shift, day x nap, day x shift x nap ⁷	Day in cycle $F_{(6, 516)} = 7.71, p < .001$	
Sleep start time	As above	Day in cycle $F_{(6, 510)} = 8.43, p < .001$	
Sleep end time	As above	Day in cycle $F_{(6, 494)} = 57.08, p < .001$	
Get up time	As above	Day in cycle $F_{(6, 593)} = 63.80, p < .001$	

⁷ Shift = shift cycle worked (includes either a K1 or K2 night shift)

Nap = napping condition on night shift (nap opportunity provided or not)

Day = day within the study period (1 to 8)

3.5.4 Results: Quality of Main Night Sleep Episodes

The final series of analyses utilising main night sleep episodes investigated whether there was a change in the quality of sleep over the study period.

Figure 3-6 displays the median fragmentation values and mean activity scores (untransformed) for each main night sleep episode across the study period. The trend of decreasing mean activity in each main night sleep up to workday 2 can be clearly seen, which is followed by an increase in these values to pre-workday levels. This trend is not as clear for the fragmentation of sleep, particularly with an apparently anomalous value evident on pre-cycle day 1.

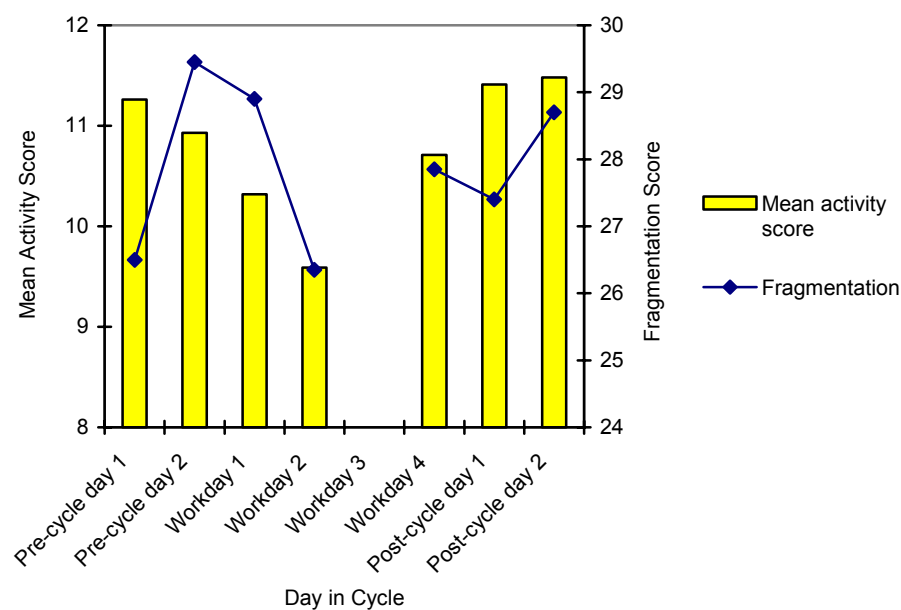


Figure 3-6: Plot of the Median Fragmentation and Mean Activity Scores for Main Night Sleep Episodes Across the Study Period.

Figure 3-7 illustrates the change in mean sleep efficiency and mean actual sleep percentage within the main night sleep episodes across the study period. As expected there is an inverse change in these values compared to the mean activity and fragmentation values. Sleep efficiency and actual sleep percentage increase across workdays, and then drop off again at the end of the work week.

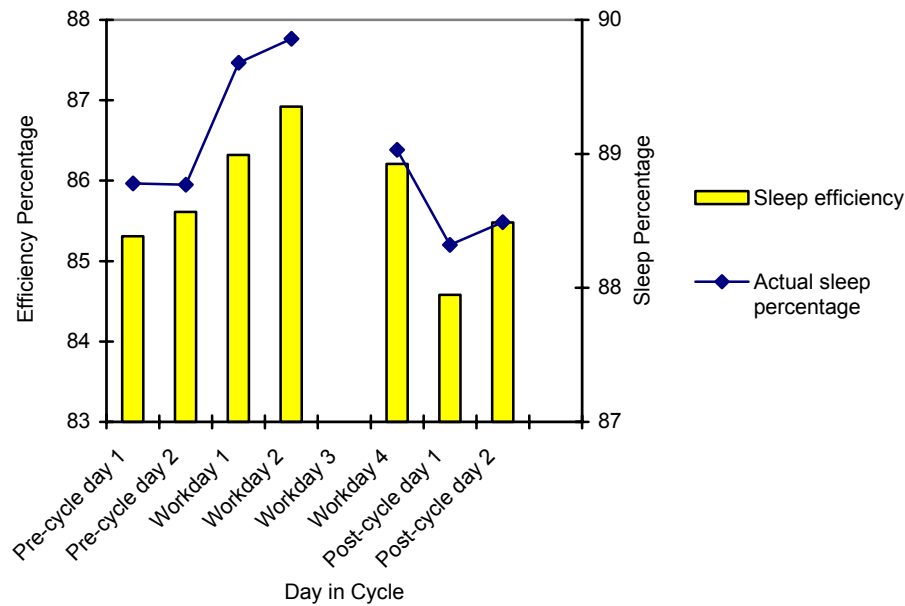


Figure 3-7: Plot of Mean Sleep Efficiency and Actual Sleep Percentage for Main Night Sleep Episodes Across the Study Period.

Table 3-9 summarises the significant main and interaction effects found in mixed model analyses that examined changes in sleep quality. Across the study period, the fragmentation of sleep did not alter. However, a significant interaction of study day by napping condition was found, as well as a significant effect for the interaction of study day by napping condition by shift type. On further investigation none of the post hoc comparisons reached significance.

The mean activity scores for main night sleep episodes were found to differ significantly between days in the study period. However, post hoc analyses identified only two significant study day differences, with the main night sleep commencing on workday 1, ($t_{(550)} = -3.65, p < .001$) and workday 2 ($t_{(587)} = -4.28, p < .001$) having lower mean activity than post-cycle day 1. In addition, although the shift type and napping condition interaction was found to be significant, no post hoc tests reached significance.

Similarly, sleep efficiency differed according to the day of the study period, indicating that there is a change in the proportion of TIB actually spent sleeping across the week studied. Further, a significant interaction of shift type with napping condition was found. Post hoc analyses identified the main night sleep commencing on workday 2 as more efficient than the sleep on post-cycle day 1 ($t_{(582)} = 4.03, p < .001$). None of the

post hoc comparisons on the interaction of shift type and napping condition reached significance.

The percentage of an assumed sleep episode that is actual sleep also changed over the study period. However, when interpreting the post hoc tests and after correcting for multiple comparisons, there were few comparisons that remained significant. Only the percentage of actual sleep on workday 1 and workday 2 was higher than during the main night sleep commencing on post-cycle day 1 ($t_{(549)} = 3.31, p = .001$ and $t_{(589)} = 3.65, p < .001$ respectively).

Table 3-9: Details and Results of Mixed Model ANCOVAs for the Sleep Quality Parameters of Main Night Sleep Episodes Over the Study Period

Dependent Variable	Fixed and Interaction Effects in Mixed Model	Significant Main Effects	Significant Interaction Effects
Fragmentation	shift, nap, day, shift x nap, day x shift, day x nap, day x shift x nap ⁸		Day in cycle x napping condition $F_{(6, 479)} = 2.24, p = .038$ Day in cycle x napping condition by shift type $F_{(6, 479)} = 2.659, p = .016$
Mean activity score	As above	Day in cycle $F_{(6, 485)} = 3.83, p = .001$	Shift type x napping condition $F_{(1, 159)} = 4.73, p = .031$
Sleep efficiency	As above	Day in cycle $F_{(6, 483)} = 3.04, p = .006$	Shift type x napping condition $F_{(1, 190)} = 7.58, p = .007$
Actual sleep percentage	As above	Day in cycle $F_{(6, 487)} = 3.20, p = .004$	

⁸ Shift = shift cycle worked (includes either a K1 or K2 night shift)
 Nap = napping condition on night shift (nap opportunity provided or not)
 Day = day within the study period (1 to 8)

3.6 Question 2: Cumulative Sleep Debt and Acute Sleep Loss

3.6.1 *Baseline Sleep Requirements*

The calculation of baseline sleep is detailed in Appendix M. The sum of all sleep that occurred between midday on one day and midday on the following day on each of the four off-duty days (pre-cycle day 1 and 2, and post-cycle days 1 and 2) was calculated. Mean baseline TIB was 8.86 hours and mean baseline assumed sleep was 8.51 hours. Baseline actual sleep averaged 7.51 hours, whereas controllers reported a sleep requirement of 8.0 hours.

3.6.2 *Analyses and Data Management*

Table 3-10 details all the dependent variables that were the basis of mixed model ANOVAs and ANCOVAs examining cumulative sleep debt and acute sleep loss amongst air traffic controllers. The various independent variables that were utilised in each model are also identified in Table 3-10. For those analyses involving the variables associated with sleep loss or gain, cumulative sleep debt, and acute sleep loss, an autoregressive structure was determined to be the most suitable covariance structure to apply. In contrast, for the analyses of cumulative sleep debt at the end of the night shift, and preparatory sleep, a compound symmetric covariance structure produced a better fitting model.

Table 3-10: Dependent and Independent Variables for Analyses Related to Cumulative Sleep Debt and Acute Sleep Loss

Dependent Variables	Independent Variables ⁹
<i>Sleep Loss or Gain and Cumulative sleep debt</i>	
TIB	Shift cycle
Assumed sleep	Nap
Actual sleep	Day
	Shift cycle x Nap
	Day x Shift cycle
	Day x Nap
	Shift cycle x Nap x Day
<i>Preparatory sleep</i>	
Bedtime	
Sleep start	
TIB	
Assumed sleep	Shift
Actual sleep	Nap
	Shift x Nap
Actual sleep time percentage	
Sleep efficiency	
Movement and fragmentation index	
Mean activity score	
<i>Acute sleep loss</i>	
TIB	Shift
Assumed sleep	Nap
Actual sleep	Time of night
	Shift x Nap
	Time of night x Shift
	Time of night x Nap
	Shift x Nap x Time of night

⁹ Shift cycle= shift cycle worked (includes either a K1 or K2 night shift)

Shift = night shift worked (either a K1 or K2)

Day = day within the study period (1 to 8)

Nap = napping condition on night shift (nap opportunity provided or not)


Baseline day = day within the study period (1, 2, 7, 8)

Time of night = time at which acute sleep debt was calculated (1st, 2nd or 3rd performance test)

3.6.3 *Results: Cumulative Sleep Debt*

Determining whether sleep episodes were restricted, and the extent and pattern of cumulative sleep loss, provides an indication as to the time in the roster cycle which may be most vulnerable to decrements in performance and increased sleepiness.

Sleep Loss or Gain Across the Study Period

The baseline values for TIB, assumed sleep and actual sleep were compared to actual TIB, assumed and actual sleep values obtained in each 24 hour period to give a value of sleep loss or gain for each day of the y. In the 24 hour period that included the night shift, the logbook values for the sleep on the night shift were used, as was the case with any other sleep episodes shorter than 1 hour.

These data were then utilised in three separate mixed model ANCOVAs to determine if sleep loss (or gain) altered significantly given the shift schedule worked, the provision or not of a napping opportunity, and the day of the week. The structure of each model, along with the results can be found in Table 3-11.

Post hoc tests determined that sleep loss (measured by TIB, assumed sleep, or actual sleep) on pre-cycle days 1 and 2, and post-cycle days 1 and 2, did not differ. Nor did sleep loss differ between the 24 hour period commencing on workday 2 and that commencing on workday 3. However, all other days differed from each other (see Table 3-23 for results).

The 24 hour period from midday on workday 3 to midday on workday 4 during the K2 shift cycle resulted in a greater loss of TIB ($t_{(725)} = 4.06, p < .001$), assumed ($t_{(726)} = 3.83, p < .001$), and actual sleep ($t_{(678)} = 4.17, p < .001$) than the same period in the K1 shift cycle. It was estimated that on the K2 shift cycle, an hour more of actual sleep was lost on average compared to the same time frame in the K1 shift cycle.

Because of these differences, the mean amount of sleep loss (or gain) in each 24 hour period across the shift cycles which include either a K1 or K2 night shift are graphed separately in Figure 3-8 and Figure 3-9. These graphs depict the mean amount of sleep lost compared to each individual's baseline sleep need for each day in the study period. Sleep loss is a negative value and sleep gain is represented by positive values.

Figure 3-9: Mean Amount of Sleep Loss (or Gain) in Each 24 Hour Period Across a Study Period that Includes a K2 Night Shift

Table 3-11: Details and Results of Mixed Model ANCOVAs Comparing the Amount of TIB, Assumed Sleep, and Actual Sleep Loss (or Gain) Over the Study Period

Dependent Variable	Fixed and Interaction Effects in Mixed Model	Significant Main Effects	Significant Interaction Effects
TIB	shift, nap, day, shift x nap, day x shift, day x nap, day x shift x nap ¹⁰	Day in cycle $F_{(7,605)} = 49.17, p < .001$	Day in cycle x shift type $F_{(7,603)} = 3.68, p < .001$
Assumed sleep	As above	Day in cycle $F_{(7,605)} = 47.10, p < .001$	Day in cycle x shift type $F_{(7,603)} = 3.11, p = .003$
Actual sleep	As above	Day in cycle $F_{(7,560)} = 48.89, p < .001$	Day in cycle x shift type $F_{(7,558)} = 3.93, p < .001$

¹⁰ Shift = shift cycle worked (includes either a K1 or K2 night shift)

Nap = napping condition on night shift (nap opportunity provided or not)

Day = day within the study period (1 to 8)

Cumulative Changes Across the Study Period

Assuming that after two full nights of unrestricted sleep an individual has repaid any previous sleep debt, controllers in the present study should have a sleep debt of zero after the sleep spanning pre-cycle day 2 to workday 1. From this time, the amount of sleep lost or gained in each 24 hour period was summed over the consecutive working days to produce a cumulative value. Because it is considered that sleep can not be stored, this cumulative value only represented an unchanging, or progressively increasing debt as opposed to also including values corresponding to a sleep “credit”.

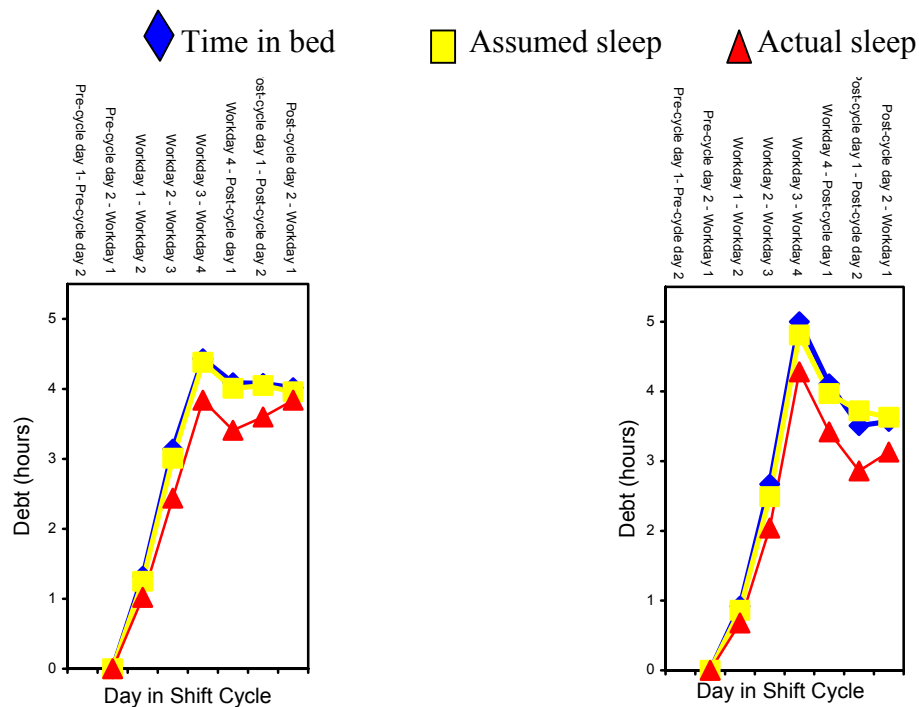


Figure 3-10: Mean Cumulative Sleep Debt Across the K1 Shift Cycle

Figure 3-11: Mean Cumulative Sleep Debt Across the K2 Shift Cycle

Figure 3-10 and Figure 3-11 display the level of cumulative sleep debt as it progressively increases across both the shift cycle containing a K1 shift and a K2 shift respectively. Table 3-12 and Table 3-13 detail the mean values for each day of the week, the range of values, as well as the extremes that are reached. Note that a debt is represented as a positive number, with increasing debt indicated by higher values. The range and standard deviation values presented in Table 3-12 and Table 3-13 show the large degree of variability that exists in the level of sleep debt accumulated by individuals. In the 24

hour period from workday 1 to workday 2, there are individuals who still maintain a zero sleep debt. However, the mean sleep debt across all sleep parameters is approximately 1 hour. In the next two consecutive 24 hour periods, from workday 2-3 and workday 3-4, almost all individuals loose sleep and for some it is a considerable amount. For instance, during a K2 shift cycle a single individual accumulated an assumed sleep debt of nearly 14 hours by midday on workday 4. In the following 24 hour period from workday 4 to post-cycle day 1, recovery sleep occurs and the mean sleep debt is lessened somewhat.

A series of mixed model ANCOVAs were calculated to investigate whether the change in cumulative sleep debt across study days was significant for each of the three sleep parameters. Details of the fixed and interaction effects included in each mixed model can be found in Table 3-14. For all three models, the days compared were from the second study day until the end of the week, i.e. values for pre-cycle day 2-workday 1 until post-cycle day 2-workday 1.

Table 3-12: Mean, Standard Deviation, and Range of Cumulative Sleep Debts for TIB, Assumed Sleep, and Actual Sleep Across a K1 Shift Cycle

Day in Study Period	TIB (h)				Assumed Sleep (h)				Actual Sleep (h)			
	<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>
Workday 1-Workday 2	1.32	0.98	0-3.88	52	1.25	0.96	0-3.38	52	1.02	0.80	0-2.68	51
Workday 2-Workday 3	3.14	1.48	0.65-6.76	52	3.01	1.53	0.31-6.80	52	2.44	1.26	0.44-5.33	50
Workday 3-Workday 4	4.43	2.30	0.33-10.90	52	4.38	2.31	0.18-9.69	52	3.84	1.72	0.96-7.98	49
Workday 4-Post-cycle day 1	4.09	2.71	0-11.49	52	4.01	2.62	0-10.87	52	3.41	1.94	0-8.32	48
Post-cycle day 1-Post-cycle day 2	4.08	2.89	0-11.85	41	4.05	2.88	0-11.83	41	3.60	2.38	0-9.92	38
Post-cycle day 2-Workday 1	4.02	3.56	0-15.46	27	3.96	3.50	0-14.52	27	3.84	3.16	0.44-13.54	26

Table 3-13: Mean, Standard Deviation, and Range of Cumulative Sleep Debts for TIB, Assumed, and Actual Sleep Across a K2 Shift Cycle

Day in Study Period	TIB (h)				Assumed Sleep (h)				Actual Sleep (h)			
	<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>
Workday 1-Workday 2	1.02	1.08	0-3.68	52	0.96	1.04	0-3.82	52	0.74	0.87	0-3.13	48
Workday 2-Workday 3	2.88	1.49	0.23-7.09	52	2.71	1.39	0.12-6.56	52	2.13	1.25	0-5.42	47
Workday 3-Workday 4	5.19	2.73	0.40-15.08	52	5.02	2.50	0.97-13.67	52	4.56	2.21	1.03-10.49	45
Workday 4-Post-cycle day 1	4.40	3.17	0-15.12	52	4.04	3.02	0-3.13	52	3.42	2.30	0-9.01	41
Post-cycle day 1-Post-cycle day 2	3.41	2.76	0-9.16	42	3.72	2.84	0-10.09	42	2.86	2.31	0-9.54	32
Post-cycle day 2-Workday 1	3.57	3.29	0-13.85	28	3.63	3.13	0-11.97	28	3.13	2.49	0-9.92	22

Table 3-14: Details and Results of Mixed Model ANCOVAs Investigating the Change in Cumulative Sleep Debt

Dependent Variable	Fixed and Interaction Effects in Mixed Model	Significant Main Effects	Significant Interaction Effects
Cumulative TIB debt	shift, nap, day, shift x nap, day x shift, day x nap, day x shift x nap ¹¹	Day in cycle $F_{(5,422)} = 77.11, p < .001$	Day in cycle x shift type $F_{(5,422)} = 3.24, p = .007$
Cumulative assumed sleep debt	As above	Day in cycle $F_{(5,424)} = 78.82, p < .001$	Day in cycle x shift type $F_{(5,425)} = 2.68, p = .02$
Cumulative actual sleep debt	As above	Day in cycle $F_{(5,378)} = 81.58, p < .001$	Day in cycle x shift type $F_{(5,378)} = 4.50, p < .001$

¹¹ Shift = shift cycle worked (includes either a K1 or K2 night shift)
 Nap = napping condition on night shift (nap opportunity provided or not)
 Day = day within the study period (1 to 8)

As can be seen in Table 3-14, the level of cumulative debt was found to differ significantly across the days within the study period, as well as show a significant shift-by-study day interaction for all three cumulative sleep parameters. For all three cumulative sleep parameters the post hoc tests indicate that the level of debt differed significantly for each day compared to every other day from pre-cycle day 2 until workday 3, although for the cumulative TIB debt, workday 3 did not differ from post-cycle day 2 and for cumulative assumed debt workday 3 did not differ from either post-cycle day 1 or 2. The post hoc tests indicate that the cumulative debt decreases significantly between workday 3 and workday 4 for all three sleep parameters. In all instances the levels of cumulative debt on workday 4, post-cycle day 1 and post-cycle day 2 did not differ from each other. The details of the significant post hoc tests can be found at the end of this chapter in Table 3-24. The level of cumulative sleep debt on successive days did not differ significantly between the two shift schedules.

3.6.4 *Preparatory Sleep*

An assessment was made of the quantity of, and the regularity with which, participants obtained preparatory sleep prior to the night shift. Sleep prior to the night shift is important in two respects. First, it will influence the level of acute sleep loss experienced, and second, the length of time since sleep was last obtained will determine the homeostatic drive for sleep.


Any sleep episode that occurred between the end of the morning shift and the beginning of the night shift was included in these analyses.

Participants slept prior to 90% of the 107 night shifts monitored. Those who slept spent a mean of 2.52 hours in bed ($SD = 0.99$, $Range = 0.28-5.15$, $N = 95$) obtaining on average 2.32 hours of assumed sleep ($SD = 0.99$, $Range = 0.28-5.03$ hrs, $N = 95$) and a mean of 2.16 hours actual sleep ($SD = .90$, $Range = 0.52-4.85$, $N = 87$). Where an individual had more than one sleep episode, the sleep length reported is the total sleep obtained, and the start time utilised in the calculations was that of the first sleep episode. Further details on the length, quality, and timing of sleep prior to either the K1 or K2 night shift can be seen in Table 3-15.

Table 3-15: Mean (or Median), Standard Deviation, and Range of the Length, Quality, and Timing of Sleep Prior to Each Type of Night Shift

Sleep Parameter	Shift Type	Descriptive Statistics			
		<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>
TIB	K1	1.99	1.18	0-4.62	53
	K2	2.27	1.28	0-5.15	54
Assumed sleep length	K1	1.85	1.16	0-4.60	53
	K2	2.10	1.26	0-5.03	54
Actual sleep length	K1	2.10	0.82	0.63-4.20	42
	K2	2.17	1.01	0.52-4.85	45
Fragmentation	K1	10.90*		3.40-55.60	42
	K2	22.50*		1.50-81.50	45
Mean activity	K1	6.59*		0.59-36.81	42
	K2	8.53*		1.14-38.58	45
Sleep efficiency	K1	85.75*		49.40-98.50	42
	K2	85.40*		47.80-97.10	45
Actual sleep percentage	K1	92.45*		71.70-99.50	42
	K2	91.30*		63.30-98.60	45
Bedtime	K1	15:57	2:20	10:10-20:18	45
	K2	17:30	2:40	10:58-22:13	50
Sleep start	K1	15:54	2:20	10:12-20:40	43
	K2	17:36	2:44	10:59-22:38	50

*median reported rather than mean, as these variables were not normally distributed

Mixed model ANOVAs were calculated to determine if the experimental condition in any way influenced the length, timing, or quality of the pre-shift p. Despite the fact that the descriptive statistics in Table 3-15 suggest that sleep prior to the K1 night shift is

shorter than that prior to the K2 night shift, none of the mixed models produced significant findings.

Comparable analyses for sleep quality variables (actual sleep percentage, sleep efficiency, and mean activity) also showed no significant differences due to shift type or napping condition. However, fragmentation of sleep did differ significantly according to the shift type, with sleep prior to the K1 night shift being less fragmented than that prior to the K2 night shift ($F_{(1,64.8)} = 5.92, p = .02$). For sleep timing there was also a significant effect of shift type, with individuals tending to go to bed and get to sleep 1.3 hours earlier when they were rostered on a K1 shift than on a K2 shift ($F_{(1,67.8)} = 12.13, p < .001$ and $F_{(1,66.5)} = 11.99, p < .001$ respectively).

3.6.5 *Results: Acute Sleep Loss*

It is not only relevant to investigate cumulative sleep debt but also to determine the degree of acute sleep loss that individuals are experiencing at critical times in the roster cycle. Like cumulative sleep debt, the level of acute sleep loss may assist in indicating time points where performance and sleepiness are the most impaired.

Calculating Acute Sleep Loss

The time points at which acute sleep loss was determined were those at which performance data were also available, this being approximately the beginning, middle and end of the night shift. To determine acute sleep loss, a comparison was made between the amount of sleep obtained in the 24 hours preceding the chosen points in time and the individual's respective baseline requirement. For example, the first performance test on K1 night shift began at approximately 2230 hours, therefore all sleep that occurred in the 24 hours previous to that time was summed and the difference between that value and their baseline sleep value was deemed acute sleep loss. An example of the sleep episodes that were spanned when calculating the sleep in the 24 hours prior to each test is graphically illustrated in Figure 3-12. As with cumulative sleep debt, an individual could not have a negative acute sleep loss (e.g. if they attained more sleep than their baseline value in a 24 hour period). Instead they were deemed to have zero debt.

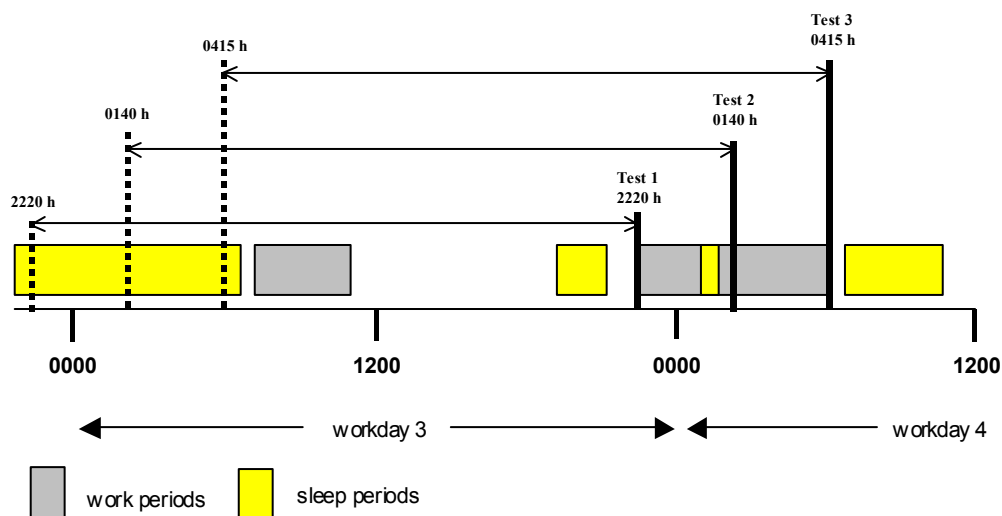


Figure 3-12: Example of Sleep Episodes Spanned When Calculating Acute Sleep Loss Prior to Each Performance Test on a K1 Night Shift

If a nap had been taken during the night shift, the acute sleep loss for the last two performance tests would be affected. Because the time frames involved in these calculations were much shorter overall than those used when looking at cumulative sleep debt (i.e. a single 24 hour period compared to 8 x 24 hour periods) it was deemed necessary to be more accurate in assessing the amount of sleep obtained during the napping opportunity. On this basis, polysomnography was used to measure the length of the nap sleep. To determine acute TIB sleep loss, actigraphically estimated TIB values plus, if relevant, the length of time analysed polysomnographically in the napping opportunity, were summed. For acute assumed sleep loss, the polysomnographic variable used was the length of time from sleep onset to the last scored epoch of sleep. Actigraphically estimated actual sleep time was added to polysomnographically determined total sleep time to calculate the acute actual sleep loss.

Acute Sleep Loss According to Shift Type and Napping Condition

Table 3-16 details the descriptive statistics for each shift type at each of the three times acute sleep loss was calculated. Mixed model ANOVA's were calculated to determine if there were shift type and/or napping condition differences in acute sleep loss values.

The time at which the debt was calculated was an additional fixed factor in each ANOVA. This factor was included in models in order to determine if the level of acute

sleep loss altered significantly across the night shift. Although the exact time of each performance test was used in calculating acute sleep loss leading up to it, within the mixed models the acute sleep loss was classified as relating to either the first, second or third performance test of the night shift. The distribution of each of the three tests in relation to time of night can be seen in Chapter 5, Table 5-1.

Further details and the results of the ANOVAs are presented in Table 3-17, which show a significant interaction of shift type with the time at which the debt was determined for all three dependent variables. Post hoc analyses identified shift type differences in all acute sleep loss values at the first and second performance test of the night. Only acute assumed sleep loss differed significantly between shifts at the last performance test of the night. In addition, as would be expected, acute TIB sleep loss differed significantly according to whether a napping opportunity was provided or not. However, neither acute assumed or actual sleep loss differed in association with the napping condition.

Finally, there was a napping condition by time of night interaction for acute TIB sleep loss, and from the post hoc test results it is apparent that only at the second performance test of the night did sleep loss differ significantly according to whether a napping opportunity was provided or not. The details of all these findings can be seen in Table 3-25 at the end of this chapter.

Table 3-16: Descriptive Statistics for Acute TIB, Assumed Sleep, and Actual Sleep Loss at the Beginning, Middle and End of the Night Shift

Test Sequence	Shift	TIB Sleep Loss (h)			Assumed Sleep Loss (h)			Actual Sleep Loss (h)		
			<i>Range</i>	<i>N</i>	<i>Median</i>	<i>Range</i>	<i>N</i>	<i>Median</i>	<i>Range</i>	<i>N</i>
1	K1	0.03	0-3.89	50	0.05	0-3.60	50	0.02	0-2.66	46
	K2	0.84	0-4.15	53	0.77	0-4.02	53	0.61	0-3.13	46
2	K1	2.70	0.12-6.19	50	2.78	0.29-6.69	50	2.53	0.56-6.34	46
	K2	4.52	0.99-8.41	53	4.73	1.59-7.88	53	4.45	1.68-7.33	46
3	K1	5.12	2.35-8.76	50	5.33	2.65-8.51	50	4.63	2.29-7.59	47
	K2	6.37	2.63-10.08	53	6.15	3.15-9.51	52	5.40	2.82-7.91	48

NB: A positive value represents a sleep debt

Table 3-17: Details and Results of Mixed Model ANOVAs Comparing the Acute TIB, and Acute Actual Sleep Loss Values Across the Night Shift

Dependent variable	Fixed and Interaction Effects in Mixed Model	Significant Main Effects	Significant Interaction Effects
TIB	shift, nap, time, shift x nap, time x shift, time x nap, time x shift x nap ¹²	Shift type $F_{(1,99.9)} = 13.39, p < .001$ Napping condition $F_{(1,99.9)} = 4.68, p = .033$ Time of test $F_{(2,195)} = 989.28, p < .001$	Shift type x Time of test $F_{(2,195)} = 25.70, p < .001$ Napping condition by Time of test $F_{(2,1950)} = 12.40, p < .001$
Assumed sleep	As above	Shift type $F_{(1,99.5)} = 13.19, p < .001$ Time of test $F_{(2,195)} = 1000.86, p < .001$	Shift type x Time of test $F_{(2,195)} = 22.42, p < .001$
Actual sleep	As above	Shift type $F_{(1,67.81)} = 29.15, p < .001$ Time of test $F_{(2,171)} = 1093.52, p < .001$	Shift type x Time of test $F_{(2,171)} = 20.00, p < .001$

¹² Shift = shift cycle worked (includes either a K1 or K2 night shift)

Nap = napping condition on night shift (nap opportunity provided or not)

Time = time of the performance test (1st, 2nd or 3rd)

3.7 Question 3: Workplace Naps and Post-Night Shift Sleep

3.7.1 Analyses and Data Management

The final question addressed in this chapter was whether the opportunity to nap during the night shift affected the timing, length, or quality of the first sleep episode after the night shift.

A series of mixed model ANOVA's were run with sleep length, quality, and timing parameters as the dependent variables and a compound covariance structure was found to best model the relationship between repeated measures. The dependent and independent variables included in this series of analyses can be seen in Table 3-18.

Table 3-18: Dependent and Independent Variables for Analyses Related to Question 3

Dependent variables	Independent variables ¹³
<i>Sleep timing</i>	
Bedtime	
Sleep start	
Sleep end	
Get up time	
<i>Sleep length</i>	
TIB	Shift
Assumed sleep	Nap
Actual sleep	Shift * Nap
<i>Sleep quality</i>	
Actual sleep time percentage	
Sleep efficiency	
Movement and fragmentation index	
Mean activity score	

3.7.2 Results: Post-Night Shift Sleep

The mean or median length, quality and timing of sleep post either the K1 or K2 night shift can be seen in Table 3-19.

¹³ Shift = night shift worked (either a K1 or K2)

Nap = napping condition on night shift (nap opportunity provided or not)

Details and results of the mixed models can be seen in Table 3-20 and the findings clearly show that the opportunity to nap had no significant effect on any of the post-night shift sleep parameters. However, the type of night shift that was worked did affect subsequent sleep. As can be seen from Table 3-19, the sleep following the K1 night shift started earlier, and was longer but of poorer quality than the sleep subsequent to the K2 night shift.

Table 3-19: Mean (or Median), Standard Deviation, and Range of the Length, Quality, and Timing of Initial Sleep After Each Type of Night Shift

Sleep Parameter	Shift Type	Descriptive Statistics			
		<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>
TIB	K1	5.20	1.33	2.05-7.65	51
	K2	4.50	1.28	1.33-7.63	53
Assumed sleep length	K1	4.96	1.30	1.67-7.63	51
	K2	4.30	1.24	1.32-7.28	53
Actual sleep length	K1	4.49	1.15	1.55-7.23	49
	K2	3.88	1.16	1.23-6.72	47
Fragmentation	K1	26.39	11.11	6.70-53.40	49
	K2	21.67	11.88	4.00-60.70	47
Mean activity	K1	11.31	6.60	0.63-31.47	49
	K2	8.91	4.77	1.62-22.85	47
Sleep efficiency	K1	85.46	6.78	69.80-98.80	49
	K2	87.93	6.62	61.70-95.50	47
Actual sleep percentage	K1	89.72	5.12	75.70-99.60	49
	K2	91.65	3.76	82.70-97.10	47
Bedtime	K1	05:21*		04:31-07:13	51
	K2	07:27*		06:31-11:01	53
Sleep start	K1	05:29*		04:34-07:16	51
	K2	07:30*		06:32-11:03	53

*median reported rather than mean, as these variables were not normally distributed

Table 3-20: Details and Results of Mixed Model ANOVAs for the Length, Quality, and Timing of the First Sleep After the Night Shift

Dependent variable	Fixed and Interaction Effects in Mixed Model	Significant Main Effects	Significant Interaction Effects
Length of TIB	shift, nap, shift*nap, ¹⁴	Shift type $F_{(1,77)} = 8.88, p = .004$	
Assumed sleep length	As above	Shift type $F_{(1,77.3)} = 7.69, p = .007$	
Actual sleep length	As above	Shift type $F_{(1,72.7)} = 4.91, p = .030$	
Fragmentation	As above	Shift type $F_{(1,70.3)} = 8.17, p = .005$	
Mean activity	As above	Shift type $F_{(1,70.2)} = 7.54, p = .008$	
Sleep efficiency	As above	Shift type $F_{(1,70.9)} = 5.20, p = .026$	
Actual sleep percentage	As above	Shift type $F_{(1,70.4)} = 5.88, p = .018$	
Bedtime	As above	Shift type $F_{(1,76.4)} = 394.12, p < .001$	
Sleep start	As above	Shift type $F_{(1,76.6)} = 362.18, p < .001$	

¹⁴ Shift = shift cycle worked (includes either a K1 or K2 night shift)

Nap = napping condition on night shift (nap opportunity provided or not)

3.8 Summary

Q1. Does the length, timing, and quality of sleep episodes change across the shift cycle worked by air traffic controllers?

There was strong, consistent evidence that the sleep of air traffic controllers changed across the working week. As hypothesised, the backward rotating shift cycle, and consequently progressively earlier starts, resulted in earlier rising times which were responsible for the reduced amount of sleep across the working week. However, contrary to the first hypothesis, the sleep start times became slightly earlier, although in no way enough to overcome the effects of the earlier rising times. It was also hypothesised that sleep quality would not alter. Although some measures of sleep quality were found to change in relation to changes in the work pattern, the findings were not as consistent as those associated with the timing and length of sleep.

Q2. Are air traffic controllers cumulatively and/or acutely sleep deprived when working the night shift?

Every air traffic controller participating in this study was found to be cumulatively sleep deprived by midday prior to commencing the night shift. The cumulative sleep debt peaked by the end of the night shift. At this point in time the maximum assumed debt experienced was just under 14 hours, although values did vary widely. Unsurprisingly, air traffic controllers also experience acute sleep loss on the night shift, although the levels are not high until the middle and end of the shift. Of greater interest is the finding that the nap opportunity does not significantly alter the degree of assumed or actual acute sleep loss, indicating that the amount of sleep obtained during the napping opportunity is small compared to the variability between individuals in their sleep in the preceding 24 hours.



Q3. Do pre-planned workplace naps affect the length, timing, and quality of post night shift sleep?

The opportunity to nap was not found to influence the length, timing or quality of sleep immediately after the night shift. However, it was determined that the type of night shift worked influenced the characteristics of the first sleep subsequent to the shift. The

earlier starting and earlier finishing shift resulted in an earlier, longer, but slightly poorer quality sleep than the later night shift.

Together the results of this chapter indicate that the shift cycle worked by air traffic controllers progressively reduces the opportunity for sleep, which in turn leads to a rapid build up of sleep debt that peaks at the end of the night shift. Both cumulative sleep debt and acute sleep loss values indicate that sleep loss is a greater issue on the K2 night shift, which is also followed by less initial recovery sleep.

Table 3-21: Results of the Post Hoc Tests for Sleep Length Parameters of Main Night Sleep Episodes Across the Study Period

Significant Differences	TIB		Assumed Sleep Length		Actual Sleep Length	
	<i>df</i>	<i>t</i>	<i>df</i>	<i>t</i>	<i>df</i>	<i>t</i>
						
Workday 1 vs Pre-cycle day 1	634	4.40**	634	4.15**	587	2.98*
Workday 1 vs Pre-cycle day 2	420	5.31**	418	4.74**	381	4.04**
Workday 1 vs Workday 2	423	5.75**	421	5.48**	387	5.25**
Workday 1 vs Workday 4	634	-6.86**	634	-6.15**	588	-5.76**
Workday 1 vs Post-cycle day 1	632	-5.31**	632	-4.47**	588	-3.64*
Workday 1 vs Post-cycle day 2	635	-5.60**	636	-5.49**	594	-4.77**
						
Workday 2 vs Pre-cycle day 1	632	10.05**	633	9.56**	588	8.13**
Workday 2 vs Pre-cycle day 2	634	11.03**	633	10.21**	587	9.32**
Workday 2 vs Workday 4	421	-12.72**	419	-11.71**	394	-10.97**
Workday 2 vs Post-cycle day 1	634	-11.06**	633	-9.97**	587	-8.92**
Workday 2 vs Post-cycle day 2	636	-10.70**	636	-10.35**	593	-9.47**

* $p < .005$ ** $p < .0001$

Table 3-22: Estimates of Differences and Results of the Post Hoc Tests for Sleep Timing Parameters of Main Night Sleep Episodes Across the Study Period

Significant Differences	↓	Bedtime			Sleep Start Time			↓	Sleep End Time			Get Up Time		
		Difference (minutes)	<i>df</i>	<i>t</i>	Difference (minutes)	<i>df</i>	<i>t</i>		Difference (minutes)	<i>df</i>	<i>t</i>	Difference (minutes)	<i>df</i>	<i>t</i>
Workday 1 vs Pre-cycle day 1		30	638	3.82**	29	632	3.63**		69	640	6.55**	74	640	7.10**
Workday 1 vs Pre-cycle day 2									60	493	6.04**	64	391	6.59**
Workday 1 vs Workday 2									78	394	7.86**	79	392	8.12**
Workday 1 vs Workday 4									72	639	-6.87**	76	639	-7.40**
Workday 1 vs Post-cycle day 1									64	616	-6.06**	71	616	-6.88**
Workday 1 vs Post-cycle day 2									74	623	-6.30**	75	622	-6.49**
	↓							↓						
Workday 2 vs Pre-cycle day 1		49	624	6.11**	50	620			147	618	13.83**	153	618	14.60**
Workday 2 vs Pre-cycle day 2		31	638	3.94**	34	632	4.34**		137	639	13.20**	143	639	13.95**
Workday 2 vs Workday 4		31	422	-4.09**	36	418	-4.72**		150	396	-15.08**	155	393	-15.90**
Workday 2 vs Post-cycle day 1		38	638	-4.88**	41	625	-5.19**		141	639	-13.54**	150	639	-14.60**
Workday 2 vs Post-cycle day 2									152	628	-12.90**	155	627	-13.29**
	↓													
Post-cycle day 2 vs Pre-cycle day 1		29	628	3.30**	29	623	3.18**							

* $p < .005$ ** $p < .001$

Table 3-23: Significant Post Hoc Comparisons for Sleep Lost (or Gained)

24 Hour Periods (starting on indicated day)	TIB		Assumed Sleep Length		Actual Sleep Length	
	<i>df</i>	<i>t</i>	<i>df</i>	<i>t</i>	<i>df</i>	<i>t</i>
Pre-cycle day 1 vs Workday 1	720	4.34**	721	4.21**	674	3.12*
Pre-cycle day 1 vs Workday 2	723	8.73**	724	8.51**	674	7.18**
Pre-cycle day 1 vs Workday 3	725	8.66**	725	9.10**	679	9.91**
Pre-cycle day 1 vs Workday 4	725	-4.35**	725	-3.87**	679	-4.62**
Pre-cycle day 2 vs Workday 1	455	4.78**	455	4.44**	406	3.73**
Pre-cycle day 2 vs Workday 2	719	9.47**	720	9.00**	672	8.10**
Pre-cycle day 2 vs Workday 3	722	9.38**	723	9.59**	673	10.86**
Pre-cycle day 2 vs Workday 4	724	-3.84**	724	-3.59**	676	-4.00**
Workday 1 vs Workday 2	456	4.26**	456	4.17**	412	3.97**
Workday 1 vs Workday 3	720	4.36**	720	4.93**	674	6.96**
Workday 1 vs Workday 4	723	-8.78**	724	-8.17**	675	-7.87**
Workday 1 vs Post-cycle day 1	737	-4.63**	738	-3.98**	691	-2.93*
Workday 1 vs Post-cycle day 2	732	-5.45**	733	-5.51**	686	-4.76**
Workday 2 vs Workday 4	720	-13.31**	720	-12.59**	673	-12.07**
Workday 2 vs Post-cycle day 1	735	-8.85**	736	-8.11**	687	-6.85**
Workday 2 vs Post-cycle day 2	732	-9.41**	733	-9.38**	685	-8.48**
Workday 3 vs Workday 4	454	-12.70**	455	-12.69**	417	-14.17**
Workday 3 vs Post-cycle day 1	735	-8.80**	736	-8.69**	691	-9.51**
Workday 3 vs Post-cycle day 2	732	-9.34**	732	-9.91**	685	-10.96**
Workday 4 vs Post-cycle day 1	491	3.56**	492	3.63**	444	4.34**

* $p < .005$ ** $p < .001$

Table 3-24: Results of Significant Post Hoc Comparisons for Cumulative TIB, Assumed Sleep, and Actual Sleep Debt

24 Hour Period (begins on specified day)	TIB		Assumed Sleep Length		Actual Sleep Length	
	<i>df</i>	<i>t</i>	<i>df</i>	<i>t</i>	<i>df</i>	<i>t</i>
Pre-cycle day 2 vs Workday 1	506	8.01**	507	7.74**	460	7.43**
Pre-cycle day 2 vs Workday 2	590	15.54**	591	15.16**	541	14.85**
Pre-cycle day 2 vs Workday 3	604	21.56**	603	21.66**	547	23.72**
Pre-cycle day 2 vs Workday 4	560	17.44**	557	17.80**	502	18.34**
Pre-cycle day 2 vs Post-cycle day 1	516	15.79**	513	16.49**	464	16.85**
Pre-cycle day 2 vs Post-cycle day 2	503	14.14**	500	14.65**	455	14.28**
Workday 1 vs Workday 2	504	12.62**	505	12.33**	456	11.90**
Workday 1 vs Workday 3	588	18.81**	589	19.06**	538	21.16**
Workday 1 vs Workday 4	604	13.76**	604	14.27**	549	14.72**
Workday 1 vs Post-cycle day 1	579	12.02**	577	12.85**	522	13.16**
Workday 1 vs Post-cycle day 2	558	10.61**	555	11.21**	501	10.84**
Workday 2 vs Workday 3	503	12.36	504	12.92**	455	15.69**
Workday 2 vs Workday 4	587	6.37**	588	7.12**	538	7.74**
Workday 2 vs Post-cycle day 1	611	5.16**	611	6.18**	555	6.63**
Workday 2 vs Post-cycle day 2	602	4.56**	599	5.30**	540	5.03**
Workday 3 vs Workday 4	503	-3.90**	504	-3.48**	458	-5.28**
Workday 3 vs Post-cycle day 1	588	-2.88*			541	-3.57**
Workday 3 vs Post-cycle day 2					562	-3.31*
Workday 4 vs Post-cycle day 1					444	4.34**

* $p < .005$ ** $p < .001$

Table 3-25: Results of Significant Post Hoc Comparisons for Acute TIB, Assumed Sleep, and Actual Sleep Loss

Significant Differences	TIB in Loss		Assumed Acute Loss		Actual Acute Loss	
	<i>df</i>	<i>t</i>	<i>df</i>	<i>t</i>	<i>df</i>	<i>t</i>
<i>K1 shift versus K2 shift</i>						
Test 1	136	-2.85*	141	-2.92*	127	-3.87**
Test 2	136	-5.46**	141	-5.39**	127	-7.26**
Test 3					124	-2.31*
<i>Nap versus no nap</i>						
Test 2	136	-3.39**				

* $p < .05$

** $p < .001$

3.9 Data Management

Transformations Used in Section 3.4

When checking the assumptions prior to the mixed model for total working hours over the eight day study period, two values were identified as outliers. The model was run twice, once with and again without the outliers. Both models produced similar output, with neither model showing significant shift or nap differences. Total work hours over the four rostered days were checked for normality and it was necessary to apply an exponential transformation (constant = 0.0001) to this data to eliminate the negatively skewed distribution.

Transformations Used in Section 3.5

For those models with TIB and assumed sleep length as the dependent variable, a single outlier was removed from the data, as the outlier represented an extremely short main night sleep episode on post-cycle day two. Removing this value did not alter the findings in the model where assumed sleep length was the dependent variable. However, the results reported do not include this value, because removing it altered the findings for TIB and secondly because it was not considered representative of sleep length compared to all other sleep length parameters available for that individual.

For the dependent variables of bedtime and sleep start, two separate models were run due to the presence of three extreme residual values. However, the findings did not alter when these values were removed from the analysis. Consequently the reported results include the outlying values.

When investigating whether there were changes in sleep start and sleep end times, two extreme outliers were identified in the data. The reported results from the mixed models contain these outlying values, as the results did not alter when these values were removed.

Fragmentation values were transformed using a square root function, as it was determined the distribution was positively skewed. For the mixed model investigating possible changes in the fragmentation of sleep over the study period, a single outlier was removed from the analysis as it displayed an extremely high value. Mixed models were run with and without this outlying value and although including it did not change the

outcome of the model, the value was assessed as being unusual for that individual. Exclusion of the outlying value also produced normally distributed residuals, whereas this was not the case when this value was included. It was therefore decided to report the results that did not include this outlying value.

The variable of mean activity was positively skewed and was transformed using the square root function. After transformation four residual values were identified as outliers, but removal of them made no difference to the results. The reported findings therefore include these values.

When analysing the efficiency of sleep over the study period, seven outlying values were identified and the mixed models were subsequently run with and without these values. The results did not differ between these two analyses and the reported findings include the outlying values.

Two clear outliers were identified in the distribution of the residual actual sleep percentage values, and the models were run with and without these values. The results of these two models differed and on this basis the results excluding the outliers have been reported.

Transformations Used in Section 3.6

For all three mixed models associated with the dependent variables of sleep loss (gain), three outlying values were identified, and each model was run with and without the outliers. However, removing them did not alter the results, therefore they are included in the reported findings.

Outliers were identified in the residual plots produced from each of the three models for cumulative sleep debt. In all instances models were re-run after removing these extreme values. For the models with cumulative TIB and assumed sleep debt as the dependent variable, removing the outliers did not alter the main findings. Therefore, the results reported for these variables include the outlying values. For the model that had cumulative actual sleep debt as a dependent variable two outlying values were removed, which in turn altered the main findings, therefore the results do not include these extreme scores.

All preparatory pre-night shift sleep quality variables produced skewed distributions, and each was transformed as necessary to correct this. Both actual sleep percentage and sleep efficiency were moderately negatively skewed, which was adjusted by reflecting the values then applying a square root function. Mean activity and fragmentation values were positively skewed, and both variables were transformed by applying a logarithmic function. Analysis of the residuals in the model of mean activity and fragmentation identified two outlying values, but re-running the mixed model after removal of these values did not alter the results.

All acute sleep loss variables were positively skewed, which was corrected by applying a logarithmic transform to each distribution.

Transformations Used in Section 3.7

Prior to these analyses it was necessary to transform all sleep quality variables to produce normally distributed data. This was achieved for actual sleep percentage and sleep efficiency by reflecting the values then applying a square root function. A square root transform removed the positive skewness seen in the distributions for mean activity and fragmentation values. Bedtime and sleep start were also both severely positively skewed, which was reduced by applying a reciprocal transform. In addition, two outlying values were identified in the residual plots from both the bedtime and sleep start models. These outliers were removed and the results reported do not include them, due to changed findings.

The first episode of sleep that occurred after the end of night shift but before 2000 hours that day was included in these analyses. On two occasions individuals didn't sleep at all during the day after the night shift, resulting in a 105 sleep episodes being considered as the first sleep post the night shift. On 17 occasions individuals had more than one sleep during the day but 15 of these occurred after midday and were therefore not included in this series of analyses.

CHAPTER 4

POLYSOMNOGRAPHIC ANALYSIS OF THE PRE-PLANNED NAPPING OPPORTUNITIES PROVIDED ON THE NIGHT SHIFT

4.1 Introduction

The focus of the initial part of this chapter is on describing the characteristics of sleep obtained during the napping opportunity and whether they are related to prior sleep or the timing of the napping opportunity. This information is relevant in subsequent analyses that address whether the length and quality of the sleep obtained during the nap will have an important influence on performance and neurophysiological alertness during the remainder of the night shift.

The second section of this chapter reports on subjective data obtained at the end of the night shift and investigates the nature of the relationship between these data and that obtained from polysomnography.

Finally, it was necessary to determine whether study participants slept at any stage during the night shift other than within the napping opportunity. The final section of this chapter describes these analyses.

The relevant research questions and hypotheses for this chapter are as follows:

- Q1. How much polysomnographically defined sleep do air traffic controllers obtain during the napping opportunities on the night shift?
- H1. It is hypothesised that due to the circadian phase at which the napping opportunity occurs, on average, air traffic controllers will be able to initiate sleep at work relatively rapidly. However, it is predicted that sleep latencies will be longer than those for a normal night's sleep due to ambient factors such as location. Hence, sleep length will be much less than the length of the sleep opportunity provided and the efficiency of the sleep will be relatively low.

- Q2. Does the length, depth or quality of the sleep obtained differ according to the level of acute sleep loss, length of prior wake, or the timing of the nap?
- H1. It is hypothesised that the amount of sleep obtained in the 24 hours prior to the napping opportunity, and the length of time since an individual last slept, will have an influence on the length and depth of sleep obtained in the nap.
- H2. Circadian influences are also expected to play a role in determining the length and structure of the sleep obtained during the napping opportunity. However, because the change in timing of the napping opportunity is not large, and both napping opportunities occur along the descending arm of the circadian temperature rhythm, the differences are not expected to be large. The expected trend, if any, would be for the later sleep to be longer, have shorter latencies and contain more SWS.
- Q3. Is there any evidence that sleep obtained during the first study night differed from sleep obtained on subsequent study nights?
- H3. Controllers who regularly napped at work slept in the same location and under similar circumstances for the nap taken as part of the study. This consistent nap related behaviour would be hypothesised to minimise any first night effects for these individuals, although for those who don't nap at work, first night effects are a possibility. In addition, the polysomnography equipment was worn for several hours prior to the napping opportunity which would have allowed individuals to become comfortable with the equipment and help reduce any disruptive effect it might have when they attempted to sleep. Nevertheless, an awareness of the equipment may contribute to a first night effect.

- Q4. Can individuals accurately assess the quantity and quality of sleep obtained during the napping opportunity?
- H4. From previous findings it is predicted that individuals will, to some extent, be able to estimate the length of sleep obtained, their sleep latencies and sleep quality.
- Q5. Do air traffic controllers ever fall asleep during the night shift outside the napping opportunity?
- H5. Given that both circadian and homeostatic processes are weighted towards sleep, and that the night shift is a time of relatively low workload, it is hypothesised that there will be occasions on which individuals fall asleep while at work.

4.2 Method

4.2.1 Measures

The details regarding the viewing, filtering, and scoring of the polysomnographic data are outlined in Chapter 2. The variables listed below are derived largely from the averaged polysomnographic data. For the sleep scoring of data outside the napping opportunity, only total sleep time in the various stages was obtained. The subjective sleep variables were recorded on the post-night shift questionnaire.

General Sleep Length and Quality Variables (Determined Via Polysomnography):

- **Analysis start time:** This represents both the beginning of the napping opportunity and the commencement of the period that was scored for sleep.
- **Total time analysed:** The length of time (minutes) that was scored polysomnographically, which was also the length of the opportunity for sleep.
- **Sleep latency:** Latency from the beginning of the nap opportunity until the first two consecutive scored epochs of sleep.

- **Sleep period:** The total time (in minutes) from the first minute of sleep until the end of the nap opportunity.
- **Wake time (during sleep period):** Amount of time (in minutes) scored as wake within the sleep period.
- **Total sleep time:** Total time (in minutes) spent in stages 1, 2, 3 and 4¹⁵.
- **Sleep efficiency (referred to sleep period):** The percentage of the sleep period that was polysomnographically scored as sleep.
- **Sleep efficiency (referred to total time for nap):** This represents the percentage of the napping opportunity that was polysomnographically scored as sleep.
- **Wake time (during total time for nap):** Total time spent awake in the napping opportunity.
- **Percentage time awake (referred to total time for nap):** Wake time expressed as a percentage of the napping opportunity.

Sleep Stage Variables (Determined Via Polysomnography):

- **Total time (NREM S1, S2, S3 or S4):** Total time (in minutes) spent in each of the 4 NREM sleep stages. This variable was not calculated if an individual had less than 1 consecutive minute of the respective sleep stage.
- **Percentage of sleep time (NREM S1, S2, S3 or S4):** Percentage of “total time analysed” spent in each respective sleep stage.
- **Percentage of sleep period (NREM S1, S2, S3 or S4):** Percentage of “sleep period” spent in each respective sleep stage.
- **Latency to first occurrence of sleep stage (NREM S1, S2, S3 or S4):** Time (in minutes) from the beginning of the nap opportunity until the first occurrence of a consecutive minute of the respective stage.

¹⁵ REM sleep was not seen in any of the polysomnographic data collected during the study, therefore all subsequent sleep variables only refer to NREM stages 1, 2, 3, and 4.

- **Latency from sleep onset to sleep stage (NREM S2, S3 or S4):** Time (in minutes) from the first 2 minutes of sleep until the first occurrence of a consecutive minute of the respective stage.
- **Number of slow wave sleep (SWS) periods:** The number of times SWS was entered within the napping opportunity.
- **Sleep stage transitions:** A count of the number of times the sleep stage changes. For example from stage 3 to stage 2, or stage 2 to wake.
- **Sleep stage transitions per hour of sleep:** An index of the number of times the sleep stage changes across an hour. Calculated by dividing the total number of sleep stage transitions by the total time scored as sleep.

Awakening and Arousal Variables (Determined Via Polysomnography):

- **Awakenings:** A count of the number of times wake was entered from sleep.
- **Awakenings per hour of sleep:** The number of awakenings represented per hour of sleep. Calculated by dividing the total number of awakenings by the total time (fraction of hour) scored as sleep.
- **Awakenings greater than 60 seconds:** A count of the number of times wake was entered from sleep for a period greater than 60 seconds.
- **Number of awakenings from stage (NREM S1 or S2):** Number of awakenings that occurred from a particular sleep stage.
- **Number of awakenings from stage (NREM S1 or S2) greater than 60 seconds:** Number of awakening that occurred from a particular sleep stage that were longer than 60 seconds.
- **Number of ASDA arousals:** A count of the number of times an ASDA arousal was scored.
- **ASDA arousal index:** Number of ASDA arousals per hour of sleep. Calculated by dividing the total number of arousals by the total time scored as sleep.

- **Stage woken from:** Stage of sleep that an individual was woken from at the end of the napping opportunity.

Subjective Sleep Variables:

- **Subjective sleep length:** Reported amount of sleep obtained during the night shift (in minutes).
- **Difficulty falling asleep:** Subjective rating of the degree of difficulty experienced when trying to fall asleep during the napping opportunity (0 = “not at all difficult”, 10 = “very difficult”).
- **Quality of sleep:** Subjective rating of the quality of sleep obtained during the napping opportunity (0 = “very poor”, 10 = “very good”).
- **Waking feeling refreshed:** Subjective rating of how refreshed the participant felt on waking from sleep obtained during the napping opportunity (0 = “not at all” (refreshed), 10 = “completely” (refreshed)).
- **Depth of sleep:** Subjective rating of the depth of sleep obtained during the napping opportunity, (0 = “light”, 10 = “deep”).
- **Degree of help in managing fatigue during remainder of night:** Subjective rating of how helpful any sleep obtained during the napping opportunity was in helping to manage fatigue across the remainder of the night shift (0 = “very unhelpful”, 10 = “very helpful”).

4.2.2 *Statistical Analyses*

Descriptive statistics have been presented in this chapter where a summary view of the overall sample, or sub-portion of the sample, is useful. Largely the mean, standard deviation and range are utilised, however, where variables are not normally distributed the median, standard error of the median, and range are presented instead.

Mixed model ANOVAs, ANCOVAs or logistic regressions were employed to investigate the predictive relationship between certain factors and the polysomnographically or subjectively generated dependent variables.

In several instances dependent variables were dichotomised due to severely skewed distributions and therefore mixed model logistic regressions were calculated rather than an ANOVA or ANCOVA. Apart from the binomial dependent variable, the model structure was equivalent to that utilised in all other instances.

Circadian Influences and Homeostatic Sleep Drive

In addition to the independent variable of night shift type, the start time of the napping opportunity, level of acute sleep loss in the 24 hours prior to commencement of the nap, and the length of time since the last sleep was obtained, were considered for inclusion in each model. The latter two variables were deemed representative of the homeostatic drive for sleep. Both acute sleep loss and prior time awake were calculated backwards from the commencement time of the napping opportunity. The amount of acute assumed sleep loss in the 24 hours prior to the nap was calculated in an identical manner, using actigraphy and logbook data, to the calculations performed in the acute sleep debt analyses as detailed in Chapter 3, section 3.6.4. Acute assumed sleep debt was calculated rather than TIB or actual sleep loss, given previous findings which indicated that these three sleep parameters do not differ very much. More data is normally available for assumed sleep, because when only logbook data are available actual sleep parameters can not be calculated, resulting in fewer values for analyses. The distribution of this variable can be seen in Figure 4-1.

The length of time since the last sleep was calculated using the sleep end time available in the actigraphy database. Generally the length of time since the last sleep was the difference between the wake up time of a short sleep taken prior to the night shift and the start of the analysed period of the napping opportunity. However, if a pre-night shift nap was not taken, then this variable was the difference between the wake up time of the sleep of the night before and the beginning of the napping opportunity. The resulting distribution can be seen in Figure 4-2.

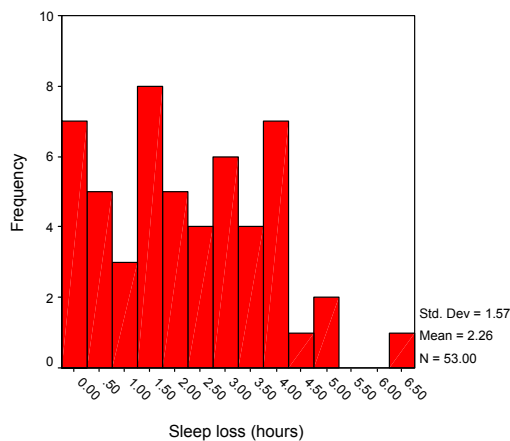


Figure 4-1: Level of Acute Assumed Sleep Loss in the 24 Hours Prior to the Napping Opportunity

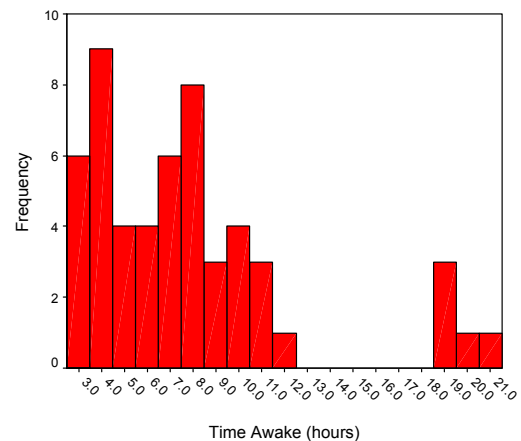


Figure 4-2: Length of Time Awake at the Commencement of the Napping Opportunity

First Night and Order Effects

Because of previous findings that the first night of sleep in a laboratory based situation is characterised by more wake, more stage 1, longer latencies to stage 3, and more sleep stage transitions (Agnew, Webb & Williams, 1966), the night number on which the napping conditions occurred was included as a fixed factor in all models in this chapter. Therefore, a napping opportunity was labelled as occurring on either the first, second, third or fourth study night.

In a within-subjects design, a potential confound is introduced through the arrangement of the study conditions (Millar, 1992). It is possible, although unlikely in the present study, that carry over effects from the initial study conditions have an influence on the findings in later study conditions. For example, greater familiarity with the polysomnography equipment and study protocol could lead to study participants obtaining more sleep in the later study nights. Counterbalancing the order in which study conditions were completed, goes some way to minimising this issue. In the present study true counterbalancing did not occur, as participants completed study conditions as they occurred within the roster. However, even a counterbalanced study design does not stop potential carry over effects from occurring. To control for potential carry-over effects, in all mixed model analyses in this chapter, a variable representing the order in which an individual completed the four study conditions was entered as a fixed factor.

Collinearity

Prior to entry into the mixed models, possible relationships between independent variables were assessed to ensure redundant variables were not included, thereby avoiding issues of multi-collinearity (Tabachnick & Fidell, 1996; Kleinbaum, Kupper, Muller & Nizam, 1998). A small eigenvalue (.005), large conditioning index (39), and variance proportions greater than 0.5, were associated with the variables of shift type and the start time of the napping opportunity. It was decided to remove the continuous variable representing the start time of the napping opportunity rather than the type of shift worked. This was for two reasons; first the start time of the napping opportunity was not a true continuous variable, with the values being bimodally distributed rather than a range of values along a continuum. Second, the start time of the napping opportunity was completely defined by the type of night shift worked (K1 or K2). However, in one instance an individual on a K2 night shift took a nap at a time that fell within the timing of naps taken on the earlier K1 night shift (see Figure 4-3 for more detail). To ensure that this nap did not alter possible findings related to the timing of the napping opportunity, all models were run with and without this case. On no occasion did the removal of this case from the analyses alter the findings. Therefore the findings reported are based on data which include this case.

Dependent Variables

In some instances there were dependent variables that were logically expected to have a linear relationship with the total amount of sleep obtained in the napping opportunity. An example of this is the number of sleep stage transitions, where the opportunity to transition from one stage to another increases with increasing time asleep. In the analyses of such variables an additional fixed factor was included that controlled for the amount of sleep that had been obtained. Where this was done, it is reported along with the findings in the results section.

In the large majority of mixed models presented in this chapter, the independent variables utilised were; sequence (the place in the sequence in which a particular experimental condition was completed), order (the arrangement of the four study conditions for an individual), the night shift type worked, the length of time since the last sleep was obtained, and the level of acute assumed sleep loss. Specific details of the structure of each model are discussed in the respective sections below.

4.3 Results: Timing and Length of the Napping Opportunity

Some variability in the timing of the nap was necessarily introduced due to the operational demands upon the study participants. An extreme example of an outlying start time can be seen in Figure 4-3 with the much earlier K2 nap, which was due to an anomalous combination of operational and staffing demands.

Of the 27 napping opportunities recorded on the K1 night shift, the mean time of commencement was 0023 hours ($SD = 00:13$, $Range = 0003-0053$ hours); while for the K2 shift the mean start time was 0222 hours (0218 hours with the outlying value included), $SD = 00:13$, $Range = 0203-0256$ hours, $N = 26$).

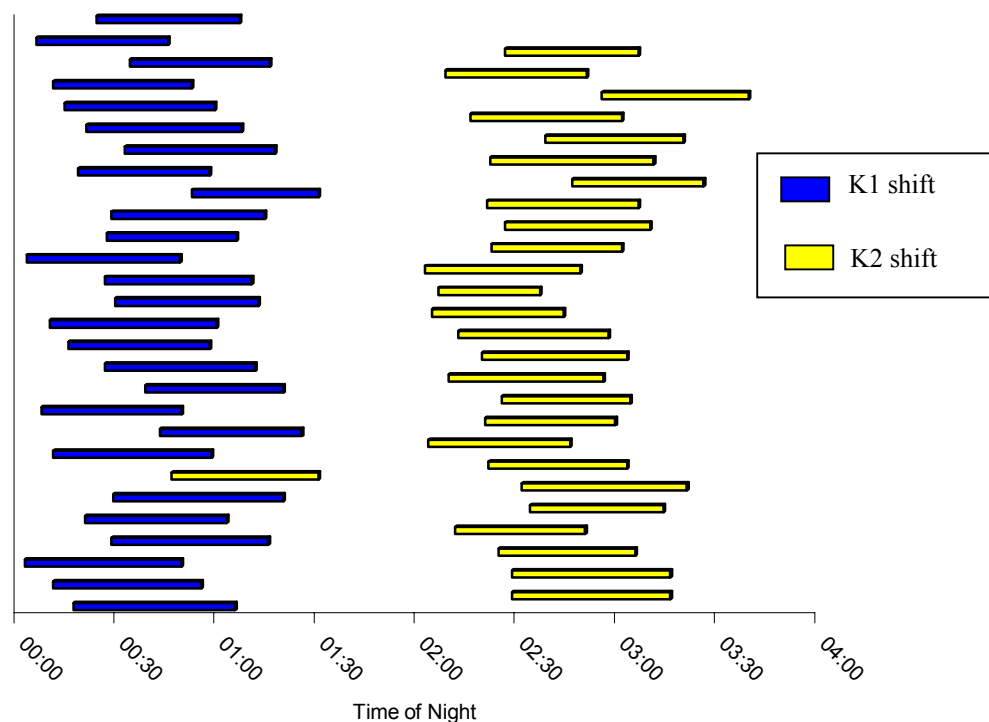


Figure 4-3: Timing and Length of the Napping Opportunity on the K1 and K2 Shift

The mean length of the periods analysed, and consequentially the amount of time individuals spent attempting to nap, was 42.99 minutes ($SD = 4.25$, $Range = 28-51$ $N = 54$). From exploratory mixed model analyses, the length of the napping opportunity provided did not differ given the timing of the nap.

4.4 Question 1: Polysomnographic Sleep During the Napping Opportunity

4.4.1 *Statistical Analyses*

The dependent variables in this section include measures related to general sleep length, sleep stage, and awakenings and arousals and were chosen for analyses in order to establish an overview of the sleep that occurred during the napping opportunity. Basic descriptive statistics are presented in this section to provide the necessary detail.

4.4.2 *Results: Sleep During the Napping Opportunity*

Sleep period was the length of time from the first minute of sleep through to the end of the napping opportunity. The mean value of this variable was determined to be 20.73 minutes ($SD = 12.68$, $Range = 0-47.50$, $N = 54$) and within this time frame a median of 1.50 minutes was wake time ($Range = 0-43.50$, $N = 54$). These values produced a mean sleep efficiency (referred to sleep period) of 90.98% ($SD = 33.78$, $Range = 0-100$, $N = 54$).

As shown in Figure 4-4 the amount of total sleep obtained by participants varied widely, from those getting no sleep through to one individual obtaining 47 minutes of sleep. Figure 4-4 displays the frequency of total sleep, with values grouped in 5 minute bins. The mean total sleep time (TST) was 17.70 minutes ($SD = 12.16$, $N = 54$). This produces to a mean sleep efficiency (referred to the total time allowed for the nap) of 41.01% ($SD = 27.31$, $Range = 0-94.90$, $N = 54$).

The five occasions on which participants obtained no sleep at all were from five separate individuals. Of these five, three obtained less than a minute sleep on the other napping occasion, while the other two had greater amounts of sleep (6 minutes and 16.75 minutes respectively).

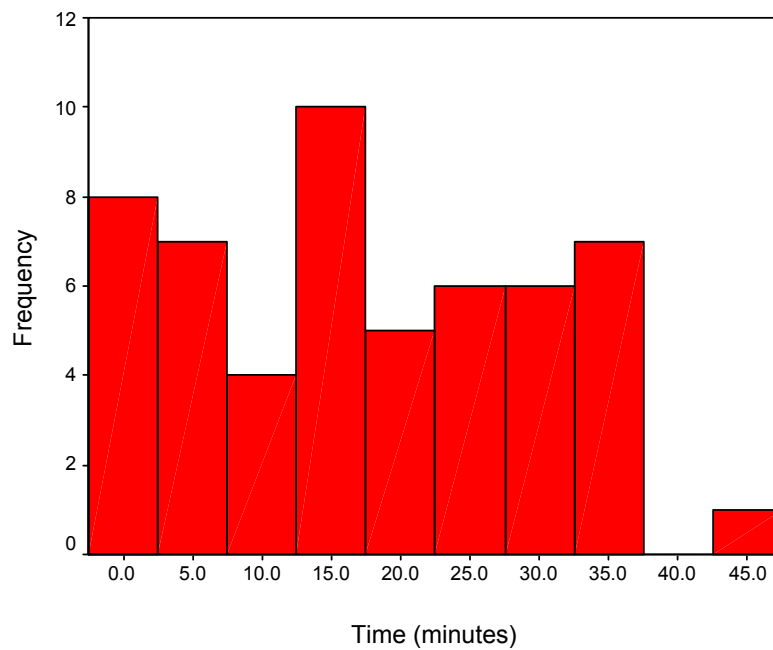


Figure 4-4: Distribution of Total Scored Sleep Time

Of those who fell asleep, the mean latency to the first minute of sleep was 19.40 minutes ($SD = 10.54$, $Range\ 2-46.75^{16}$, $N = 48$). Across the entire napping opportunity, participants were awake for a mean of 25.28 minutes ($SD = 12.15$, $Range = 2-50$, $N = 54$). This equates to a mean percentage time awake of 58.96 % ($SD = 27.31$, $Range = 5.10-100$, $N = 54$), the distribution of which can be seen in Figure 4-5. Again values in this plot are grouped in bins, with each bin representing 5%.

¹⁶ This high value relates to a single occasion when a study participant had a napping opportunity of 51 minutes.

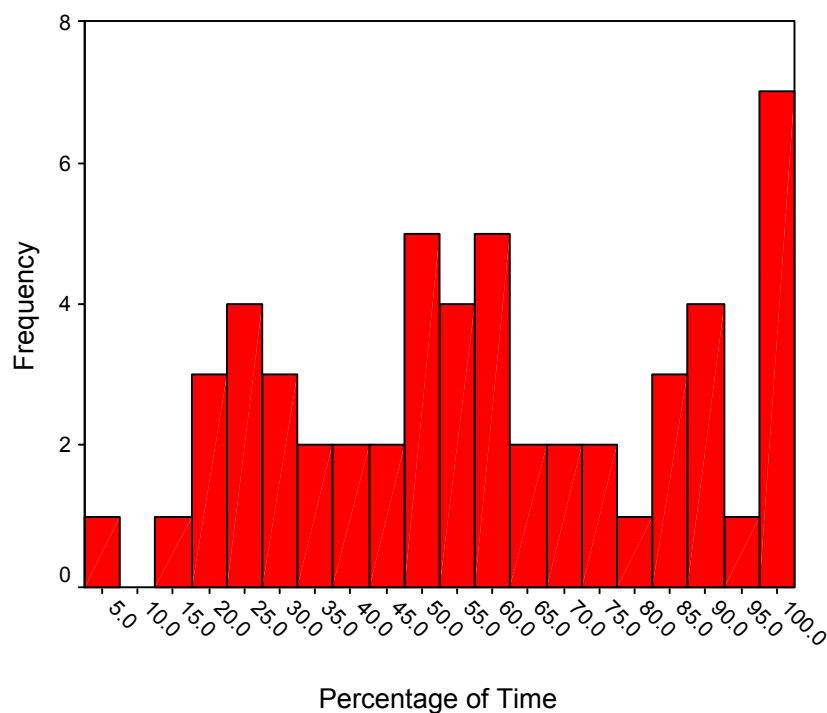


Figure 4-5: Distribution of Percentage of Napping Opportunity Scored as Wake



4.4.3 Results: Sleep Stage Information

In this section the amount of time spent in each sleep stage (in minutes) was investigated, along with the percentage of the total sleep this represented. Total time in a stage was considered because of possible dose-dependent relationships the different stages may have with performance and alertness outcomes, while the percentage of TST that a stage represented was examined to determine if architectural changes in the nap sleep occurred as a consequence of the different levels of the independent variables of interest.

Table 4-1: Mean (or Median), Standard Deviation, and Range for Sleep Length and Efficiency Variables on Each Shift Type for Stage 1 and 2 Sleep (Values in Brackets are for the Sub-Group of Individuals who Entered that Sleep Stage)

	Stage 1								Stage 2							
	K1 Shift				K2 Shift				K1 Shift				K2 Shift			
	<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>
Total time (minutes)	4.24	2.65	0.25-10.0	24	5.66	3.09	0.50-12.25	25	12.79 (15.58)	9.15 (7.67)	0-30.50 (3.25-30.50)	24 (20)	12.60 (13.70)	7.92 (7.26)	0-24 (1.75-24)	25 (23)
Percentage of total sleep time	24.89*		4.50 -100	24	29.35*		3.63-100.00	25	70.38* (74.08)*		0-95.50 (28.91-95.50)	24 (20)	62.50* (64.89)*		0-96.37 (14.40-96.37)	25 (23)
Percentage of napping opportunity	8.67*		0.56-24.10	24	14.74*		1.14-26.92	25	31.70* (37.64)*		0-64.89 (7.45-64.89)	24 (20)	31.15* (33.52)*		0-58.33 (4.12-58.33)	25 (23)
Latency to first occurrence of stage (minutes)	22.79	11.21	6.00-46.75	20	16.19	9.79	2.00-37.25	21	24.58	9.29	10-43	20	23.10	9.78	5.5-42.50	23
Latency to stage from sleep onset (minutes)	-	-	-	-	-	-	-	-	4.00*		0.50-9.50	19	5.00*	5.23	0.75-23.50	23

* Variables were not normally distributed; therefore medians are presented in place of the mean.

Table 4-2: Median and Range for Sleep Length and Efficiency Variables on Each Shift Type for Stage 3 Sleep (Values in Brackets are for the Sub-Group of Individuals who Entered that Sleep Stage)

Stage 3						
	K1 Shift			K2 Shift		
	<i>Median</i>	<i>Range</i>	<i>N</i>	<i>Median</i>	<i>Range</i>	<i>N</i>
Total time (minutes)	0 (2.75)	0-5.50 (0.50-5.50)	24 (9)	0 (1.50)	0-23.00 (0.25-23.00)	25 (11)
Percentage of total sleep time	0 (7.43)	0-16.42 (2.33-16.42)	24 (9)	0 (4.51)	0-48.94 (1.06-48.94)	25 (11)
Percentage of napping opportunity	0 (5.79)	0-13.29 (1.08-13.29)	24 (9)	0 (3.61)	0-46.46 (0.55-46.46)	25 (11)
Latency to first occurrence of stage (minutes)	42.50	18.50-46.50	7	31.50	21.00-45.75	3
Latency to stage from sleep onset (minutes)	31.00	12.00-33.50	7	21.25	19.00-21.50	3

In Table 4-1 and Table 4-2 the values for the two shift types are presented separately. Of those who had some sleep during the napping opportunity, a mean of 4.96 minutes ($SD = 2.94$, $Range = 0.25-12.25$, $N = 49$) was scored as stage 1 sleep, which was a median of 27.46% ($Range = 3.63-100$, $N = 49$) of the sleep obtained during the nap, or alternatively 9.44% of the napping opportunity ($Range = 0.56-26.92$, $N = 49$). Nearly 3 times as much stage 2 sleep was scored, with the mean being 12.79 minutes ($SD = 8.46$, $Range = 0-30.50$, $N = 49$). This in turn represents a median of 66.14% ($Range = 0-96.37$, $N = 49$) of the nap sleep and 31.16% of the entire nap opportunity ($Range = 0-64.89$, $N = 49$). When considering only individuals who entered stage 2 sleep, rather than all those that slept during the nap, then the mean amount of stage 2 obtained was 14.57 minutes ($SD = 7.43$, $Range = 1.75-30.50$, $N = 43$). This was a median of 67.61% of the sleep scored during the nap ($Range = 14.40-96.37$, $N = 43$) and 33.79% of the time spent attempting to sleep ($Range = 4.12-64.89$, $N = 43$).

For stage 3 sleep, a median of 0 minutes sleep ($Range = 0-23$, $N = 49$) was obtained when considering all those who initiated sleep during the napping opportunity. For those who entered stage 3, a median of 1.50 minutes was obtained ($Range = 0.25-23$, $N = 20$), which

was a median of 5.47% (*Range* = 1.06-48.94, *N* = 20) of the total sleep scored during the nap and 3.73% of the entire napping opportunity (*Range* = 0.55-46.46, *N* = 20).

Figure 4-6 and Figure 4-7 display example sleep histograms. Figure 4-6 shows the sleep structure typically found in the napping opportunity. Such sleep starts approximately half way through the napping opportunity and contains predominantly stage 2 sleep, with short periods of stage 1 following brief awakenings. Figure 4-7 represents a “longer sleep”, characterised by the presence of some stage 3 sleep towards the end of the napping opportunity.

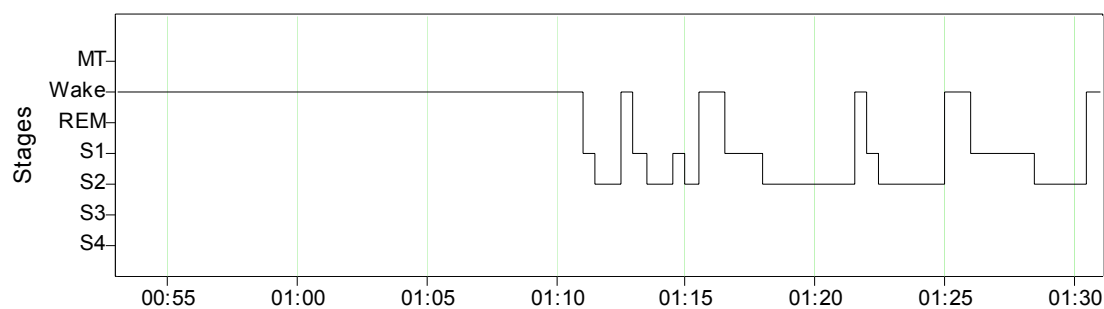


Figure 4-6: Sample Sleep Histogram Showing Predominantly Stages 1 and 2 Sleep

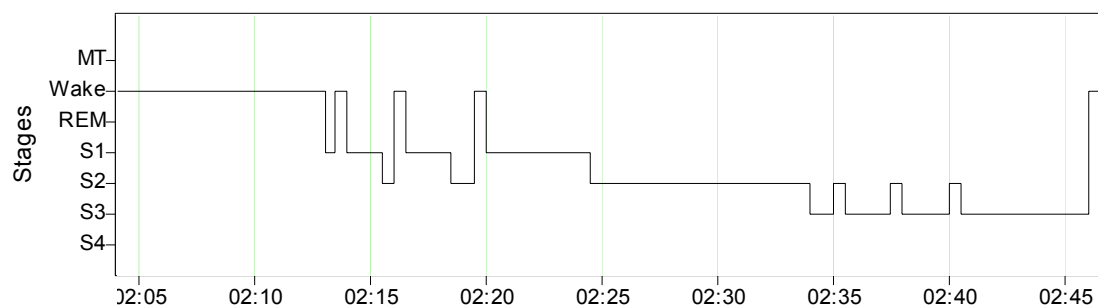


Figure 4-7: Sample Sleep Histogram Showing Stage 1, 2, and 3 Sleep

It was not possible to calculate descriptive statistics for stage 4 sleep variables, as only two people on four separate occasions (once each on both night shifts) were scored as entering stage 4 sleep. On three of these occasions S4 was entered for a minute or less. For these three occasions the stage 4 sleep comprised approximately 3% of the total sleep time and 2% of the napping opportunity. On the fourth occasion that an individual entered stage 4 sleep, it was for considerably longer (13.50 minutes), and

accounted for 42% of the total sleep time and 34% of the napping opportunity. This particular instance occurred on a K2 night shift and was subsequent to an extended period of prior wake (18.6 hours) and with a relatively high acute sleep debt (3.8 hours). One of the other occurrences of stage 4 sleep took place after a prolonged period of wakefulness (21 hours), and another with a high acute sleep debt (5.1 hours). The fourth occasion on which stage 4 sleep was entered was not extreme with regard to either prior wake time or acute sleep debt.

Latency from the beginning of the nap opportunity to the first 60 seconds of stage 1 sleep was 19.41 minutes ($SD = 10.90$, $Range = 2-46.75$, $N = 41$), while to stage 2 sleep the mean latency was 23.78 minutes ($SD = 9.47$, $Range = 5.50-43.00$, $N = 43$) and to stage 3, 36.83 minutes ($SD = 10.06$, $Range = 18.50-46.50$, $N = 10$). From sleep onset the median latency to stage 2 was 4.00 minutes ($Range = 0.50-23.50$, $N = 42$) and a mean of 25.08 minutes to the first occurrence of stage 3 sleep ($SD = 7.37$, $Range = 12.00-33.50$, $N = 10$).

On all occasions individuals entered sleep through stage 1, therefore only the latencies to stages 2 and 3 from sleep onset are detailed in Table 4-1 and Table 4-2. It should be noted, that for these latencies as well as for the latencies to the first occurrence of each sleep stage, the values are calculated to the first **minute** of each particular stage. In addition, in these calculations sleep onset was considered to be the first two minutes of unbroken sleep, which influences the determination of the latencies from sleep onset to stage 2 and 3 sleep. If an individual had less than two minutes consecutive sleep and/or less than one minute in a sleep stage, these particular variables could not be calculated. This explains the lower number of individuals included in these calculations. In addition, if a 30 second period of a particular stage occurred in isolation, then this short period was not utilised in the calculation of the latencies. Requiring three consecutive epochs of sleep for determining latencies is standard practice (Carskadon, 1989). Because, in this instance the sleep is of short duration, and under less than ideal conditions, it was considered a practical compromise to require at least one minute, or two consecutive epochs, of a stage in order to calculate latencies.

In Table 4-2 the median latencies to S3 on each shift type are presented, and Figure 4-8 displays the distribution of latencies for those who reached stage 3 for at least one consecutive minute or longer.

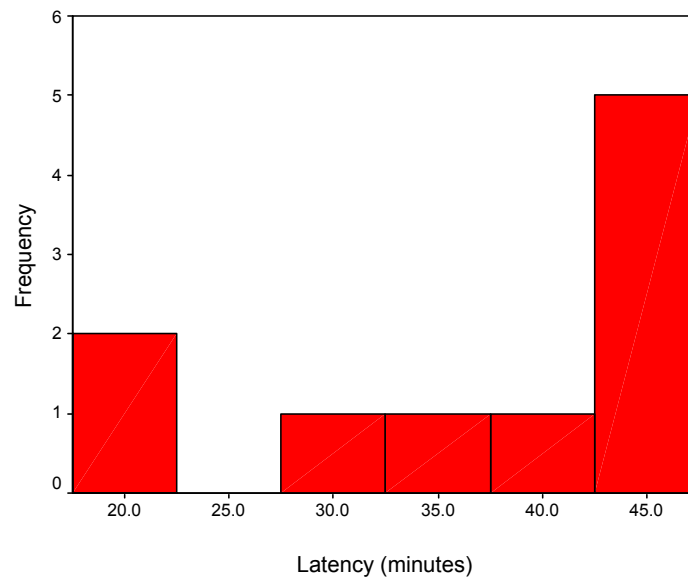


Figure 4-8: Latency to the First Minute of Stage 3 Sleep From the Commencement of the Napping Opportunity

The median number of sleep stage transitions during the sleep period was 8.50 (*Range* = 1-27.50) and across the period of an hour this represents a median stage transition rate of 25.45 (*Range* = 2.05-70.00).

4.4.4 Results: Awakenings and Arousals

For those who fell asleep, the median number of awakenings was 2.00 (*Range* = 1-5.50¹⁷, $N = 49$), which equates to a median value of 5.90 awakenings per hour (*Range* = 0-66.82, $N = 49$). In addition, of those who fell asleep, 25 individuals experienced awakenings greater than 1 minute in length (52%).

Table 4-3 displays the median number of awakenings per hour of sleep that occurred from stage 1 and 2, as well as the median number of awakenings per hour greater than 60 seconds from both these sleep stages.

¹⁷ Because the final values of sleep variables were averaged across the results from the two individuals who sleep scored the data, it was possible to get fractional values.

Table 4-3: Mean, Standard Error, and Range of the Number of Awakenings from Stage 1 and 2 Sleep Per Hour

Stage	<i>Median</i>	<i>Range</i>	<i>N</i>
Stage 1	.02	0-2.00	49
Stage 1 greater than 60 secs awake	0	0-1.00	49
Stage 2	.06	0-.26	43
Stage 2 greater than 60 secs awake	0	0-0.15	43

ASDA arousals were scored and the mean number recorded was 5.0 ($SD = 2.84$, $Range = 0-12.50$, $N = 49$), the distribution of which can be seen in Figure 4-9. Across the period of an hour the median was 15.33 ($Range = 0-96.00$).

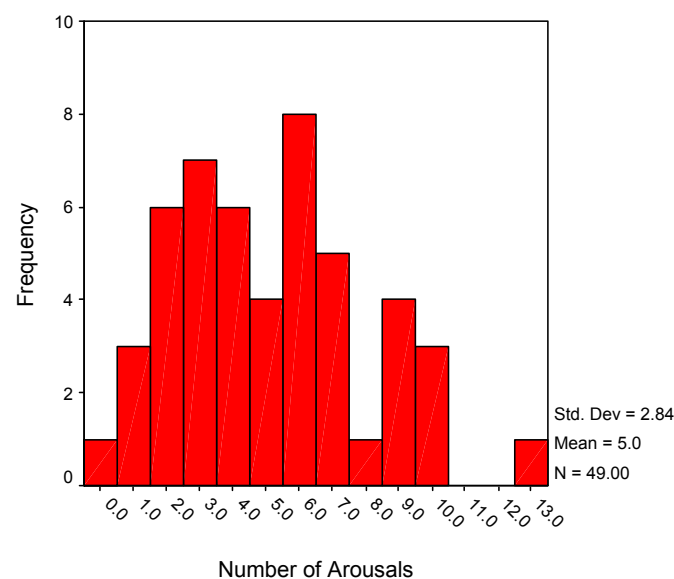
**Figure 4-9: Distribution of the Number of ASDA Scored Arousals**

Figure 4-10 displays the stage of sleep an individual was woken from, and alternatively if they were already awake at the end of the napping opportunity or had not slept at all. It can be seen that individuals were mostly woken from stage 2 sleep. On 20 occasions participants were scored as entering stage 3 and possibly also stage 4 sleep, although in only nine instances did they wake from either of these SWS stages. Six of these occurred at the end of an early nap and the remaining three were on waking from the later nap.

Furthermore, in seven instances individuals woke spontaneously before the end of the napping opportunity.

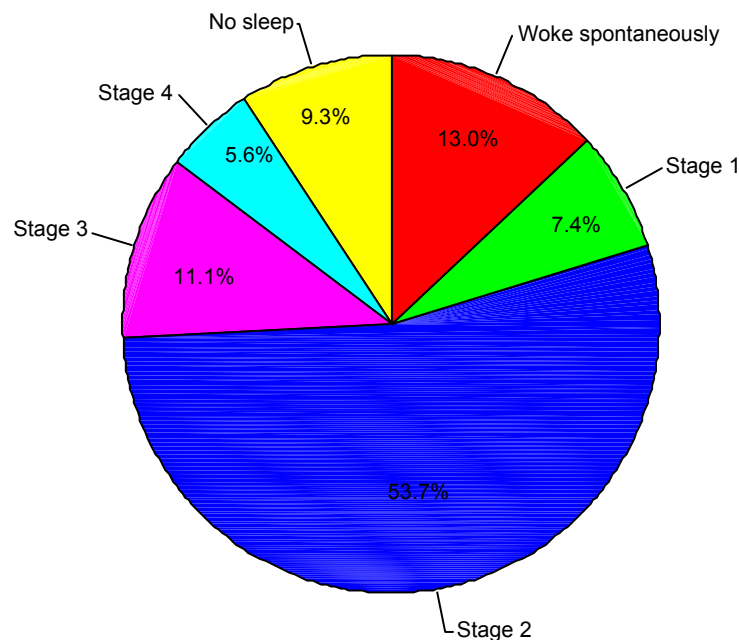


Figure 4-10: Stage of Sleep at the End of the Napping Opportunity

4.5 Question 2: Length, Depth and Quality of Sleep Depending on Acute Sleep Loss, Prior Wake, and the Timing of the Nap

Question 3: First Night Effects

4.5.1 Statistical Analyses

Mixed model ANCOVAs or logistic regressions analyses were performed to determine if there were differences for each of the respective sleep related variables due to a variety of independent variables detailed in Table 4-4. In all mixed model ANCOVAs a compound symmetric covariance structure was theoretically most suitable, as well as providing the best fit between repeated measures.

The mixed model undertaken for stage 3 sleep (minutes) differed from that for stages 1 and 2 sleep. This was due to the fact that on only 20 occasions individuals reached stage 3 sleep, and in most cases the amount of stage 3 sleep obtained was not great, as can be seen from data in Table 4-2. This produced a distribution that was severely positively

skewed due to the large number of zero values. In this instance it was considered more appropriate to dichotomise the variable with the two levels representing no stage 3 sleep and some stage 3 sleep respectively. A mixed model logistic regression was then run using almost identical independent variables included in the models for stage 1 and 2 sleep. The changes included the removal of the variable representing the order in which the four experimental conditions were completed, as the model had difficulties running. This was thought to be due to the sparse amount of data associated with each level of order. In addition, a variable representing the total amount of sleep obtained was included because entry into stage 3 sleep is dependent on sleep being maintained for a certain period of time.

Models incorporating the dependent variables of the percentage of the total sleep time stage 3 sleep represented, and the percentage of the sleep period this stage comprised were not run. Mixed model ANCOVAs were again not appropriate due to the distribution of this data. Nor was dichotomisation of these variables appropriate in order to allow mixed model logistic regressions to run, as these would produce identical results to that for the number of minutes in stage 3 sleep. Further, for the variables latency to stage 3 sleep from the commencement of the napping opportunity, and latency to stage 3 sleep from sleep onset, mixed models could not be run due to the small amount of data available ($N = 10$). It was also not possible to calculate mixed models with the stage 4 sleep variables, due to the very small amount of data.

In the model for absolute number of sleep stage transitions, and the large majority of variables associated with awakenings and arousals, an additional fixed factor was included that controlled for the amount of sleep that had been obtained. This was necessary as the opportunity for such events would be expected to, and was found to, increase in a linear manner with increasing total sleep time.

Due to a severely skewed distribution, with a large number of zero values, the number of awakenings greater than 1 minute in length was dichotomised into a binomial variable with the two levels representing zero and greater than zero awakenings greater than 1 minute. In this case, a mixed model logistic regression was run rather than an ANCOVA.

The same principle was applied to the number of awakenings from stage 1 and 2 sleep greater than one minute. Both variables were dichotomised due to the large number of

zero values creating severely skewed distributions and consequently mixed model logistic regressions were run.

Table 4-4: Dependent and Independent Variables for Analyses Related to the Length, Depth and Quality of Sleep Depending on Acute Sleep Loss, Prior Wake, and the Timing of the Nap

Dependent Variables	Independent Variables ¹⁸
<i>General sleep length variables</i>	
Sleep period	
Wake time (during sleep period)	
Sleep efficiency (referred to sleep period)	Sequence
Total sleep time	Order
Sleep efficiency (referred to total time for nap)	Shift type
Sleep latency	Prior wake
Wake time (during total time for nap)	Acute debt
Percentage time awake (referred to total time for nap)	
<i>Sleep stage variables</i>	
Total time (S1, S2)	
Percentage of sleep time (S1, S2)	Sequence
Percentage of sleep period (S1, S2)	Order
Latency to first occurrence of stage (S1, S2,)	Shift type
Latency from sleep onset to sleep stage (S2)	Prior wake
Sleep stage transitions	Acute debt
Sleep stage transitions per hour of sleep	
Occurrence of S3 (entered or not)	Sequence
	Shift type
	Prior wake
	Acute debt
	Total sleep time

¹⁸ Sequence = place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

Order = the arrangement of the four study conditions for an individual (1 to 18)

Shift type = night shift worked (K1 or K2)

Prior wake = length of time awake prior to start of napping opportunity

Acute debt = acute sleep loss in the 24-hours prior to the start of the napping opportunity

Total sleep = amount of polysomnographically calculated sleep scored during the napping opportunity

Dependent Variables	Independent Variables ¹⁸
Sleep stage transitions	Sequence Order Shift type Prior wake Acute debt Total sleep time
<i>Awakening and arousal variables</i>	
Awakenings	Sequence
Awakenings greater than 60 seconds	Order
Number of awakenings from stage (S1 or S2)	Shift type
Number of awakenings from stage (S1 or S2) > 60s	Prior wake
Number of ASDA arousals	Acute debt
Stage woken from	Total sleep time
Awakenings per hour of sleep	Sequence
ASDA arousal index	Order Shift type Prior wake Acute debt

4.5.2 Results: Sleep During the Napping Opportunity

In the mixed model ANCOVAs including the length of the sleep period and sleep efficiency (referred to sleep period) as dependent variables, no significant findings were seen. However, the model with wake time (during sleep period) as the dependent variable produced a significant effect according to the amount of acute sleep loss preceding the napping opportunity (see Table 4-5 for details and results). The slope of the regression estimate reflects the expected direction of the relationship, with greater levels of acute sleep loss resulting in a decrease in the percentage of wake time during the sleep period.

In further mixed models, the length of time awake prior to the napping opportunity just failed to reach significance for the dependent variable of total sleep time. However, it was a significant factor for sleep efficiency (referred to total time for nap), with the estimates from the model indicating that longer time frames since the last sleep produced more efficient sleep.

The results of the mixed model investigating the relationship between the independent sleep loss and timing variables and sleep latency suggest that the length of time awake prior to the napping opportunity decreases sleep latency. Further, prior wakefulness was a significant effect in the models for wake time (during total time for nap) and the percentage time awake (referred to total time for nap). The estimates of the regression lines indicate that both the amount of time awake in the napping opportunity and the percentage of time awake decreased with increasing prior wakefulness.

Table 4-5: Details and Results of Mixed Model ANCOVAs for Polysomnographic Sleep Parameters

Dependent Variable	Fixed and Continuous Effects in Mixed Model	Significant Main Effects
Length of napping opportunity	sequence, order, shift type, prior wake, acute debt ¹⁹	
Sleep period	As above	
Wake time (during sleep period)	As above	Acute debt $F_{(1, 21.8)} = 5.88, p = .024$
Sleep efficiency (referred to sleep period)	As above	
Total sleep	As above	
Sleep efficiency (referred to total time for nap)	As above	Prior wake $F_{(1, 18.2)} = 7.75, p = .012$
Sleep latency	As above	Prior wake $F_{(1, 17.7)} = 11.03, p = .004$
Wake time (during total time for nap)	As above	Prior wake $F_{(1, 27.7)} = 4.51, p = .043$
Percentage time awake (referred to total time for nap)	As above	Prior wake $F_{(1, 26)} = 5.66, p = .025$

¹⁹ Sequence = place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

Order = the arrangement of the four study conditions for an individual (1 to 18)

Shift type = night shift worked (K1 or K2)

Prior wake = length of time awake prior to start of napping opportunity

Acute debt = acute sleep loss in the 24 hours prior to the start of the napping opportunity

Although there were no statistically significant differences between the total sleep obtained, time awake, and sleep latency according to the shift worked there was a tendency for the earlier sleep (on the K1 night shift) to be shorter, have a longer latency and greater time awake values than the later sleep. These patterns can be seen in Table 4-6, which presents the mean values, standard deviation and range on each night shift type.

Table 4-6: Mean, Standard Deviation, and Range for Total Sleep Length, Sleep Efficiency, Total Wake Time, and Sleep Latency on Each Night Shift Type

	Early Napping Opportunity (K1 Shift)				Late Napping Opportunity (K2 Shift)			
	<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>
Total sleep time (minutes)	16.73	12.28	0-37	27	18.69	12.19	0-47	27
Sleep efficiency (referred to total time for the nap)	44.32	23.90	2.60-81	24	47.85	25.41	1.15-94.90	25
Total time awake (minutes)	27.40	12.18	7.50-50	27	23.17	11.96	2.50-47.50	27
Sleep latency (minutes)	21.75	10.88	6-46.75	23	17.76	10.17	2-37.25	25

4.5.3 Results: Sleep Stage Information

As with the variables in section 4.5.2 above, mixed models were calculated to determine if differences in the various stages of NREM sleep were created due to the effects of sequence, order, shift type worked, the length of time awake, and the level of acute sleep loss at the beginning of the napping opportunity. In all instances variables did not differ significantly according to the shift type worked. However, the values for each shift type were previously presented separately in Table 4-1 and Table 4-2 (pg. 180 and 181) in order for the areas of interest in the data to be visible. The mixed model analyses that produced significant effects are detailed below in Table 4-7.

Table 4-7: Details and Results of Mixed Model ANCOVAs for Sleep Stage Related Polysomnographic Parameters

Dependent Variable	Fixed and Continuous Effects in Mixed Model	Significant Main Effects
Occurrence of S3 (stage entered or not)	Sequence, shift type, prior wake, acute debt, total sleep ²⁰	Shift $F_{(1, 37.5)} = 7.35, p = .010$ Acute debt $F_{(1, 26.3)} = 6.64, p = .016$ Total sleep $F_{(136.8)} = 13.74, p < .001$
Latency to first 60 seconds of S1	Sequence, order, shift type, prior wake, acute debt	Prior wake $F_{(1, 9.27)} = 31.95, p < .001$
Latency to first 60 seconds of S2	As above	Prior wake $F_{(1, 16.5)} = 8.56, p = .010$
Number of sleep stage transitions	Sequence, order, shift type, prior wake, acute debt, total sleep	Total sleep $F_{(1, 13)} = 19.74, p < .001$

²⁰ Sequence = place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

Order = the arrangement of the four study conditions for an individual (1 to 18)

Shift type = night shift worked (K1 or K2)

Prior wake = length of time awake prior to start of napping opportunity

Acute debt = acute sleep loss in the 24 hours prior to the start of the napping opportunity

Total sleep = amount of polysomnographically calculated sleep scored during the napping opportunity

The shift type worked, amount of acute sleep loss and total sleep obtained were significant factors in the model with S3 sleep as the dependent variable. Regression estimates from the models indicate that a nap taken later in the night (on the K2 night shift), and after greater acute sleep loss, will more likely result in entry to stage 3 sleep. Not surprisingly longer periods of sleep were also related to the increased incidence of stage 3 sleep.

The results from the mixed model for the latency to the first 60 consecutive seconds of stage 1 sleep showed a significant effect for the amount of time awake prior to the nap. The factor “prior time awake” was also identified as significant in the model with latency to the first occurrence of stage 2 sleep as the dependent variable. In both these models the estimates of the regression slope suggested that longer periods awake prior to the napping opportunity result in shorter latencies to both the first occurrence of stage 1 and stage 2 sleep.

In the first model for absolute number of sleep stage transitions, an additional fixed factor was included that controlled for the amount of sleep that had been obtained. This was necessary as the opportunity for such events would be expected to, and was found to, increase in a linear manner with increasing total sleep time. Only the effect of total sleep time during the nap reached significance, and the estimate of the slope of the regression line indicated that as total sleep time increased so too did the number of sleep stage transitions.

4.5.4 *Results: Awakenings and Arousals*

For the ANCOVA that modelled factors associated with the number awakenings per hour, the sequence in which a particular experimental condition was completed was significant. Examination of the post hoc t tests point towards a napping opportunity occurring on the first study night of the four as having significantly more awakenings per hour than if the napping opportunity occurred on the fourth and final study night ($t_{(15,2)} = -3.28, p = .005$).

As can be seen from Table 4-8 the amount of acute sleep loss experienced influenced the number of awakenings greater than 1 minute. However, the slope of the regression line was not in the expected direction, as it indicated that greater acute sleep loss was more likely to produce awakenings of 1 minute or greater.

In the mixed model ANCOVA for the number of ASDA arousals the variable representing the length of the sleep period was found to be significant. Not surprisingly the estimated slope of the regression line indicated an increasing number of arousals with increased total sleep time. For the mixed model for number of ASDA arousals per hour the effect of sequence of study nights was significant, and in analysing the post hoc t tests, study night 1 was found to have more awakenings per hour than study night 3 ($t_{(20.1)} = 2.99, p = .007$), and 4 ($t_{(21.8)} = 2.84, p = .010$).

The final mixed model logistic regression run in this section was to determine if waking from SWS or not differed according to the various independent variables. As can be seen in Table 4-8 the shift type worked, level of acute sleep debt, and total sleep length were all significant factors in this model. The regression line estimates suggest that later and longer sleeps resulted in individuals waking from deeper sleep stages, as do greater levels of acute sleep loss.

Table 4-8: Details and Results of Mixed Model ANCOVAs for Awakenings and Arousals

Dependent Variable	Fixed and Continuous Effects in Mixed Model	Significant Main Effects
Number of awakenings per hour	sequence, order, shift type, prior wake, acute debt ²¹	Sequence $F_{(3,15)} = 4.11, p = .026$
Number of awakenings greater than 1 minute	As above plus total sleep	Acute debt $F_{(1,15)} = 5.19, p = .038$
Number of ASDA arousals	As above	Total sleep $F_{(1,33.6)} = 5.34, p = .027$
ASDA arousal index	As above minus total sleep	Sequence $F_{(3,20.3)} = 4.44, p = .015$ Shift $F_{(1,17)} = 7.73, p = .013$
Stage woken from (SWS or not)	As above plus total sleep	Acute debt $F_{(1,17)} = 7.08, p = .017$ Total sleep $F_{(1,17)} = 15.16, p = .0018$

²¹ Sequence = place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

Order = the arrangement of the four study conditions for an individual (1 to 18)

Shift type = night shift worked (K1 or K2)

Prior wake = length of time awake prior to start of napping opportunity

Acute debt = acute sleep loss in the 24 hours prior to the start of the napping opportunity

Total sleep = amount of polysomnographically calculated sleep scored during the napping opportunity

4.6 Question 4: Subjective Estimates of Nap Sleep

4.6.1 *Statistical Analyses*

Mixed models ANCOVAs were calculated to determine if there were differences for each of the respective dependent subjective variables due to the shift type on which the napping opportunity occurred. As can be seen from Table 4-9 these models also included the length of prior wakefulness and the level of acute sleep loss.

A second series of models were then run to determine whether individuals' subjective estimates of their nap sleep were related to their polysomnographically defined sleep variables. The structure of these analyses are also detailed in Table 4-9. All dependent variables, apart from stage 3 sleep, were continuous. A mixed model logistic regression was run for the model including stage 3 sleep, with stage 3 dichotomised as either present or absent.

In all models a compound symmetric covariance structure provided the best fit between repeated measures, while also being the theoretically the most appropriate.

Table 4-9: Dependent and Independent Variables for Analyses Related to the Subjective Estimate of Nap Sleep

Dependent Variables	Independent Variables ²²
<i>Subjective sleep variables</i>	
Subjective sleep length	
Quality of sleep	Sequence
Difficulty falling asleep	Order
Depth of sleep	Shift type
Waking feeling refreshed	Prior wake
Degree of help in managing fatigue during remainder of night	Acute debt
<i>Objective sleep variables</i>	
Total sleep	Sleep length
Sleep latency	Falling asleep
Amount of stage 1 sleep (minutes)	Quality
Amount of stage 2 sleep (minutes)	Refreshed
Stage 3 sleep (entered or not)	Depth of sleep
Number of ASDA arousals	Usefulness in managing sleep

4.6.2 Results: Subjective Data on the Length and Quality of Sleep

No significant differences were found between ratings made on the two types of night shift worked, therefore the summary data presented in Table 4-10 are for all napping opportunities irrespective of the type of night shift worked.

²² Sequence = place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

Order = the arrangement of the four study conditions for an individual (1 to 18)

Shift type = night shift worked (K1 or K2)

Prior wake = length of time awake prior to start of napping opportunity

Acute debt = acute sleep loss in the 24 hours prior to the start of the napping opportunity

Sleep length = subjective estimate of sleep length

Falling asleep = rating of difficulty falling asleep

Quality = rating of quality of sleep

Refreshed = rating of how refreshed an individual felt on waking

Depth of sleep = rating of depth of sleep

Usefulness in managing fatigue = rating of how helpful the sleep was in helping to manage fatigue across the remainder of the night shift

Table 4-10: Mean, Standard Deviation, and Range of Subjective Sleep Length and Quality Variables

Dependent Variable	<i>Mean</i>	<i>SD</i>	<i>Range</i>	<i>N</i>
Sleep length (minutes)	16.69	11.35	0-35	54
Difficulty falling asleep (0 = not at all difficult, 10 = very difficult)	5.97	2.90	6-10	53
Quality of sleep (0 = very poor, 10 = very good)	3.80	2.84	0-9.7	53
Waking feeling refreshed (0 = not at all, 10 = completely)	4.48	2.13	0-9	53
Depth of sleep (0 = light, 10 = deep)	2.91	2.64	0-8.4	53
Degree of help in managing fatigue during remainder of night (1 = very unhelpful, 10 = very helpful)	5.63	2.24	0-9.5	53

Table 4-11: Details and Results of Mixed Model ANCOVAs for Subjective Sleep Variables

Dependent Subjective Variable	Fixed and Continuous Effects in Mixed Model	Significant Main Effects
Sleep length (minutes)	sequence, order, shift type, prior wake, acute debt ²³	Prior wake $F_{(1, 28)} = 7.44, p = .011$
Difficulty falling asleep	As above	Prior wake $F_{(1, 24.6)} = 8.35, p = .008$ Sequence $F_{(3, 21.9)} = 3.21, p = .043$
Quality of sleep	As above	Order $F_{(18, 8.05)} = 3.37, p = .042$ Prior wake $F_{(1, 27)} = 13.82, p < .001$
Depth of sleep	As above	Prior wake $F_{(1, 26.9)} = 10.18, p = .003$

²³ Sequence = place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

Order = the arrangement of the four study conditions for an individual (1 to 18)

Shift type = night shift worked (K1 or K2)

Prior wake = length of time awake prior to start of napping opportunity

Acute debt = acute sleep loss in the 24 hours prior to the start of the napping opportunity

As can be seen in Table 4-11, the length of prior wake was found to be significant for several variables. The slope of the regression estimates from each of the models indicated that longer periods awake resulted in higher subjective estimates of the amount of sleep obtained during the napping opportunity, reduced difficulty falling asleep, better quality sleep, and a deeper sleep.

The variables of sequence and order also reached significance in the model for subjective sleep quality. Analysis of the post hoc t tests indicated that the nap on the first study night of the four was rated as being of poorer quality than the nap on the second study night ($t_{(21,1)} = -2.98, p = .007$). However, when looking at the post hoc t tests for the variable of order, no comparisons reached significance.

Although there were no significant differences due to the type of shift worked there was a tendency for the later sleep on the K2 night shift to be reported as slightly longer, initiated more easily, of better quality, more refreshing, deeper, and more useful in assisting individuals to manage their fatigue across the remainder of the night shift. Despite these trends it must be re-emphasised that these differences were not statistically significant and were small (less than 1 on the VAS).

Individuals had also been asked to record the location in which they had napped. Ninety three percent of the naps were taken in an office or equivalent area (such as the sick bay, conference room, or room that contained technical equipment). In all these instances the room could be darkened and was isolated from disruptions. Two individuals on four napping occasions chose to sleep on a couch in the main radar room. Despite the levels of ambient noise and light, on all four occasions these individuals slept.

The next series of mixed model ANCOVAs were completed to determine if individuals' subjective estimates of their nap sleep were related to their polysomnographically defined sleep variables. The structure and main findings of these analyses are detailed in Table 4-12.

As seen in Table 4-12 subjective estimates of sleep length were related to polysomnographically scored sleep, sleep latency and the amount of stage 2 sleep obtained. In all these instances the direction of the relationship was as expected, with longer subjective estimates associated with greater periods of polysomnographically scored sleep, shorter latencies and more stage 2 sleep.

Table 4-12: Details and Results of Mixed Model ANCOVAs Investigating the Relationship Between Subjective Estimates of Sleep and Polysomnographically Determined Parameters

Dependent Variable (polysomnographically generated)	Fixed Effects in Mixed Model	Significant Main Effects
Total sleep	Sleep length, falling asleep, quality, refreshed, depth of sleep, usefulness in managing sleep ²⁴	Subjective sleep length $F_{(1,38.5)} = 8.27, p = .007$
Sleep latency	As above	Subjective sleep length $F_{(1,34.2)} = 5.52, p = .025$
Amount of stage 1 sleep (minutes)	As above	Sleep quality $F_{(1,38.3)} = 4.26, p = .046$
Amount of stage 2 sleep (minutes)	As above	Subjective sleep length $F_{(1,44.8)} = 6.27, p = .016$
Stage 3 sleep (entered or not)	As above	Usefulness in managing fatigue $F_{(1,32.2)} = 4.16, p = .050$
Number of ASDA arousals	As above	No significant effects

²⁴ Sleep length = subjective estimate of sleep length

Falling asleep = rating of difficulty falling asleep

Quality = rating of quality of sleep

Refreshed = rating of how refreshed an individual felt on waking

Depth of sleep = rating of depth of sleep

Usefulness in managing fatigue = rating of how helpful the sleep was in helping to manage fatigue across the remainder of the night shift

An individual's rating of the quality of sleep and the usefulness of the nap sleep in managing fatigue across the remainder of the night shift was related to the occurrence of stage 1 and 3 sleep respectively. A nap was considered better quality when less stage 1 sleep was present and rated as more useful in helping manage fatigue when stage 3 sleep was scored.

However, as can be seen from the Bland-Altman plot (Bland & Altman, 1986) displayed in Figure 4-11, that although there may be a statistical relationship between subjective estimates of sleep length and polysomnographic scored sleep, the agreement between the two measures is actually poor (*mean difference* = 1.02, *SD of differences* = 9.51). Given that the distribution of the differences between sleep length measured via polysomnography and subjective estimates is normal, then it would be expected that 95% of the differences lie between 2 standard deviations of the mean difference. This results in what is termed the “limits of agreement”, which range between +20.04 minutes and -18 minutes. Thus, subjectively an individual is likely to rate their sleep length in a range between 20 minutes longer and 18 minutes shorter than polysomnographically determined sleep length.

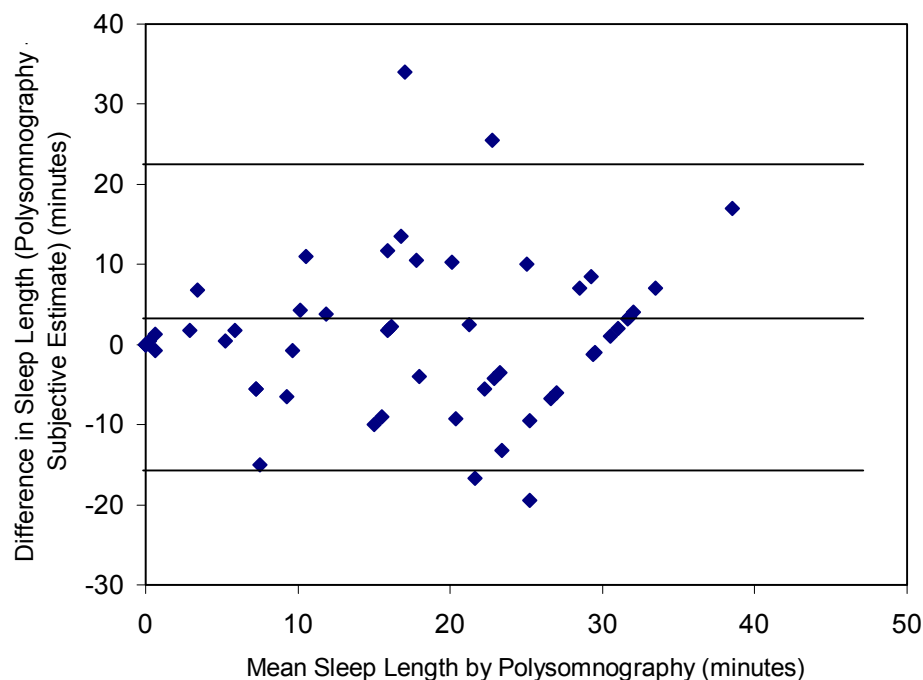


Figure 4-11: Polysomnographically Scored Sleep Length Plotted Against the Difference Between Polysomnographically Determined Sleep Length and Subjectively Reported Sleep Length

4.7 Question 5: Sleep Outside the Napping Opportunity

4.7.1 *Statistical Analyses*

As detailed in the method section of this chapter, the night shift (excluding the nap opportunity) was scored separately for possible sleep. This was completed by a single trained sleep scorer who applied the criteria of Rechtschaffen and Kales (1968) and was followed by validation from a second trained sleep scorer, who independently checked 20% of the recordings.

4.7.2 *Results: Sleep on the Night Shift Outside the Napping Opportunity*

Only on two occasions was sleep, as defined by the standard criteria of Rechtschaffen and Kales (1968) seen outside the napping opportunity. Both these occasions were a minute in total and occurred on two separate study nights for two separate individuals.

On one occasion sleep was seen at approximately 0500 hours on a late starting (K2) night shift where no nap opportunity was provided. In this instance two 30 second epochs of stage 1 were scored approximately 5 minutes apart.

On the second occasion, an individual entered stage 1 for 60 seconds at approximately 0515 hours on a late starting (K2) night shift. No nap opportunity had been provided.

Prior, during, and after entry to stage 1 sleep, both individuals had a large number of SEMs. Neither of these individuals had unusual sleep debts or sleep histories leading up to the night shift. One individual had a cumulative sleep debt of 2.37 hours at midday prior to the night shift, an acute debt of 0.77 hours by the start of the night shift, and had slept for 3.25 hours between the early morning shift and starting the night shift. The second individual had a cumulative debt of 2.67 by midday preceding the night shift, an acute debt of 1.10 hours on starting the night shift, and had slept for 1.1 hours the afternoon before the night shift.

4.8 Summary

Q1. How much polysomnographically defined sleep do air traffic controllers obtain during the pre-planned napping opportunities on the night shift?

It was hypothesised that because of the circadian phase in which the napping opportunity occurred that sleep would be initiated relatively quickly. However, of those who fell asleep, the mean latency to the first minute of sleep was actually very long at 19.40 minutes. It was also hypothesised that the length of the sleep would be much less than the length of the sleep opportunity provided and the efficiency of the sleep would be relatively low. This was the case with the mean time between the first minute of sleep and last epoch of sleep being 20.73 minutes and within this time frame a mean total 17.70 minutes of sleep occurred. Consequently, a low mean sleep efficiency (referred to the total time allowed for the nap) of 41.01% was found.

Q2. Does the length, depth or quality of the sleep obtained differ according to the timing of the nap?

As was hypothesised, the amount of sleep obtained in the 24 hours prior to the napping opportunity and the length of time since an individual last slept did influence variables representing the structure of the nap sleep. Homeostatic factors were seen to effect the amount and percentage of time awake, sleep efficiency, sleep latency, and whether deep sleep was entered and woken from. Although circadian influences were also expected to play a role in determining the length and structure of the sleep obtained during the napping opportunity, few variables were seen to differ between the napping opportunities on the two night shifts. The later sleep was more likely to result in stage 3 sleep and individuals waking from deep sleep.

Q3. Is there any evidence that sleep obtained during the first study night differs from sleep obtained on subsequent study nights?

There was little evidence of a first night effect. Only the number of awakenings per hour and number of ASDA arousals per hour differed according to the night shift on which the polysomnographic recording occurred. Furthermore, the first night differed only from one other night (either the third or fourth study night). Therefore, there is little support for a first night effect.

Q4. Can individuals accurately assess the quantity and quality of sleep obtained during the napping opportunity?

Although there is a statistically significant relationship between the various subjective estimates of the facets of nap sleep and the polysomnographically derived variables, the relationship is not strong. For example for sleep length, an individual may rate their sleep as being anywhere between 20 minutes longer or 18 minutes shorter than it was determined to be from the polysomnographic data.

Q5. Do air traffic controllers ever fall asleep during the night shift outside the napping opportunity?

This is very rare. Across the 107 night shifts monitored, only two individuals on two separate occasions were seen to fall asleep and on each occasion it was for no more than 1 minute.

4.9 Data Management

Transformations Used in Section 4.5

The distributions of wake time during the sleep period, and sleep efficiency (referred to sleep period) were found to be severely positively and negatively skewed respectively.

The variable, wake time during the sleep period, was log transformed to produce a normal distribution, while the reciprocal of sleep efficiency was calculated to correct the severe negative skewness.

Prior to running the mixed model analyses both S1 and S2 sleep as a percentage of TST were transformed. A logarithmic transform corrected the positively skewed distribution of S1 sleep as a percentage of TST, while S2 sleep as a percentage of TST was reflected then a square root function applied.

The latency to stage 2 sleep from sleep onset was positively skewed, which was corrected by applying an inverse transform.

The total number of sleep transitions that occurred during the napping opportunity and the number of transitions per hour this represented were both transformed using a square root function to eliminate the positively skewed distribution.

A log transform was applied to the dependent variable number of awakenings, as a means of minimising the severe positive skewness. For the same reason, the number awakenings per hour was transformed by applying a reciprocal function. Further, two outlying values were removed from this distribution after analysis of the residuals. The reported results do not include these values due to changed results on the removal of the outliers.

Both the number of awakenings from stage 1 and stage 2 were transformed using a logarithmic function to reduce the severely positively skewed distribution. The number of ASDA arousals per hour was positively skewed, which was corrected by applying a log transform.

Transformations Used in Section 4.6

Prior to running the models pertaining to this section a square root function was applied to the dependent variables, depth and quality of sleep, to remove the slight positive skewness in each of these distributions.

CHAPTER 5

PERFORMANCE CHANGES ACROSS THE NIGHT SHIFT IN CONJUNCTION WITH NAPPING

5.1 Introduction

This chapter presents findings from the performance tests completed by study participants during the night shift. It focuses on the influence of the workplace nap on performance. In addition, other circadian and sleep related factors that could theoretically affect performance were considered in the analyses presented here.

The first section of this chapter focuses on performance at the start of the night shift. The subsequent sections focus on the second and third performance tests, which occurred after the napping opportunity. The analyses for these tests thus compare the nap and no nap conditions.

Although sleep is largely seen as having a positive influence, from an operational perspective there is concern surrounding the occurrence of sleep inertia. This is examined in analyses addressing the possible effects of key aspects of sleep architecture on performance closest to the napping opportunity.

The last sections of this chapter determine whether, in the operational context of the present study, sleep improves subsequent performance. The first of these analyses addresses the relationship between the amount of sleep obtained during the napping opportunity and performance subsequent to the nap. However, individuals have no way of knowing how long they will sleep for. The second set of analyses therefore addresses whether there are benefits of napping, irrespective of the amount of sleep obtained. This question is addressed so that recommendations can be made to individual workers. Finally, it is considered how a range of factors, including circadian and homeostatic components, affects performance in the later stages of the night shift.

Thus, the respective research questions addressed in this chapter, and the associated hypotheses, are as follows:

- Q1. Are there sleep and circadian factors that are reliably associated with performance levels and subjective sleepiness at the start of the night shift?

- H1A. It is hypothesised that increased prior wake, greater acute sleep loss and greater cumulative sleep debt will produce poorer performance and higher subjective sleepiness at the beginning of the night shift, just as these factors would at other times in the circadian cycle.
- H1B. Because the performance tests at the start of the night shift are relatively close in time to each other, irrespective of the night shift they were completed on, it is hypothesised that the night shift worked will not produce differences in performance and subjective sleepiness.
- Q2. Are the effects of the napping opportunity on subsequent performance and subjective sleepiness related to the architecture of the sleep obtained?
- H2A. Based on previous research, it is hypothesised that deeper, less fragmented sleep will result in improved post-nap performance.
- H2B. Because the nap sleep is short and the deeper stages of sleep are infrequently reached, it is hypothesised that sleep inertia will not be evident in post-nap performance.
- Q3. Are the effects of the napping opportunity on subsequent performance and subjective sleepiness related to the quantity of sleep obtained in dose-dependent manner?
- H3. It is believed that the main effect of sleep obtained during the napping opportunity would be to diminish the homeostatic drive for sleep. Performance subsequent to the nap would therefore be improved with greater amounts of nap sleep. However, the influence of circadian phase is expected to produce a sustained decline in performance across the night shift irrespective of the amount of sleep obtained at work. The outcome is hypothesised to be a more gradual reduction in performance decline for those who sleep more, compared to a steeper decline in performance for those who obtain little or no sleep.

- Q4. Are the effects of the napping opportunity on subsequent performance and subjective sleepiness independent of the architecture and quantity of sleep obtained?
- H4. It is hypothesised that performance improvements will be related to the amount of sleep obtained in the napping opportunity.
- Q5. Do prior sleep patterns or circadian phase predict performance and subjective sleepiness subsequent to the workplace nap on the night shift?
- H5A. It is hypothesised that increased prior wake, greater acute sleep loss and cumulative sleep debt will produce poorer performance and higher subjective sleepiness at each performance test, through the influence of an increased homeostatic drive for sleep.
- H5B. The timing of performance tests is hypothesised to produce changes in performance, with tests occurring closest to the circadian nadir showing the poorest performance.

5.2 Method

5.2.1 Measures

The following selected dependent variables, generated by the *REACT*TM software from each performance test, were utilised in this chapter.

Performance variables

- **Number of reaction events:** This variable is a count of the number of “correct” responses in a single test.
- **Mean reaction time:** This is the mean of all reaction events across a single test. Due to a small number of slow reaction times skewing average performance, and the resulting increase in variance, the distribution of this variable within a testing session requires transformation. Dinges et al. (1987) have shown that a reciprocal transform reduces the influence of the extreme responses and

increases the likelihood of observing subtle shifts in performance at the faster end of the reaction time continuum. On this basis, in all instances in the present study, raw values were transformed using a reciprocal function, then the mean of these transformed values employed. The PVT analysis programme, *REACT*TM, calculates the mean in this manner, rather than calculating the mean of raw values and then applying a reciprocal transform. Thus, all calculations are on reaction speed, rather than reaction time.

- **Median reaction time:** The median response time across a single test was also utilised as a measure of overall performance. This measure of central tendency was used in addition to mean performance because of the skewed distributions that generally occurred within each performance test, sometimes even after a reciprocal transform was applied to raw values. The *REACT*TM programme calculates the median from untransformed raw values, and the resulting distribution of medians required reciprocal transformation.
- **Standard deviation:** Increased variability in responding is a well-known effect associated with sleep loss (Williams, Lubin & Goodnow, 1959; Angus & Heslegrave, 1985; Dinges et al., 1987) and to this end the standard deviation of mean reaction speed was employed as a measure of performance variability.
- **Fastest reaction time:** As mentioned in Chapter 1, one facet of performance change due to increasing sleep loss is a relatively modest decline in the fastest responses. This end of the responding continuum has been termed the “optimum response” domain (Dinges, 1991), and can be represented by the mean of the fastest 10% of reaction events (Dinges & Powell, 1989). Again due to the distribution of values, the mean of the fastest 10% of values were reciprocally transformed.
- **Slowest reaction time:** The slowest 10% of reaction events encapsulates the worst performance that has occurred across a testing session. This value included events exceeding 500 ms, and was reciprocally transformed.
- **Slope:** An aspect of performance decrement commonly recognised in association with sleep loss is the inability to maintain a constant rate of responding across the entire duration of a task. This time-on-task decrement has

been demonstrated in tests as short as 10 minutes (Dinges, 1992) and can be captured by the linear regression slope fitted to reaction events across a test. To minimise the influence of extreme reaction events, it is calculated using the reciprocal reaction times on a minute-by-minute basis. A negative regression line represents overall performance decline across the testing session, while a positive regression line indicates improvement.

- **Intercept:** The y intercept of the linear regression line calculated using the reciprocal reaction times for each minute of a test.
- **Percentage change:** Change in the regression line from beginning to end of the test.
- **Lapses:** Lapses were also included as a measure of increased variability in responding. The count of the number of responses more than twice the baseline mean has previously been utilised to capture increased unevenness in performance. In the present study the number of responses greater than 500 ms (twice the mean) were summed. The number of lapses was dichotomised into lapses or no lapses due the severely skewed distribution.
- **Total errors:** A sum of the number of all errors, which includes false responses, incorrect key usage, false starts using the correct key, false starts using the incorrect key, and instances where the key was held down. As with lapses the variable was dichotomised into two levels representing errors, or no errors.

Subjective estimates of sleepiness

- **Pre- and post-test sleepiness rating:** Rating on the 10 point VAS before and after each test.

5.2.2 *Statistical Analyses*

As in previous chapters, descriptive statistics have been generated and are presented in order to provide information on the general characteristics of a variable. Where normal distributions are evident the mean and standard deviation are given, while the median

and standard error of the median were considered more informative if the data did not show a Gaussian distribution.

Linear mixed models were again used, and the specific dependent and independent variables for analyses are detailed in the respective sections. However, in general the independent variables included in analyses are as discussed below.

Homeostatic Sleep Drive

Levels of cumulative sleep debt and acute sleep loss, as calculated in Chapter 3, were utilised. Cumulative sleep debt was represented by the levels of debt at midday prior to the start of the night shift. Acute sleep loss and prior time awake were both calculated backwards from the commencement time of each performance test. Cumulative and acute assumed sleep values were utilised rather than TIB or actual sleep debt for the same reasons as outlined in Chapter 3.

Circadian Influences

In this study, the shift type (early K1 or late K2 night shift) and test number (1st, 2nd, or 3rd) capture the timing of tests, as well as allowing the assessment of performance changes between shifts and across the respective shift (further detail of the timing of the three tests is given in section 5.3, Table 5-1).

Because the napping opportunity occurred prior to the second and third performance tests, it was deemed inappropriate to investigate changes across all three performance tests and at the same time include in the model the amount of sleep obtained during the napping opportunity.

Sleep Architecture Variables

Where the relationship of architectural aspects of sleep to performance were of interest, the sleep stage woken from, occurrence of S3 sleep, and the number of arousals were included in analyses, in addition to the amount of sleep obtained during the napping opportunity, shift type worked, and the length of prior wakefulness. The reason for selecting these specific variables is further detailed in section 5.5.1.

In the analyses where the focus was on which factors best predicted performance subsequent to the napping opportunity, performance at the start of the night shift was

considered for inclusion as a covariate, along with other independent variables discussed above.

Homogeneity of Variance

Homogeneity of variance between performance at the three test times was tested using F_{\max} , which is the ratio of the largest cell variance to the smallest. This was considered more appropriate than using formal tests of homogeneity of variance, such as Levene's test of equality of variance, which are considered too highly influenced by non-normality (Tabachnick & Fidell, 1996). At $p < .05$, the variance at time 2 differed from the variance at time 3, $F_{(102,103)} = 1.52$, and the variance at time 1 differed from the variance at time 3, $F_{(102,103)} = 1.94$, although the variance at time 1 and 2 did not differ significantly. Given this expected finding, heterogeneity of variance between an individual's performance at different times was modelled in analyses where the results from more than one performance test session were considered.

Collinearity

For all models, possible multi-collinearity between independent variables was checked to prevent the statistical problems created by including redundant variables in analyses (Tabachnick & Fidell, 1996). For variables influencing performance at the beginning of the night shift, and the architectural facets of sleep that influence performance, the eigenvalues, conditioning indexes and variance proportions did not indicate collinearity. However, for variables associated with the architectural aspects of sleep, there was concern about the relatively high levels of correlation between nap sleep time (plus and minus stage 1) and the stage of sleep woken from and the occurrence of stage 3 sleep. Pearson correlation coefficients were $r = 0.58, p < .0001$ and $r = 0.72, p < .0001$ respectively. To ensure that multi-collinearity was not influencing outcomes, these analyses were run with and without nap sleep time. In all instances the results remained unchanged, therefore nap sleep time was included in the reported results.

The variables considered important in predicting performance subsequent to the napping opportunity were also tested for multi-collinearity. As expected, the amount of sleep obtained during the napping opportunity and the length of prior wakefulness were found to be highly correlated, $r = -0.74, p < .0001$. Because the focus of this study was on the effect of sleep obtained at work, it was decided to remove the variable representing length of prior wakefulness. There was also found to be a relationship between

cumulative sleep debt and performance at the beginning of the night shift. A small eigenvalue, large conditioning index, and variance proportions greater than 0.5, were associated with these variables. Because of the likely importance of cumulative sleep debt (Ferrara & Gennaro, 2001) it was decided to retain this variable and remove performance at the beginning of the night shift. Further collinearity diagnostics indicated no redundancy between the remaining variables.

Transformations

Throughout this chapter many dependent variables required transforming to meet the mathematical assumptions of linear mixed modelling (see Figure 5-1). In such instances, the differences between the least-squares means obtained from the mixed model output can not be transformed back to represent meaningful relationships between the original levels of the variables. The differences have therefore not been reported, although the relative relationships are.

Figure 5-1 shows the relationship between the mean and standard deviation for each subject in each test. The increased variability, due to longer response times can be clearly seen. This illustrates the need to transform the raw response time data, to allow statistical analysis of mean performance.

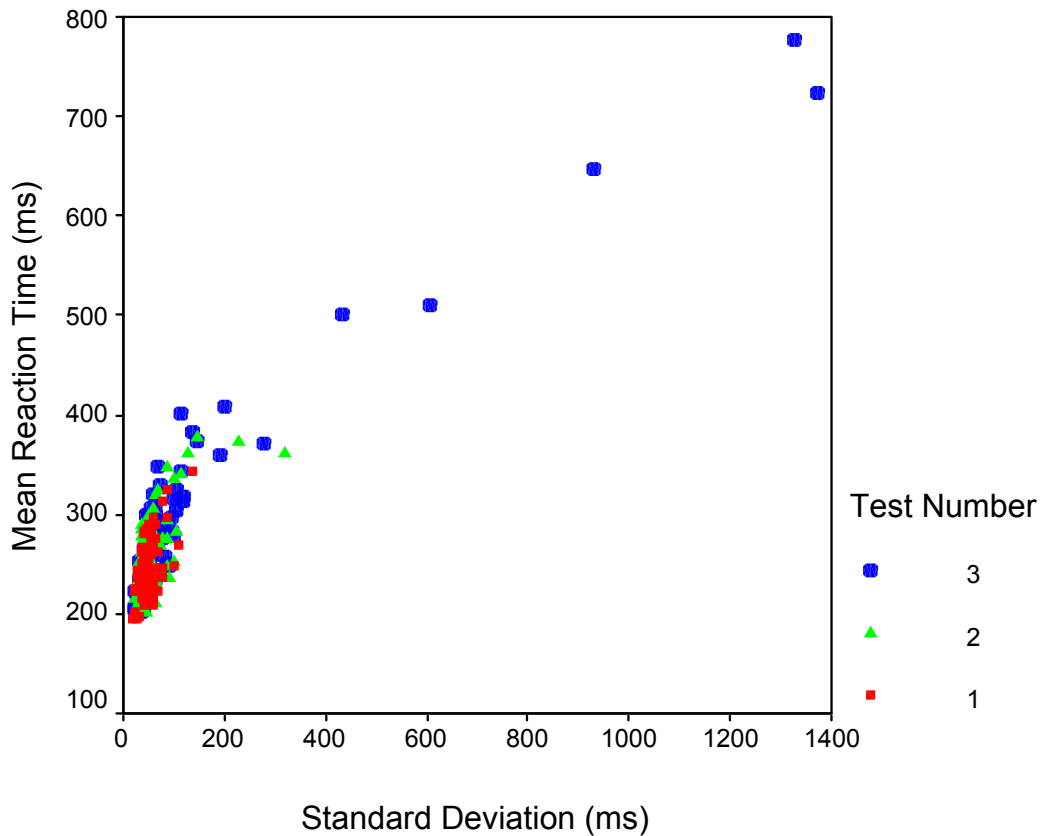


Figure 5-1: Relationship Between Mean and Standard Deviation Across Tests

5.3 Test Details

On average, the PVT recorded 96 correct reaction time values from each individual across the 10 minute duration of each of the 308 tests completed as part of the study (*Range* = 84-104)²⁵. The mean clock time each test was performed is detailed below in Table 5-1. Like the timing of the napping opportunities, operational demands influenced the timing of the performance tests, introducing some variability. However, as can be seen in Figure 5-2 the performance tests on each shift type were closely grouped in time according to the particular test number. As expected there was some overlap in time between tests completed at the end of the K1 (early) night shift and those in the middle of the K2 (late) night shift.

²⁵ See Chapter 2, section 2.2.2, regarding the total number of reaction events presented versus the total number of correct reaction events recorded.

Table 5-1: Mean, Standard Deviation, and Range of Performance Test Times for Each Shift Type.

Shift	Test 1				Test 2				Test 3			
	<i>Mean (hours)</i>	<i>SD</i>	<i>Range</i>	<i>N</i>	<i>Mean (hours)</i>	<i>SD</i>	<i>Range</i>	<i>N</i>	<i>Mean (hours)</i>	<i>SD</i>	<i>Range</i>	<i>N</i>
K1 (early)	2233	0:28	2204-0016	51	0200	0:27	0128-0320	51	0431	0:25	0403-0538	51
K2 (late)	2350	0:24	2325-0117	53	0354	0:25	0306-0415	53	0643	0:22	0622-0744	52

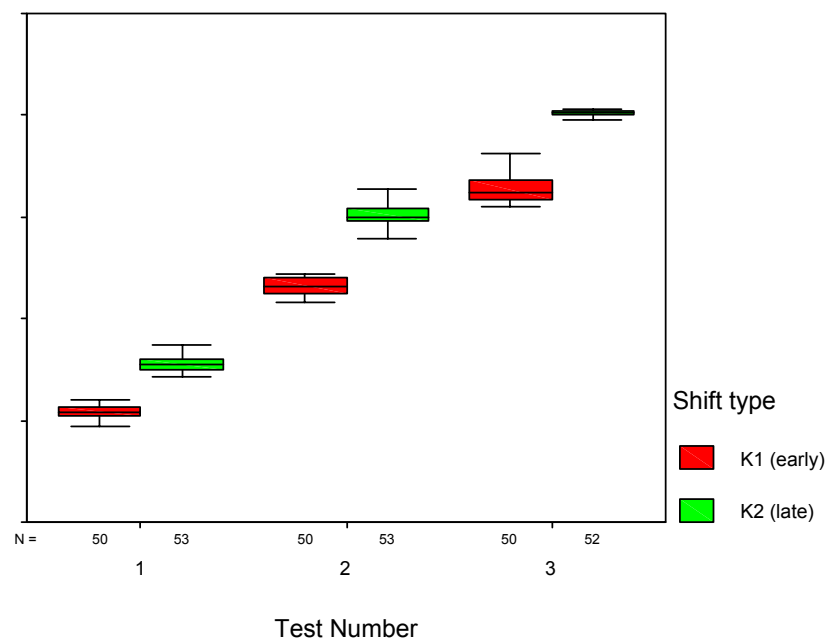


Figure 5-2: Box Plot of Median, Middle 50% of Values, and Standard Errors of Clock Times at Which Performance Tests Were Completed



The distributions of prior wakefulness and levels of acute sleep loss prior to each performance test are presented in Figure 5-3 to Figure 5-8. Values for both shift types are combined in these plots. The data for the separate shifts can be seen in Table 3-16, Chapter 3.

Figure 5-3 displays a severely positively skewed distribution due to a number of individuals having acute debts of zero, producing a median acute debt of 0.47 hours (*Range* = 0-4.02). At the second performance test all individuals have some degree of acute sleep debt, with a mean of 3.77 hours ($SD = 2.94$, *Range* = 0.29-7.88), which by the third performance test reaches a mean of 5.74 hours ($SD = 1.50$, *Range* = 2.65-9.51).

Due to some participants not sleeping between the morning and night shift, there is a bimodal distribution in the length of prior wakefulness at the time of the first performance test, as seen in Figure 5-6. This distribution becomes trimodal for both subsequent performance tests (Figure 5-7 and Figure 5-8) due to the study protocol allowing some participants to nap after the first performance test. Because of these distributions, this variable was categorised into either two or three levels of prior wakefulness, each representing distinctly different sleeping behaviour. In analyses for question 1, there were two levels of prior wake, less than 16 hours since sleep occurred,

and 16 hours or longer since the last sleep. For analyses associated with question 5, the three categories were four hours or less since the last sleep, greater than 4 hours but less than 16 hours since sleep, and 16 hours or longer since the last sleep.

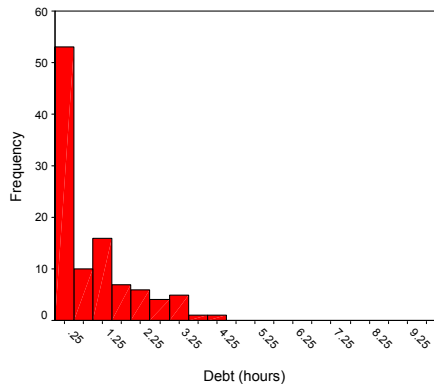


Figure 5-3: Level of Acute Assumed Sleep Debt at First Test.

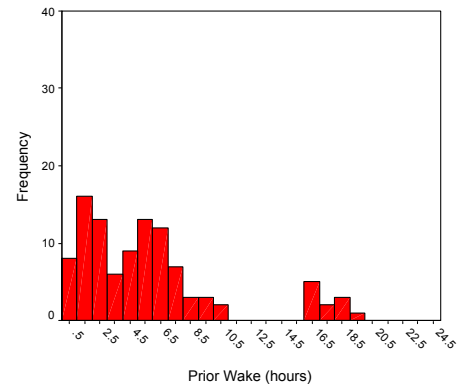


Figure 5-6: Length of Prior Wakefulness at First Test

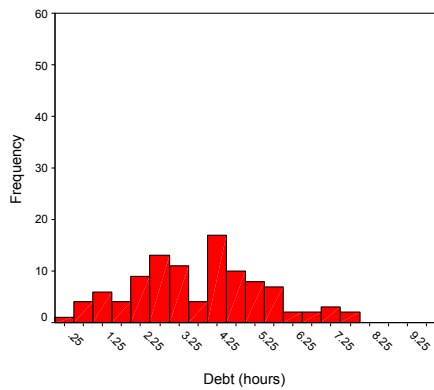


Figure 5-4: Level of Acute Assumed Sleep Debt at Second Test

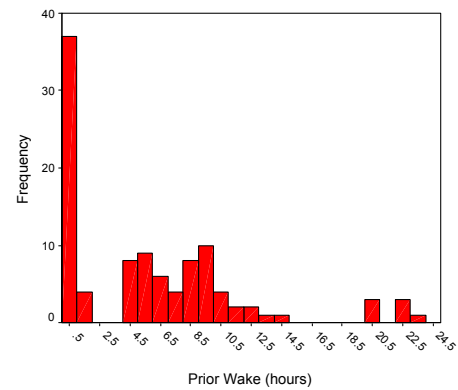


Figure 5-7: Length of Prior Wakefulness at Second Test

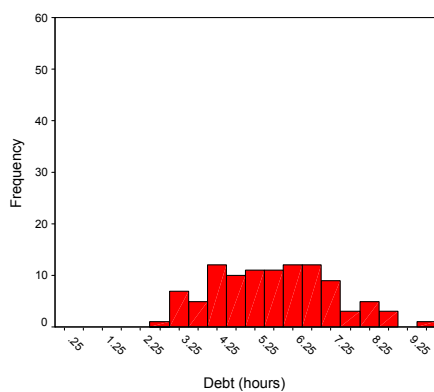


Figure 5-5: Level of Acute Assumed Sleep Debt at Third Test

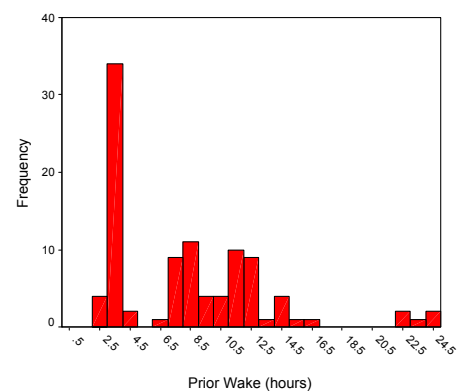


Figure 5-8: Length of Prior Wakefulness at Third Test

5.4 Question 1: Factors Affecting Performance and Subjective Sleepiness at the Start of the Night Shift

5.4.1 Analyses and Data Management

The variables included in these analyses are outlined below in Table 5-1 and in all instances a compound symmetric covariance structure was stipulated between repeated measures. Either mixed model logistic regressions or ANCOVAs were employed depending on the dependent variable.

Table 5-2: Dependent and Independent Variables for Analyses Related to Performance and Sleepiness at the Start of the Night Shift

Dependent Variables	Independent Variables ²⁶
<i>Performance variables</i>	
Mean reaction time	
Median reaction time	
Standard deviation	
Fastest 10% responses	
Slowest 10% responses	
Slope of regression line	Shift
Intercept of regression line	Prior wake
Percentage change in reaction time	Acute sleep debt
Number of lapses	Cumulative sleep debt
Total number of errors	
<i>Subjective estimates of sleepiness</i>	
Pre-test sleepiness rating	
Post-test sleepiness rating	

5.4.2 Results: Performance at the Start of the Night Shift

In all instances the shift type worked, length of prior wakefulness, and the level of acute sleep loss did not reach significance in the analyses where performance measures on the first test were the dependent variables.

²⁶ Shift = night shift worked (either a K1 or K2)

Prior wake = length of time awake prior to start of performance test (≥ 16 hrs or < 16 hrs)

Acute sleep debt = acute sleep loss in the 24 hours prior to the start of the performance test

Cumulative sleep debt = level of cumulative sleep debt at midday prior to commencing the night shift

However, the level of cumulative sleep debt was found to be statistically significant in the models where mean performance, median performance, and the intercept of the regression line were the dependent variables. In contrast to the expected findings, higher levels of cumulative sleep debt were associated with faster mean and median performance. In addition greater levels of cumulative sleep debt produced higher intercept values, indicating faster initial performance. These findings can be seen in Table 5-3.

Further investigations were conducted in an attempt to understand the nature of this anomalous relationship. Consideration was given to the idea that the amount of sleep obtained between the morning and afternoon shift was an important factor and that it could be inversely related to the level of cumulative sleep debt at midday. For example, individuals with higher cumulative sleep debts at midday prior to the night shift may attempt to compensate for their loss of sleep by getting as much sleep as they could between the morning and night shifts. This might explain the unexpected direction of the relationship between cumulative sleep loss at midday and performance at the start of the night shift. The amount of sleep obtained prior to the night shift is taken into account in the calculation of acute sleep loss. However, because the acute sleep loss calculation considered the amount of sleep obtained in the last 24 hours, the pre-night shift sleep only represents a fraction of the sleep considered. Because of this, any possible relationship between the length of pre-night shift sleep and performance at the commencement of the night shift would be unlikely to be seen.

Mixed model analyses were re-run to include a newly calculated variable that represented cumulative debt at midday prior to the night shift plus the amount of sleep obtained between midday and the start of the night shift. Acute sleep debt was excluded from these analyses due to concerns about collinearity. The findings from these analyses were not dissimilar to those of the first series of analyses with the new cumulative sleep debt value also showing a significant positive relationship with performance at the beginning of the night shift, and still not in the direction expected.

Table 5-3: Details and Results of Mixed Model ANCOVAs for Performance and Subjective Sleepiness at the Start of the Night Shift

Dependent Variable	Independent Variables	Significant Main Effects
Mean reaction time	shift, prior wake, acute sleep debt, cumulative sleep debt ²⁷	Cumulative debt $F_{(1, 75.2)} = 6.75, p = .011$
Median reaction time	As above	Cumulative debt $F_{(1, 75.47)} = 6.29, p = .014$
Intercept of regression line (of reciprocal mean)	As above	Cumulative debt $F_{(1, 82.6)} = 8.73, p = .004$
Pre-test sleepiness rating	As above	Shift $F_{(1, 77.9)} = 6.80, p = .011$ Prior wake $F_{(1, 89.1)} = 15.43, p < .001$
Post-test sleepiness rating	As above	Shift $F_{(1, 77.3)} = 9.64, p = .003$ Prior wake $F_{(1, 86.9)} = 7.30, p = .008$

²⁷ Shift = night shift worked (K1 or K2)

Prior wake = length of time awake prior to start of performance test (≥ 16 hours, or < 16 hours)

Acute sleep debt = acute sleep loss in the 24 hours prior to the start of the performance test

Cumulative sleep debt = level of cumulative sleep debt at midday prior to commencing the night shift

Given that these further models did not produce findings that could help explain the unexpected relationship, the data were looked at more closely by fitting a regression line to each individual's performance data. There were two distinct groups of individuals, those who had a clear positive relationship between increasing cumulative sleep debt and improved performance and a smaller group with the opposite relationship. There were also several individuals ($N = 5$) whose performance remained almost unchanged with increasing cumulative sleep debt.

Comparisons between these two groups (excluding individuals with unchanging performance) were made regarding the performance at the beginning of the night, cumulative sleep debt (calculated by adding debt at midday to sleep between midday and the start of the night shift), baseline sleep values, subjective baseline sleep need, age, sex, and length of experience as an air traffic controller. The results of independent t tests showed that cumulative sleep debt at midday prior to the night shift, cumulative sleep debt at the start of the night shift, the amount of sleep obtained between the morning and night shift, and subjective baseline sleep need, did not differ between these two groups. However, those who showed the unexpected relationship, between increasing cumulative sleep debt and better performance, were found to be on average faster at the beginning of the night shift ($t_{(79)} = 3.59, p = .001$), have higher actigraphically determined baseline sleep values ($t_{(79)} = 2.81, p = .006$), be younger ($t_{(82)} = -7.43, p < .001$), less experienced ($t_{(82)} = -7.86, p < .001$), and female ($t_{(82)} = 2.92, p = .005$) compared to those who showed worsening performance with increasing cumulative sleep debt.

Subjective ratings of sleepiness prior to and after the performance test were also utilised as dependent variables in two separate models. As can be seen in Table 5-3, both the shift type worked and the length of prior wakefulness were found to be significant for these variables.

In both instances the estimates of the least-squares means indicate greater sleepiness pre and post the first test on a K2 night shift compared to the first test on the K1 night shift, as well as greater sleepiness with periods of prior wakefulness in excess of 16 hours.

5.5 Question 2: The Influence of Sleep Architecture on Performance and Subjective Sleepiness

5.5.1 *Analyses and Data Management*

In order to look more closely at possible relationships between characteristics of sleep obtained at work and subsequent performance, a subset of data, from shifts on which individuals were allowed to nap, was developed. This resulted in a dataset of 51 cases, 3 less than the total number of naps (54) due to incomplete performance data on these occasions.

Only data from the second performance test were utilised, on the basis that if there were significant effects of sleep structure on performance they would be most apparent in performance that occurred closest to the sleep episode.

Sleep quality can be captured in a number of ways but both depth and continuity have been considered key components (Wesensten, Balkin & Belenkey, 1999; Bonnet, 2000). In the present study sleep quality was indexed through the number of arousals, and the occurrence (or not) of stage 3 sleep.

There is currently debate surrounding the usefulness of considering stage 1 sleep, which is thought to have little recuperative value. When determining total sleep time, the amount of stage 1 sleep is normally summed along with the amount of time in other sleep stages. Wesensten et al (1999) has demonstrated that excluding stage 1 sleep from total sleep time strengthens the relationship between sleep time and subsequent performance and alertness. Because many of the workplace naps in the present study are comprised entirely of stage 1 sleep it was considered important to address this issue. Therefore models were run with total sleep during the napping opportunity represented as the sum of all sleep stages and again minus the amount of stage 1 sleep obtained.

Although the present study was not designed to detect the occurrence of sleep inertia, variables that capture aspects of this effect were included in the analyses examining the performance of individuals prior to returning to work after napping. This was done in case sleep inertia had persisted for a prolonged period of time and because of the possible effects on operational performance. The sleep stage an individual wakes from has been postulated as influential in determining the severity of sleep inertia (Dinges, 1990). Therefore the polysomnographically derived variable representing whether an

individual woke from SWS or not was included in the models examining performance following the workplace nap.

Estimates of the time course of sleep inertia range widely, varying between 1 minute (Webb & Agnew, 1964) and approximately 4 hours (Dijk, Jewett, Czeisler & Kronauer, 1999). If these upper limits of inertia are accurate, then this has important ramifications for operational air traffic controllers. The time since waking was therefore modelled in relation to post-nap performance. In this instance length of prior wake, for those who slept until the end of the napping opportunity, was the difference between the end of the nap and the timing of the performance test. The few individuals who woke before the end of the napping opportunity had prior wake values calculated from their last scored epoch of sleep until the performance test. Alternatively, if they didn't sleep at all, prior wake was the difference between their last actigraphically recorded sleep prior to work and the performance test. For the model in which nap sleep time was represented as total sleep minus the amount of stage 1, prior wake was calculated from the last epoch of sleep other than stage 1 sleep. Individuals whose nap sleep time minus stage 1 was zero minutes had prior wake values calculated from the difference between their last actigraphically recorded sleep prior to work and the performance test.

Finally, to account for the potential influence of circadian factors, the shift type worked, and by extension the timing of the test, was included in these models.

These independent variables and the relevant dependent variables are listed in Table 5-4²⁸. In this instance prior wake values were kept as continuous rather than categorical, because of the lack of distinct groups. In addition, the large majority of prior wake values were less than an hour. Nap sleep duration and number of ASDA arousals were also continuous variables while the shift type worked, stage 3 sleep, and sleep stage woken from were dichotomous variables.

²⁸ As in most analyses there is always the potential to investigate a greater number of main effects and to include various interaction effects in the models. For example, for this question, the interaction of prior wake with the amount of sleep obtained, or stage woken from, or time of the napping opportunity could have been included in order to further investigate aspects of sleep that may influence sleep inertia. In this instance, there is some evidence sleep inertia is most severe after longer periods of sleep and when waking from SWS (Dinges, 1990). There is also contradictory evidence about the influence of circadian phase on the severity of sleep inertia (Dinges, et al., 1985). Despite the possible theoretical interest of including these interaction effects, the statistical ramifications would have been a further reduction in statistical power in a sample where there were only 51 observations. The philosophy with which models were established was that effects should be included only when sound theoretical evidence supported their inclusion, and that the statistical power of tests should be maximised so that significant effects had the best chance of being identified. In balancing these factors the decision was made to focus on the main effects of interest. Although the importance of sleep inertia is recognised, it was not the main focus of the study.

Table 5-4: Dependent and Independent Variables for Analyses Related to the Effects of Nap Sleep Architecture on Performance and Subjective Sleepiness

Dependent Variables	Independent Variables ²⁹
<i>Performance variables</i>	
Mean reaction time	
Median reaction time	
Standard deviation of mean	
Fastest 10% responses	
Slowest 10% responses	Shift
Slope of regression line	Nap sleep (plus or minus S1)
Intercept of regression line	Number of ASDA arousals
Percentage change in reaction time	S3 sleep
Number of lapses	Sleep stage woken from
Total number of errors	Prior wake (s1 sleep considered or not)
<i>Subjective estimates of sleepiness</i>	
Pre-test sleepiness rating	
Post-test sleepiness rating	

As with the analyses for Question 1, mixed model ANCOVAs were employed and for investigating lapses and the total number of errors mixed model logistic regression were utilised due to the dichotomous dependent variables. In all instances a compound symmetric covariance structure was not only theoretically most appropriate, but also provided the best model fit between repeated measures.

5.5.2 Results: The Influence of Architectural Aspects of Sleep

When the amount of stage 1 sleep obtained is removed from total sleep time, the mean sleep length of the napping opportunity is reduced from 17.70 minutes (for the entire 54 naps) to 13.75 minutes ($SD = 10.76$, $Range = 0-42.50$, $N = 51$). Excluding stage 1 sleep

²⁹ Shift = night shift worked (either a K1 or K2)

Nap sleep = Polysomnographic scored total sleep during the napping opportunity (which included or excluded the amount of stage 1 sleep)

Number of ASDA arousals = Number of ASDA arousals per hour

S3 sleep = Whether stage 3 sleep was obtained (entered or not)

Sleep stage woken from = Woke from SWS or not

Prior wake = Period of time from end of napping opportunity to start of performance test (which included or excluded the amount of stage 1 sleep)

also had obvious implications for the calculation of prior wakefulness. The resulting median length of prior wakefulness was 0.83 hours (*Range* = 0.42-22.18, *N* = 51), compared to 0.79 hours (*Range* = 0.42-22.18, *N* = 51) when stage 1 sleep is considered.

The findings of the mixed models ANCOVAs can be seen in Table 5-5. Largely, the various architectural components of sleep were found not to significantly affect subsequent performance. The only exception to this was the intercept of the regression line (for mean reaction time). In this instance, initial performance was faster after stage 3 sleep had occurred than when it had not.

Because of this finding, a further model was run to determine if the effect of S3 sleep on the intercept of the regression line was carried through until the third performance test of the night. This additional model did not produce any significant effects.

A more consistent finding was the significant effect of the shift type worked. Both median reaction time and the standard deviation of the mean were found to vary significantly according to the shift worked. From the estimates of the least-squares means, slower and more variable performance was seen in tests completed on the K2 night shift. Tests completed on the K2 night shift, also produced greater pre- and post-test sleepiness.

Table 5-5: Details and Results of Mixed Model ANCOVAs Investigating the Effect of Architectural Aspects of Sleep on Performance and Subjective Sleepiness

Dependent Variable	Independent Variables ³⁰	Significant Main Effects
Median reaction time	shift, nap sleep, ASDA arousals, S3, stage woken from, prior wake	Shift $F_{(1, 18.4)} = 4.44, p = .049$
Standard deviation (of mean reaction time)	As above	Shift $F_{(1, 19.1)} = 5.03, p = .037$
Intercept of regression line (of mean reaction time)	As above	S3 sleep $F_{(1, 21.8)} = 4.95, p = .037$
Pre-test sleepiness	As above	Shift $F_{(1, 18.7)} = 6.28, p = .022$
Post-test sleepiness rating	As above	Shift $F_{(1, 17.1)} = 8.62, p = .009$

³⁰ Shift = night shift worked (either a K1 or K2)

Nap sleep = Polysomnographic scored total sleep during the napping opportunity (which included or excluded the amount of stage 1 sleep)

Number of ASDA arousals = Number of ASDA arousals per hour

S3 sleep = Whether stage 3 sleep was obtained (entered or not)

Sleep stage woken from = Woke from SWS or not

Prior wake = Period of time from end of napping opportunity to start of performance test (which included or excluded the amount of stage 1 sleep)

In all instances substituting the variable of nap sleep including the amount of stage 1, for nap sleep minus stage 1 made no difference to the significant outcomes. F and p values altered slightly, but those independent variables that were significant remained so. The same applied for prior wakefulness, with no difference in findings whether the amount of stage 1 sleep was accounted for or not.

5.6 Question 3: Effect of the Duration of Nap Sleep on Performance and Subjective Sleepiness

5.6.1 *Analyses and Data Management*

To answer this particular question the focus was restricted to the main and interaction effects of the variables listed in Table 5-6.

These particular interaction effects were included to determine whether sleep obtained during the napping opportunity affected performance differently depending on the proximity of the test to the napping opportunity and the time of night at which the test was completed.

As with the previous set of analyses, two series of models were run, one with nap sleep representing the sum of all four sleep stages and then again with nap sleep calculated without the amount of stage 1 sleep that occurred.

An autoregressive covariance structure was determined to provide the best fit between repeated measures, while random variation due to individual variability was also accounted for.

Table 5-6: Dependent and Independent Variables for Analyses Related to the Effects of Nap Sleep Duration on Subsequent Performance and Subjective Sleepiness

Dependent Variables	Independent Variables ³¹
<i>Performance variables</i>	
Mean reaction time	
Median reaction time	
Standard deviation of mean	
Fastest 10% responses	
Slowest 10% responses	Shift
Slope of regression line	Test number
Intercept of regression line	Nap sleep (plus or minus S1)
Percentage change in reaction time	Shift x Test number
Number of lapses	Shift x Nap sleep
Total number of errors	Test number x Nap sleep
	Shift x Test number x Nap sleep
<i>Subjective estimates of sleepiness</i>	
Pre-test sleepiness rating	
Post-test sleepiness rating	

5.6.2 *Results: Influence of the Amount of Sleep on Subsequent Performance and Subjective Sleepiness.*

Figure 5-9 displays a plot of the raw mean reaction time data versus the amount of sleep obtained at work (during the napping opportunity) for each shift type at test number 2 and 3. A regression line has been fitted to each shift type, test number combination.

³¹ Shift = Night shift worked (either a K1 or K2)

Test number = Number of performance test (either 2nd or 3rd)

Nap sleep = Polysomnographic scored total sleep during the napping opportunity (which included or excluded the amount of stage 1 sleep)

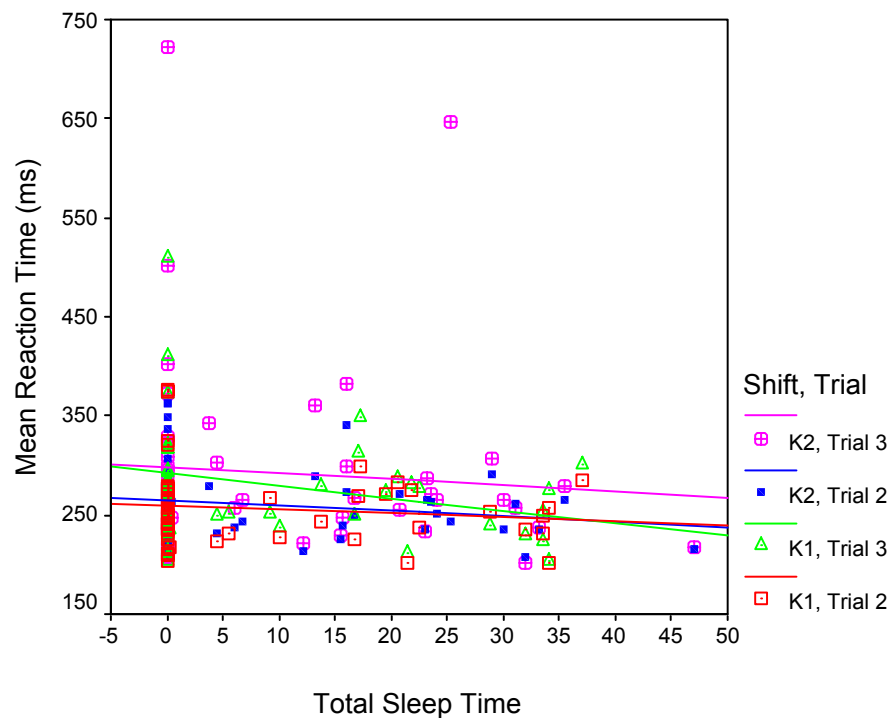


Figure 5-9: Plot of Raw Mean Reaction Time Data Versus Total Sleep Obtained in Napping Opportunity

Details and results of significant analyses investigating whether the amount of sleep obtained during the napping opportunity produced performance changes are outlined in Table 5-7. As can be seen from the results, sleep obtained during the nap was seen to significantly improve most facets of reaction time performance. However, fewer measures of performance improved when sleep stages 2-4 were used to calculate nap sleep length than when stages 1-4 were utilised.

Table 5-7: Details and Results of Mixed Model ANCOVAs and Logistic Regressions for the Effect of Sleep on Performance and Subjective Sleepiness Subsequent to the Nap

Dependent Variable	Independent Variables ³²	Significant Main Effects	Significant Interaction Effects
Mean reaction time	shift, test no, nap sleep, shift x test no, shift x nap sleep, test no x nap sleep, shift x test no x nap sleep	Nap sleep $F_{(1, 71.7)} = 6.26, p = .015$	
Median reaction time	As above	Nap sleep* $F_{(1, 71.8)} = 5.01, p = .028$	
Standard deviation (of reciprocal mean)	As above		Shift x Test number x Nap sleep $F_{(1, 81.2)} = 6.25, p = .014$
Fastest 10%	As above	Nap sleep* $F_{(1, 71.7)} = 4.70, p = .033$	
Slowest 10%	As above	Nap sleep $F_{(1, 69.4)} = 7.13, p = .009$	Shift x Test number x Nap sleep $F_{(1, 75.7)} = 5.65, p = .020$
Intercept of regression line (of reciprocal mean)	As above	Nap sleep* $F_{(1, 71.5)} = 5.07, p = .027$	
Lapses	As above	Test number $F_{(1, 52)} = 9.31, p = .0041$	
Pre-test sleepiness rating	As above	Shift $F_{(1, 55.8)} = 9.09, p = .004$ Test number $F_{(1, 35.5)} = 23.02, p < .001$ Nap sleep $F_{(1, 63.1)} = 9.21, p = .004$	
Post-test sleepiness rating	As above	Shift $F_{(1, 57.1)} = 7.03, p = .010$ Test number $F_{(1, 36.4)} = 22.59, p < .001$ Nap sleep $F_{(1, 63.9)} = 15.10, p < .001$	Test number x Nap sleep $F_{(1, 82.6)} = 12.00, p < .001$

* Did not reach significance when total sleep was represented by the sum of stages 2-4

³² Shift = Night shift worked (either a K1 or K2)

Test number = Number of performance test (either 2nd or 3rd)

Nap sleep = Polysomnographic scored total sleep during the napping opportunity (which included or excluded the amount of stage 1 sleep)

Overall, the estimates from each mixed model indicate that greater amounts of sleep (all stages) during the workplace nap resulted in faster mean and median reaction times. The fastest 10% and slowest 10% of responses were also faster with increasing amounts of nap sleep, as was the intercept of the regression line fitted to each performance test.

For mean reaction time, median reaction time, the fastest 10% of reaction events, and the intercept of the regression line, the interaction of nap sleep length with shift type and test number was not found to be significant. Despite this, the examination of least-square mean estimates suggested differential effects within the shift by test number combinations with performance at the end of the K1 night shift showing the greatest improvement. To see these areas of interest in the data, the plot of estimated mean reaction time performance against sleep obtained during the nap is displayed in Figure 5-10. The mean reaction time values have been transformed back to their original scale, so that larger values indicate slower responses.

In Figure 5-11 the slowest 10% of responses is plotted against the amount of sleep obtained during the napping opportunity. In this particular model the interaction of the amount of sleep obtained with the shift type and test number was identified as statistically significant. However, it was not possible to identify where the differences were. The differences between test number 2 and 3 for each night shift type did not reach statistical significance. Rather, changes between test 2 and 3 were different on the two shift types. Tests of the slopes of the regression lines for the predicted relationship between nap sleep and performance on each shift type and test number combination were assessed and, after correcting for multiple comparisons, only the slope of the regression line for a K1 night shift, test number 3 was significantly different from zero. The regression line for the K2 night shift, test number 2 did not quite reach significance.

The shift type by test number by nap sleep interaction was also found to be significant in the model including the standard deviation of mean performance speed. However, none of the post hoc *t* tests reached significance for the combinations of interest.

Both Figure 5-10 and Figure 5-11 use the estimates from the model to show the predicted relationship between performance and the amount of sleep obtained, in contrast to the raw data seen in Figure 5-9. This was done in order to see more clearly the nature of the relationship between these two variables when accounting for the other variables and interactions stipulated in the model.

In order to understand the overall relationship between performance and the amount of sleep obtained, a further two models were run using mean performance and slowest 10% of responses as the dependent variables, where all interactions between the amount of sleep obtained during the nap and the other variables of shift type and test number were removed. The outcome of this model produced an estimate of the improvement in mean and slowest performance in relation to the amount of sleep obtained. Given that for mean performance the estimate was a reciprocal transform of the original scale, the variable was transformed back in order to make sense of the estimate value. It is realised that this is not a linear transformation, therefore the range in values for each 10 minute increment in sleep on each shift-by-test combination have been reported. In a similar way the slowest 10% of responses had originally been transformed using a reciprocal function followed by a square root. The estimates from the models were transformed back to the original scale, and the range of values is also given due to the non-linear nature of the reverse transform.

For every ten minutes of sleep obtained, up to a total of 47 minutes, mean performance was found to improve by approximately 2.65 ms (*Range* = 2.38-2.96). Therefore, in the second half of the night shift an individual who had obtained no sleep would be approximately 10.6 ms slower on average in responding to a stimulus than if they had obtained 40 minutes of sleep during the napping opportunity.

For the slowest 10% of responses, every 10 minutes of sleep produced an improvement of approximately 14.57 ms (11.92-17.72), so that a 40 minute sleep would improve the average duration of the slowest 10% of responses by 58 ms compared to no sleep.

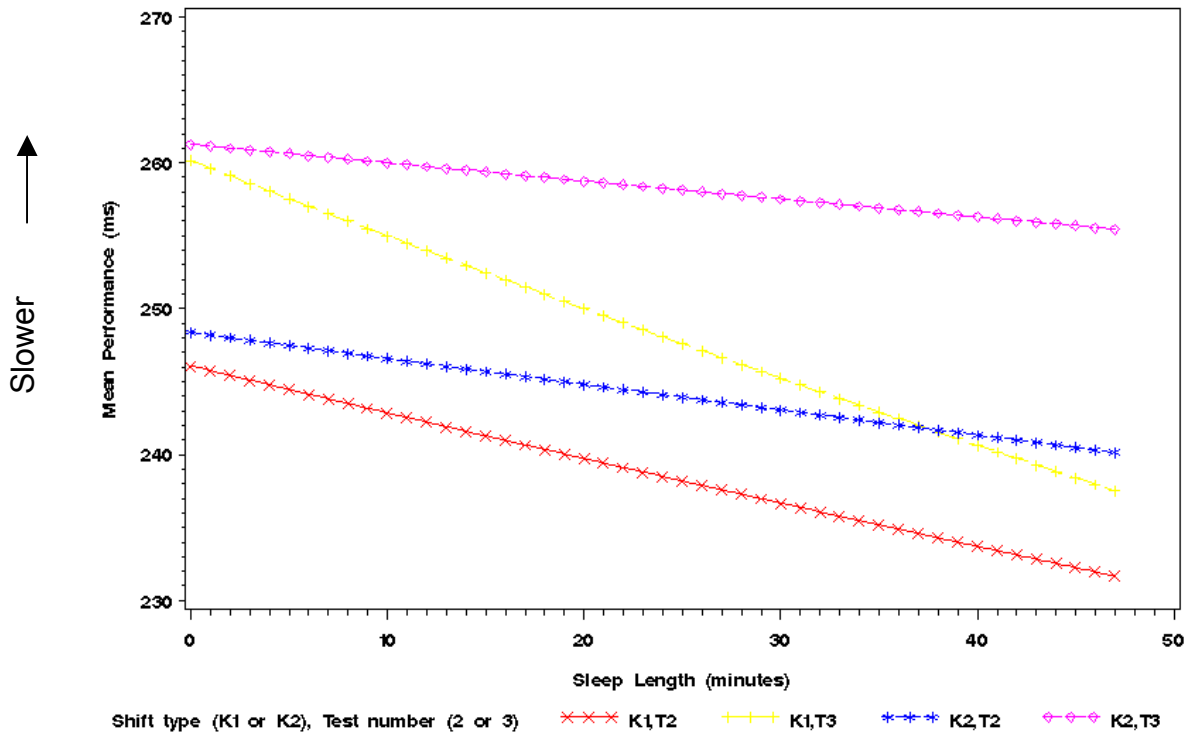


Figure 5-10: Best-Fitted Mean Reaction Time Versus Amount of Sleep During Napping Opportunity for Each Shift Type and Test Number

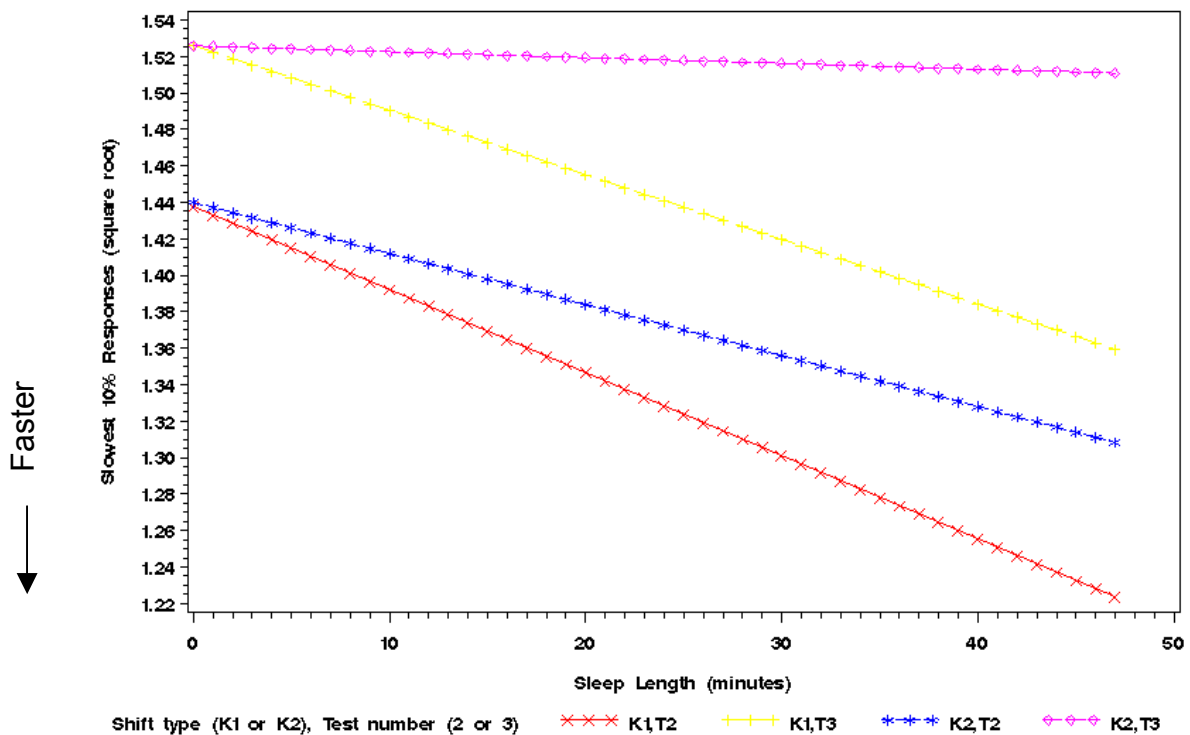


Figure 5-11: Best-Fitted Slowest 10% of Responses Versus Amount of Sleep During Napping Opportunity for Each Shift Type and Test Number

5.7 Question 4: The Effect of a Nap Opportunity on Performance and Subjective Sleepiness Independent of the Architecture and Quantity of Sleep Obtained

5.7.1 *Analyses and Data Management*

As with analyses associated with Question 3, only the independent variables that defined the design of the experiment, and whether a napping opportunity was provided or not were included in the model. A list of these variables, with the respective interaction effects are listed Table 5-8.

Again, as with the models in Question 3, the various interaction effects were stipulated to determine if the napping opportunity produced differential effects given the temporal relationship of the performance test to the napping opportunity and timing differences in the tests on the two different night shifts.

An autoregressive covariance structure was applied between repeated measures. Random variation due to individual variability was also accounted for.

Table 5-8: Dependent and Independent Variables for Analyses Related to the Effects of the Napping Opportunity on Performance and Subjective Sleepiness Independent of the Architecture and Quantity of Sleep Obtained

Dependent Variables	Independent Variables ³³
<i>Performance variables</i>	
Mean reaction time	
Median reaction time	
Standard deviation of mean	
Fastest 10% responses	
Slowest 10% responses	Shift
Slope of regression line	Test number
Intercept of regression line	Nap
Percentage change in reaction time	Shift x Test number
Number of lapses	Shift x Nap
Total number of errors	Test number x Nap
	Shift x Test number x Nap
<i>Subjective estimates of sleepiness</i>	
Pre-test sleepiness rating	
Post-test sleepiness rating	

5.7.2 Results: Influence of the Napping Opportunity on Performance

The spread of reaction times at each test for each of the four study conditions is presented in Figure 5-12. This has been done to illustrate the general changes in overall reaction time performance across the different night shifts depending on whether a napping opportunity was provided or not. The box plot in Figure 5-12 displays the median of the mean as the central horizontal bar, and the box represents the inter-quartile range, which contains 50% of the values. The whiskers extend from the box to the highest and lowest values. The box plot shows generally increasing medians of the mean performance, and perhaps more marked, an increasing spread of mean performance across the three performance tests. The “no nap” study conditions can be seen to have slower mean performance in contrast to the napping condition on the K1

³³ Shift = Night shift worked (either a K1 or K2)

Test number = Number of performance test (either 2nd or 3rd)

Nap = Whether a napping opportunity was provided or not.

night shift by the third performance test, but the improvement is less marked at the second test.

Because a significant interaction between shift type, test number and the amount of sleep obtained during the nap was found for the slowest 10% of responses in the previous series of analyses, the relationship between the slowest 10% of responses under each study condition for each performance test has been graphed in Figure 5-13. The most obvious change in the mean of the slowest 10% of responses is the slowing by the third performance test of the night. This is the case for all study conditions, other than the K1 night shift with a napping opportunity. In this study condition the slowest responses appear to remain relatively unchanged across the night shift. In contrast, on the K2 night shift, the napping opportunity seems to improve the slowest responses at test 2, but this improvement is not completely maintained through to the end of the night shift.

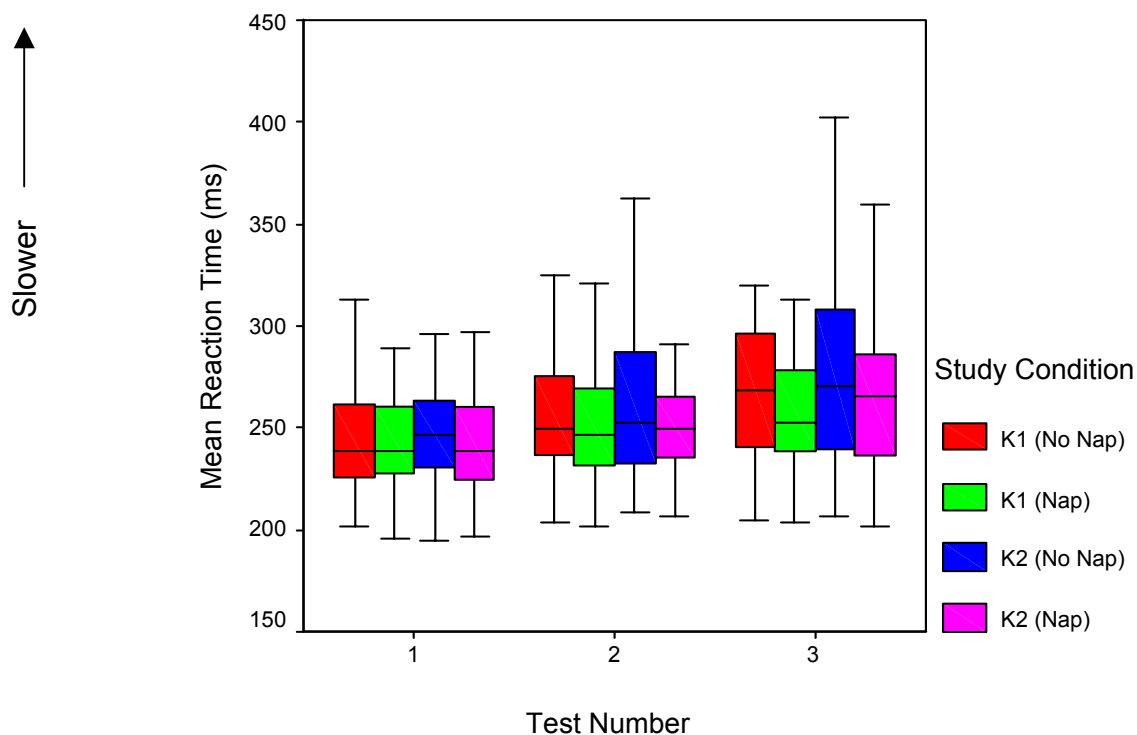


Figure 5-12: Box Plot of Mean Reaction Time For Each Test in the Four Study Conditions

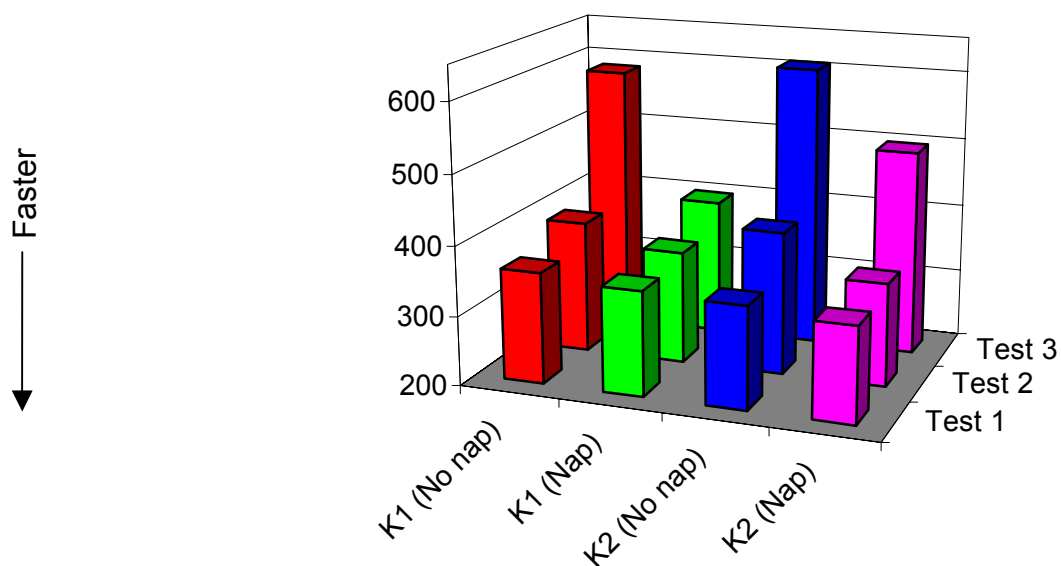


Figure 5-13: Mean of the Slowest 10% of Reaction Events For Each Test in the Four Study Conditions

Table 5-9: Details and Results of Mixed Model ANOVAs and Logistic Regressions for the Effect of Sleep on Performance and Subjective Sleepiness Subsequent to the Nap

Dependent Variable	Independent Variables ³⁴	Significant Main Effects	Significant Interaction Effects
Mean reaction time	shift, test no, nap, shift x test no, shift x nap, test no x nap, shift x test no x nap	Nap $F_{(1, 68.5)} = 7.60, p = .008$	
Median reaction time	As above	Nap $F_{(1, 68.8)} = 6.72, p = .012$	
Fastest 10%	As above	Nap $F_{(1, 67.9)} = 4.09, p = .047$	
Slowest 10%	As above	Nap $F_{(1, 64.6)} = 5.92, p = .018$	
Lapses	As above	Test number $F_{(1, 52)} = 13.48, p < .001$	Test number x Shift $F_{(1, 145)} = 4.03, p = .046$
		Shift $F_{(1, 55.4)} = 15.77, p < .001$	Test number x Nap $F_{(1, 145)} = 4.76, p = .031$
Pre-test sleepiness rating	As above	Test number $F_{(1, 30.6)} = 20.71, p < .001$	
		Nap $F_{(1, 54.6)} = 13.22, p < .001$	
Post-test sleepiness rating	As above	Shift $F_{(1, 53.7)} = 16.57, p < .001$	Test number x Nap $F_{(1, 78.9)} = 9.74, p = .003$
		Test number $F_{(1, 28.5)} = 16.48, p < .001$	
		Nap $F_{(1, 52.9)} = 13.92, p < .001$	

³⁴ Shift = Night shift worked (either a K1 or K2)

Test number = Number of performance test (either 2nd or 3rd)

Nap = Whether a napping opportunity was provided or not.

Details and results from the mixed model ANOVAs can be found in Table 5-9. As with the total amount of sleep obtained during the nap opportunity, the nap opportunity itself produces significant improvements in mean and median reaction time performance. There is also a significant effect for the fastest and slowest 10% of responses. In all these instances the estimates obtained from the model output indicate that the napping opportunity results in faster responding.

Whether an individual lapsed or not showed a significant effect for the test number, and test number by shift type interaction. The shift type by nap opportunity interaction was also found to reach significance. The results from post hoc t tests indicate that lapses are generally more likely at test number 3. In addition, there is an increased likelihood of lapsing at test number 3 on a K1 night shift than at test number 2 on a K2 night shift ($t_{(145)} = 3.27, p = .001$) and at test number 3 rather than at test number 2 on a K2 night shift ($t_{(145)} = -3.95, p < .001$). None of the other shift type by test number interactions reached significance after correcting for multiple comparisons. Nor did any of the shift type by nap opportunity interactions reach significance.

For the subjective estimates of sleepiness, the night shift worked, test number, and whether a nap was provided or not, all resulted in significant changes in ratings both pre and post the performance test. Not having a nap, working the later night shift, and ratings taken at test number 3, produced greater estimates of sleepiness. For post-test sleepiness there was a significant interaction of the test number by nap opportunity. Those t tests that reached significance are displayed in Table 5-10.

Table 5-10: Results of Significant Post Hoc Comparisons for Nap Opportunity by Test Number Interaction

Significant Differences		
	df	t
Nap at Test 2 versus Nap at Test 3	36.4	2.65*
Nap at Test 2 versus No nap at Test 3	62.5	5.40**
Nap at Test 3 versus No nap at Test 3	96.3	4.82**
No Nap at Test 2 versus No nap at Test 3	36.3	4.92**

* $p < .05$

** $p < .001$

5.8 Question 5: Influence of Prior Sleep, Circadian Phase, and Nap Sleep on Performance and Subjective Sleepiness

5.8.1 Analyses and Data Management

In these series of analyses a range of factors that could potentially influence performance outcomes were considered in an effort to determine whether there were variables that were predictive of performance changes in the latter stages of the night shift. These dependent variables included those considered in earlier sets of analyses, such as the shift type worked, test number, interaction of these variables, and the amount of sleep obtained during the napping opportunity. In addition the level of acute sleep loss and cumulative sleep debt experienced were included in the models.

The amount of sleep outside the napping opportunity was originally intended to be a dependent variable in these models, but because sleep was only seen for a minute on two occasions in two individuals, it was not included.

An autoregressive covariance structure was stipulated between repeated measures, with allowance for random variation due to individual variability.

Table 5-11: Dependent and Independent Variables for Analyses Related to the Influence of Prior Sleep, Circadian Phase, and Nap Sleep on Performance and Subjective Sleepiness

Dependent Variables	Independent Variables ³⁵
<i>Performance variables</i>	
Mean reaction time	
Median reaction time	
Standard deviation	
Fastest 10% responses	
Slowest 10% responses	Shift
Slope of regression line	Test number
Intercept of regression line	Shift x Test number
Percentage change in reaction time	Nap sleep
Number of lapses	Acute debt
Total number of errors	Cumulative debt
<i>Subjective estimates of sleepiness</i>	
Pre- test sleepiness rating	
Post- test sleepiness rating	

5.8.2 Results: Factors that Determine Performance and Subjective Sleepiness

In previous analyses both pre- and post-test sleepiness were consistently related to the timing of tests, as well as the napping opportunity. Figure 5-14 displays the median ratings of sleepiness for each study condition across consecutive tests. Medians have been plotted due to non-normally distributed data. They show an overall tendency for increasing sleepiness across the night shift and to a lesser extent the difference due to the nap opportunity and shift type worked.

³⁵ Shift = night shift worked (either a K1 or K2)

Test number = Number of performance test (either 2nd or 3rd)

Nap sleep = Polysomnographic scored total sleep during the napping opportunity (which included or excluded the amount of stage 1 sleep)

Acute debt = acute sleep loss in the 24 hours prior to the start of the performance test

Cumulative debt = level of cumulative sleep loss at midday prior to commencing the night shift

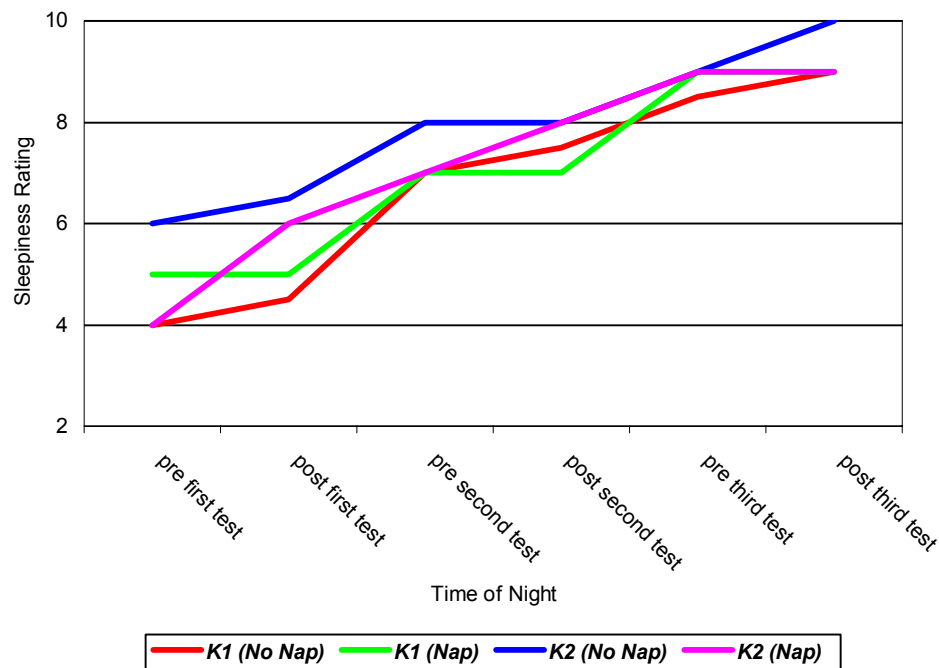


Figure 5-14: Median Ratings of Subjective Sleepiness Pre and Post the Performance Tests on Each Study Condition

Those independent variables for which significant effects were found from the mixed model ANCOVAs, are listed in Table 5-12 along with details of the models. When controlling for the level of acute sleep loss and cumulative sleep debt, the amount of sleep obtained during the napping opportunity was significant for mean reaction time performance and the slowest 10% of responses. The estimates from the models were in the expected direction, with greater amounts of sleep relating to faster mean performance and the slowest responses also being faster.

The test number was significantly related to the presence or absence of lapses, indicating an increased likelihood of lapsing at the end of the night shift as opposed to around the middle of the shift. Test number was also significant in the models for pre- and post-test sleepiness ratings, as were the shift type worked, amount of sleep obtained during the napping opportunity, and the level of acute sleep loss experienced. Both pre- and post-test sleepiness ratings made on the earlier starting night shift, and at test number 2, were lower. Greater amounts of sleep during the napping opportunity and lower levels of acute sleep loss prior to each test also produced lower ratings of sleepiness.

Table 5-12: Details and Results of Mixed Model ANCOVAs and Logistic Regressions for the Effect of Prior Sleep, Circadian Phase, and Nap Sleep on Performance and Subjective Sleepiness

Dependent Variable	Independent Variables ³⁶	Significant Main Effects	Significant Interaction Effects
Mean reaction time	shift, test number, nap sleep, acute sleep loss, cumulative debt	Nap sleep $F_{(1, 70.4)} = 4.46, p = .038$	
Slowest 10%	As above	Nap sleep $F_{(1, 69.7)} = 4.59, p = .036$	
Lapses	As above	Test number $F_{(1, 52)} = 6.50, p = .014$	
		Shift $F_{(1, 61.2)} = 4.47, p = .039$	
Pre-test sleepiness rating	As above	Test number $F_{(1, 44.7)} = 8.39, p = .006$	
		Nap sleep $F_{(1, 62.1)} = 5.68, p = .020$	
		Acute debt $F_{(1, 118)} = 4.88, p = .029$	
		Shift $F_{(1, 64.7)} = 4.37, p = .041$	
Post-test sleepiness rating	As above	Test number $F_{(1, 50)} = 5.03, p = .029$	
		Nap sleep $F_{(1, 65)} = 9.03, p = .004$	
		Acute debt $F_{(1, 116)} = 7.16, p = .009$	

³⁶ Shift = night shift worked (either a K1 or K2)

Test number = Number of performance test (either 2nd or 3rd)

Nap sleep = Polysomnographic scored total sleep during the napping opportunity (which included or excluded the amount of stage 1 sleep)

Acute debt = acute sleep loss in the 24 hours prior to the start of the performance test

Cumulative debt = level of cumulative sleep loss at midday prior to commencing the night shift

5.9 Summary

Q1. Are there sleep and circadian factors that are reliably associated with performance levels and subjective sleepiness at the start of the night shift?

Results indicated that neither prior wake nor acute sleep loss produced significant changes in performance for the first psychomotor vigilance test of the night. Surprisingly, and contrary to what was hypothesised, greater levels of cumulative sleep debt were seen to produce faster mean and median reaction time performance. Further analyses conducted to explain this unexpected relationship identified a subgroup of younger, less experienced, predominantly female controllers, who showed faster responding overall. For subjective estimates of sleepiness, longer periods of prior wakefulness and ratings made on the later starting night shifts resulted in greater reported sleepiness.

Q2. Are the effects of the napping opportunity on subsequent performance and subjective sleepiness related to the architecture of the sleep obtained?

Overall, none of the architectural aspects of sleep were found to be related to performance or subjective sleepiness. The only exception was the occurrence of stage 3 sleep, which produced faster initial reaction time performance. This was the only evidence supporting the possible occurrence of sleep inertia. A more consistent result was an effect of the night shift worked, with the later starting night shift producing slower, more variable performance and higher sleepiness ratings.

Q3. Are the effects of the napping opportunity on subsequent performance and subjective sleepiness related to the quantity of sleep obtained in a dose-dependent manner?

Greater amounts of nap sleep were significantly related to faster reaction time performance. This effect was maintained until the end of both types of night shift. While not statistically robust, there was a trend for sleep on the earlier night shift to be more beneficial than on the later shift, particularly by the end of the shifts.

Q4. Are the effects of the napping opportunity on subsequent performance and subjective sleepiness independent of the architecture and quantity of sleep obtained?

The napping opportunity was related to an improvement in reaction time performance. From an operational perspective this provides support for recommending that a 40

minute napping opportunity be taken during the rest break on the night shift, even if controllers are sometimes unable to sleep.

Q5. Are there factors that predict performance and subjective sleepiness subsequent to the pre-planned workplace nap on the night shift?

It was determined that the amount of sleep obtained during the nap opportunity was more predictive of mean and slowest reaction time performance, than sleep loss or timing factors. However, for extremely long reaction events the time of night was the key predictive variable. In contrast, for sleepiness ratings, both the amount of sleep and time of night were seen to be important, in addition to the amount of acute sleep loss individuals were experiencing.

5.9.1 *Data Management*

Transformations Used in Section 5.4

A large number of the variables listed in Table 5-2 were only ever utilised as transformed variables. In addition to these previously transformed variables, pre-test sleepiness was transformed prior to these analyses using a square root function due to a moderately, positively skewed, distribution.

Transformations Used in Section 5.5

The distribution of post-test sleepiness ratings was found to negatively skewed, which was corrected by applying a logarithmic function to these values.

Transformations Used in Section 5.6

Several variables required transforming to produce distributions that met the assumptions of linear mixed models. The slowest 10% of reciprocal values was negatively skewed, which was corrected by reflecting and applying a square root function. An identical transform was applied to both the percentage change in reaction time across a performance test and pre-test sleepiness ratings. Post-test sleepiness ratings were severely negatively skewed, which was reduced by reflecting values then applying a log transform.

Transformations Used in Section 5.7

Identical variables to those used in Question 3 were utilised in these analyses, therefore the transformations applied are discussed above.

Transformations Used in Section 5.8

Again, the independent variables utilised in Question 3 were the focus of these analyses. The pertinent transformations are discussed above. In the model for the percentage change in reaction time across the test, two outliers were identified from the residual plot. Removing these two values did not alter the findings therefore they were included in the reported results.

CHAPTER 6

NEUROPHYSIOLOGICAL ALERTNESS DURING THE NIGHT SHIFT

6.1 Introduction

This chapter focuses on the relationship between sleep obtained during the napping opportunity and neurophysiological measures of alertness taken during the latter portion of the night shift. One of the prominent changes seen when alertness decreases is increased power in certain frequencies below 25 Hz, generally in the 1 to 9 Hz range (see Chapter 1). Increased power in the alpha and theta frequencies is mentioned most often when the traditional frequency bands are the focus of analyses, while when single frequencies are investigated those around 3-4 Hz and 14-15 Hz have been associated with either an increased homeostatic drive and/or lapses in performance.

Another consistent neurophysiological change that is associated with decreased alertness is the occurrence of slow rolling eye movements. It is still debatable exactly where on the sleepiness continuum they occur, with some authors viewing them as an early sign of sleepiness, while others suggest they represent a relatively late sign of sleep onset. For both SEMs and the EEG changes with increasing sleepiness a wide range of individual variability exists. Thus, comparing data between individuals is difficult and group baselines are likely to be inappropriate.

The neurophysiological changes that occur with sleepiness are not subject to the lack of reliability of subjective measures, making them potentially the most useful means of assessing sleepiness currently available. However, their limitations must be carefully considered and taken into account during the measurement, analysis, and interpretation of data.

The following research questions and associated hypotheses are addressed in this chapter:

- Q1. Once artefact is removed from the EEG data, how much data remains to objectively assess alertness during the last hour of the night shift?
- H1. Because the data were recorded under ambulatory conditions, artefact due to muscle movement is likely to be prevalent. This is in addition to other

sources of artefact, such as eye blinks and movement of the electrode leads. Although every effort was made to minimise artefact from all sources, it is expected that more than 50% of data will be rejected due to artefact.

- Q2. Does the amount of sleep obtained during workplace naps improve, or limit a decline in, neurophysiological alertness in a dose-dependent manner?
- H2. It is hypothesised that the nap sleep will reduce the homeostatic drive for sleep, and therefore symptoms of lowered neurophysiological alertness in a dose-dependent manner. The nap sleep is also expected to make the circadian trough in alertness less pronounced.
- Q3. Are there specific architectural aspects of sleep obtained during the napping opportunity that influence neurophysiological alertness?
- H3. Because of the reduced effectiveness of shorter, broken sleep it is hypothesised that longer, better quality sleep will result in fewer signs of decreased neurophysiological alertness in the latter part of the night shift.
- Q4. Does the opportunity to nap assist in improving, or limiting a decline, in neurophysiological alertness, independent of the amount of sleep obtained?
- H4. It is hypothesised that the effectiveness of the napping opportunity in improving neurophysiological alertness will be due to the sleep obtained during this time frame.
- Q5. Do prior sleep patterns or circadian phase influence neurophysiological alertness on the night shift?

- H5A. It is hypothesised that increased prior wake, greater acute sleep loss and cumulative sleep debt will produce signs of diminished neurophysiological alertness through the influence of the increased homeostatic drive for sleep.
- H5B. Neurophysiological alertness across the night shift is expected to vary due to the influence of changing circadian phase. It is hypothesised that the lowest levels of alertness will be seen at and around the circadian nadir.

6.2 Method

6.2.1 Measures

The two physiological signals on which the assessment of alertness was based were the EEG and EOG. The location of the electrodes and the details of how the data were recorded have been covered at length in Chapter 2.

Information regarding the particular channels used for determining alertness, the time frames involved, and the processes applied in analysing the data are also presented in detail in Chapter 2.

Absolute power was used in all analyses (amplitude squared), as within-subject comparisons were only ever made (Fisch, 1991). Based on the reviewed literature it was decided to initially group the spectral power into the traditional frequency bands. In reviewing the compressed spectral arrays, extremely large, unexpected bursts of power were seen around 1 Hz. Another research group utilising the same equipment had previously reported to the author that they had seen unexplained artefact at very low frequencies (J. Axelsson, personal communication, September 1, 2000). After communicating with the manufacturer it was suggested lead movement could account for artefact in these frequencies, but such movement was not obvious in the visual screening of EEG data. Nevertheless, a conservative approach was taken, and it was decided to limit the delta band to no less than 1.5 Hz.

As well as grouping spectral data into bands, single frequencies were utilised as dependent variables (see below). Because spectral output was produced in 0.195 Hz steps, those frequency values closest to the frequency or band of interest were summed to produce a spectral value.

The following variables, generated from the EEG and EOG data, were used in analyses:

EOG variables

- **Occurrence of Slow Rolling Eye Movements (SEMs):** A dichotomous variable, with levels representing whether SEMs occur or not.

EEG variables

- **Delta Power:** Sum of spectral power between 1.56 and 3.91 Hz (log transformed).
- **Theta Power:** Sum of spectral power between 4.10 and 7.81 Hz (log transformed).
- **Alpha Power:** Sum of spectral power between 8.01 and 11.91 Hz (log transformed).
- **Beta Power:** Sum of spectral power between 12.11 and 15.82 Hz (log and square root transform applied).
- **Single Frequencies:** Sum of spectral power between the following frequencies

Frequency	Upper and lower limits (Hz)
2	2.15-2.93
3	3.12-3.91
4	4.10-4.88
5	5.08-5.86
6	6.05-6.84
7	7.03-7.81
8	8.01-8.98
9	9.18-9.96
10	10.16-10.94
11	11.13-11.91

Frequency	Upper and lower limits (Hz)
12	12.11-12.89
13	13.09-13.87
14	14.06-14.84
15	15.04-15.82
16	16.02-16.99

6.2.2 *Statistical Analyses*

In order to capture and provide an understanding of the frequency and pattern of changes in neurophysiological alertness, both graphical methods and simple descriptive statistics are presented. Where normal distributions exist, the mean, standard deviation, and range are given. If departures from normality were identified, the median and range is reported instead.

Linear mixed models were again the most appropriate statistic to employ because both random factors and repeated measures could be accounted for. Based on the reviewed literature it was expected that individual variability in neurophysiological alertness would be evident, thus making it important to allow random variation due to the individual to be modelled. In addition, this method of analysis enabled the pattern of variance between repeated observations within individuals to be modelled.

Because the questions are similar to those addressed in Chapter 5, many of the mixed models are similar in structure. The specific details of each model are detailed in the relevant results sections. General issues that are relevant to several questions are considered below.

First Night Effect

In Chapter 4, a variable representing the night number on which a study condition was completed was included as a fixed factor in all models. This was done because it had previously been demonstrated that the first night of sleep in a laboratory differed in structure and length from subsequent nights (Agnew, 1966). To this author's knowledge such differences have not been demonstrated in the recording of waking neurophysiological data. However, given the strong first night effect when recording

sleep in laboratory studies, and despite the lack of a first night effect for the nap sleep in the present study, it was considered important to determine if an individual's neurophysiological alertness changed in association with their progression through the four study nights.

Many of the analyses associated with Chapter 4 had also included a variable representing the arrangement of study conditions. This was because a potential confound, due to carry over effects, exists in a within-subjects cross over design. However, it was found that a number of the mixed models that included this variable could not be run, possibly because only a few individuals were associated with each level of the variable. In addition, models requiring more computational capacity were less likely to run with this independent variable included. Due to the expected computational demands of the analyses in the present chapter, this variable was therefore excluded from models.

Homeostatic Sleep Drive and Circadian Influences

When the levels of cumulative and/or acute sleep loss were used in analyses, the values calculated in Chapter 3 were utilised. As in all prior instances actigraphically determined assumed sleep values have been used, rather than TIB or actual sleep values.

The timing of events was usually modelled by the night shift worked (early K1 or late K2 night shift). An additional timing variable was considered for the analysis of SEMs, namely whether they occurred in the first half of the night shift (up to the 2nd performance test, but excluding the nap opportunity), or the second half of night shift. In those analyses where the influence of nap sleep was investigated, only occurrences of SEMs in the second half of the night shift (after the nap) were considered.

As discussed in Chapter 2, due to the enormous amount of EEG data recorded and the time involved in removing artefact and analysing the data, it was decided for the purposes of the present study to restrict the focus to the last hour of each night shift. The time of each 1 hour block of data considered in these analyses can be seen in Figure 6-1.

Sleep Architecture Variables

The same sleep quality measures were included as for the analyses in Chapter 5, namely: the sleep stage woken from, occurrence of S3 sleep, and the number of arousals. The basis for selecting these variables has been outlined in Chapter 5.

As in Chapter 5, models where the amount of sleep obtained in the napping opportunity was included as an independent variable were run once with the total amount of sleep minus the amount of stage 1 sleep obtained, and again with nap sleep length comprising the sum of all four sleep stages.

Collinearity

To remove the statistical problems created by including redundant independent variables in analyses, each model was checked for multi-collinearity.

Eigenvalues, conditioning indexes and variance proportions indicated collinearity between the amount of sleep obtained and several other sleep architecture variables. Because the interest was in the architectural aspects of sleep, the amount of sleep obtained during the nap was removed from these models. However, there were still indications of collinearity between prior wakefulness and sleep architecture variables, and high correlations between the stage of sleep woken from and whether stage 3 sleep was entered. To get the models to run it was necessary to remove both the length of prior wakefulness and the sleep stage woken from.

The variables included in the model for predicting alertness (Question 5) were also tested for multi-collinearity. The eigenvalues, conditioning indexes, and variance proportions indicated collinearity between the length of prior wakefulness and the amount of sleep obtained during the napping opportunity. Since the nap was the main focus of this study, length of prior wakefulness was excluded from these analyses. In addition, there was collinearity indicated between the shift type worked and the sleep loss variables. The choice was made to focus on prior sleep loss, since the timing of the two night shifts did not differ greatly.

Transformations

A number of study participants did not produce any SEMs, resulting in a severely skewed overall distribution. This variable was therefore dichotomised with the two levels representing the presence or absence of SEMs.

Spectral power in the standard frequency bands (delta, theta, and alpha), and the single frequencies was log transformed to reduced the positive skewness of the distribution and to meet the mathematical assumptions of the statistical test employed. A log function

and square root function were applied to beta power to minimise the impact of the skewed distribution.

As discussed in the previous chapter, such transformations result in the differences between least-squares means being difficult to interpret, therefore only the direction of any significant relationship has been indicated rather than the size of the difference.

6.3 Question 1: Artefact Removal

6.3.1 *Timing of 1 Hour Blocks*

A plot of the timing of the 1 hour block at the end of each night shift can be seen in Figure 6-1. These are closely grouped according to the type of shift worked. On average, the last hour of the K1 shift spanned 0316 to 0416 hours, with the start of this 1 hour block ranging between 0255 and 0351 hours. The last hour of the K2 shift started on average at 0529 hours (range = 0516-0552) and finished at 0629 hours.

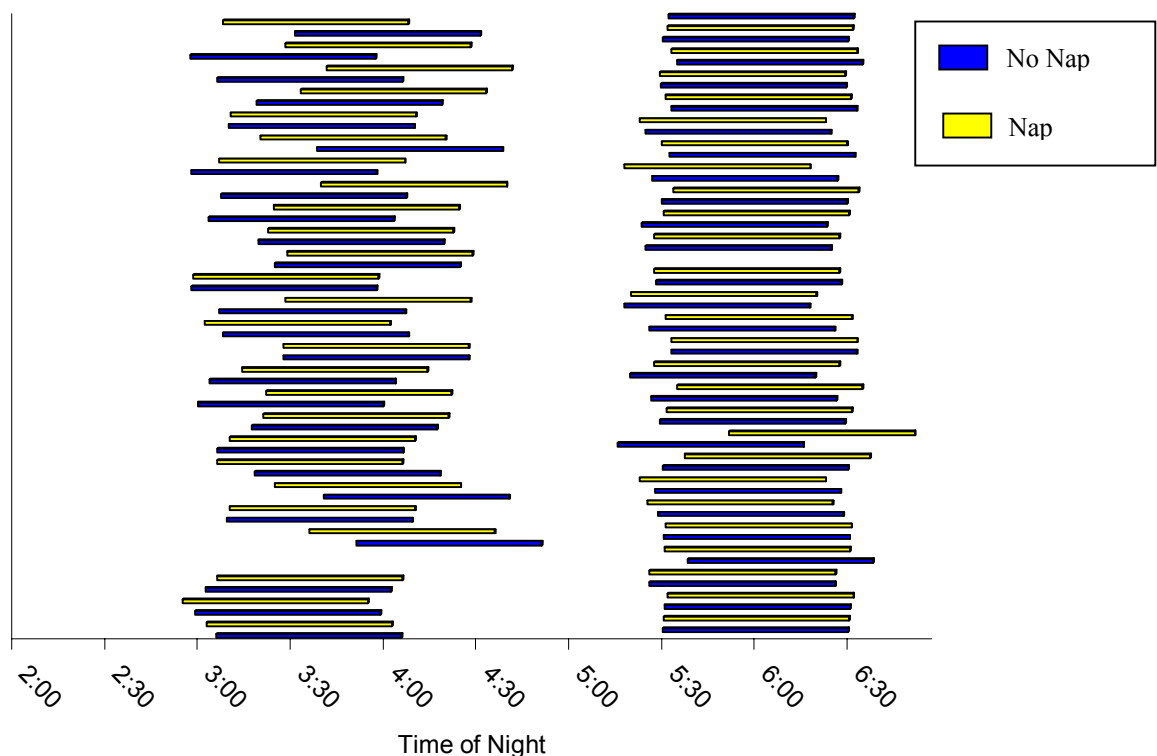


Figure 6-1: Timing of 1 Hour Episodes of EEG Analysed for Neurophysiological Alertness.

6.3.2 *Amount and Pattern of EEG Data Accepted and Rejected*

From the 105, 1 hour blocks of EEG data recorded from the O2-Oz channel, a median of 10.74% of data were considered to be artefact free. Figure 6-2 displays the percentage of artefact free data for each of the 105 recordings, with the minimum value being 0.36% and the maximum 67.52%. A mixed model ANOVA was conducted to determine if the percentage of data accepted differed according to the night shift worked, the napping condition, or the interaction of these variables. The model produced no significant findings.

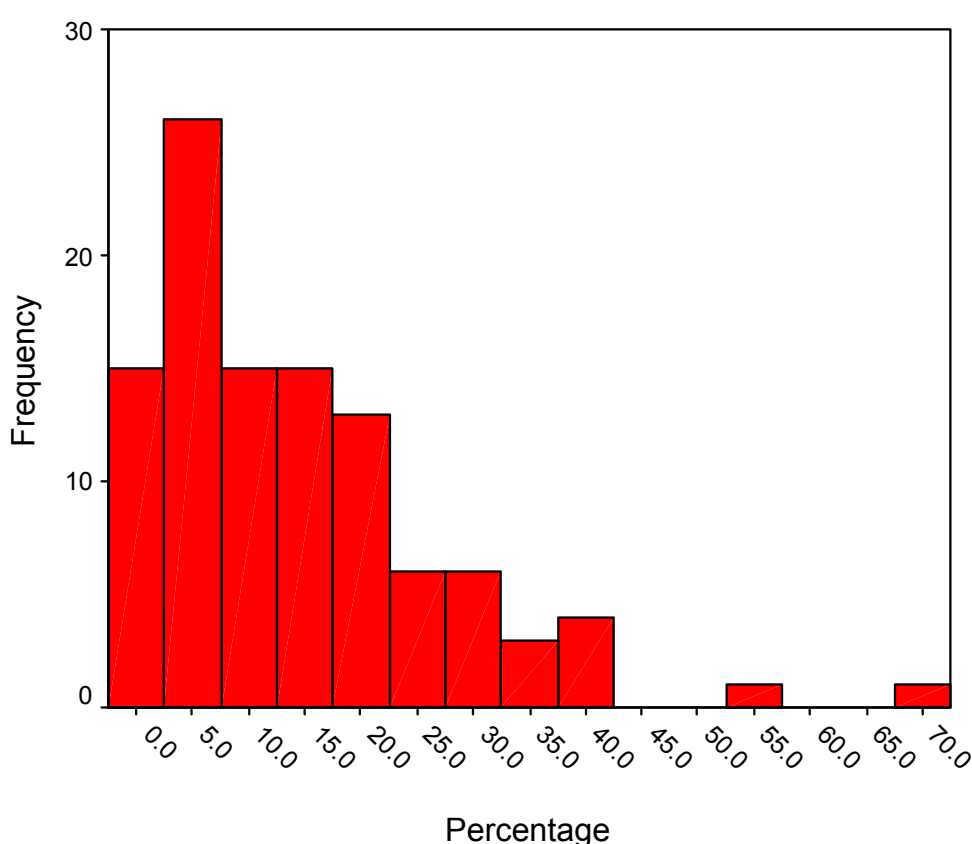


Figure 6-2: Percentage of 1 Hour EEG Recording (Channel O2-Oz) Found to be Artefact Free

A previous investigation (J. Gale, et al., personal communication, January 20, 2002) did not find any pattern in artefact rejection. To graphically illustrate the quantity and random pattern of artefact free data, each individual epoch of artefact free data for each recording has been plotted in Figure 6-3 to Figure 6-5.

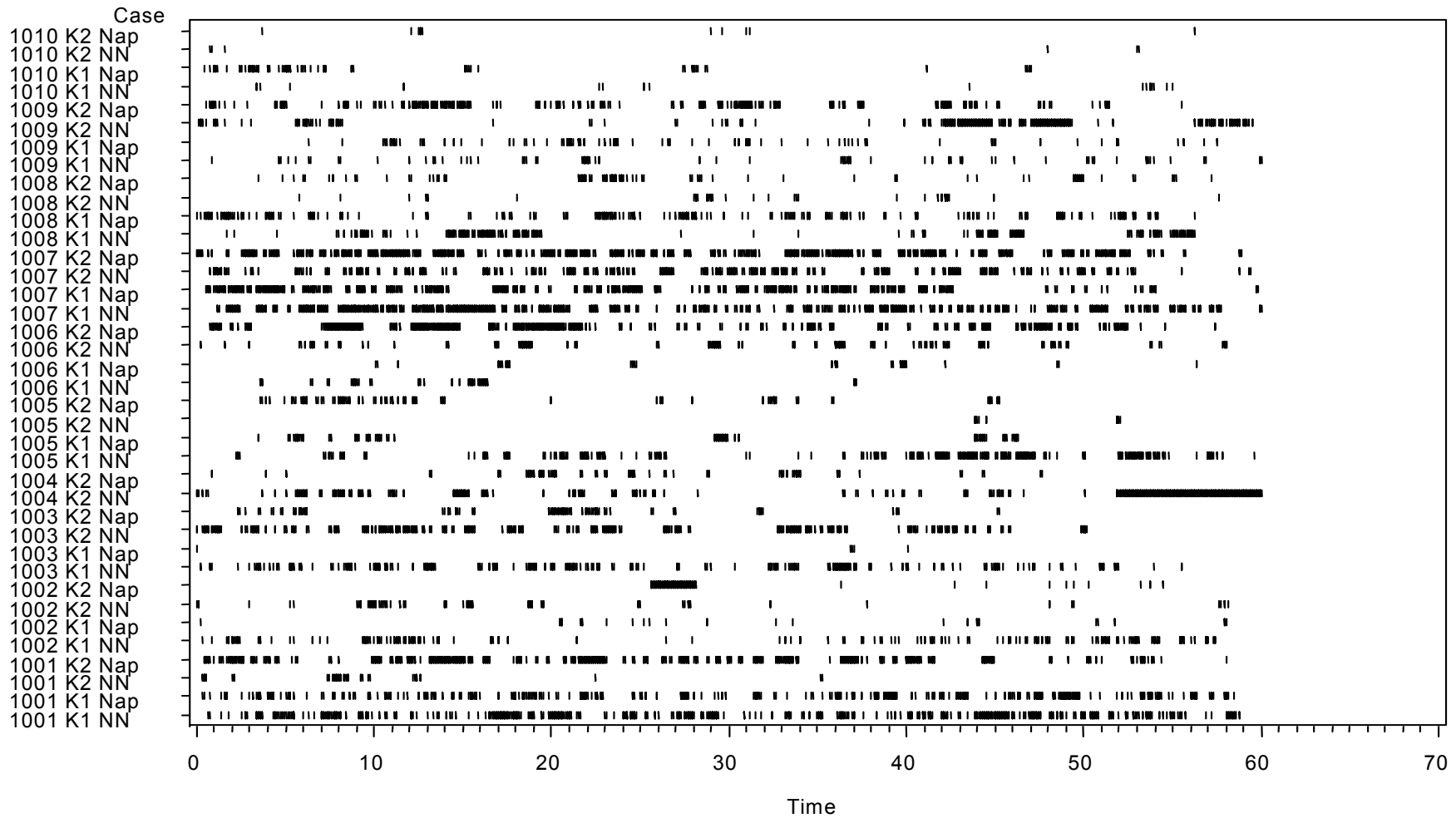


Figure 6-3: Artefact Free Data for Participants 1001-1010 During the Last Hour of Each Study Condition

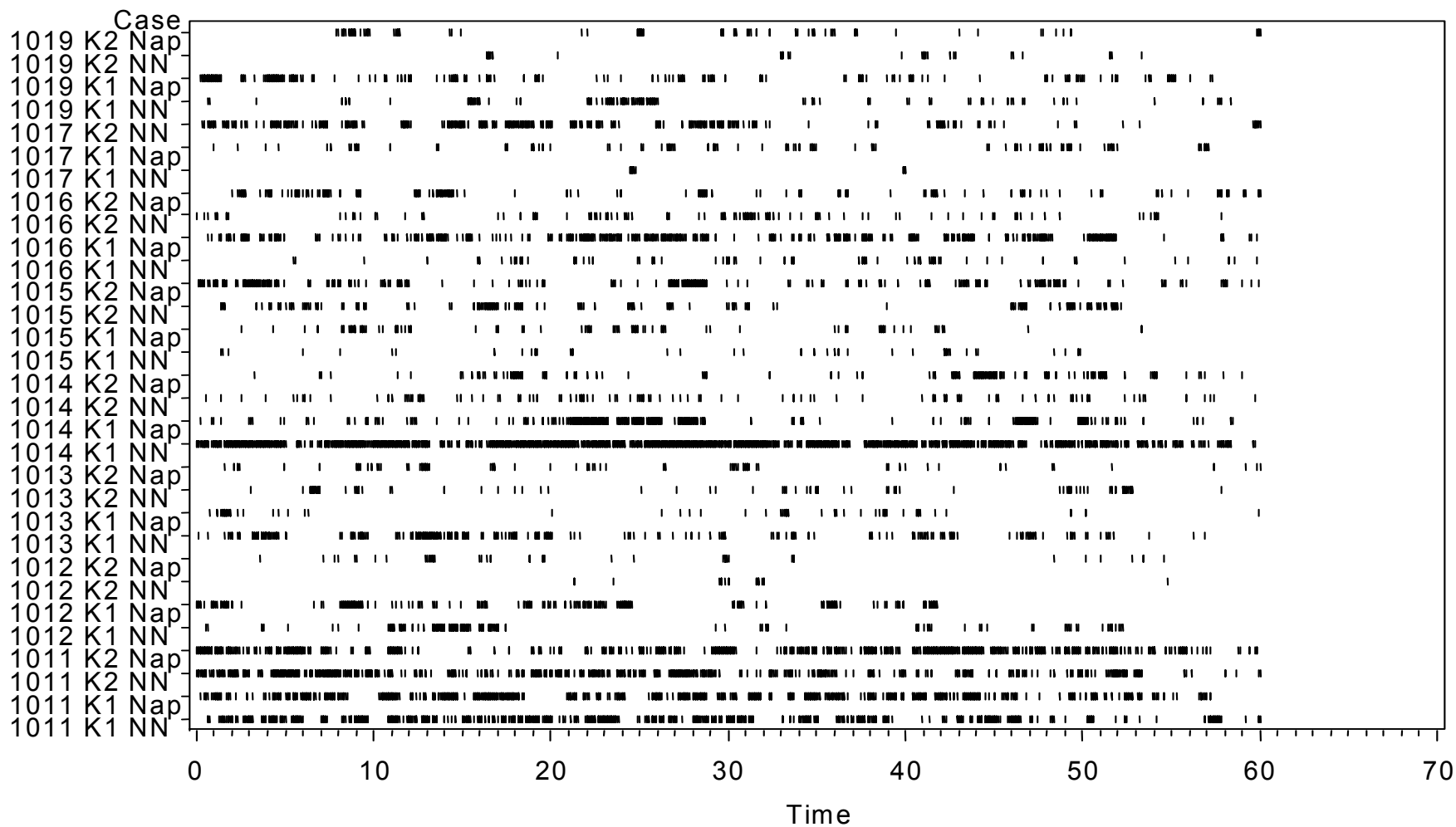


Figure 6-4: Artefact Free Data for Participants 1011-1019 During the Last Hour of Each Study Condition

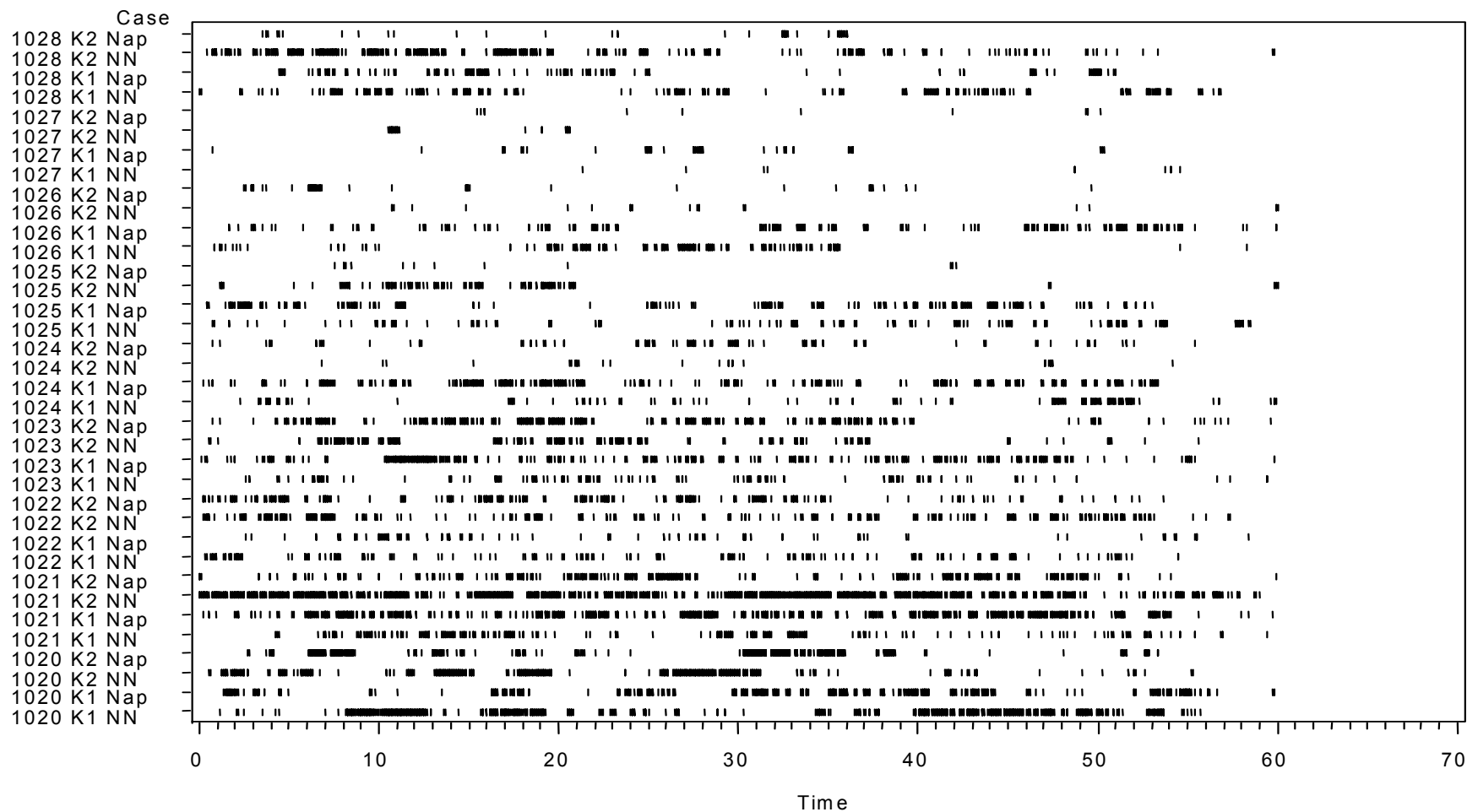


Figure 6-5: Artefact Free Data for Participants 1020-1028 During the Last Hour of Each Study Condition

Because of the large amount of data rejected due to artefact, the power in 5.12 second epochs was summed over one minute periods for subsequent analyses. Despite this there were still many individuals without complete data. Therefore, subsequent analyses for questions 2, 4, and 5, in this chapter were run with two groups of data, one set which included all individuals and a second set with data from nine individuals (participants 1107, 1009, 1011, 1013, 1014, 1016, 1020, 1021 and 1023). These nine individuals had greater amounts of data in each of the four study conditions compared to other participants, and the data were relatively evenly distributed across the 1 hour block ($M = 23\%$ of analysable EEG data).

6.4 Question 2: Effect of the Duration of Nap Sleep on Neurophysiological Alertness

6.4.1 *Analyses and Data Management*

The independent variables chosen for these analyses are listed in Table 6-1. The interaction of shift type with quantity of sleep was included in order to determine whether sleep affected alertness differently, depending on the time of night at which alertness was measured.

As with the analyses conducted in Chapter 5, two series of models were run, one with nap sleep representing the sum of all four sleep stages and then again with nap sleep calculated without the amount of stage 1 sleep that occurred.

A compound symmetric covariance structure was determined to provide the best fit between repeated measures (each study condition) for those models investigating the occurrence of SEMs, while also accounting for random variation due to individual variability. For models associated with EEG dependent variables, an autoregressive covariance structure was the best fit between each repeated EEG measure.

Table 6-1: Dependent and Independent Variables for Analyses Related to the Effect of Nap Sleep Duration on Neurophysiological Alertness

Dependent Variables	Independent Variables ³⁷
<i>SEM variables</i>	
Occurrence of SEMs	
<i>EEG variables</i>	
Delta band	Shift
Theta band	Nap sleep (plus or minus S1)
Alpha band	Sequence
Beta band	Shift x Nap sleep (plus or minus S1)
Single frequencies (2-16 Hz)	

6.4.2 Results: Influence of the Amount of Sleep on Neurophysiological Alertness.

Figure 6-6 to Figure 6-8 are examples of the change in spectral power seen in 3 study participants across each of the four study conditions³⁸. In all instances the 3 individuals slept during their napping opportunities, obtaining on average 30.6 minutes of sleep (range = 22.5-47 minutes).

The plot for participant 1007 shows two bursts of power in the alpha range at the end of the K1 shift on which some sleep was obtained. In comparison, at the end of the K1 shift without a nap numerous bursts of power in the upper theta/lower alpha range occur, albeit of a lower amplitude, which suggests that the effect of the nap may be to reduce the frequency of such events. However, the end of the K2 night shift during which a nap took place, looks similar to the plot of the K1 shift where no nap was allowed. This may indicate that the nap was less effective in limiting a decline in alertness at the end of the K2 night shift. The frequent bursts of upper theta/lower alpha power are also seen at the end of the K2 shift without a nap, although on this shift bursts of power in lower frequencies (approximately 3-4 Hz) are also observed. For this individual these changes suggest that sleepiness is first seen with increased upper

³⁷ Shift = Night shift worked (either a K1 or K2)

Nap sleep (plus or minus S1) = Polysomnographic scored sleep during the napping opportunity (either including or excluding the amount of stage 1 sleep)

Sequence = Place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

³⁸ The spectral arrays were constructed using a purpose built LabVIEW programme that plotted power in 0.195 Hz increments for each epoch of artefact free data during the last hour of the night shift.

theta/lower alpha power, followed by increased power in the upper delta range of frequencies.

The pattern of change is quite different for participant 1022. After a nap on the K1 night shift, there are numerous bursts of increased spectral power below 4 Hz, while on the same shift after no sleep, the bursts of power at the lower frequencies still occur but they extend into the lower alpha frequencies. On the K2 shifts increased power in the lower frequencies is not prominent. Instead bursts of power around 7-8 Hz are seen in the napping condition, but not the no-nap condition. In the latter conditions, one large burst in the alpha range is seen, along with several smaller bursts of power around 2-3 Hz (which are difficult to see due to the large peak in the alpha band). The changes across all four study conditions are more difficult to interpret than for the previous individual. When looking at the differences between the two K1 shifts, increased sleepiness seems to be displayed by increased power in the upper theta/lower alpha band. However, the opposite occurs in the K2 shifts, with less theta/alpha power and more delta power seen when no sleep is allowed.

The changes seen in Figure 6-8, for participant 1023 are more similar to those seen for participant 1007. However, the scale is not the same, because in order to view the data it was necessary to apply a log transform to the power values. This was due to several extremely large peaks of power. The difference between the K1 shift with and without sleep is marginal, with possibly a few more bursts of power in frequencies in the upper theta/lower alpha band without sleep. The K2 shift with a nap also has peaks at 8-10 Hz, while the K2 shift with no nap shows bursts of increased power across all frequencies plotted.

Probably the most striking aspect of the spectral arrays from these 3 individuals are the differences. There is no clear pattern of change between the four study conditions, although a greater number of bursts of increased power are seen in those conditions where individuals would be expected to be sleepier. Further, increased power is generally seen in the upper theta band/lower alpha (~7-8 Hz), and/or upper delta frequencies (~3-4 Hz).

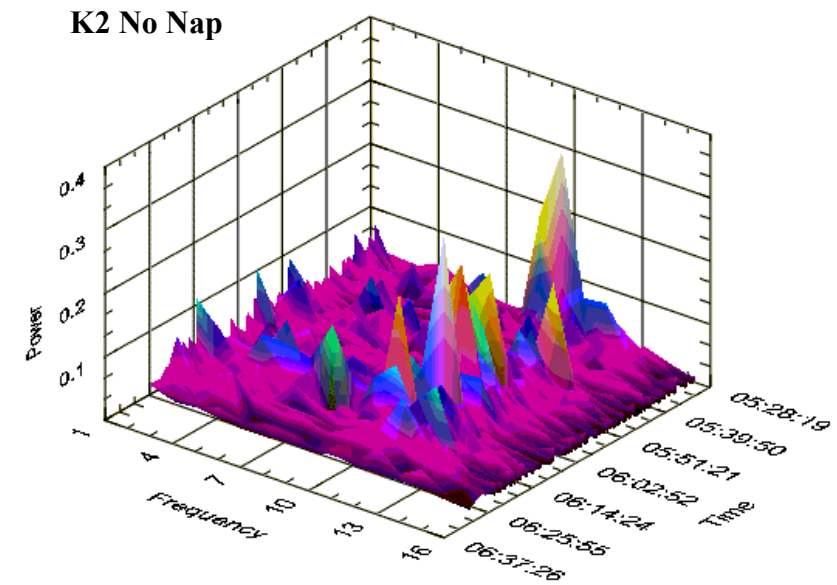
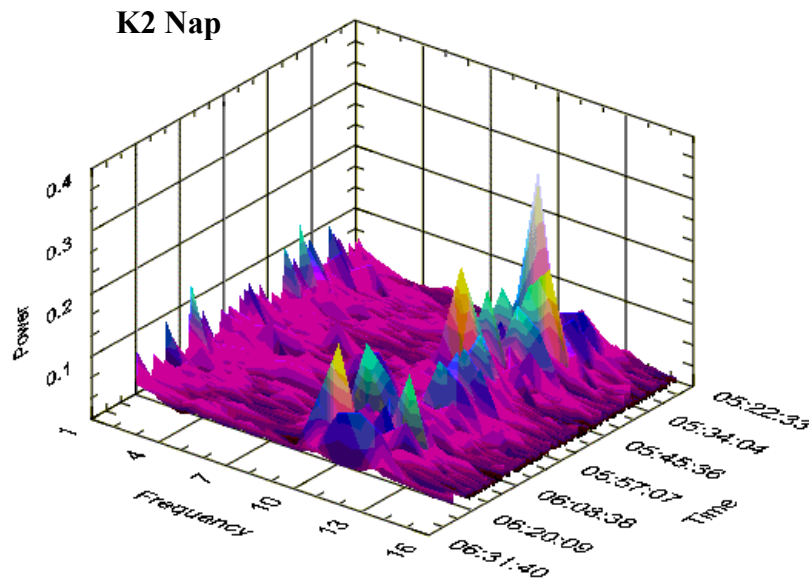
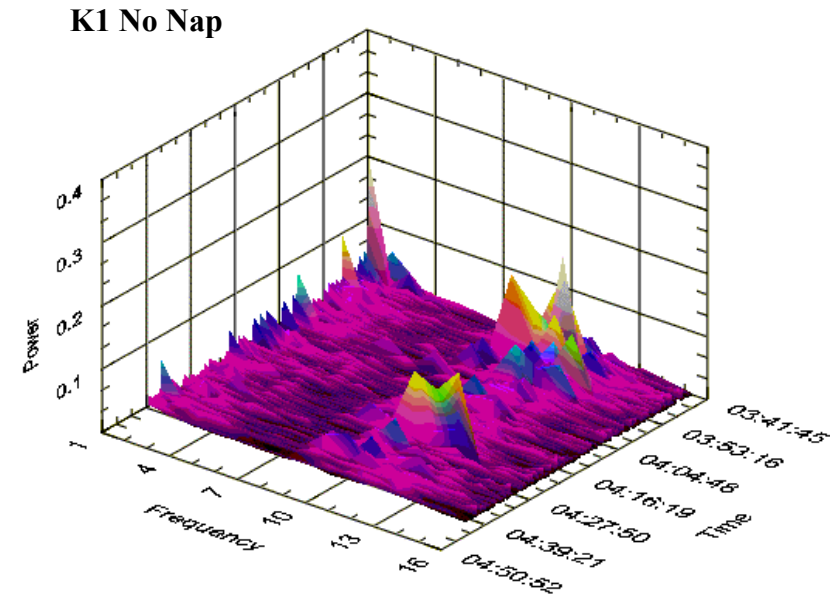
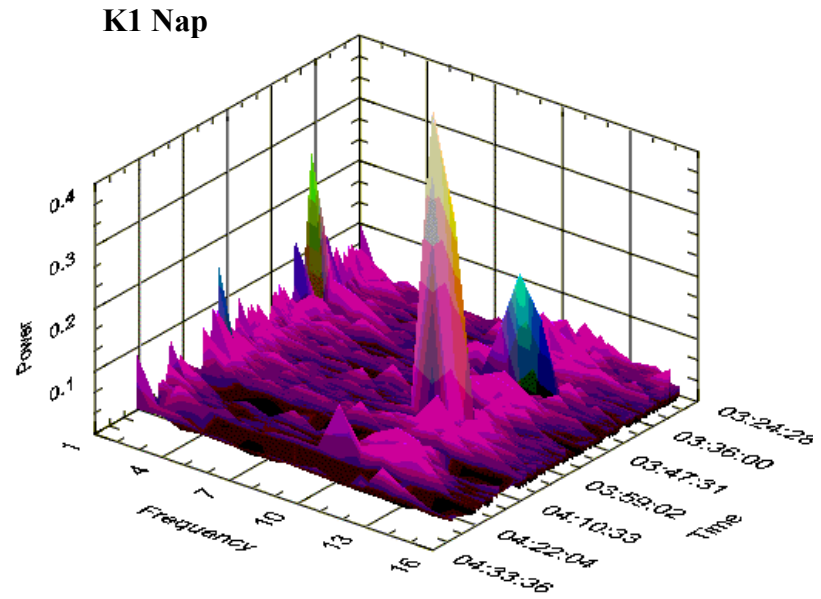


Figure 6-6: Compressed Spectral Arrays for Study Participant 1007 in Each of the Four Study Conditions

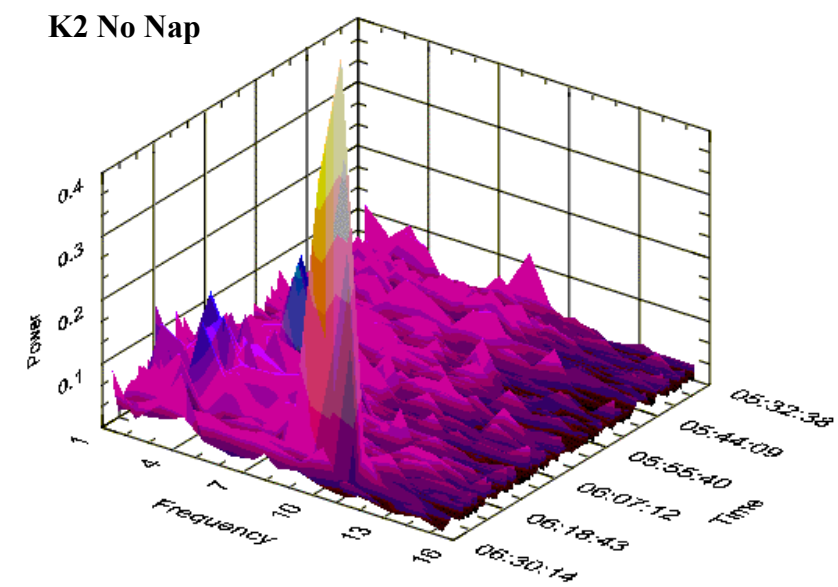
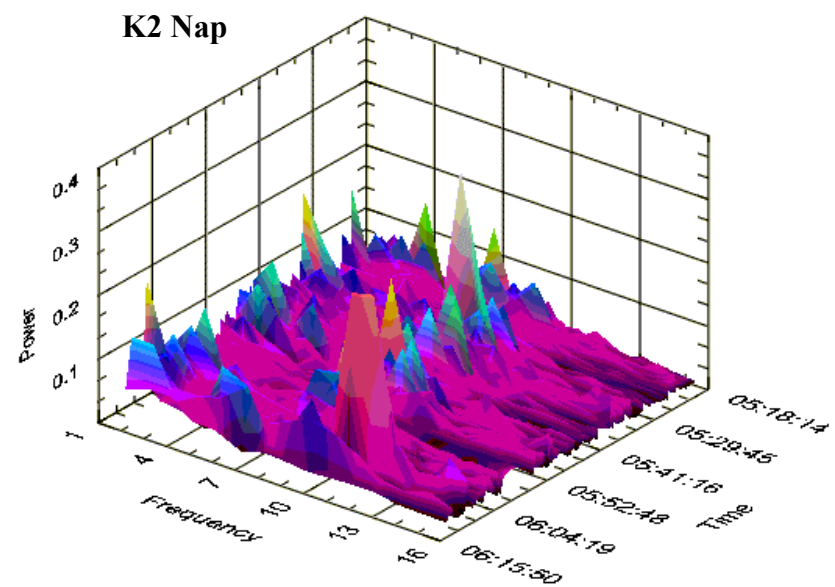
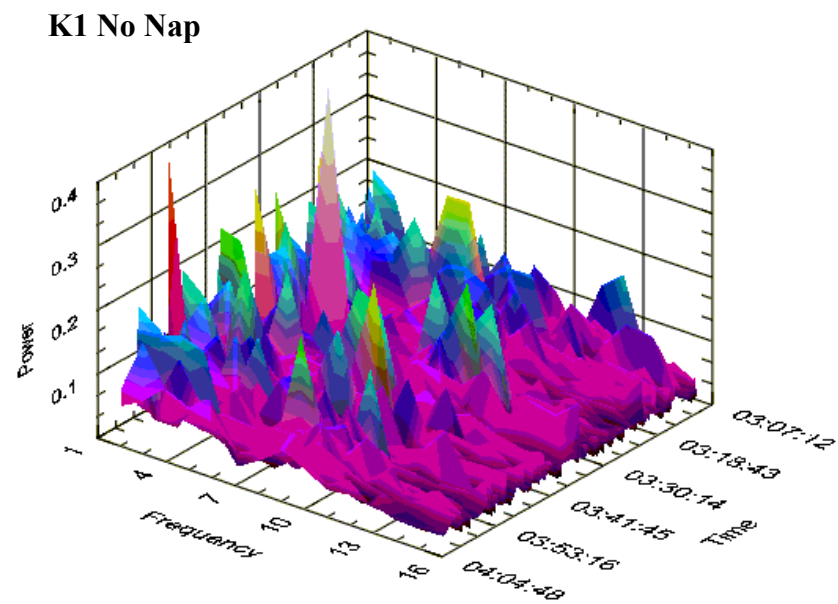
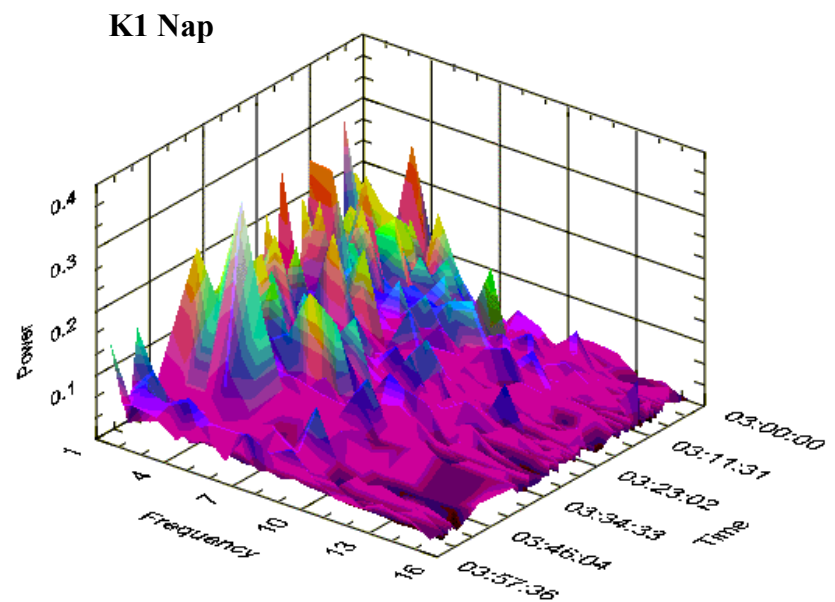


Figure 6-7: Compressed Spectral Arrays for Study Participant 1022 in Each of the Four Study Conditions

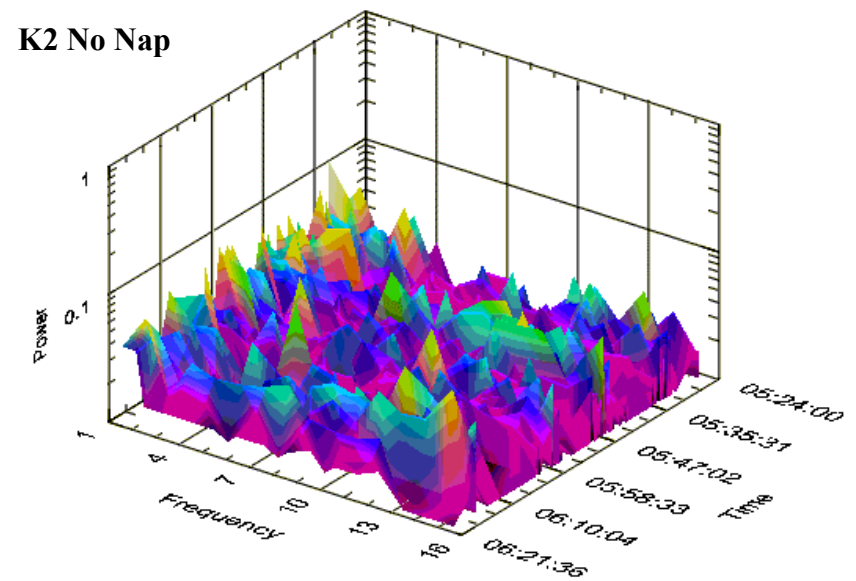
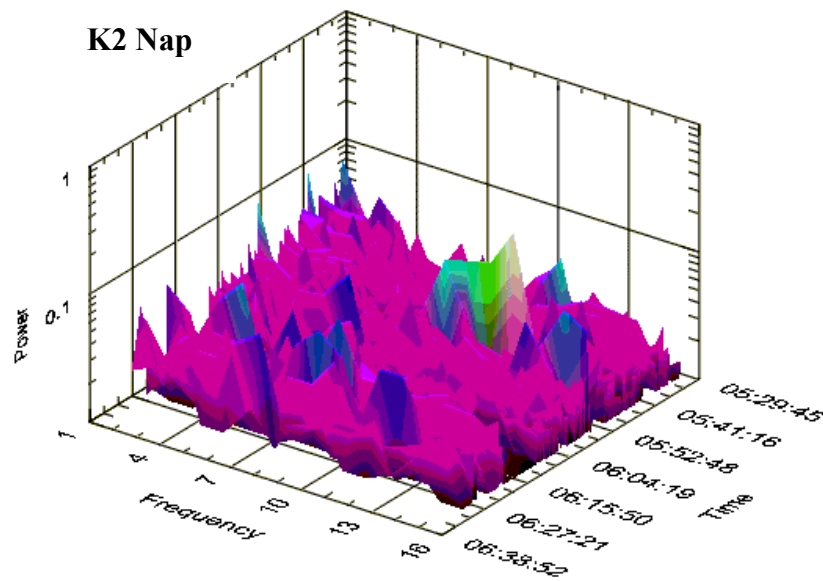
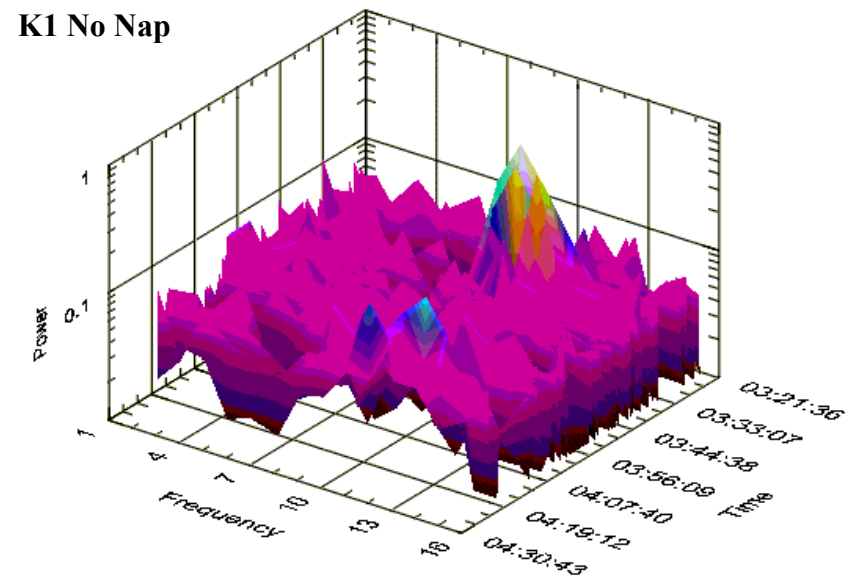
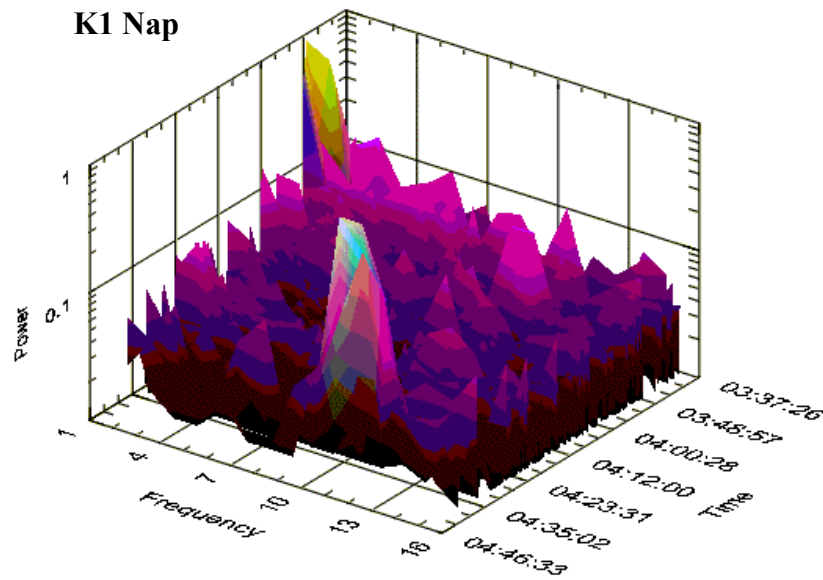


Figure 6-8: Compressed Spectral Arrays for Study Participant 1023 in Each of the Four Study Conditions

Table 6-2: Details and Results of Mixed Model ANCOVAs and Logistic Regressions for the Effect of Nap Sleep on Neurophysiological Alertness

Dependent Variable	Independent Variables ³⁹	Significant Effects for Analyses Including all Participants	Significant Effects for Analyses with Nine Participants
Occurrence of SEMs	shift, nap sleep, sequence, shift x nap sleep	Nap sleep $F_{(1, 72)} = 5.67, p = .020$	
Delta power	As above	Shift $F_{(1, 2757)} = 76.37, p < .001$ Nap sleep $F_{(1, 3071)} = 19.96, p < .001$ Sequence $F_{(3, 2930)} = 10.44, p < .001$	Shift $F_{(1, 703)} = 106.71, p < .001$ Nap sleep $F_{(1, 769)} = 125.21, p < .001$ Sequence $F_{(3, 879)} = 30.23, p < .001$ Shift*Nap sleep $F_{(1, 686)} = 22.01, p < .001$
Theta power	As above	Shift $F_{(1, 2692)} = 92.35, p < .001$ Nap sleep $F_{(1, 2982)} = 36.95, p < .001$ Sequence $F_{(3, 2841)} = 30.58, p < .001$	Shift $F_{(1, 751)} = 68.26, p < .001$ Nap sleep $F_{(1, 815)} = 124.11, p < .001$ Sequence $F_{(3, 916)} = 45.01, p < .001$ Shift $F_{(1, 760)} = 78.39, p < .001$
Alpha power	As above	Shift $F_{(1, 2896)} = 64.91, p < .001$ Nap sleep $F_{(1, 3153)} = 54.31, p < .001$ Sequence $F_{(3, 3038)} = 22.12, p < .001$	Nap sleep $F_{(1, 825)} = 117.91, p < .001$ Sequence $F_{(3, 928)} = 53.94, p < .001$ Shift*Nap sleep $F_{(1, 749)} = 14.90, p < .001$
Beta power	As above	Shift $F_{(1, 2818)} = 69.72, p < .001$ Nap sleep $F_{(1, 2991)} = 70.45, p < .001$ Sequence $F_{(3, 2925)} = 16.81, p < .001$	Shift $F_{(1, 766)} = 79.68, p < .001$ Nap sleep $F_{(1, 827)} = 132.49, p < .001$ Sequence $F_{(3, 916)} = 66.01, p < .001$ Shift*Nap sleep $F_{(1, 768)} = 8.61, p = .003$

³⁹ Shift = Night shift worked (either a K1 or K2)

Test number = Number of performance test (either 2nd or 3rd)

Nap sleep (plus or minus S1) = Polysomnographic scored sleep during the napping opportunity (either including or excluding the amount of stage 1 sleep)

Sequence = Place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

SEMs: All Participants

Findings from the logistic regression where the occurrence of SEMs was the dependent variable indicate that the amount of sleep obtained during the napping opportunity significantly alters the occurrence of SEMs. Regression estimates indicate that greater quantities of sleep decrease the likelihood of SEMs occurring.

Because the variable “occurrence of SEMs” is dichotomous, it was not possible to determine how much sleep was required to minimise the occurrence of SEMs. In an effort to do this a further series of analyses were undertaken with a sub-group of data that included only those individuals who showed SEMs. By doing this the independent variables percentage of time spent in SEMs, the total number of SEM events, and the length of the longest SEM event could be modelled as continuous independent variables. The mixed models had identical dependent variables to those used for investigating the occurrence of SEMs, but they were ANCOVAs rather than logistic regressions. There were no significant relationships found between the dependent variables and percentage of time spent in SEMs, the total number of SEM events, or the length of the longest SEM event.

EEG: All Participants, Standard Frequency Bands

As can be seen in Table 6-2 the total amount of sleep obtained, as well as the night shift worked, were significant predictors for each frequency band tested. The slope of the regression estimates indicated that power in all bands declined with greater amounts of sleep during the napping opportunity. Least-square means also suggest that higher spectral power occurs in all frequency bands at the end of the K2 night compared to the K1 shift.

The night shift number also reached significance. Post hoc tests for each power band showed that the 2nd study night was significantly different from all other nights (see Table 6-9 at the end of the chapter for the findings). The direction of the difference was the same in all instances, with lower power seen on the 2nd study night compared to all other nights. In only one other instance was an additional significant post hoc test found, and that was in the analysis where theta power was the dependent variable. In this case spectral power on the 1st study night was significantly lower than the 3rd study night.

Analyses using data from all participants were repeated limiting nap sleep time to time spent in stages 2-4. The findings were identical, with the exception of those associated with theta power, which indicated a significant shift by nap sleep interaction ($F_{(1, 2871)} = 4.22, p < .040$).

EEG: Nine Participants, Standard Frequency Bands

Because of the large amount of EEG data excluded due to artefact, all analyses were re-run with the sub-group of nine individuals who had the most complete, and evenly spread, data across the four study conditions. The findings associated with the analyses that included nap sleep (all stages) are presented in Table 6-2. It can be seen that the factors that reached significance are the same as those in the analyses run with the total group of study participants. In addition, the shift type by sleep quantity interaction reached significance for all frequencies other than theta power. The effects that were significant remained unchanged when only time spent in sleep stages 2-4 was considered.

EEG: All Participants, Single Frequencies

Mixed model analyses were completed for each frequency between 2 and 16 Hz. Because of the large number of statistics generated and the similarity of the findings to those of the four frequency bands, the statistical details are presented at the end of this chapter (Table 6-10). Analyses using the data from all study participants and where nap sleep (all stages) was included, show identical findings to those for the four frequency bands calculated using the same parameters. For every frequency the amount of sleep obtained during the nap, the shift type worked, and the night shift number reached significance. Greater amounts of sleep during the nap resulted in lower spectral power in each frequency and lower spectral power was also seen in the last hour of the K1 night shift compared to the K2 shift. The post hoc tests indicated that the 2nd study night had lower spectral power than all other study nights across all frequencies. For frequencies between and including 5-8 Hz, the 1st study night also resulted in lower spectral power than the 3rd study night.

When nap sleep (all stages) was replaced with nap sleep (stages 2-4) the large majority of findings remained the same for the single frequencies. The only exception was that the interaction of nap sleep amount with shift type reached significance for 4-6, 13, and 16 Hz.

EEG: Nine Participants, Single Frequencies

Analyses for each single frequency were also repeated using data from the sub-group of nine individuals. The findings for analyses completed with nap sleep (all stages) can be seen in Table 6-10 at the end of this chapter. The amount of sleep obtained during the nap, the night shift worked and the interaction of these terms were again significant for all frequencies. The only exception was at 5 and 6 Hz, where the interaction of nap sleep length and night shift were not significant. The night number was again significant for all frequencies, with the 2nd study night consistently resulting in lower spectral power than all other study nights. These analyses were repeated with nap sleep (stages 2-4) and the findings matched those seen with nap sleep (all stages). The only difference was that for frequencies from 4 to 7 Hz the interaction of nap sleep amount and the night shift worked did not reach significance.

6.5 Question 3: The Influence of Sleep Architecture on Neurophysiological Alertness

6.5.1 *Analyses and Data Management*

The 53 night shifts where naps occurred were included in this data set, which excludes the one napping opportunity with a corrupt EEG/EOG file. The variables included in these models are outlined in Table 6-3. The basis for including these particular variables in this set of analyses has been previously outlined in Chapter 5.

All the models where the occurrence of SEMs was the dependent variable were run twice, once with nap sleep calculated as the sum of all four sleep stages, and then again minus stage 1 sleep. In addition, the values for prior wakefulness are the same as those for the performance data (the length of time between the end of sleep during the nap and the beginning of the 2nd performance test). For naps that included only stage 1 sleep, this was calculated from the end of the last sleep prior to beginning the night shift to the start of the second neurophysiological recording of the night.

Due to issues of collinearity fewer independent variables were included in the models associated with EEG power. The final dependent and independent variables used in the models are listed in Table 6-3. Nap sleep duration and the number of ASDA arousals indexed across an hour were continuous variables. The occurrence of stage 3 sleep and whether individuals woke from SWS or not were both dichotomous variables.

Mixed model ANCOVAs were employed for investigating the relationship between EEG power and sleep architecture variables, with an autoregressive structure providing the best fit between repeated EEG measures. A mixed model logistic regression was utilised for predicting the presence or absence of SEMs, and a compound symmetric covariance structure was modelled between repeated measures.

Table 6-3: Dependent and Independent Variables for Analyses Related to the Effects of Nap Sleep Architecture on Neurophysiological Alertness

Dependent Variables	Independent Variables ⁴⁰
<i>SEM variables</i>	
Occurrence of SEMs	Shift
	Nap sleep (plus or minus S1)*
<i>EEG variables</i>	ASDA arousals
Delta power	S3 sleep
Theta power	Sleep stage woken from*
Alpha power	Prior wake (s1 sleep considered or not)*
Beta power	Sequence

* Variables excluded from analyses with EEG dependent variables due to collinearity.

6.5.2 Results: Architectural Aspects of Sleep and Neurophysiological Alertness

SEMs: All Participants

The findings of the Logistic Regression and ANCOVAs can be seen in Table 6-4, and indicate no significant effects for the model in which the occurrence of SEMs was the dependent variable.

EEG: All Participants, Standard Frequency Bands

The models where frequency bands were the dependent variables produced relatively consistent findings, but few significant effects for the sleep architecture variables. Across all frequency bands, lower spectral power was again seen on the K1 night shift. In

⁴⁰ Shift = night shift worked (either a K1 or K2)

Nap sleep = Polysomnographic scored sleep during the napping opportunity (either including or excluding the amount of stage 1 sleep)

ASDA arousals = Number of ASDA arousals per hour in the nap sleep

S3 sleep = Whether stage 3 sleep was obtained (entered or not) during the nap

Sleep stage woken from = Woke from SWS or not

Prior wake = Period of time from end of napping opportunity (or previous sleep if no nap sleep was obtained) to start of 2nd half of the night, or the 1 hour block of EEG data (either including or excluding stage 1 sleep)

Sequence = Place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

addition, the sequence in which study nights were completed was consistently a significant variable. Generally the 2nd study night resulted in lower spectral power than all other study nights, and the 4th study night had lower power than the 1st and 3rd study night. In most instances the power on nights 1 and 3 did not differ, except for the beta power band where the 3rd study night produced lower power than the 1st study night. The results of the significant post hoc tests can be seen in Table 6-11.

The occurrence of stage 3 sleep significantly predicted beta power, with least-square means indicating that those who obtain stage 3 sleep during the nap opportunity had lower levels of beta power in the last hour of the night shift.

EEG: All Participants, Single Frequencies

The findings of the analyses for the single frequencies are reported in Table 6-12 and show that the night shift worked and study night number are again consistent significant effects for most frequencies. At 3, 12, and 16 Hz the type of night shift worked did not reach significance, but for all other frequencies the last hour of the K1 night shift was associated with lower spectral power than the K2 night shift.

In all instances, the post hoc tests for the night number showed lower power across all frequencies on the 2nd study night. For all frequencies, other than 12, 14, and 16 Hz, the 4th study night also resulted in lower power than either the 1st or 3rd study night. The 1st and 3rd study did not differ significantly from each other.

At 3 and 12 Hz the number of ASDA arousals per hour scored during the nap sleep significantly influenced power values. The regression estimates indicated that a greater number of arousals (or poorer quality sleep) resulted in increased power at these frequencies. At 4 and 6 Hz the occurrence of stage 3 sleep was a significant effect, and least-square mean values implied that the occurrence of stage 3 during the nap lead to greater power at 4 Hz, while at 6 Hz the reverse effect was seen, with stage 3 sleep during the nap resulting in lower spectral power.

Table 6-4: Details and Results of Logistic Regression and ANCOVAs Investigating the Effect of Architectural Aspects of Sleep on Neurophysiological Alertness

Dependent Variable	Independent Variables ⁴¹	Significant Main Effects
Occurrence of SEMs	shift, nap sleep*, ASDA arousals, S3 sleep, stage woken from*, prior wake*, sequence	
Delta power	As above	Shift $F_{(1, 1157)} = 13.92, p < .003$ Sequence $F_{(3, 1163)} = 51.28, p < .001$
Theta power	As above	Shift $F_{(1, 1223)} = 32.20, p < .001$ Sequence $F_{(3, 1224)} = 88.21, p < .001$
Alpha power	As above	Shift $F_{(1, 1216)} = 10.04, p = .002$ Sequence $F_{(3, 1219)} = 78.38, p < .001$
Beta power	As above	Shift $F_{(1, 1227)} = 17.61, p < .001$ S3 sleep $F_{(1, 1263)} = 3.97, p = .047$ Sequence $F_{(3, 1228)} = 94.68, p < .001$

* Variables excluded from analyses with EEG dependent variables due to collinearity.

⁴¹ Shift = night shift worked (either a K1 or K2)

Nap sleep = Polysomnographic scored sleep during the napping opportunity (either including or excluding the amount of stage 1 sleep)

ASDA arousals = Number of ASDA arousals per hour in the nap sleep

S3 sleep = Whether stage 3 sleep was obtained (entered or not) during nap

Sleep stage woken from = Woke from SWS or not

Prior wake = Period of time from end of napping opportunity (or previous sleep if no nap sleep was obtained) to start of 2nd half of the night, or the 1 hour block of EEG data (inc or exc S1 sleep)

Sequence = Place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

6.6 Question 4: The Effect of a Nap Opportunity on Neurophysiological Alertness Independent of the Architecture and Quantity of Sleep Obtained

6.6.1 Analyses and Data Management

As in Chapter 5, this question was included because an individual cannot predict if, or how long, he/she will sleep in a given nap opportunity. Only the independent variables that defined the design of the experiment, and whether a napping opportunity was provided or not were included in the model. A list of these variables, with the respective interaction effects are listed Table 6-5.

A series of mixed model ANOVA's and Logistic Regressions were undertaken. In the Logistic Regression, a compound symmetric covariance structure modeled the relationship between measures taken repeatedly on the four study nights. In the ANOVAs associated with the EEG dependent variables, an autoregressive structure provided the best fit between the repeated EEG measures.

Table 6-5: Dependent and Independent Variables for Analyses Related to the Effects of the Napping Opportunity on Neurophysiological Alertness Independent of the Architecture and Quantity of Sleep Obtained

Dependent Variables	Independent Variables ⁴²
<i>SEM variables</i>	
Occurrence of SEMs	
<i>EEG variables</i>	
Delta power	Shift
Theta power	Nap
Alpha power	Sequence
Beta power	Shift x Nap
Single frequencies (2-16 Hz)	

⁴² Shift = Night shift worked (either a K1 or K2)

Nap = Whether a napping opportunity was provided or not.

Sequence = Place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

6.6.2 Results: Napping Opportunity and Neurophysiological Alertness

SEMs: All Participants

Figure 6-9 displays the percentage of time across each half of the night shift, under each study condition, that individuals were scored as having SEMs. A logistic regression was run to determine if the occurrence of SEMs in the 2nd half of the night shift was influenced by the napping opportunity. The findings listed in Table 6-6 show the opportunity to nap was the only variable to reach significance, and estimates of least-square means indicated the nap opportunity decreased the likelihood of SEMs occurring.

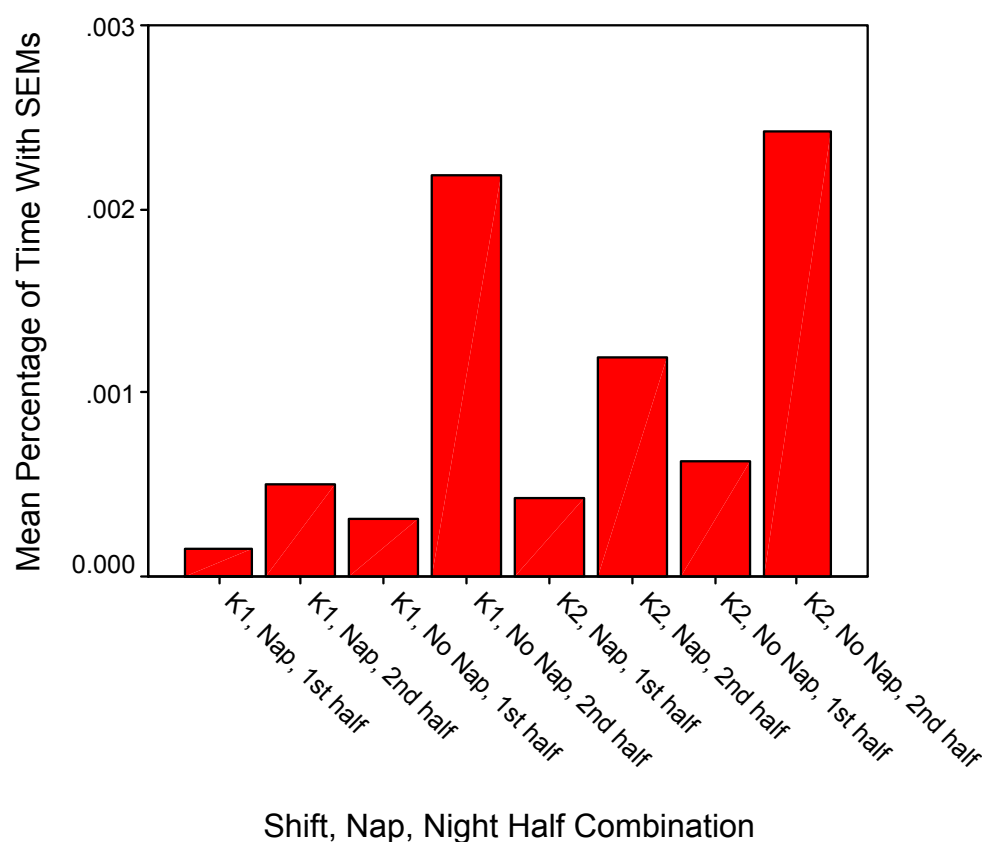


Figure 6-9: Percentage of Time Scored with SEMs During Each Study Condition

EEG: All Participants, Standard Frequency Bands

The results of the mixed models ANCOVA's investigating the effect of the nap opportunity on spectral power in each frequency band can be seen in Table 6-6. In all instances, the nap opportunity and shift type worked were significant, with the nap opportunity and the K1 (early) night shift resulting in lower spectral power across all

frequencies. Further, the interaction of the night shift worked with the nap opportunity was significant for all power bands. The significant post hoc tests are listed in Table 6-13, which generally show that the K2 no nap condition results in greater spectral power compared to all other study conditions. A further consistent finding is that the K1 no nap condition results in lower spectral power than the K2 nap condition.

As in the analyses for Question 2, the night number was also consistently significant. The differences of the least-square means for theta and alpha power indicated that the 2nd study night again produced lower spectral power compared to all other nights. Details of the post hoc analyses are listed in Table 6-14. For delta power the 1st night differed significantly from both nights 2 and 3, while for beta power night 1 differed only from night 2.

EEG: Nine Participants, Standard Frequency Bands

These analyses were repeated with the smaller group of nine individuals. The significant findings, and the post hoc analyses, produced the same results as those from all the study participants.

EEG: All Participants, Single Frequencies

The findings for the single frequencies were similar to those calculated for the four frequency bands. The night shift worked and study night number were significant for all frequencies. The opportunity to nap reached significance for all frequencies other than 7,8, and 13-16 Hz, while the interaction of the nap opportunity with the night shift worked did not reach significance at 5 Hz. The direction of the differences were consistent, with the nap opportunity (where significant) and the K1 night shift resulting in lower power compared to not napping and the K2 night shift respectively. Details of these findings can be found in at the end of this chapter.

The interaction of the napping opportunity with the night shift worked, for each of the single frequencies, produced similar findings to those seen in the four frequency bands. For all frequencies, other than 16 Hz, higher spectral power was seen on the K2 shift when no nap was allowed compared to all other study conditions. At 4-6, 8, and 13 Hz the K1 nap condition produced lower spectral power than the K2 nap condition, and as was seen in the four frequency bands, the K1 no nap condition resulted in lower spectral power than the K2 nap condition. These latter post hoc tests were significant for 2, 4, 5, 7-9, and 11-15 Hz.

The reliable finding that spectral power was lower on the 2nd study night compared to all other study night was again seen in the post hoc tests for each frequency.

EEG: Nine Participants, Single Frequencies

As was undertaken for the four frequency bands, the analyses for each single frequency were repeated using data from the nine individuals with a greater amount of EEG data. Findings were relatively consistent with those calculated using all study participants. For each frequency, the opportunity to nap, type of night shift worked, study night number, and the interaction of napping opportunity with night shift were significant. The only exceptions were 13-16 Hz. At these frequencies, the opportunity to nap was not a significant effect. Post hoc tests generally reflected those seen in the larger group of participants.

Table 6-6: Details and Results of Mixed Model ANOVAs and Logistic Regressions for the Effect of the Napping Opportunity on Neurophysiological Alertness

Dependent Variable	Independent Variables ⁴³	Significant Main Effects	Significant Interaction Effects
Occurrence of SEMs	shift, nap, sequence, shift x nap,	Nap $F_{(1, 26)} = 6.43, p = .018$	
Delta power	As above	Shift $F_{(1, 515)} = 44.78, p < .001$ Nap $F_{(1, 506)} = 5.50, p = .019$ Sequence $F_{(3, 509)} = 4.79, p = .003$	Shift by Nap $F_{(1, 506)} = 14.44, p < .001$
Theta power	As above	Shift $F_{(1, 464)} = 45.86, p < .001$ Nap $F_{(1, 455)} = 4.92, p = .027$ Sequence $F_{(3, 457)} = 5.46, p = .001$	Shift by Nap $F_{(1, 457)} = 7.05, p = .008$
Alpha power	As above	Shift $F_{(1, 497)} = 33.67, p < .001$ Nap $F_{(1, 488)} = 5.60, p = .018$ Sequence $F_{(3, 491)} = 6.34, p < .001$	Shift by Nap $F_{(1, 490)} = 12.05, p < .001$
Beta power	As above	Shift $F_{(1, 377)} = 33.82, p < .001$ Nap $F_{(1, 370)} = 8.00, p = .005$ Sequence $F_{(3, 373)} = 3.98, p = .008$	Shift by Nap $F_{(1, 372)} = 13.44, p < .001$

⁴³ Shift = Night shift worked (either a K1 or K2)

Nap = Whether a napping opportunity was provided or not.

Sequence = Place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

6.7 Question 5: Influence of Prior Sleep, Circadian Phase, and Nap Sleep on Neurophysiological Alertness

6.7.1 Analyses and Data Management

Several factors that would theoretically be expected to influence an individuals' level of neurophysiological alertness were included in this last series of models. Variables used in earlier models were included along with the level of acute sleep loss and cumulative sleep debt experienced. The various dependent and independent variables are listed in Table 6-7.

As with previous models, a compound symmetric covariance structure was stipulated between repeated measures in the model for the occurrence of SEMs, while an autoregressive structure provided the best between repeated EEG measures.

Table 6-7: Dependent and Independent Variables for Analyses Related to the Influence of Prior Sleep, Circadian Phase, and Nap Sleep on Neurophysiological Alertness

Dependent Variables	Independent Variables ⁴⁴
<i>SEM variables</i>	
Occurrence of SEMs	
<i>EEG variables</i>	Shift
	Nap sleep
	Sequence
Delta power	Acute sleep loss
Theta power	Cumulative debt
Alpha power	
Beta power	

6.7.2 Results: Factors that Determine Neurophysiological Alertness

SEMs: All Participants

The independent variables, for which significant effects were found, are listed in Table 6-8. The amount of sleep obtained during the nap opportunity had a significant

⁴⁴ Shift = night shift worked (either a K1 or K2)

Nap sleep = Polysomnographic scored sleep during the napping opportunity (either including or excluding the amount of stage 1 sleep)

Sequence = Place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

Acute sleep loss = acute sleep loss in the 24 hours prior to the start of the 1 hour EEG block

Cumulative debt = level of cumulative sleep loss at midday prior to commencing the night shift

influence in almost all instances, including the occurrence of SEMs. Where the amount of sleep obtained had a significant effect, the solution estimate indicated a relationship in the expected direction, with greater amounts of sleep reducing the occurrence of SEMs.

EEG: All Participants, Standard Frequency Bands

Greater amounts of sleep also related to lower power in the four frequency bands of interest. In addition, the level of acute sleep loss was a significant effect in each of the models for spectral power. In all instances the regression solution estimates indicated that greater levels of acute sleep loss related to higher levels of spectral power.

The sequence in which the study nights were completed was again a significant effect. Details of the post hoc tests (for the models including nap sleep (all stages) and all participants) are listed at the end of the chapter in Table 6-16. Where the post hoc tests reached significance, it was the 2nd study night that differed from all other nights, having lower spectral power.

In the models where nap sleep (all stages) was substituted with nap sleep (stages 2-4) the significant effects remained almost identical. The only difference was in the models for delta and theta power, in which cumulative sleep loss was also significant (delta = $F_{(1, 2938)} = 27.73, p < .001$, theta = $F_{(1, 2846)} = 14.79, p < .001$). The regression estimate indicated that greater cumulative sleep debt led to higher delta and theta power.

EEG: Nine Participants, Standard Frequency Bands

Table 6-8 also displays the significant findings for models that were run using data from the nine individuals who had more complete EEG data. They indicate very similar findings to those seen with the entire group of study participants, except that the amount of sleep obtained during the nap opportunity did not reach significance for the alpha and beta bands. Where effects are significant, the direction of the relationships are the same as those for the larger group of participants.

Using the data from the smaller number of participants, models were also re-run after substituting nap sleep (stages 2-4) for nap sleep (all stages). Again the findings generally matched those of the larger group, but for all frequency bands increased cumulative sleep debt was associated with increased spectral power (delta = $F_{(1, 508)} = 12.28, p < .001$, theta = $F_{(1, 525)} = 16.14, p < .001$, alpha = $F_{(1, 537)} = 12.75, p < .001$, beta = $F_{(1, 534)} = 20.60, p < .001$).

EEG: All Participants, Single Frequencies

The findings for the analyses for single frequencies calculated with all participants and using nap sleep (all stages) can be seen in at the end of this chapter. The results indicate that the amount of sleep obtained during the nap has a significant effect for all frequencies other than 7, 15, and 16 Hz. The regression estimates suggest that greater amounts of sleep lead to lower spectral power. The level of acute sleep loss was significantly related to spectral power for all frequencies other than 15 Hz. In all instances greater amounts of acute sleep loss produced higher spectral power. At 5 Hz and 6 Hz the level of cumulative sleep loss also reached significance, and as expected greater levels of cumulative sleep loss result in greater spectral power. The study night number was, in each instance, highly significant and post hoc tests indicated the 2nd study night had lower power than all other study nights.

When the analyses for single frequencies were re-calculated using nap sleep (stages 2-4) in place of nap sleep (all stages) the findings were very similar. The main differences were that nap sleep duration was significant for all frequencies, and that cumulative sleep debt was a significant effect at 3-5 Hz. Study night 2 was again consistently different from other study nights, having lower spectral power.

EEG: Nine Participants, Single Frequencies

The analyses that included nap sleep (all stages) were repeated with data from the nine individuals with the highest proportion of EEG data. The findings closely matched those of the larger group under the same conditions. However, nap sleep (all stages) did not show a significant effect for a greater number of frequencies (7-9, and 12-16 Hz), and cumulative sleep debt was significant for 11 Hz, as well as 5 and 6 Hz. This series of analyses were re-calculated using nap sleep (stages 2-4) in place of nap sleep (all stages) and the findings were identical to those using nap sleep (all stages) and the smaller group of participants.

Table 6-8: Details and Results of Mixed Model ANCOVAs and Logistic Regressions for Analyses Related to the Influence of Prior Sleep, Circadian Phase, and Nap Sleep on Neurophysiological Alertness

Dependent Variable	Independent Variables ⁴⁵	Significant Effects for Analyses Including all Participants	Significant Effects for Analyses with Nine Participants
Occurrence of SEMs	shift*, nap sleep, sequence, acute sleep loss, cumulative debt	Nap sleep $F_{(1, 68)} = 4.80, p = .032$	
Delta power	As above	Nap sleep $F_{(1, 438)} = 3.97, p = .047$ Acute sleep loss $F_{(1, 474)} = 8.48, p = .004$ Sequence $F_{(3, 456)} = 4.08, p = .007$	Nap sleep $F_{(1, 188)} = 7.71, p = .006$ Acute sleep loss $F_{(1, 193)} = 26.38, p < .001$ Sequence $F_{(3, 192)} = 18.35, p < .001$
Theta power	As above	Nap sleep $F_{(1, 408)} = 4.30, p = .039$ Acute sleep loss $F_{(1, 440)} = 11.85, p < .001$ Sequence $F_{(3, 424)} = 7.52, p < .001$	Nap sleep $F_{(1, 168)} = 5.09, p = .025$ Acute sleep loss $F_{(1, 172)} = 32.27, p < .001$ Sequence $F_{(3, 171)} = 21.50, p < .001$
Alpha power	As above	Nap sleep $F_{(1, 450)} = 10.39, p = .001$ Acute sleep loss $F_{(1, 487)} = 12.61, p < .001$ Sequence $F_{(3, 468)} = 7.63, p < .001$	Acute sleep loss $F_{(1, 179)} = 37.15, p < .001$ Sequence $F_{(3, 179)} = 18.69, p < .001$
Beta power	As above	Nap sleep $F_{(1, 337)} = 16.87, p < .001$ Acute sleep loss $F_{(1, 366)} = 5.63, p = .018$ Sequence $F_{(3, 349)} = 3.79, p = .011$	Acute sleep loss $F_{(1, 161)} = 29.46, p < .001$ Sequence $F_{(3, 160)} = 19.66, p < .001$

* Due to collinearity the variable for night shift type was removed from models with EEG dependent variables

⁴⁵ Shift = night shift worked (either a K1 or K2)

Nap sleep = Polysomnographic scored sleep during the napping opportunity (either including or excluding the amount of stage 1 sleep)

Sequence = Place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

Acute debt = acute sleep loss in the 24 hours prior to the start of the 1 hour EEG block

Cumulative debt = level of cumulative sleep loss at midday prior to commencing the night shift

6.8 Summary

Q1. Once artefact is removed from the EEG data how much data remains to objectively assess alertness during the last hour of the night shift?

Greater amounts of EEG data than expected were excluded from further analysis due to artefact. The amount remaining for analysis varied from 0.36-67.52%, with a median of 10.74% of data determined to be artefact free.

Q2. Does the amount of sleep obtained during pre-planned workplace naps assist to improve, or limit a decline, in neurophysiological alertness in a dose-dependent manner?

Greater amounts of sleep during the night shift reduced the occurrence of SEMs, an accepted sign of decreased alertness, and resulted in lower spectral power in the delta, theta, alpha and beta frequency bands and across all single frequencies below 16 Hz. Sleep on the earlier night shift (K1) was more effective in improving alertness at the end of the night shift, than was sleep on the later night shift (K2).

Q3. Are there specific architectural aspects of sleep obtained during the pre-planned napping opportunity that influence neurophysiological alertness?

The occurrence of stage 3 sleep during the nap resulted in lower beta power, and lower power at 6 Hz, but higher power at 4 Hz, at the end of the night shift. In addition, a greater number of ASDA arousals scored during the nap opportunity were related to greater subsequent power at 3 and 12 Hz. None of the sleep architecture variables were related to the occurrence of SEMs. These findings suggest that better quality sleep (as indexed by deeper sleep and fewer arousals) can positively influence EEG signs of neurophysiological alertness.

Q4. Does the opportunity to nap assist in improving, or limiting a decline, in neurophysiological alertness independent of the amount of sleep obtained?

The opportunity to nap was found to decrease the occurrence of SEMs and result in lower spectral power across all four frequency bands and most single frequencies below 13 Hz. The end of the K2 night shift, when no nap opportunity had occurred, was found to produce the greatest EEG signs of lowered neurophysiological alertness compared to all other conditions. Operationally, these findings strongly support the recommendation for a 40 minute napping opportunity on the night shift.

Q5. Do prior sleep patterns or circadian phase influence neurophysiological alertness on the night shift?

Acute sleep loss was found to significantly affect almost all the EEG parameters representing neurophysiological alertness. Greater levels of acute sleep loss consistently resulted in increased power across the four frequency bands and single frequencies below 16 Hz. The effect of cumulative sleep debt was a less consistent finding, but in a number of instances greater levels of debt were related to increased spectral power.

Table 6-9: Results of Significant Post Hoc Comparisons Between Night Shift Number For Each Power Band in Analyses Investigating the Effect of Nap Sleep on the Power in Standard EEG Frequency Bands

Sequence	Delta		Theta		Alpha		Beta	
	<i>df</i>	<i>t</i>	<i>df</i>			<i>t</i>	<i>df</i>	<i>t</i>
Night 1 versus Night 2	2984	4.33**	2920	5.05**	3081	5.38**	2959	5.93**
Night 1 versus Night 3			2897	-3.55**				
Night 2 versus Night 3	2958	-4.76**	2882	-8.92**	3061	-7.68**	2933	-4.64**
Night 2 versus Night 4	2910	-4.43**	2844	-7.41**	3022	-5.83**	2913	

* $p < .01$

** $p < .001$

Table 6-10: Details and Results of Mixed Model ANCOVAs Investigating the Effect of Nap Sleep on the Power in Single EEG Frequencies

Dependent Variable	Independent Variables ⁴⁶	Significant Effects for Analyses Including all Participants	Significant Effects for Analyses with Nine Participants
2 Hz	shift, nap sleep, sequence, shift x nap sleep	Shift $F_{(1, 2758)} = 55.92, p < .001$ Nap sleep $F_{(1, 3086)} = 23.46, p < .001$ Sequence $F_{(3, 2934)} = 6.96, p < .001$	Shift $F_{(1, 640)} = 67.70, p < .001$ Nap sleep $F_{(1, 705)} = 117.18, p < .001$ Sequence $F_{(3, 818)} = 21.01, p < .001$ Shift*Nap sleep $F_{(1, 621)} = 22.13, p < .001$
3 Hz	As above	Shift $F_{(1, 2661)} = 50.07, p < .001$ Nap sleep $F_{(1, 3062)} = 29.46, p < .001$ Sequence $F_{(3, 2864)} = 13.13, p < .001$	Shift $F_{(1, 624)} = 75.07, p < .001$ Nap sleep $F_{(1, 684)} = 89.94, p < .001$ Sequence $F_{(3, 824)} = 28.11, p < .001$ Shift*Nap sleep $F_{(1, 573)} = 13.94, p < .001$
4 Hz	As above	Shift $F_{(1, 2555)} = 80.75, p < .001$ Nap sleep $F_{(1, 2966)} = 26.75, p < .001$ Sequence $F_{(3, 2770)} = 18.00, p < .001$	Shift $F_{(1, 634)} = 76.14, p < .001$ Nap sleep $F_{(1, 698)} = 90.67, p < .001$ Sequence $F_{(3, 829)} = 33.27, p < .001$ Shift*Nap sleep $F_{(1, 590)} = 4.12, p = .043$
5 Hz	As above	Shift $F_{(1, 2481)} = 70.59, p < .001$ Nap sleep $F_{(1, 2919)} = 47.58, p < .001$ Sequence $F_{(3, 2709)} = 25.48, p < .001$	Shift $F_{(1, 643)} = 58.88, p < .001$ Nap sleep $F_{(1, 708)} = 100.74, p < .001$ Sequence $F_{(3, 833)} = 35.30, p < .001$
6 Hz	As above	Shift $F_{(1, 2580)} = 49.44, p < .001$ Nap sleep $F_{(1, 2936)} = 39.14, p < .001$ Sequence $F_{(3, 2779)} = 29.17, p < .001$	Shift $F_{(1, 660)} = 60.90, p < .001$ Nap sleep $F_{(1, 723)} = 91.96, p < .001$ Sequence $F_{(3, 853)} = 44.22, p < .001$
7 Hz	As above	Shift $F_{(1, 2702)} = 54.16, p < .001$ Nap sleep $F_{(1, 3057)} = 54.16, p < .001$ Sequence $F_{(3, 2890)} = 16.84, p < .001$	Shift $F_{(1, 678)} = 79.71, p < .001$ Nap sleep $F_{(1, 740)} = 58.43, p < .001$ Sequence $F_{(3, 876)} = 38.47, p < .001$ Shift*Nap sleep $F_{(1, 629)} = 8.43, p = .004$

⁴⁶ Shift = Night shift worked (either a K1 or K2)

Test number = Number of performance test (either 2nd or 3rd)

Nap sleep (plus or minus S1) = Polysomnographic scored sleep during the napping opportunity (either including or excluding the amount of stage 1 sleep)

Sequence = Place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

Dependent Variable	Independent Variables ⁴⁶	Significant Effects for Analyses Including all Participants	Significant Effects for Analyses with Nine Participants
8 Hz	As above	Shift $F_{(1, 2884)} = 53.25, p < .001$ Nap sleep $F_{(1, 3146)} = 24.07, p < .001$ Sequence $F_{(3, 3027)} = 18.85, p < .001$	Shift $F_{(1, 702)} = 82.04, p < .001$ Nap sleep $F_{(1, 770)} = 77.72, p < .001$ Sequence $F_{(3, 889)} = 41.21, p < .001$ Shift*Nap sleep $F_{(1, 674)} = 10.11, p = .002$
9 Hz	As above	Shift $F_{(1, 2943)} = 49.39, p < .001$ Nap sleep $F_{(1, 3189)} = 28.73, p < .001$ Sequence $F_{(3, 3073)} = 14.23, p < .001$	Shift $F_{(1, 695)} = 71.15, p < .001$ Nap sleep $F_{(1, 763)} = 80.07, p < .001$ Sequence $F_{(3, 876)} = 48.67, p < .001$ Shift*Nap sleep $F_{(1, 671)} = 13.52, p < .001$
10 Hz	As above	Shift $F_{(1, 2756)} = 45.44, p < .001$ Nap sleep $F_{(1, 3109)} = 54.72, p < .001$ Sequence $F_{(3, 2936)} = 12.48, p < .001$	Shift $F_{(1, 614)} = 86.70, p < .001$ Nap sleep $F_{(1, 670)} = 82.67, p < .001$ Sequence $F_{(3, 815)} = 38.36, p < .001$ Shift*Nap sleep $F_{(1, 554)} = 20.29, p < .001$
11 Hz	As above	Shift $F_{(1, 2566)} = 57.41, p < .001$ Nap sleep $F_{(1, 2967)} = 75.23, p < .001$ Sequence $F_{(3, 2777)} = 12.35, p < .001$	Shift $F_{(1, 626)} = 83.37, p < .001$ Nap sleep $F_{(1, 689)} = 89.30, p < .001$ Sequence $F_{(3, 822)} = 42.40, p < .001$ Shift*Nap sleep $F_{(1, 580)} = 20.36, p < .001$
12 Hz	As above	Shift $F_{(1, 2577)} = 62.06, p < .001$ Nap sleep $F_{(1, 3002)} = 45.24, p < .001$ Sequence $F_{(3, 2794)} = 18.87, p < .001$	Shift $F_{(1, 661)} = 83.31, p < .001$ Nap sleep $F_{(1, 726)} = 80.34, p < .001$ Sequence $F_{(3, 846)} = 57.83, p < .001$ Shift*Nap sleep $F_{(1, 627)} = 26.58, p < .001$
13 Hz	As above	Shift $F_{(1, 2573)} = 53.44, p < .001$ Nap sleep $F_{(1, 2939)} = 19.08, p < .001$ Sequence $F_{(3, 2776)} = 11.80, p < .001$	Shift $F_{(1, 647)} = 95.13, p < .001$ Nap sleep $F_{(1, 714)} = 55.21, p < .001$ Sequence $F_{(3, 837)} = 62.08, p < .001$ Shift*Nap sleep $F_{(1, 608)} = 23.89, p < .001$
14 Hz	As above	Shift $F_{(1, 2631)} = 59.99, p < .001$ Nap sleep $F_{(1, 3021)} = 16.27, p < .001$ Sequence $F_{(3, 2834)} = 15.66, p < .001$	Shift $F_{(1, 588)} = 95.17, p < .001$ Nap sleep $F_{(1, 649)} = 36.99, p < .001$ Sequence $F_{(3, 781)} = 55.20, p < .001$ Shift*Nap sleep $F_{(1, 536)} = 21.31, p < .001$

Dependent Variable	Independent Variables ⁴⁶	Significant Effects for Analyses Including all Participants	Significant Effects for Analyses with Nine Participants
15 Hz	As above	Shift $F_{(1, 2581)} = 53.26, p < .001$ Nap sleep $F_{(1, 2996)} = 9.27, p = .002$ Sequence $F_{(3, 2792)} = 16.48, p < .001$	Shift $F_{(1, 612)} = 103.67, p < .001$ Nap sleep $F_{(1, 676)} = 35.98, p < .001$ Sequence $F_{(3, 800)} = 59.20, p < .001$ Shift*Nap sleep $F_{(1, 572)} = 21.92, p < .001$
16 Hz	As above	Shift $F_{(1, 2671)} = 43.79, p < .001$ Nap sleep $F_{(1, 3069)} = 5.23, p = .022$ Sequence $F_{(3, 2868)} = 5.23, p < .001$	Shift $F_{(1, 600)} = 86.40, p < .001$ Nap sleep $F_{(1, 663)} = 38.49, p < .001$ Sequence $F_{(3, 786)} = 70.68, p < .001$ Shift*Nap sleep $F_{(1, 564)} = 20.33, p < .001$

Table 6-11: Results of Significant Post Hoc Comparisons Between Night Shift Number For Analyses Investigating the Relationship Between Architectural Aspects of Sleep and Power in the Standard EEG Frequency Bands

Sequence	Delta		Theta		Alpha		Beta	
	<i>df</i>	<i>t</i>	<i>df</i>			<i>t</i>	<i>df</i>	<i>t</i>
Night 1 versus Night 2	1239	8.70**	1260	12.25**	1262	12.19**	1257	14.72**
Night 1 versus Night 3							1129	2.93*
Night 1 versus Night 4	1275	3.15*	1329	2.61*	1303	2.61*	1296	5.13**
Night 2 versus Night 3	1092	-7.92**	1176	-10.90**	1169	-9.85**	1189	-8.46**
Night 2 versus Night 4	1314	-2.77*	1358	-5.82**	1336	-5.80**	1327	-4.96**
Night 3 versus Night 4	1104	5.26**	1173	4.64**	1173	3.48**	1189	3.07*

* $p < .01$

** $p < .001$

Table 6-12: Details and Results of Mixed Model ANCOVAs For Analyses Investigating the Relationship Between Architectural Aspects of Sleep and Power in Single EEG Frequencies

Dependent Variable	Independent Variables ⁴⁷	Significant Effects for Analyses
2 Hz	shift, ASDA arousals, S3 sleep, sequence	Shift $F_{(1, 1097)} = 7.22, p = .007$ Sequence $F_{(3, 1120)} = 30.69, p < .001$
3 Hz	As above	ASDA arousals $F_{(1, 1014)} = 5.30, p = .022$ Sequence $F_{(3, 1231)} = 31.43, p < .001$
4 Hz	As above	Shift $F_{(1, 1189)} = 26.35, p < .001$ S3 sleep $F_{(1, 1143)} = 3.92, p = .049$ Sequence $F_{(3, 1196)} = 48.07, p < .001$
5 Hz	As above	Shift $F_{(1, 1338)} = 42.62, p < .001$ Sequence $F_{(3, 1323)} = 69.00, p < .001$
6 Hz	As above	Shift $F_{(1, 1201)} = 13.24, p < .001$ S3 sleep $F_{(1, 1320)} = 4.75, p = .029$ Sequence $F_{(3, 1195)} = 70.05, p < .001$
7 Hz	As above	Shift $F_{(1, 1203)} = 14.43, p < .001$ Sequence $F_{(3, 1211)} = 55.59, p < .001$
8 Hz	As above	Shift $F_{(1, 1183)} = 13.16, p < .001$ Sequence $F_{(3, 1193)} = 57.30, p < .001$
9 Hz	As above	Shift $F_{(1, 1151)} = 15.11, p < .001$ Sequence $F_{(3, 1151)} = 57.75, p < .001$
10 Hz	As above	Shift $F_{(1, 1063)} = 9.78, p = .002$ Sequence $F_{(3, 1062)} = 40.92, p < .001$
11 Hz	As above	Shift $F_{(1, 1322)} = 5.12, p = .024$ Sequence $F_{(3, 1298)} = 54.00, p < .001$
12 Hz	As above	ASDA arousals $F_{(1, 1022)} = 4.95, p = .026$ Sequence $F_{(3, 1223)} = 67.30, p < .001$
13 Hz	As above	Shift $F_{(1, 1154)} = 7.06, p = .008$ Sequence $F_{(3, 1157)} = 63.13, p < .001$
14 Hz	As above	Shift $F_{(1, 1130)} = 4.27, p = .039$ Sequence $F_{(3, 1125)} = 59.23, p < .001$
15 Hz	As above	Shift $F_{(1, 1331)} = 8.35, p = .039$ Sequence $F_{(3, 1293)} = 52.95, p < .001$
16 Hz	As above	Sequence $F_{(3, 1140)} = 60.21, p < .001$

⁴⁷ Shift = night shift worked (either a K1 or K2)

ASDA arousals = Number of ASDA arousals per hour in the nap sleep

S3 sleep = Whether stage 3 sleep was obtained (entered or not) during the nap

Sequence = Place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

Table 6-13: Results of Significant Post Hoc Comparisons Between Night Shift Type For Analyses Investigating the Effect of the Nap Opportunity on Power in the Standard EEG Frequency Bands

Shift, Nap Combination	Delta		Theta		Alpha		Beta	
	<i>df</i>	<i>t</i>	<i>df</i>			<i>t</i>	<i>df</i>	<i>t</i>
K1 Nap versus K2 Nap			454	-3.04*				
K1 Nap versus K2 No nap	513	-6.34**	464	-6.31**	497	-5.73**	378	-6.06**
K1 No nap versus K2 Nap	508	-3.22**	455	-3.63**	489	-2.55**		
K1 No nap versus K2 No nap	518	-7.36**	467	-6.63**	501	-6.51**	380	-6.66**
K2 Nap versus K2 No nap	514	-4.26**	462	-3.38**	496	-4.05**	375	-4.51**

* $p < .01$ ** $p < .001$

Table 6-14: Results of Significant Post Hoc Comparisons Between Night Shift Number For Analyses Investigating the Effect of the Nap Opportunity on Power in the Standard EEG Frequency Bands

Sequence	Delta		Theta		Alpha		Beta	
	<i>df</i>	<i>t</i>	<i>df</i>			<i>t</i>	<i>df</i>	<i>t</i>
Night 1 versus Night 2	515	3.51**	464	3.01*	498	3.61**	376	2.95*
Night 1 versus Night 3	525	2.82**						
Night 2 versus Night 3			460	-3.12*	492	-3.30*		
Night 2 versus Night 4			445	-3.04**	476	-3.66**		

* $p < .01$

** $p < .001$

Table 6-15: Details and Results of Analyses Investigating the Effect of the Nap Opportunity on Power in Single EEG Frequencies.

Dependent Variable	Independent Variables ⁴⁸	Significant Effects for Analyses
2 Hz	shift, nap, sequence, shift*nap	Shift $F_{(1, 595)} = 39.15, p < .001$ Nap $F_{(1, 582)} = 10.24, p = .001$ Shift*nap $F_{(1, 582)} = 16.99, p < .001$ Sequence $F_{(3, 586)} = 4.99, p = .002$
3 Hz	As above	Shift $F_{(1, 698)} = 33.43, p < .001$ Nap $F_{(1, 684)} = 10.32, p = .001$ Shift*nap $F_{(1, 683)} = 16.49, p < .001$ Sequence $F_{(3, 688)} = 5.43, p = .001$
4 Hz	As above	Shift $F_{(1, 641)} = 69.48, p < .001$ Nap $F_{(1, 628)} = 6.70, p = .011$ Shift*nap $F_{(1, 628)} = 5.15, p = .024$ Sequence $F_{(3, 632)} = 5.03, p = .002$
5 Hz	As above	Shift $F_{(1, 659)} = 58.62, p < .001$ Nap $F_{(1, 646)} = 10.50, p = .011$ Sequence $F_{(3, 650)} = 7.37, p < .001$
6 Hz	As above	Shift $F_{(1, 630)} = 32.64, p < .001$ Nap $F_{(1, 618)} = 7.95, p = .005$ Shift*nap $F_{(1, 618)} = 3.91, p = .048$ Sequence $F_{(3, 622)} = 9.09, p < .001$
7 Hz	As above	Shift $F_{(1, 641)} = 34.14, p < .001$ Shift*nap $F_{(1, 629)} = 9.10, p = .003$ Sequence $F_{(3, 633)} = 6.02, p < .001$
8 Hz	As above	Shift $F_{(1, 574)} = 37.48, p < .001$ Shift*nap $F_{(1, 563)} = 9.44, p = .002$ Sequence $F_{(3, 566)} = 6.14, p < .001$
9 Hz	As above	Shift $F_{(1, 583)} = 29.13, p < .001$ Nap $F_{(1, 572)} = 4.46, p = .035$ Shift*nap $F_{(1, 573)} = 7.82, p = .005$ Sequence $F_{(3, 576)} = 5.61, p < .001$
10 Hz	As above	Shift $F_{(1, 689)} = 33.33, p < .001$ Nap $F_{(1, 675)} = 10.27, p = .001$ Shift*nap $F_{(1, 674)} = 15.50, p < .001$ Sequence $F_{(3, 679)} = 6.61, p < .001$

⁴⁸ Shift = Night shift worked (either a K1 or K2)

Nap = Whether a napping opportunity was provided or not.

Sequence = Place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

Dependent Variable	Independent Variables ⁴⁸	Significant Effects for Analyses
11 Hz	As above	Shift $F_{(1, 684)} = 43.09, p < .001$ Nap $F_{(1, 669)} = 12.59, p < .001$ Shift*nap $F_{(1, 670)} = 15.57, p < .001$ Sequence $F_{(3, 674)} = 6.96, p < .001$
12 Hz	As above	Shift $F_{(1, 619)} = 35.79, p < .001$ Nap $F_{(1, 607)} = 4.24, p = .041$ Shift*nap $F_{(1, 607)} = 18.32, p < .001$ Sequence $F_{(3, 611)} = 9.00, p < .001$
13 Hz	As above	Shift $F_{(1, 620)} = 44.04, p < .001$ Shift*nap $F_{(1, 609)} = 12.20, p < .001$ Sequence $F_{(3, 612)} = 6.38, p < .001$
14 Hz	As above	Shift $F_{(1, 666)} = 35.79, p < .001$ Shift*nap $F_{(1, 653)} = 13.18, p < .001$ Sequence $F_{(3, 656)} = 7.50, p < .001$
15 Hz	As above	Shift $F_{(1, 636)} = 35.98, p < .001$ Shift*nap $F_{(1, 623)} = 17.59, p < .001$ Sequence $F_{(3, 623)} = 7.54, p < .001$
16 Hz	As above	Shift $F_{(1, 617)} = 32.70, p < .001$ Shift*nap $F_{(1, 605)} = 11.88, p < .001$ Sequence $F_{(3, 609)} = 11.15, p < .001$

Table 6-16: Results of Significant Post Hoc Comparisons Between Night Shift Number For Analyses Investigating the Effects of Sleep Related Factors on Power in the Standard EEG Frequency Bands

Sequence	Delta		Theta		Alpha		Beta	
	<i>df</i>	<i>t</i>	<i>df</i>			<i>t</i>	<i>df</i>	<i>t</i>
Night 1 versus Night 2	465	2.93*	430	2.64*	475	2.67*		
Night 1 versus Night 3								
Night 2 versus Night 3			426	-3.59**	469	-3.72**		
Night 2 versus Night 4	443	-2.84*	413	-4.49**	454	-4.47**	340	-3.33**

* $p < .01$

** $p < .001$

Table 6-17: Details and Results of Mixed Model ANCOVAs for Analyses Investigating the Effects of Sleep Related Factors on Power in Single EEG Frequencies.

Dependent Variable	Independent Variables ⁴⁹	Significant Effects for Analyses	
2 Hz	nap sleep, sequence, acute sleep loss, cumulative debt	Nap sleep	$F_{(1, 492)} = 9.32, p = .002$
		Acute sleep loss	$F_{(1, 535)} = 8.10, p = .005$
		Sequence	$F_{(3, 513)} = 3.84, p = .011$
3 Hz	As above	Nap sleep	$F_{(1, 614)} = 7.28, p = .007$
		Acute sleep loss	$F_{(1, 665)} = 6.89, p = .011$
		Sequence	$F_{(3, 643)} = 7.03, p < .001$
4 Hz	As above	Nap sleep	$F_{(1, 553)} = 4.04, p = .045$
		Acute sleep loss	$F_{(1, 600)} = 9.38, p = .002$
		Sequence	$F_{(3, 579)} = 7.76, p < .001$
5 Hz	As above	Nap sleep	$F_{(1, 577)} = 8.29, p = .004$
		Acute sleep loss	$F_{(1, 624)} = 14.49, p < .001$
		Cumulative debt	$F_{(1, 631)} = 4.24, p = .040$
		Sequence	$F_{(3, 604)} = 11.31, p < .001$
6 Hz	As above	Nap sleep	$F_{(1, 579)} = 8.34, p = .004$
		Acute sleep loss	$F_{(1, 625)} = 14.31, p < .001$
		Cumulative debt	$F_{(1, 631)} = 4.20, p = .041$
		Sequence	$F_{(3, 605)} = 13.63, p < .001$
7 Hz	As above	Acute sleep loss	$F_{(1, 626)} = 10.57, p = .001$
		Sequence	$F_{(1, 626)} = 8.88, p < .001$
8 Hz	As above	Nap sleep	$F_{(1, 517)} = 4.08, p = .044$
		Acute sleep loss	$F_{(1, 560)} = 12.43, p < .001$
		Sequence	$F_{(3, 539)} = 8.75, p < .001$
9 Hz	As above	Nap sleep	$F_{(1, 527)} = 8.57, p = .004$
		Acute sleep loss	$F_{(1, 571)} = 10.65, p = .001$
		Sequence	$F_{(3, 550)} = 6.92, p < .001$
10 Hz	As above	Nap sleep	$F_{(1, 619)} = 20.93, p < .001$
		Acute sleep loss	$F_{(1, 672)} = 7.36, p = .007$
		Sequence	$F_{(3, 647)} = 6.96, p < .001$

⁴⁹ Shift = night shift worked (either a K1 or K2)

Nap sleep = Polysomnographic scored sleep during the napping opportunity (either including or excluding the amount of stage 1 sleep)

Sequence = Place of the study night in the sequence of four study nights (1st, 2nd, 3rd, or 4th)

Acute sleep loss = acute sleep loss in the 24 hours prior to the start of the 1 hour EEG block

Cumulative debt = level of cumulative sleep loss at midday prior to commencing the night shift

Dependent Variable	Independent Variables ⁴⁹	Significant Effects for Analyses
11 Hz	As above	Nap sleep $F_{(1, 623)} = 25.50, p < .001$ Acute sleep loss $F_{(1, 675)} = 15.49, p < .001$ Sequence $F_{(3, 653)} = 8.02, p < .001$
12 Hz	As above	Nap sleep $F_{(1, 555)} = 17.58, p < .001$ Acute sleep loss $F_{(1, 603)} = 5.55, p = .019$ Sequence $F_{(3, 582)} = 7.98, p < .001$
13 Hz	As above	Nap sleep $F_{(1, 549)} = 7.05, p = .008$ Acute sleep loss $F_{(1, 596)} = 6.70, p = .011$ Sequence $F_{(3, 575)} = 6.08, p < .001$
14 Hz	As above	Nap sleep $F_{(1, 590)} = 5.59, p = .018$ Acute sleep loss $F_{(1, 640)} = 7.36, p = .007$ Sequence $F_{(3, 620)} = 9.07, p < .001$
15 Hz	As above	Sequence $F_{(3, 586)} = 7.51, p < .001$
16 Hz	As above	Acute sleep loss $F_{(1, 591)} = 5.90, p = .015$ Sequence $F_{(3, 571)} = 10.99, p < .001$

CHAPTER 7

DISCUSSION

The main aim of this study was to determine the effectiveness of a pre-planned nap on the night shift for improving the performance and neurophysiological alertness of air traffic controllers. To do this, it was considered necessary to understand how the shift schedule of air traffic controllers influenced their sleep across the working week prior to the night shift, the exact quantity and structure of sleep obtained at work, and whether the napping opportunity affected sleep subsequent to the night shift. These questions were addressed in an operational air traffic control environment by monitoring 28 controllers across four weeks of work.

This chapter begins by discussing the limitations that need to be taken into account when considering the findings of this study. The subsequent sections then address the findings of the study on a chapter-by-chapter basis. Given the study findings, recommendations are made for operational air traffic controllers, the employing organisation, and the regulating body. Finally, further research needs, highlighted by the present study, are discussed.

7.1 Study Limitations

7.1.1 *Generalisability of Findings*

The job of an area controller has many features in common with other controlling positions both nationally and internationally. Nevertheless, many factors also vary, such as the type of traffic controlled, features associated with the airspace, the particular separation standards employed, the general working environment, and at an international level, the arrangement of work. Greater care is required in generalising the findings to controlling positions more removed from that of area control. This is particularly so with respect to work-schedule findings.

Terminal controlling is not dissimilar from the position of area controller, but non-radar positions, and controlling in overseas environments, have less in common with area control. Given that 80% of area controllers who were asked to participate in the study did so, the assumption can be made that the findings are representative of this occupational group.

Although napping was found to have positive effects for reaction time performance and neurophysiological alertness, it is not known how these changes relate to operational air traffic control performance. However, given that vigilance is critical to the task of controlling, the findings should have some relevance. Furthermore, the measures of neurophysiological alertness were recorded while controllers were operationally active.

Measuring operational air traffic control performance continues to be a complicated and evolving issue. Air traffic control is a complex task, and what to measure, when to measure it, and by whom, are all research questions that as yet have not been sufficiently addressed. Often there are a number of different ways of achieving identical outcomes in a controlling situation and individual controllers favour different methods. The time taken to perform tasks, number of aircraft movements, or proximity of aircraft to each other, all seem like possible operational measures, but they in no way capture the complexity of the task and in fact may misrepresent what is occurring. Simulated air traffic control tasks exist, but they are often not sensitive to changes in alertness (Della-Rocco & Cruz, 1995), and it has not been shown how they relate to operational performance. As our understanding of the changes in complex cognitive performance with declining alertness advances, it is likely that tests that more closely reflect operational performance, or actual components of operational performance, will become available as well-validated and sensitive measures.

The general findings associated with napping support, and add to, the large body of napping literature. Furthermore, all individuals working at night face the same physiological challenges therefore the findings of the present study also have some relevance to other occupational groups.

7.1.2 *Missing Data*

The amount of missing actigraphy, logbook and PVT data was minimal. Only 1.6% of actigraphy and/or logbook entries were missing entirely. For 12.9% of the days across which actigraphy was collected, only logbook data were available. Of the 321 PVT tests completed, 96% provided complete sets of summary statistics. In all instances the missing data were random. Despite the small amount of data lost there is still the potential for misrepresentation to occur when not all values are available. However, given the long duration of the study, the number and complexity of the measures taken,

and therefore the level of commitment required of study participants, it is considered that the amount of data lost was surprisingly small.

7.1.3 *Variability in Experimental Features*

This study was testing an intervention. However, because the study was undertaken in an operational environment, where operational needs have priority, it was impossible to control the exact timing and duration of certain events. Examples include the length and timing of the napping opportunity, the timing of the performance tests, and the timing of the 1 hour blocks of EEG data at the end of each night shift.

The variability in the length of the nap opportunity was not great, with only a few individuals having significantly less or more time to sleep. The lower values in the range were due to an individual discontinuing their nap after suddenly waking, and in another instance being unable to sleep at all. The upper values in the range were likely to be due to slight inaccuracies in timing the nap as well as a few participants continuing to nap after the experimenter had woken them. Despite the variability in the timing of these events, in all instances the timing of the naps, performance tests and 1 hour blocks of EEG data significantly differed according to the night shift worked.

Variability in the conditions under which the data are collected is an inherent weakness of field research. However, one of the strengths of field research is that interventions are tested under “noisy” conditions. They provide a link between the rigorous but unrealistic setting of laboratory studies, and the less predictable real world. Nevertheless, napping behaviour seen in the present study cannot be presumed to be entirely unaffected by the study context. Although every effort was made to allow study participants to nap under the conditions they would, or could, normally nap, the equipment worn and presence of the experimenter may well have influenced controllers’ behaviour.

7.1.4 *Sleep Measures*

Limitations of Actigraphically Determined Sleep Parameters

Although actigraphy correlates well with polysomnographic measures of sleep, the agreement declines as the quality and quantity of sleep diminishes (Ancoli-Israel, 2000). For the particular actiwatch used in the present study, it has recently been shown that there is a slight overestimation of total sleep time and sleep efficiency (Kushida et al.,

2001). In the present study, a consistent approach in analyses that related to sleep length was to utilise all three actigraphy sleep length parameters (TIB, assumed and actual sleep) in separate mixed models in order to see if changes were consistent across the different measures of sleep length. Generally, identical findings were present across all three sleep length parameters, with TIB and assumed sleep length reflecting each other most closely. Generally for night sleep length, actual sleep was approximately an hour less than TIB, and assumed sleep length was close to, but slightly shorter than TIB. However, the greatest differences between these variables was found when all values were high and the smallest difference seen when they were at their lowest.

In the present study, longer episodes of sleep were repeatedly found to be of poorer quality. There are often reasonable physiological reasons for the shorter sleep episodes to be of better quality, such as the short sleep prior to the K1 night shift also occurring close to a circadian time of greater sleep propensity. In addition, it has previously been demonstrated that shorter sleep episodes show normal amounts of deep sleep and less of all other stages, which may explain the improved quality (Åkerstedt, 1991; Carskadon & Roth, 1991). However, in the present study there were also occasions where slightly longer sleep episodes occurred at times of greater sleep propensity, but were still found to be of poorer quality than shorter sleep episodes further from the circadian nadir. For example, the morning sleep after the K1 night shift occurred after a period of sleep deprivation and around the circadian low point in body temperature. Both these factors would be expected to assist in producing quality sleep. However, this sleep was found to be of significantly poorer quality than the later but shorter sleep after the K2 night shift. Because of the repeated association of poorer quality sleep with longer sleep episodes, irrespective of the timing of the sleep, and the less stable nature of the sleep quality findings, caution should be applied when interpreting these findings.

Baseline Sleep Calculation

There is no ideal way of determining individual sleep need. In the present study the amount of sleep obtained on multiple days off was recorded, it was ensured that this sleep had not been restricted by work demands, and the quantity of sleep was determined via objective methods. The assumption then has to be made that this quantity of sleep relates to individual sleep need. Ferrara and De Genarro (2001), in a recent review, reinforce the concept that individual baseline sleep values should be determined. Unfortunately, they don't suggest how best to do this.

In comparing the mean baseline sleep requirement calculated using actigraphy in the present study to other populations, it is found to be relatively high. Previous studies report individuals obtaining on average 7.4 hours of sleep per night (Carskadon, 1994), or for a New Zealand population, 7.5 hours per night (R. Harris, personal communication, December 2001). A recent study of New Zealand anaesthetists (Millar, 2001), using similar methods to the present study, reported an average baseline sleep value of 7.7 hours. In the present study, the actigraphically determined values were also slightly higher than the subjective baseline sleep requirements. These findings suggest that the baseline values calculated from the air traffic controllers in the present study might be a slight overestimation of individual sleep need.

If this were the case, using these values would lead to the calculation of a “false” or “higher” sleep debt than actually exists. This would help explain the unexpected relationship between cumulative sleep debt and performance that was seen at the commencement of the night shift. However, when cumulative sleep debt was recalculated using subjective estimates of baseline sleep need, these findings did not alter. Furthermore, the relationship seen between cumulative sleep debt and spectral power of nap sleep was in the expected direction.

Given the data collected in the present study, it is impossible to know the real sleep need of individuals. In fact, unless study participants were monitored across a large number of consecutive episodes of unrestricted sleep, it is probably impossible to accurately gauge individual baseline sleep need.

7.1.5 *Quantity of Artefact Free Neurophysiological Data*

Despite efforts made to reduce the occurrence of artefact in the recordings, there was still concern surrounding the pattern and quantity of artefact in the EEG channels. A separate in-depth examination of a random subset of data from 12 controllers, found that there was no temporal pattern to the occurrence of artefact, and that significantly more data were retained from the Oz-O2 channel compared to all other single channels (18% versus 5-17%) (J. Gale, personal communication, January 20, 2002).

In the present study, the median amount of data retained from Oz-O2 during the last hour of the night shift was only 10.74%, although plots showed that these data were evenly spread across the 1 hour blocks. It was initially thought that the amount of data remaining would be insufficient to detect changes in alertness as a result of the napping

opportunity, but this was not the case. Further, when analyses were repeated using only 9 individuals with the greatest amount of analysable EEG data, the main findings did not alter. However, the issue remains as to whether such a limited amount of data can be said to represent what was occurring in the last hour of the night shift. This issue is not specific to the present study, but unfortunately other published workplace studies provide minimal information on the amount of EEG data rejected as artefact, and do not address the issue of possible biases that this might introduce (e.g. Torsvall & Åkerstedt, 1987; Gillberg, Kecklund & Åkerstedt, 1996).

In the present study, artefact that rendered the EEG data unsuitable for analysis did not always affect the viewing of SEMs in the EOG channels. Nonetheless, on many occasions an eye movement was not scored as an SEM because of artefact obscuring part of the trace. The percentage of “possible” SEMs that were excluded because of the conservative scoring approach taken was not calculated. It can only be said anecdotally, that a much greater portion of the EOG channel was useful, compared to the EEG channel.

7.2 The Sleep Consequences of Backward, Rapidly-Rotating Schedules

7.2.1 *Rosters Versus Actual Work Patterns*

There was considerable variability in the number of hours controllers actually worked, over both the eight day study period and across the four rostered working days. This was primarily due to a few controllers working on their days off, and others working less than their four rostered shifts. Overall, controllers worked slightly fewer hours than they were rostered to work and the total hours of work did not differ between the two shift cycles. This was in spite of the K2 shift cycle being rostered to be 2 hours shorter than the K1 shift cycle (26 versus 28 hours). The difference was largely due to controllers on the K2 shift cycle working longer than was rostered on the early morning and night shifts. These extended shifts have the potential to limit sleep opportunities between the early morning and night shift, and the recovery sleep subsequent to the night shift.

7.2.2 *Sleep Changes Across the Work Week*

In order to understand the factors that might influence the usefulness of a nap on the night shift, actigraphy and log book data were used to assess the length, timing, and

quality of sleep across the rapidly backward rotating shift cycle worked by air traffic controllers.

Examination of main sleep episodes clearly showed that the amount of sleep obtained progressively decreased across working days. Sleep length parameters returned to their pre-work levels on subsequent days off, when work no longer impinged on sleep time. On days off, controllers spent nearly 9 hours in bed each night, slept for an assumed period of between 8.2 and 8.6 hours, and obtained over 7 hours of actual sleep. The main sleep on the night of the first workday was significantly shorter than all other days, other than the following night (workday 2). TIB decreased to less than 8 hours and actual sleep to less than 7 hours. Prior to the early morning shift, the shortest main night sleep times occurred. Individuals averaged 6.9 hours in bed, with 6.6 hours of assumed sleep and less than 6 hours actual sleep.

Recent studies of American air traffic controllers working a similar counter-clockwise rotating shift reported that individuals obtained on average between 5.6 and 6.3 hours sleep prior to the early morning shift (Cruz & Della-Rocco, 1995a; 1995b; Della-Rocco & Cruz, 1995). This is similar to the actual sleep findings of the present study. However, the data may not be directly comparable as the American controllers made self reports of their sleep length as opposed to the actigraphically collected data in the present study.

There was also a great deal of individual variability in the TIB, assumed sleep, and actual sleep values of night sleep. This variability decreased over the sequence of shifts possibly due to increasing restrictions on sleep length imposed by the work pattern.

As expected, the shortening of sleep occurred largely through the progressively earlier rising times enforced by shift start times moving backward. The sleep episode preceding the day shift on workday 2 (average start time 0650 or 0725 hours) was shorter than any other study day, except the sleep preceding the early morning shift on workday 3 (average start time 0630 hours).

It was expected that bedtimes and particularly sleep starts would remain unchanged due to circadian influences making the earlier initiation of sleep difficult. However, the sleep prior to the early morning shift (on workday 3) began 35-50 minutes earlier than sleep on off-duty days. The earlier bedtimes can possibly be explained by controllers making an effort to compensate for the increasingly earlier rising times. The ability to fall asleep

earlier prior to the early morning shift (on workday 3) may indicate an increased homeostatic drive that assisted air traffic controllers in initiating their sleep at a slightly earlier time in the circadian cycle. Despite these changes in behaviour, the sleep prior to the early morning shift and the sleep of the night prior, were the shortest of the week.

The sleep quality variables exhibited less consistent results. Sleep fragmentation did not show a statistically significant change across the study, although mean activity, sleep efficiency, and actual sleep percentage did vary over the eight day study period.

Generally the shorter sleeps, commencing on workday 1 and workday 2, were of slightly better quality than sleep on the first rostered day off after the working week (post-cycle day 1). The raw data indicated that sleep quality was also poorer on the other off-duty days but the difference did not reach statistical significance.

The large majority of air traffic controllers obtained some sleep between the early morning shift and the start of the night shift. The amount of sleep was not great (2.3 hours of assumed sleep and 2.2 hours of actual sleep) but was comparable to that obtained by North American air traffic controllers under similar circumstances. In the North American studies values range from 2.3 hours (Rhodes et al., 1996) to 3.75 hours of sleep (Schroeder, Rosa & Witt, 1998).

The results of the analyses on sleep prior to the night shift indicate that individuals did not shorten or lengthen their pre-shift sleep in anticipation of whether or not they would be napping during the night shift. However, depending on the night shift, there was a difference in the timing and quality of sleep. Sleep prior to the later starting K2 shift began around 1730 hours as compared to the earlier sleep which started on average at 1600 hours. This earlier sleep therefore occurs during a time of greater sleep propensity (Dijk & Edgar, 1999; Roehrs, Carskadon, Dement & Roth, 2000), which may explain the improved quality. In addition, although sleep length was not found to statistically differ according to the shift worked, mean values suggest that when individuals worked the later starting night shift their sleep tended to be slightly longer. This may contribute to the difference in the fragmentation of sleep, with the slightly longer sleep found to be more disturbed.

7.2.3 *Sleep Loss*

Compared to baseline sleep measures, total sleep in consecutive 24 hour periods decreased across the working week, with the greatest sleep loss associated with the 24

hour period from midday prior to a night shift to midday after the night shift. Sleep loss during this time period was about 1 hour greater on the K2 than the K1 shift cycle.

Part of this difference may be due to the arbitrary cut-off at midday, chosen for calculating sleep per 24 hours. The K2 night shift finished later and therefore afforded less time before midday, than did the K1 night shift. The difference may also be due to sleep following the K2 night shift occurring at a later circadian phase, and thus being more likely to be terminated by the circadian wake drive (Gander, Rosekind & Gregory, 1998b). In the following 24 hours (midday workday 4 to midday post-cycle day 1) controllers obtained similar amounts of sleep on both shift cycles, which was more than they averaged on baseline (off-duty) days.

It is important to note that right across the week, the levels of cumulative debt varied enormously among controllers. Yet despite the wide variability, all individuals on all occasions developed some level of sleep debt in the period workday 3 to workday 4 and only one individual on one occasion did not already have a sleep debt by the time they began the night shift.

As expected, the cumulative sleep debt did not return to zero even after two days off. Previous studies indicate that sleep is not recovered hour for hour (Gander et al., 1998a). Instead recovery sleep includes a greater proportion of the deeper stages of sleep (Daan, Beersma & Borbely, 1984).

The large number of individuals who had no acute sleep loss preceding the first performance test of the night was somewhat unexpected, given the truncated sleep on the preceding night and the small amount of sleep obtained between the morning and night shift. However, the way this value was calculated provides some explanation. The first performance test of the night occurred at approximately 2230 or 0000 hours, which was around the start of the main sleep period 24 hours earlier. Therefore the length of this sleep period (which had previously been shown to be the shortest of the week) combined with sleep that was taken between the morning and night shift, amounted to as much or more than average baseline sleep. Whether this split sleep is as effective as a single consolidated period of sleep is unclear (Åkerstedt, 1998).

7.3 Characteristics of Nap Sleep

7.3.1 *Amount and Quality of Sleep Obtained*

Polysomnographic analyses (Chapter 4) indicated that, on average, controllers took approximately half the napping opportunity to initially fall asleep, resulting in sleep efficiency, being relatively poor. After sleep was initiated, about $\frac{3}{4}$ of the remaining time available for napping was spent asleep. It is important to note, however, that there was considerable individual variability. For example, sleep latencies ranged from 2 minutes through to 47 minutes, and total sleep duration ranged from zero through to 47 minutes.

The data suggest that certain individuals consistently experienced much greater difficulty initiating and maintaining sleep in such a context. The reasons for this are likely to be many and varied. Possibilities include: reluctance to fall asleep because of a dislike of feeling of sleep inertia on waking; concern about being called back to work at short notice; sufficient good quality sleep prior to the night shift (although the findings indicated that this was unlikely); finding the sleeping facilities uncondusive to sleep; and differences in sensitivity to disturbance by the monitoring equipment or other aspects of the experimental protocol.

An expected finding was the significance of the length of the nap sleep in influencing the occurrence of stage 3 sleep and the sleep stage individuals woke from. It was found that for 6 of the 10 individuals who entered stage 3 sleep for a minute or longer, latency to this event was 40 minutes or more. If the occurrence of sleep inertia is, as it has been suggested by previous research, linked to the depth of sleep, and/or sleep stage on wakening, then this finding adds weight to the need to limit the napping opportunity strictly to 40 minutes. However, on 10 of the 20 occasions in the present study when individuals entered stage 3 sleep, it was not maintained for more than a consecutive minute (although the total amount of stage 3 could still have been greater than this). Given the paucity of data on the consequences of a small amount of SWS for sleep inertia, there is a need for further investigation in this area.

The frequency and occurrence of sleep stage transitions, awakenings, and arousals were investigated as indicators of the quality of nap sleep. It is difficult to know whether these sleep quality measures were normal or not, given the paucity of published data regarding the structure of short periods of sleep. Sallinen et al. (1998) report that during a 50 minute night time sleep opportunity in the laboratory, the average number of awakenings

was fewer than one. In the present study the average was 2 (Chapter 4, section 4.5.4). This difference is likely to be related to the markedly different conditions under which sleep was attempted. In the present study, the average number of awakenings per hour was 6, and the average number of sleep stage transitions per nap was 8 (or 25 per hour), while the average number of ASDA arousals was 5, which produces an hourly index of 39. These relatively high values imply that the nap sleep was fragmented and therefore not of good quality.

It has previously been shown by Agnew et al. (1966) that on the first night sleeping in a laboratory-based situation, and wearing polysomnography equipment, the structure of sleep is altered compared to subsequent nights. This effect was naturally controlled for in the present study through study conditions being completed as they occurred within the roster (resulting in 18 sequences of the study conditions) as well as testing for a possible effect in all models. If there was a genuine “first night effect”, it would be expected that the variable representing night shift number (labelled sequence) would have been more frequently identified as a significant factor. In particular, effects would have been expected for the amount of wake time, stage 1, and number of sleep stage transitions, as these variables have previously been identified as those showing clear first night effects (Agnew et al., 1966). In the present study, a greater number of awakenings per hour were identified as occurring during naps taken on the first study night compared to the last, while the subjective quality sleep of a sleep taken on the first night was considered poorer than that on the second. It would be expected that, if there were an actual first night effect, then the values on the first night would consistently differ from those on several subsequent nights. On this basis it is thought unlikely that there was any systematic change in sleep related parameters as an individual progressed through the four study nights.

The subjective data on the nap sleep indicated that controllers had some difficulty falling asleep, that their sleep was thought to be relatively light, and of poor quality. Yet, they also reported that the sleep was both moderately refreshing and helpful in assisting them to manage their fatigue across the remainder of the night shift. In support of the polysomnographic data, the trend in the subjective data was for the sleep on the K1 night shift to be reported as slightly shorter, longer to initiate, better quality, more refreshing, and less useful in assisting individuals to manage their fatigue across the remainder of the

night shift. Despite these trends none of these differences are statistically significant and in all instances the estimated differences between K1 and K2 night shifts were small.

Because of the intrusiveness of recording sleep at work via polysomnography, it was of interest to determine whether individuals could accurately estimate certain polysomnographic aspects of their sleep. Longer subjective estimates of the nap sleep were associated with longer polysomnographically scored sleep, shorter sleep latencies and more stage 2 sleep. In addition, a nap was rated as better quality when less stage 1 sleep was scored, and rated as more useful in helping manage fatigue when stage 3 sleep had occurred. However, these significant statistical relationships do not reflect highly accurate associations. A Bland-Altman plot showed that the level of agreement between the two measures of sleep length was poor, with individuals likely to estimate their sleep duration as between 18 minutes less and 20 minutes more than the polysomnographic value.

Further, even if individuals could accurately state the amount of sleep they had obtained, other sleep related variables, such as the amount of stage 2 sleep or sleep latency, could not be accurately determined from the subjective estimate of sleep length despite their statistical relationship. Thus, subjective estimates of sleep related variables are considered to provide little useful information about the actual quantity and quality of sleep obtained.

This is not intended to imply that subjective estimates have no value. They provide critical information about how individuals' perceive their sleep, which in turn is likely to influence their future behaviour (Minors & Waterhouse, 1987). If, for example, an individual experienced difficulty falling asleep, they perceived their sleep as light, of poor quality, and thought it was of little use in managing fatigue, then this negative perception may alter the likelihood of them making the effort to nap on subsequent night shifts. On the other hand, the lack of accuracy of subjective estimates can be demonstrated for educative purposes. This reinforces the necessity of objectively measuring sleep, particularly given the relationships between short, poor quality, sleep and performance and neurophysiological alertness.

On only two occasions was sleep scored outside the napping opportunity. This is a relatively small amount given that both circadian and homeostatic processes would have been encouraging sleep. It is also unlikely the controllers' workload was always high

enough to provide sufficient stimulation to mask these circadian and homeostatic effects. However, it must also be noted that for individuals undertaking a safety critical task, the 30 second epoch across which changes must be seen in order to qualify as sleep, is a relatively long period of time.

7.3.2 *Comparisons With Other Napping Studies*

Because there are few other studies where sleep in an operational environment has been recorded, it is not clear whether the amount of sleep obtained during the napping opportunity is typical. Table 7-1 lists the sleep efficiency, latency, and percentage of total sleep time in each sleep stage reported, in four other studies which have presented the architecture of sleep of a similar length and at similar times of the day as in the present study. The design of these studies has been discussed previously in Chapter 1, section 1.5.2.

In the only other operational study, conducted on the flight decks of long haul aircraft by Rosekind et al. (1994), the average amount of total sleep obtained by individuals during a 40 minute napping opportunity was approximately 26 minutes, which relates to a mean sleep efficiency of 64%. However, the total sleep time in the Rosekind et al. (1994) study was calculated only for those individuals who actually slept. In the present study total sleep time had a mean value of 17.70 minutes, but with data from the non-sleepers excluded the mean increased to 19.92 minutes.

In addition, the flight deck napping study had a mean reported latency (to the first 1.5 minutes of sleep) of approximately 6 minutes, while in contrast the mean latency in the present study was 19 minutes (to the first minute of sleep). This much longer latency may be taken to suggest that the air traffic controllers are either not as sleep deprived and/or fatigued as the long haul flight crew. However, although it took controllers longer to fall asleep, they remained asleep for all but approximately 4 minutes of the remaining napping opportunity. The amount of time spent awake between sleep onset and the final waking for long-haul flight crew is not reported but can be estimated from the data provided. In the 40 minute flight deck napping opportunity, an average of 6 minutes elapsed prior to sleep onset, and 26 minutes were taken up by sleep, resulting in another 8 minutes of wake either interspersed between bouts of sleep or occurring after waking but before the end of the napping opportunity.

Table 7-1: Sleep Efficiency, Latency, and Percentage of TST in each Sleep Stage Reported in the Present, and Four Other Studies.

Study	Sleep Efficiency (compared to TST)	Latency (minutes)	% TST Stage 1	% TST Stage 2	% TST SWS	Mean Age of Study Participants (years)
Present study	41%	19	27%	9%	2%	36
Rosekind et al. (1994) average of all flight decks naps	64%	6	30%	60%	8%	42
Gillberg (1984) nap at 2100 and 0430 hours	69% and 96%	8 and 2	30% and 13%	38% and 42%	30% and 34%	No mean given Range 20-25
Sallinen et al. (1998) nap at 0100 and 0350	74% and 90%	12 and 4	11% and 9%	49% and 45%	40% and 41%	No mean given Range 31-52
Rogers et al. (1989) nap at 0200 hours	94%	21	4%	40%	48%	25

Thus, although the long-haul flight crew fell asleep more quickly than the controllers, it is suggested that they did not stay asleep. Therefore, although shorter latencies would indicate a greater drive for sleep, the consolidated (or not) structure of sleep may indicate otherwise. Alternatively, it is possible that the cockpit seat is less conducive to sleep maintenance than lying horizontal in a quiet place of one's choice.

In the present study, it was determined that stage 2 was the predominant sleep stage, comprising about 58% of the total sleep time, while approximately 35% of the sleep period consisted of stage 1 sleep. Deeper stages of sleep were scored in less than half the total napping opportunities. On average, stage 3 sleep made up approximately 6% of the sleep, while stage 4 sleep was scored only very occasionally. REM sleep was not observed at all.

When comparing the architecture of the nap sleep to that seen in the flight deck napping study, the results are not dissimilar. Again however, the differences in polysomnographic metrics make direct comparisons difficult. In the flight deck study, the percentage of each sleep stage was compared to the duration of time from sleep start until the cessation of the sleep (which may have also included wake), while in the present study the comparisons were made with total polysomnographically scored sleep. For sleep stage variables, this difference is unlikely to produce large discrepancies when comparing findings, although few other values were directly comparable because of this.

Three other studies have investigated the efficacy of short naps during the night in laboratory based settings (Gillberg, 1984; Rogers et al., 1989; Sallinen et al., 1998) and although the timing and length of the napping opportunity in these studies differs somewhat from those in the present study, it is possible to make some comparison between the length and structure of the sleep in a highly controlled situation compared to a real-world setting. Gillberg (1984) allowed subjects the opportunity to sleep for 60 minutes at either 2100 or 0430 hours after limiting sleep the night before to 4 hours. Latency was measured as the time to the first 20 seconds of stage 1 sleep, and the value reported is much shorter than in the present study. However, the controlled conditions and less conservative criteria for sleep onset would be expected to be influential. In addition, the electrode site utilised was occipital (O_2-P_4) resulting in a different view of the amount of alpha present, which is critical in determining sleep onset. Further, Gillberg's study excluded 5 of the 12 individuals from the mean latency calculations, as they could not be precisely scored for sleep onset.

The sleep structure of the earlier nap in Gillberg's study, as opposed to the later nap, more closely resembles the sleep architecture found in the present study, although the air traffic controllers had more stage 2 sleep and much less SWS than in either of the laboratory based naps. Gillberg also scored a small amount of REM sleep.

The findings reported from Sallinen et al. (1998) were recorded during 50 minute sleep opportunities commencing at either 0100 or 0350 hours on a pseudo night shift. Prior to the study period, the participants slept in the laboratory but throughout the night. Therefore they were theoretically sleep satiated before beginning the study protocol⁵⁰. Like the findings from Gillberg's study, the sleep efficiency and latency suggest greater ease of initiating and maintaining sleep than in the present study. In marked contrast to the flight deck napping study, and the present study, the amount of SWS obtained was very much higher. However, it was similar to the amount of stage 3 and 4 obtained in the other laboratory-based studies. Sallinen et al. also reports no REM sleep in the early nap and a very small amount in the latter nap.

The final study mentioned in Table 7-1 is that conducted by Rogers et al. (1989) which was designed to compare a 1 hour nap at 0200 with the effects of caffeine. Again the sleep efficiency and percentage of sleep time in the various stages is similar to the other laboratory studies. The only difference was the long latency reported to sleep onset, which at first glance is very similar to that found in the present study. However, Rogers et al. (1989) defined sleep onset as the first 5 minutes of unbroken stage 2 sleep, which is extremely conservative criterion that makes comparison with other studies highly questionable.

Overall, these comparisons indicate that the sleep obtained by the air traffic controllers was less efficient and lighter than sleep obtained in laboratory based studies. This is not particularly surprising and is likely to be due to ambient factors in an operational setting continuously "bumping" an individual up into wakefulness or the lighter stages of sleep. It is clear that the structure of the sleep obtained by air traffic controllers most closely resembles that of the pilots in the flight deck napping study. However, care must be taken in all instances when making such comparisons due to the varying definitions of sleep related values or the lack of clarity when indicating how measures were calculated.

⁵⁰ Sallinen et al. (1998) also allowed a 30 minute sleep opportunity in a separate study condition but these results are not discussed here as the 50 minute condition is closer in length to the actual mean length of the napping opportunity controllers obtained.

7.3.3 *Influence of Nap Timing*

It is interesting to note that in the polysomnographic data in the present study, a consistent but statistically non-significant trend was identified, indicating the sleep obtained earlier in the night (on the K1 shift) was shorter, had a longer latency and therefore also resulted in greater time awake and lower sleep efficiency values than the later sleep (on the K2 night shift). When focussing on the architectural changes in sleep structure, stages 1 and 2 showed no statistically significant differences associated with the shift worked (and consequently also the timing of the nap). However, stage 3 sleep was more likely to be entered when the nap was taken later in the night, and although a mixed model ANOVA could not be run due to the small amount of data, the descriptive statistics suggested that stage 3 sleep may be entered more rapidly when individuals were napping later in the night (on the K2 shift). In addition, it was found that participants were more likely to wake from SWS when a nap was taken at a later time.

In comparing time-related changes to the studies previously mentioned, both Gillberg (1984) and Sallinen et al. (1998) found significant differences in the length of sleep obtained between an early and late nap. Both studies also noted significantly shorter latencies later in the night, while Gillberg also found a significant decrease in the amount of stage 1 sleep scored in the later nap. The changes in total sleep length over time are supported by the trend seen in the present study, as is the decreased latency to sleep onset. The decrease in stage 1 sleep seen by Gillberg is in contrast to the trend seen in the present study, however, it should be noted that in Gillberg's study the naps were separated by 7.5 hours, with the later one occurring at 0430 hours.

The spread in start times of the napping opportunity in the present study may be one of the reasons that the difference due to the timing of the napping opportunity only occasionally reached statistical significance. Even without the spread in times, the planned difference in the timing of the napping opportunities was only 2 hours, which was not expected to create statistically significant differences given, that in the studies by Gillberg (1984) and Sallinen et al. (1998) the time differences between naps was greater and still few variables were found to significantly differ. In the present study, the latest napping opportunity on the K1 night shift started just before 0100 hours and the earliest napping opportunity on the K2 night shift began at 0200 hours (not including the outlying napping opportunity that occurred on a K2 night shift). The increased likelihood of entry into S3 and waking from SWS on the later naps, despite the very small

time difference between napping opportunities, indicates it is important to consider when a napping opportunity will take place. This is particularly so considering that the hours of midnight to 0300 hours fall on the steeply descending portion of the circadian cycle that would be partly responsible for producing these time related differences (Carskadon, 1994). On the other hand, it should also be noted that, where differences exist, the absolute change was small. The importance of these time-linked differences, and their size, needs to be considered in relation to their potential consequences for performance and neurophysiological alertness (see below).

7.3.4 *Influence of Prior Sleep Patterns*

What was more pronounced was the frequent finding of the influence of prior wakefulness and acute sleep debt in determining the structure of the nap sleep. Sleep latency, efficiency, wake time during the nap, and subjective estimates of sleep were all identified as influenced by the length of prior wakefulness, while wake time during the sleep period, occurrence of stage 3 sleep, and the likelihood of waking from SWS were seen to be related to the levels of acute sleep loss. Both these factors are expected to influence the homeostatic drive for sleep and one possible reason that they were indicated as more influential than the timing of the nap (ie: circadian influence) may be the wider range of values.

7.3.5 *Effects on Subsequent Sleep*

It was demonstrated that the opportunity to nap on the night shift did not affect the length, timing, or quality of post-night shift sleep. Instead a consistent difference was seen between sleep after the different night shifts, with the K1 shift followed by earlier, longer, but slightly poorer quality sleep than the K2 shift. The longer sleep on the K1 shift is likely to be related to the earlier initiation time, which was on average 0520 hours. It has been estimated that on average the circadian low point in body temperature for an individual adjusted to a diurnal pattern is 0500 hours and that about 6 hours after this point in time the circadian wakeup signal makes sleep continuation difficult (Gander et al., 1998b). Sleep after the K1 shift was approximately 5 hours in length, and therefore ends nearly 6 hours after the low point in body temperature, which suggests sleep may be curtailed by the circadian wake up signal. This also explains the even shorter sleep post the K2 night shift, which started on average at 0730 hours and therefore less sleep occurs before individuals reach the circadian wakeup signal.

7.4 Performance

7.4.1 *Performance at the Beginning of the Night Shift*

Prior wake, acute sleep loss, cumulative sleep debt and the shift type worked were investigated as predictors of performance at the beginning of the night shift. These analyses produced a number of unexpected findings. Firstly, it was somewhat surprising that neither the length of prior wakefulness or acute sleep loss influenced performance on the first reaction time test of the night. Even less expected and more difficult to interpret was the finding that some aspects of performance were seen to improve with increasing levels of cumulative sleep debt.

The lack of a relationship between prior wake, acute sleep loss and performance may be explained in part by the timing of the test. Both Colquhoun (1971) and Monk (1997) have found vigilance performance to peak in the evening, with Monk demonstrating a peak at 2200 hours. In the present study the reaction time tests occurred from around this point in time until nearly 0100 hours. The circadian related peak in vigilance performance in the earlier part of this time frame may assist those who had been awake for longer and/or were experiencing an acute sleep debt, to compensate. It is also likely that higher levels of motivation associated with starting work, and the lack of a time-on-duty effect, assisted individuals to counteract the effects of prior wakefulness and acute sleep loss at this time.

Such factors may also have contributed to the anomalous relationship seen between cumulative sleep debt and performance. In addition, it is possible that using values of cumulative sleep debt calculated up to midday prior to the night shift was misleading. Several additional models were run in an attempt to understand these relationships. However, in all instances cumulative sleep debt, whether calculated at midday or including sleep prior to the night shift, maintained the unexpected relationship with performance. Furthermore, the amount of sleep obtained between the morning and night shift was not significantly related to performance at the beginning of the night shift.

Closer examination of each controller's data revealed two quite distinct groups of individuals. The larger group showed a consistent positive relationship between cumulative sleep debt and performance, while a smaller group showed a negative relationship that indicated, as expected, worsening performance with increasing

cumulative debt. Five individuals showed almost no change in performance with changing cumulative sleep debt and were excluded from the subsequent investigations.

When comparing the two groups it was determined that individuals displaying the unexpected relationship had on average faster overall reaction times and higher baseline sleep values (calculated from the actigraphy data). Mean cumulative sleep debt, degree of acute sleep loss, and subjectively estimated sleep needs did not differ between the two groups. However, controllers who showed the anomalous effects were significantly younger, less experienced, and more likely to be females.

These differences, particularly the age, (which was confounded with lower experience), may explain the overall faster reaction times of this group (Thackery & Touchstone, 1981; Becker & Milke, 1998). In addition, their younger age may have assisted them in compensating for any cumulative sleep debt they experienced. However, it is likely their age, and in turn the lifestyle associated with younger controllers, might be more important in explaining the differences in baseline sleep values between these two groups. Younger controllers, with fewer family commitments, are likely to have more time to sleep during their days off. It is somewhat surprising that they are also likely to be female. It is also possible that the baseline values determined from sleep on days off, overestimated the amount of sleep an individual required to feel fully rested. If this was the case and such values were then compared to the amount of sleep obtained on work days, a “false” cumulative debt would be produced. Controllers might then not be as sleep deprived as first thought, possibly explaining the anomalous relationship with performance.

In contrast to the performance variables, prior wakefulness and the timing of the test influenced subjective estimates of sleepiness at the beginning of the night shift, with the differences due to these factors most obvious for ratings made after the performance test. These findings suggest that subjective estimates of sleepiness were more sensitive than reaction time performance to small changes in circadian phase, and to the homeostatic drive for sleep created through extended prior wakefulness under these particular conditions.

7.4.2 *Performance After the Napping Opportunity*

The only dependent variable systematically influenced by the nap sleep architecture was initial reaction time (measured by the average intercept of regression lines fitted across

the 10 minutes of the tests). Initial reaction time was faster when the nap included stage 3 sleep than when it did not. This was the only finding suggesting the benefit of SWS for performance. Because no further models produced this finding, particularly those relating to overall performance or slowest performance, there is very little support for this effect. Nevertheless, the possible benefit of a very small amount of stage 3 sleep warrants further investigation, particularly in association with the consequences for sleep inertia.

The most consistent predictor of performance and subjective sleepiness in the 2nd performance test was the timing of the test. However, few models produced any significant findings, suggesting that even the relationship between the timing of the test and performance outcomes is not particularly strong. Even the independent variable of nap sleep length did not reach statistical significance. This may be because most individuals in this sub-set of data had some sleep during the nap and the range of values was not wide enough to see the influence of the amount of sleep obtained.

In contrast, the analyses that addressed the relationship between the amount of sleep obtained in the nap and performance at both the 2nd and 3rd tests included a great number of cases where no sleep was obtained. These analyses showed that many aspects of performance were improved with greater amounts of sleep. Given the relatively small amount of sleep obtained this is a noteworthy finding.

Assessment of the estimated regression lines showed that at the end of a K1 night shift and middle of a K2 night shift, there was a tendency for a stronger relationship between the amount of sleep obtained during the nap and improved performance. The trend associated with the end of a K1 night shift is visible in the plots of the estimates (Figure 5-10 and Figure 5-11). They suggest that sleep obtained at work is most beneficial by the end of an early starting (K1) night shift compared to the end of the K2 night shift. Both the end of a K1 night shift and middle of a K2 night shift occurred during the earlier part of the circadian nadir (0300-0500 hours), and after less time-on-task. This possibly indicates that the reduced homeostatic drive for sleep (due to the nap) counters the circadian effect. The lack of an effect at the end of the K2 night shift could be due to the positive consequences of the nap having worn off, allowing circadian effects to be unmasked, and/or that time-on-task effects associated with a longer shift had become more important.

The models described in Chapter 5, section 5.6.2 suggest that for every 10 minutes of sleep an individual obtained during the nap opportunity, their mean performance would be approximately 2.65 ms faster. As expected, this is a small improvement compared to the changes in reaction time seen across long periods of sleep deprivation (Doran, Van Dongen & Dinges, 2001). For the slowest 10% of responses, 10 minutes of sleep made a much a larger difference, improving responses by 14.57 ms on average. Thus, after 40 minutes of sleep, slowest performance improved by just over 58 ms on average.

It could be assumed that faster reaction time performance equates to somewhat better performance in the controlling environment, but accuracy of responding would also be a crucial part of operational performance. In this instance greater amounts of sleep did not relate to fewer errors. However the sensitivity of the error measure used is of debatable relevance to changes in operational performance.

People do not know ahead of time how much sleep they might be able to get during a nap opportunity. However, it was demonstrated that the opportunity to nap improved many facets of reaction time performance independent of the amount or type of sleep obtained. Sleep patterns prior to the night shift did not predict any features of reaction time performance. Although, subjective ratings of sleepiness were significantly related to the level of acute sleep loss experienced and the time of the test. Taken together, these findings suggest that, for reaction time performance, sleep obtained at work has more influence than the sleep obtained prior to work (acute sleep loss and cumulative sleep debt). This highlights the value of workplace napping on the night shift.

Across the majority of analyses, the timing of the test was the most consistent predictor of the occurrence of performance lapses, with the end of the night shift being the most likely time for them to occur. This finding reinforces the importance of circadian related performance changes and suggests the nap sleep has little effect in improving performance at this end of the continuum. On the other hand, the variable of slowest 10% of responses was most consistently improved by the nap, which has also been reported in previous studies (Dinges, Whitehouse, Orne & Orne, 1988). The fact that performance lapses were represented as a binomial variable might reduce the sensitivity of these analyses to detect relationships.

7.5 Neurophysiological Alertness at the End of the Night Shift

Compressed spectral arrays were used to visualise possible changes that were occurring in the EEG data as a result of lowered neurophysiological alertness. The most striking aspect of these presentations was the individual variability across the four study conditions. This concurs with the findings from the literature of wide individual variability in EEG changes associated with changing levels of alertness. There was no clear shift in frequencies between either the nap and no nap conditions, or between the late and early shifts. A greater number of bursts of increased power were seen in those conditions where individuals would be expected to be sleepier and increased power was generally seen in the upper theta band/lower alpha ($\sim 7\text{-}8$ Hz), and/or upper delta frequencies ($\sim 3\text{-}4$ Hz).

After reviewing the literature it was initially proposed that only a few single frequency models might be run, using frequencies identified from the literature, for example 3 and 4 Hz. However, the variability seen in the compressed spectral arrays did not strongly identify any single frequency. Therefore, it was decided to run analyses with every single frequency below 16 Hz, in addition to the analyses in the four standard frequency bands.

7.5.1 *Influences of Napping and Shift Timing*

The amount of sleep obtained during the napping opportunity was seen to reduce the likelihood of SEMs occurring, and to decrease spectral power in all the standard frequency bands and most frequencies between 2 and 16 Hz. These findings remained relatively unchanged when analyses were re-run excluding the quantity of stage 1 sleep in the nap, or when analyses were repeated using only the data from 9 individuals with the greatest proportion of analysable EEG data. This robust, consistent, dose-dependent effect clearly supports the usefulness of sleep for reducing the occurrence of features in the EEG that are likely to be associated with lowered neurophysiological alertness.

A further consistent finding across all EEG frequency bands and single frequencies was the influence of the night shift worked, with lower alertness on the K2 night shift. This finding was independent of whether the data used were from all study participants or only those 9 with the greatest amount of analysable EEG data. Furthermore, the interaction of shift type with the quantity of nap sleep (or the nap/no-nap opportunity) was significant in a large number of analyses.

These findings suggest that circadian phase and/or time-on-task effects strongly influenced changes in spectral power. The 1 hour block of data at the end of the K2 night shift was on average from 0529-0629 hours, over 2 hours later than the average end of the K1 night shift. In addition, the K2 night shift was longer than the K1 shift by an average of 39 minutes. The greater time-on-task, in conjunction with the influence of the circadian nadir, might also have contributed to the significantly increased spectral power seen at the end of the K2 shift.

Where the interaction of sleep duration and shift type was significant, the estimates indicated that on the K1 night shift, greater amounts of sleep had a stronger affect on EEG power than on the K2 night shift. For the interaction of the nap opportunity with shift type, similar findings were seen. At the end of the K2 night shift when no nap opportunity had occurred, spectral power was significantly greater than under the other study conditions. Findings also indicated that in some instances, at the end of the K1 night shift with no nap, spectral power was lower than at the end of the K2 night shift after a nap had occurred. These findings further support the strength of the circadian effect and suggest that it potentially has a greater influence on spectral power than the napping opportunity.

There are some inconsistencies in the findings associated with the interaction of the amount of nap sleep with the night shift worked. For the group as a whole, this interaction was generally only significant for the theta band, or single frequencies in the theta range. However, when only the data from the 9 individuals were used, the tendency was for the interaction effect to be significant for most bands and frequencies, other than theta or frequencies in the theta range. This discrepancy in findings is not easy to interpret, but it may be due to individual differences in the patterns associated with decreasing alertness. It is possible that there were some individuals for whom changes in the theta band were the most sensitive measure to both the combined effect of the nap and changes in circadian phase. For others, the theta band may be less sensitive, with alertness-related changes occurring across a greater range of frequencies.

The finding that stage 3 sleep results in decreased power in the beta band, and at 6 Hz, is of some interest, particularly since the amount of stage 3 sleep obtained during the nap was relatively small. In addition, a greater number of ASDA arousals were related to increased power at 3 and 12 Hz. Given the small amount of EEG data available, this is a relatively surprising finding, as it was not expected that the changes in the EEG data

would be pronounced enough to be seen. These findings possibly indicate that sleep quality is important for determining the efficacy of the nap and that there may be a strong relationship between stage 3 sleep/ASDA arousals and improved alertness. Alternatively, the EEG is highly sensitive to variations in alertness. However, it should also be considered that these findings are artefacts. The occurrence of stage 3 sleep was related to increased power at 4 Hz, and the p values for many of these findings did not greatly exceed .5. Also because of the large number of models run, some of these statistical relationships may be due to chance alone (i.e. type I errors occurred).

7.5.2 *Influence of Prior Sleep Patterns*

Increasing acute sleep loss was consistently related to greater spectral power, demonstrating the importance of individuals obtaining sufficient sleep prior to commencing the night shift. In addition, greater cumulative sleep debt also led to increased spectral power in the delta and theta bands when nap sleep (stage 2-4) was used in the model rather than nap sleep (all stages). The findings from the smaller group of 9 participants showed that all the standard frequency bands were significantly affected by cumulative sleep debt, but again only in those models where nap sleep (stage 2-4) was included. Analyses of the 1 Hz frequency bands confirm the relationship between increasing cumulative sleep debt and greater spectral power.

These effects of acute sleep loss and cumulative sleep debt on spectral power stand in contrast to their lack of effects on performance. This discrepancy could be due to EEG spectral power being more sensitive to homeostatic influences than the performance measure used.

7.5.3 *Patterns Across Study Nights*

There was no consistent evidence for a “first night effect” (awareness of the equipment and study procedure resulting in increased alertness), as study nights one, three, and four seldom differed from each other on any of the variables examined. On the other hand, in almost all models, the second study night had lower spectral power than all other study nights. There is no obvious explanation for this phenomenon, given that study conditions (K1/K2, nap/no nap) were completely randomly.

7.6 Recommendations

Because this study was conducted in an operational environment, it was considered important to supply feedback to study participants, the participating organisation, and the regulatory body that oversees their activities.

7.6.1 *Recommendations for the Individual*

Air traffic controllers are strongly encouraged to make obtaining sufficient sleep a priority across the working week, and particularly prior to the night shift. This recommendation is based on the high levels of prior wake, acute sleep loss, cumulative sleep debt, and the small amount of pre-shift sleep seen in association with the night shift.

It is recognised that some of the findings from the present study contradict, or at least do not support, this recommendation. For example prior wake, acute sleep loss, and pre-shift sleep duration were not found to relate to performance at the beginning of night. It is also recognised that for some individuals, higher cumulative sleep debts related to faster performance at the start of the night shift. Possible reasons for these findings have been previously discussed.

It is also important to bear in mind that these findings only relate to the beginning of the night shift. Both greater acute sleep loss and cumulative debt were found to result in signs of lower neurophysiological alertness at end of the night shift. These prior sleep factors are also possibly more important in determining alertness than any sleep obtained at work. In addition, increased acute sleep loss and prior wake were seen to increase the likelihood of an individual entering stage 3 sleep during the nap sleep and waking from SWS, which should be avoided due to the increased likelihood and severity of sleep inertia.

It is also important for air traffic controllers to be aware that their subjective estimates of the quantity and quality of sleep they obtain at work are likely to be inaccurate. Therefore, they should not be discouraged from attempting to nap on subsequent occasions after experiencing what they feel is little or poor quality sleep.

The findings of the present study indicate that the greater the amount of sleep obtained during the nap, the less that performance and neurophysiological alertness declined by the end of the night shift. Even stage 1 sleep was demonstrated as beneficial, particularly

for reaction time performance. Thus, controllers should be encouraged to get any sleep that they can during work breaks. Without further information on the magnitude and time-course of sleep inertia after waking from naps of this duration, it is recommended that at the present time sleep should be limited to 40 minutes, and the greatest amount of time possible should be allowed between waking and returning to work.

Air traffic controllers should also be aware that the end of the K2 shift is likely to be a time of greatest vulnerability to a decline in performance and neurophysiological alertness, particularly since the positive influence of the nap may have dissipated and circadian and time-on-task effects are likely to be unmasked.

7.6.2 *Organisational Recommendations*

Management also needs to recognise that the end of a night shift, particularly the K2 shift, is a time when controllers may be most vulnerable to decreased performance and alertness. Although the operational significance of these changes is unknown, the potential exists for information to be missed when neurophysiological alertness is low. This is supported by the finding that poorest performance, evidenced by the greatest number of lapses, occurred at this time.

Furthermore discrepancies were noted between what is rostered and what is worked. Controllers were working for longer on the K2 night shift and the early morning shift that precedes it, than was rostered. The longer K2 shift occurred because air traffic controllers often arrange between themselves for the person working the K1 night shift to finish work earlier than rostered, leaving the person working the K2 shift to work a slightly longer shift. This also normally requires an air traffic controller to work for longer than 2 hours on the radar position at the end of the K2 shift despite the fact that regulations forbid exceeding 2 hours on position without a break. However, this informal arrangement is supported because it allows the person working the K1 shift to get home earlier, thus to some extent minimising the negative effects of the night shift for one person. Because all controllers are rostered on both night shifts, everyone has a turn at going home early, and on another occasion, working for longer.

Because the end of the K2 night shift is likely to be where the safety margin becomes the narrowest, it is recommended that procedures surrounding the K2 night shift be reviewed. Consideration needs to be given to limiting the maximum length of the night shift, particularly the K2 shift, and also the length of the time allowed on the radar

position when working at night. Currently, the hours of work limitations for air traffic controllers do not distinguish between maximum duty hours at night versus day. However, the additional demands of night work need to be taken into account if roster changes are planned. For example, rostering a controller to come into work earlier in order to take over from the person working the K2 night shift will have flow on effects, as they will normally be the person who is working the following night shift. Therefore, it is also necessary to look at the features of the early morning shift that precedes the night shift when reviewing what occurs on the night shift.

A relatively small percentage of the total study group completed “call-back” shifts on rostered days off. Within the employing organisation, a system tracks the number of “call-backs” worked in order to avoid the same individuals always being asked to work on their rostered days off. The study was not run over an entire year, during which it might be expected that all controllers would cycle through being at the top of the “call-back” list. However, it was run over a relatively long time frame (approximately 7 months) so it would be expected that the majority of individuals work “call-backs”. This was not the case, suggesting that this system is possibly not being utilised to its full capacity.

Because of the level of acute sleep loss, cumulative sleep debt, and short recovery sleep after the K2 night shift, it is recommended that “call-backs” are not worked after this shift. In fact, considering the levels of cumulative debt on both night shifts, any “call-back” without sufficient recovery sleep would not be recommended.

It was found that several air traffic controllers had difficulty falling asleep during the nap opportunity at work, and the average latency to the first minute of sleep was long. The number of ASDA arousals and awakenings was also high. One reason for the long latencies, poor quality sleep, and in some instances complete lack of sleep, may be the quality of the napping facilities. There is currently no area specifically set aside for napping. Some controllers use a sick bay area, but due to its location in the radar centre, many controllers complained that it was very noisy. Other controllers sleep on portable beds in office space, but computer noise and people passing by can cause disruptions.

Because there is a dose-response relationship between the amount of sleep obtained and the positive effects on performance and alertness, the more quality sleep people can get during the 40 minute napping opportunity the better. It is therefore recommended that

the napping environment be improved. A noise attenuated room, that is temperature controlled, able to be completely darkened, and has a phone for contacting or waking the napping controller would be ideal.

Although there is no way of knowing how much sleep an individual will obtain before they attempt to nap at work, the finding that the opportunity to nap related to improved performance and alertness provides the necessary support for a pro-napping policy at the management level. However, if napping during work breaks is officially sanctioned, then procedures for doing so must be developed and made clear. The protocol that was used for napping in the present study worked well, and it included features such as the napping controller informing the controller on position where they were sleeping and when they could be expected back. The maximum recommended length of the nap should be stated, as well as the need to take the nap early in the work break in order to allow the greatest amount of time between waking and returning to work. Better means of communication between the napping controller and the controller on position needs to be provided and used. A paging system or a telephone could be used to wake the napping controller and provide means of contact if they are required urgently.

7.6.3 *Regulatory Recommendations*

Currently the hours of work of air traffic controllers are only outlined in their employment contract. However, there are efforts underway by the Civil Aviation Authority of New Zealand (CAANZ) to regulate controllers' hours of work. Instead of stipulating maximum work hours and the minimum length of breaks, the CAANZ is putting in place regulations that would allow the air traffic control provider to develop a fatigue management programme. Such a programme is developed by the air traffic control provider and approved by the CAANZ, and would allow the air traffic control provider more flexibility in the determining hours of work than would rigid regulations. However, it would also require them to have in place a range of strategies to monitor and reduce fatigue in the controlling environment. The findings of the present study suggest that the regulator should encourage the air traffic control provider to include pre-planned workplace naps in their fatigue management programme.

7.7 Future Research

7.7.1 *Scoring Workplace Sleep and Alertness*

On only two occasions (each for 1 minute) in the present study was sleep scored outside the napping opportunity at a time when it was not expected. Numerous changes indicating stage 1 sleep were seen but for an insufficient period of time to qualify as such according to the criteria of Rechtschaffen and Kales (1968). Hori (1994) and Ogilvie (1991) have shown that there are fleeting changes during the sleep onset process that are not detected when using the relatively coarse standard definitions of stage 1 sleep. Hori (1994) determined that 9 discrete stages occur prior to stage 2 sleep, which are best detected using epochs of 5 seconds in length. Wright and McGown (2001) analysed polysomnographic recordings from long-haul flight crew in an effort to determine the quantity of sleep, and frequency with which signs of sleep occurred. In this study, the criteria of Rechtschaffen and Kales (1968) were predominantly utilised, but in epochs of 5 seconds duration.

Participants in the present study, as in any working environment, would normally be attempting to remain awake. Therefore, any signs of sleep that do occur are likely to be brief. Furthermore, work tasks, or environmental stimuli would be likely to interrupt any sleep that does occur. To detect the short and fleeting changes into the early stages of sleep, the use of a more sensitive system, focussed on shorter epochs, might be better suited in such a context. However, the methods of Hori (1994) and Wright (2001) are currently not widely used or accepted as standard practice for scoring sleep.

It may also be that determining the spectral power in the EEG is a more efficient and informative process than using the labels associated with the traditional sleep stages. On the other hand, the traditional sleep stages have the advantage of being recognised and understood by both researchers and lay people.

One of the major problems associated with analysis of neurophysiological data is the need to exclude epochs containing artefact, and as has been demonstrated, artefact (particularly due to eye and muscle movement) is extensive in ambulatory data. This seriously limits the usefulness of this method despite its demonstrated sensitivity. However, in the present study, short controlled periods of neurophysiological data were also recorded as part of the Alpha Attenuation Test (AAT). It was decided early on in the analyses that this data set would not be included as part of this thesis. This decision

was based on the large amount of data already included, and it was deemed important to investigate what was occurring while air traffic controllers were operational. It would be useful to analyse the data collected during the AAT and compare the findings to those from the ambulatory data.

Further, there are many possible ways in which the EEG data could be analysed. The use of both traditional frequency bands and single frequencies in the present study was done to try and gain a more complete picture of these changes. Nevertheless, one of the downfalls of this approach was that the level of change in the bands or frequencies could not be determined. One means of doing this would be to look at the frequency with which epochs exceed baseline power values by a certain degree (100%, 200% or 300%). This is also known as burst analysis (Åkerstedt, Kecklund & Knutsson, 1991; Kecklund & Åkerstedt, 1993). This process has the added advantage of not requiring multiple epochs of data to be aggregated and therefore might also be more sensitive. The main reason this approach was not employed in the present study was because of difficulties determining an appropriate baseline value. Further consideration needs to be given to this form of data analysis.

In summary, there are a number of possible methods that may be used in the future to examine neurophysiological alertness in a working environment. All of the methods discussed here, sleep scoring in short epochs, spectral analysis of ambulatory data, and spectral analysis of data recorded in controlled conditions, warrants further investigation. A comparison of these methods would be useful, as well as clarification of the most suitable means for collecting and analysing such data. With the interest in measuring neurophysiological alertness under working conditions continuing to grow, there is a strong need to develop standard methods.

7.7.2 *Automated Artefact Detection*

A choice was made to visually screen the EEG data in the present study for artefact, rather than use any of the available automated artefact detection and rejection methods. This was largely due to the lack of proven superiority to visual screening. However, one of the single largest tasks in this study was the visual screening of the EEG data for artefact. Furthermore the large amount of artefact that was removed limits the usefulness of the EEG as a continuous measure of alertness.

Automated artefact detection methods would certainly be worth considering if they could at least match the sensitivity and specificity of the human scorer, reduce the time required for screening, and possibly increase the amount of clean data available for analysis. One complex computational method currently becoming more widely used is Independent Components Analysis (ICA). Using the combined data recorded from multiple EEG, EOG and EMG channels, this method mathematically determines the various components that make up the combined signal independently of the channel they were recorded from. Therefore, signals such as the EOG and EMG that might be recorded in the EEG channels as artefact can be separated out. However, this method is not without its drawbacks. Until recently it required an advanced understanding of digital signal processing technology, and a large computational capacity. However, more recently sub-programmes are becoming available that can be used by individuals with less expertise in the subject area.

7.7.3 *Use of EOG versus EEG*

In many instances it is not possible, or desirable, to record multiple channels of neurophysiological data. It is therefore worth considering whether one neurophysiological measure is more useful than another for determining alertness. In terms of the quantity of data available, the present study suggests that a greater portion of the EOG channel is useful. It would be useful to make a statistical comparison between the quantity of data excluded in the EEG and EOG channels. However, for the purposes of this thesis the percentage of the EOG channel that was excluded due to artefact was not determined.

Individual variability in neurophysiological measures of alertness is another issue. In the present study, SEMs were not found to occur in all individuals. However, this may be due to the level of alertness seen, as Santamaria and Chiappa (1987) state that classic drowsiness is always accompanied by SEMs. Furthermore, when they do occur, SEMs are consistently related to diminished alertness. The reviewed literature, and the compressed spectral arrays plotted in the present study, suggests that changes in the EEG with alertness are extremely variable between individuals. In addition, the exact EEG changes that signify decreased alertness for an individual are not known. Instead, the general changes that occur are better understood.

The sensitivity of the neurophysiological measures is also of importance. The findings from the present study indicate that EEG data are more sensitive to both circadian and homeostatic influences than the EOG. However, the lack of sensitivity of the SEM variable may be because the variable was dichotomised, while the EEG variables could be transformed to produce normal distributions.

In summary, there are pros and cons with both measures. In situations where a large amount of artefact is expected to occur, the EOG signal may provide a more representative picture of events. If however, sensitivity of the measure is the primary concern then recording an EEG channel will be a higher priority.

7.7.4 *Sleep Inertia*

The present study took a rudimentary approach to examining the occurrence of sleep inertia, as this was not the focus of the investigation. Fortunately, air traffic controllers also have a relatively long break during the night shift, that allows a 40 minute nap to be followed by approximately an hour of wake before they must return to work. Many working environments could not allow such an extended period of time between a nap and returning to work, and therefore the issue of sleep inertia would be of greater concern.

Currently, there is much literature on the severity and time course of sleep inertia after waking from a night of sleep, or after waking from shorter episodes of sleep but under conditions of prolonged wakefulness. There is a need for further information on the severity and time course of sleep inertia after short (less than 1 hour) episodes of sleep, and the factors that influence its severity and time course. This is critical information if napping is to be implemented in a wider range of working environments.

7.8 Conclusion

This study has provided further evidence on the sleep consequences of the backward, rapidly rotating shift cycle. High levels of cumulative sleep debt were shown to accrue across the working week, resulting in the night shift occurring under less than ideal conditions.

The disparity of the self-reports of study participants on the quantity and quality of sleep obtained at work with the data obtained polysomnographically, highlighted the need to

use objective measures to make such assessments. It was also proven that this type of technology is easily utilised in an operational context.

The findings clearly demonstrated that, despite sleep being short and of relatively poor quality, it improved the reaction time performance and neurophysiological alertness of air traffic controllers on the night shift. By using a valid and reliable measure of performance and a sensitive measure of neurophysiological functioning, the effect of a minimal quantity of sleep has been demonstrated despite the “noise” of a field setting. These findings therefore provide a link between laboratory studies of napping and the actual work environment.

The study findings fully support management endorsing a 40 minute napping opportunity for area controllers. With further information on the occurrence of sleep inertia after short episodes of sleep, these findings may also be generalised to other operational environments.

REFERENCES

- Achermann, P., & Borbély, A. A. (1992). Combining different models of sleep regulation. *Journal of Sleep Research*, 1, 144-147.
- Achermann, P., Werth, E., Dijk, D.-J., & Borbély, A. A. (1995). Time course of sleep inertia after nighttime and daytime sleep episodes. *Archives Italiennes de Biologie*, 134, 109-119.
- Aeschbach, D., Matthews, J. R., Postolache, T. T., Jackson, M. A., Giesen, H. A., & Wehr, T. A. (1997). Dynamics of the human EEG during prolonged wakefulness: Evidence for frequency-specific circadian and homeostatic influences. *Neuroscience Letters*, 239(2-3), 121-124.
- Agnew, H. W., Webb, W. B., & Williams, R. L. (1966). The first night effect: an EEG study of sleep. *Psychophysiology*, 2, 263 - 266.
- Aikin, M., & Gensler, H. (1996). Adjusting for multiple testing when reporting research results: The Bonferroni vs Holm methods. *American Journal of Public Health*, 86(5), 726-728.
- Åkerstedt, T., & Torsvall, L. (1981). Shiftwork. Shift dependent well-being and individual differences. *Ergonomics*, 24, 265-273.
- Åkerstedt, T., & Torsvall, L. (1985). Napping in shift work. *Sleep*, 8, 105-9.
- Åkerstedt, T., & Gillberg, M. (1986). A dose-response study of sleep loss and spontaneous sleep termination. *Psychophysiology*, 23(3), 293-297.
- Åkerstedt, T., Torsvall, L., & Gillberg, M. (1987). Sleepiness in shiftwork. A review with emphasis on continuous monitoring of EEG and EOG. *Chronobiology International*, 4(2), 129-140.
- Åkerstedt, T., & Gillberg, M. (1990). Subjective and objective sleepiness in the active individual. *International Journal of Neuroscience*, 52(1-2), 29-37.

- Åkerstedt, T. (1991). Sleepiness at work: Effects of irregular work hours. In T. H. Monk (Ed.), *Sleep, Sleepiness, and Performance* (pp. 129-152). New York: John Wiley and Sons Ltd.
- Åkerstedt, T., Kecklund, G., & Knutsson, A. (1991). Manifest sleepiness and the spectral content of the EEG during shift work. *Sleep*, 14, 221-215.
- Åkerstedt, T., Hume, K., Minors, D., & Waterhouse, J. (1993). Regulation of sleep and naps on an irregular schedule. *Sleep*, 16, 736-743.
- Åkerstedt, A., Hume, K., Minors, D., & Waterhouse, J. (1994a). The meaning of good sleep: A longitudinal study of polysomnography and subjective sleep quality. *Journal of Sleep Research*, 3, 152-158.
- Åkerstedt, T., Hume, K., Minors, D., & Waterhouse, J. (1994b). The subjective meaning of good sleep: An intraindividual approach using the Karolinska Sleep Diary. *Perceptual & Motor Skills*, 79(1, Pt 1), 287-296.
- Åkerstedt, T., & Folkard, S. (1995). Validation of the S and C components of the three-process model of alertness regulation. *Sleep*, 18(1), 1-6.
- Åkerstedt, T., Hume, K., Minors, D., & Waterhouse, J. (1997). Good sleep: Its timing and physiological sleep characteristics. *Journal of Sleep Research*, 6(4), 221-229.
- Åkerstedt, A. (1998). Is there an optimal sleep-wake pattern in shift work? *Scandinavian Journal of Work, Environment, & Health*, 24(Suppl. 3), 18-27.
- Åkerstedt, T., & Landström, U. (1998). Work place countermeasures of night shift fatigue. *International Journal of Industrial Ergonomics*, 21, 167-178.
- Åkerstedt, T., Kecklund, G., & Hörte, L.-G. (2001). Night driving, season, and the risk of highway accidents. *Sleep*, 24, 401-406.
- American Sleep Disorders Association (1992). EEG arousals: Scoring rules and examples. *Sleep*, 15(2), 174-184.

- Ancoli-Israel, S. (2000). Actigraphy. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and Practice of Sleep Medicine*. (3rd ed., pp. 1295-1301). Philadelphia: W. B. Saunders Company.
- Andersen, J. E. (1970). *Three-Shift Work - A Socio-Medico Investigation*. Copenhagen: Teknisk Forlag.
- Angiboust, R., & Gouars, M. (1972). Tentative d'evaluation de l'efficacité operationelle dur personnel de l'aeronautique militaire au cours de veilles nocturnes. In W. P. Colquhoun (Ed.), *Aspects of Human Efficiency: Diurnal Rhythm and Loss of Sleep* (pp. 151-170). London: English Universities Press.
- Angus, R. G., & Heslegrave, R. J. (1985). Effects of sleep loss on sustained cognitive performance during a command and control simulation. *Behavior Research Methods, Instruments, & Computers*, 17(1), 55-67.
- Angus, R. G., Heslegrave, R. J., & Myles, W. S. (1995). Effects of prolonged sleep deprivation, with and without chronic physical exercise, on mood and performance. *Psychophysiology*, 22, 276-282.
- Arendt, J., Stone, B., & Skent, D. (2000). Jet lag and sleep disruption. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and Practice of Sleep Medicine* (3rd ed., pp. 591-599). Philadelphia: W. B. Saunders Company.
- Aschoff, J., Fatranská, M., & Giedke, H. (1971). Human circadian rhythms in continuous darkness: Entrainment by social cues. *Science*, 171, 213-215.
- Aschoff, J. (1994). Naps as integral parts of the wake time within the human sleep-wake cycle. *Journal of Biological Rhythms*, 9(2), 145-55.
- Aserinsky, E., & Kleitman, N. (1953). Regularly occurring periods of eye motility, and concomitant phenomena, during sleep. *Science*, 118, 273-274.
- Badia, P., Wright, K. P. J., & Wauquier, A. (1994). Fluctuations in single-hertz EEG activity during transition to sleep. In R. D. Ogilvie & J. R. Harsh (Ed.), *Sleep Onset: Normal and Abnormal Processes* (pp. 201-218). Washington, DC: American Psychological Association.

- Baranski, J. V., Pigeau, R. A., & Angus, R. G. (1994). On the ability to self-monitor cognitive performance during sleep deprivation: A calibration study. *Journal of Sleep Research*, 3, 36-44.
- Becker, J. T., & Milke, R. M. (1998). Cognition and aging in a complex work environment: Relationships with performance among air traffic control specialists. *Aviation, Space, & Environmental Medicine*, 69(10), 944-951.
- Belyavin, A., & Wright, N. A. (1987). Changes in electrical activity of the brain with vigilance. *Electroencephalography and clinical Neurophysiology*, 66, 137-144.
- Bennington, J. H. (2000). Sleep homeostasis and the function of sleep. *Sleep*, 23(7), 959-966.
- Bjerner, B. (1949). Alpha depression and lowered pulse rate during delayed actions in a serial reaction test: A study of sleep deprivation. *Acta Psychologica Scandinavica*, 19(Suppl. 65), 1-93.
- Bjerner, B., Holm, A., & Swensson, A. (1955). Diurnal variation of mental performance. A study of three-shift workers. *British Journal of Industrial Medicine*, 12, 103-110.
- Blagrove, M., Alexander, C., & Horne, J. A. (1995). The effects of chronic sleep reduction on the performance of cognitive tasks sensitive to sleep deprivation. *Applied Cognitive Psychology*, 9(1), 21-40.
- Bland, J. M., & Altman, D. G. (1986). Statistical methods for assessing agreement between two methods of clinical measurement. *The Lancet*, February 8, 307-310.
- Boivin, D. B., Duffy, J. F., Kronauer, R. E., & Czeisler, C. A. (1996). Dose-response relationships for resetting of human circadian clock by light. *Nature*, 379, 540-542.
- Bonnefond, A., Muzet, A., Winter-Dill, A.-S., Bailloeuil, C., Bitouze, F., & Bonneau, A. (2001). Innovative working schedule: Introducing one short nap during the night shift. *Ergonomics*, 44(10), 937-945.
- Bonnet, M. H. (1986). Performance and sleepiness as a function of frequency of sleep disruption. *Psychophysiology*, 23, 263-271.

- Bonnet, M. H. (1991). The effect of varying prophylactic naps on performance, alertness and mood throughout a 52-hour continuous operation. *Sleep*, 14(4), 307-315.
- Bonnet, M. H., & Arand, D. L. (1994a). Impact of naps and caffeine on extended nocturnal performance. *Physiology & Behavior*, 56(1), 103-109.
- Bonnet, M. H., & Arand, D. L. (1994b). The use of prophylactic naps and caffeine to maintain performance during a continuous operation. *Ergonomics*, 37(6), 1009-1020.
- Bonnet, M. H., & Arand, D. L. (1995). We are chronically sleep deprived. *Sleep*, 18(10), 908-911.
- Bonnet, M. H., Gomez, S., Wirth, O., & Arand, D. L. (1995). The use of caffeine versus prophylactic naps in sustained performance. *Sleep*, 18(2), 97-104.
- Bonnet, M. H. (2000). Sleep Deprivation. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and Practice of Sleep Medicine*. (3rd ed., pp. 53-71). Philadelphia: W. B. Saunders Company.
- Borbély, A. A., & Achermann, P. (1992). Concepts and models of sleep regulation: An overview. *Journal of Sleep Research*, 1, 63-79.
- Borbély, A. A., & Achermann, P. (1999). Sleep homeostasis and models of sleep regulation. *Journal of Biological Rhythms*, 14(6), 557-568.
- Broughton, R., & Hasan, J. (1995). Quantitative topographic electroencephalographic mapping during drowsiness and sleep onset. *Journal of Clinical Neurophysiology*, 12(4), 372-386.
- Broughton, R. J. (1983). The seista: Social or biological phenomenon? *Sleep Research*, 12, 28.
- Broughton, R. J. (1989). Chronobiological aspects and models of sleep and napping. In D. F. Dinges & R. J. Broughton (Eds.), *Sleep and Alertness: Chronobiological, Behavioral, and Medical Aspects of Napping* (pp. 71-98). New York: Raven Press.
- Brown, R. C. (1949). The day and night performance of teleprinter switchboard operators. *Occupational Psychology*, 23, 121-126.

- Bruck, D., & Pisani, D. L. (1999). The effects of sleep inertia on decision-making performance. *Journal of Sleep Research*, 8(2), 95-103.
- Cajochen, C., Brunner, D. P., Kräuchi, K., Graw, P., & Wirz-Justice, A. (1995). Power density in theta/alpha frequencies of the waking EEG progressively increases during sustained wakefulness. *Sleep*, 18(10), 890-894.
- Cajochen, C., Khalsa, S. B., Wyatt, J. K., Czeisler, C. A., & Dijk, D.-J. (1999). EEG and ocular correlates of circadian melatonin phase and human performance decrements during sleep loss. *American Journal of Physiology*, 277(3), R640-649.
- Cajochen, C., Knoblauch, V., Kräuchi, K., Renz, C., & Wirz-Justice, A. (2001). Dynamics of frontal EEG activity, sleepiness and body temperature under high and low sleep pressure. *NeuroReport*, 12(10), 2277-2281.
- Caldwell, J. A., Jr., & Caldwell, J. L. (1998). Comparison of the effects of zolpidem-induced prophylactic naps to placebo naps and forced rest periods in prolonged work schedules. *Sleep*, 21(1), 79-90.
- Campbell, K. B. (2000). Introduction to special section: Information processing during sleep onset and sleep. *Canadian Journal of Experimental Psychology*, 54(4), 209-218.
- Carrier, J., Monk, T. H., Buysse, D., & Kupfer, D. (1997). Sleep and morningness-eveningness in the 'middle' years of life (20-59 y). *Journal of Sleep Research*, 6, 230-237.
- Carskadon, M. A., & Dement, W. C. (1977). Sleep tendency: An objective measure of sleep loss. *Sleep Research*, 6, 200.
- Carskadon, M. A., & Dement, W. C. (1981). Cumulative effects of sleep restriction on daytime sleepiness. *Psychophysiology*, 18(2), 107-113.
- Carskadon, M. A., & Dement, W. C. (1982). Nocturnal determinants of daytime sleepiness. *Sleep*, 5, S73-S81.
- Carskadon, M. A., & Roth, T. (1991). Sleep restriction. In T. H. Monk (Ed.), *Sleep, Sleepiness, and Performance* (pp. 155 - 167). Chichester: John Wiley & Sons.

- Carskadon, M. A., & Dement, W. C. (1992). Multiple sleep latency tests during the constant routine. *Sleep*, 15(5), 396-399.
- Carskadon, M. A. (1994). Measuring daytime sleepiness. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and Practices of Sleep Medicine*. (2nd ed., pp.961-966) Philadelphia: W. B. Saunders Company.
- Carskadon, M. A., & Dement, W. C. (2000). Normal human sleep: An overview. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and Practice of Sleep Medicine*. (3rd ed., pp. 16-25). Philadelphia: W. B. Saunders Company.
- Carskadon, M. A., & Rechtschaffen, A. (2000). Monitoring and Staging Human Sleep. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and Practice of Sleep Medicine*. (3rd ed., pp. 1197-1215). Philadelphia: W. B. Saunders Company.
- Colquhoun, W. P. (1971). *Biological rhythms and human performance*. London: Academic Press.
- Comperatore, C. A., & Krueger, G. P., Eds. (1990). Circadian rhythm desynchronis, jet lag, shift lag, and coping strategies. *Occupational Medicine: State of the Art Reviews*. Philadelphia, Hanley & Belfus, Inc.
- Corsi-Cabrera, M., Arce, C., Ramos, J., Lorenzo, I., & Guevara, M. A. (1996). Time course of reaction time and EEG while performing a vigilance task during total sleep deprivation. *Sleep*, 19(7), 563-569.
- Costa, G., Schallenberg, G., Ferracin, A., & Gaffuri, E. (1995). Psychophysical conditions of air traffic controllers evaluated by the standard shiftwork index. *Work & Stress*, 9(2/3), 281-288.
- CRATCOH (1990). *Report of a Committee on Regulation of Air Traffic Controllers' Hours to the Civil Aviation Authority*. Cheltenham, UK: Civil Aviation Authority.
- Cruz, C. E., & Della-Rocco, P. S. (1995a). *Investigation of sleep patterns among air traffic control specialists as a function of time off between shifts in rapidly rotating work schedules*. 8th International Symposium on Aviation Psychology, Ohio: Ohio State University.

- Cruz, C. E., & Della-Rocco, P. S. (1995b). *Sleep patterns in air traffic controllers working rapidly rotating shifts: A field study*. (DOT/FAA/AM-95/12). Oklahoma City: Civil Aeromedical Institute, Federal Aviation Administration.
- Cruz, C. E., Della-Rocco, P. S., & Hackwaorth, C. (2000). Effects of quick rotating shift schedules on the health and adjustment of air traffic controllers. *Aviation, Space, & Environmental Medicine*, 71(4), 400-407.
- Czeisler, C. A., Duffy, J. F., Shanahan, T. L., Brown, E. N., Mitchell, J. F., Rimmer, D. W., Ronda, J. M., Silva, E. J., Allan, J. S., Emens, J. S., Dijk, D.-J., & Kronauer, R. E. (1999). Stability, precision, and near 24-hour period of the human circadian pacemaker. *Science*, 284(5423), 2177-2181.
- Czeisler, C. A., & Khalsa, S. B. S. (2000). The human circadian timing system and sleep-wake regulation. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and Practice of Sleep Medicine*. (3rd ed., pp. 353-375). Philadelphia: W. B. Saunders Company.
- Daan, S., Beersma, D. G. M., & Borbely, A. A. (1984). Timing of human sleep: Recovery process gated by a circadian pacemaker. *American Journal of Physiology*(246), R161-R178.
- Dahlgren, K. (1981). Adjustment of circadian rhythms to rapidly rotating shiftwork: A field study of two shift systems. In A. Reinberg, N. Vieux & P. Andlauer (Eds.), *Night and Shiftwork: Biological and Social Aspects* (pp. 357-364). New York: Pergamon Press.
- Daniel, R. S. (1967). Alpha and theta EEG in vigilance. *Perceptual & Motor Skills*, 25, 697-703.
- Davis, H., Davis, P. A., Loomis, A. L., Harvey, E. N., & Hobart, G. A. (1938). Human brain potentials during the onset of sleep. *Journal of Neurophysiology*, 1, 24-38.
- De Gennaro, L., Ferrara, M., Ferlazzo, F., & Bertini, M. (2000). Slow eye movements and EEG power spectra during wake-sleep transition. *Clinical Neurophysiology*, 111, 2107-2115.

- Della-Rocco, P. S., & Cruz, C. E. (1995). *Shift work, age, and performance: Investigation of the 2-2-1 shift schedule used in air traffic control facilities I. The sleep/wake cycle*. (DOT/FAA/AM-95/19). Oklahoma City: Civil Aeromedical Institute, Federal Aviation Administration.
- Della-Rocco, P. S. (1999). *The Role of Shift Work and Fatigue in Air Traffic Control Operational Errors and Incidents*. (DOT/FAA/AM-99/02). Washington, DC: Federal Aviation Administration, Office of Aviation Medicine.
- Della-Rocco, P. S., Comperatore, C. A., Caldwell, L., & Cruz, C.E. (2000). *The effects of napping on night shift performance*. (DOT/FAA/AM-00/10). Oklahoma City, Oklahoma, Civil Aeromedical Institute, Federal Aviation Administration.
- Dement, W. C., & Greenberg, S. (1966). Changes in total amount of stage 4 sleep as a function of partial sleep deprivation. *Electroencephalography & Clinical Neurophysiology*, 20, 523-526.
- Dement, W. C. (2000). History of sleep physiology and medicine. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and Practice of Sleep Medicine*. (3rd ed., pp. 1-14). Philadelphia: W. B. Saunders Company.
- Devoto, A., Lucide, F., Volani, C., & Bertini, M. (1999). Effects of different sleep reductions on daytime sleepiness. *Sleep*, 22, 336-343.
- Dijk, D. J., Beersma, D. G., & Daan, S. (1987). EEG power density during nap sleep: Reflection of an hourglass measuring the duration of prior wakefulness. *Journal of Biological Rhythms*, 2(3), 207-219.
- Dijk, D.-J., & Czeisler, C. A. (1995). Contribution of the circadian pacemaker and the sleep homeostat to sleep propensity, sleep structure, electroencephalographic slow waves, and sleep spindle activity in humans. *The Journal of Neuroscience*, 15(5), 3526-3538.
- Dijk, D.-J. (1997). Light, circadian rhythms, and the homeostatic regulation of human sleep. In J. M. Kinney & H. N. Tucker (Eds.), *Physiology, Stress, and Malnutrition: Functional Correlates, Nutritional Intervention* (pp. 55-78). Philadelphia: Lipponcott-Raven.

- Dijk, D.-J., Duffy, J. F., Riel, E., Shanahan, T. L., & Czeisler, C. A. (1999). Ageing and the circadian and homeostatic regulation of human sleep during forced desynchrony of rest, melatonin and temperature rhythms. *Journal of Physiology*, 516, 611-627.
- Dijk, D.-J., & Edgar, D. M. (1999). Circadian and homeostatic control of wakefulness and sleep. In F. W. Turek & P. C. Zee (Eds.), *Regulation of Sleep and Circadian Rhythms* (pp. 111-147). New York: Marcel Dekker, Inc.
- Dijk, D.-J., Jewett, M. E., Czeisler, C. A., & Kronauer, R. E. (1999). Reply to Technical Note: Nonlinear interactions between circadian and homeostatic processes: Models or metrics? *Journal of Biological Rhythms*, 14(6), 604-605.
- Dijk, D.-J., Duffy, J. F., & Czeisler, C. A. (2000). Contribution of circadian physiology and sleep homeostasis to age-related changes in human sleep. *Chronobiology International*, 17, 285-311.
- Dinges, D. F., Orne, E. C., Evans, F. J., & Orne, M. T. (1981). Performance after naps in sleep-conducive and alerting environments. In L. Johnson, D. Tepas, W. Colquhoun & M. J. Colligan (Eds.), *Biological Rhythms, Sleep and Shiftwork: Advances in Sleep Research* (pp. 539-522) New York: Spectrum.
- Dinges, D. F., Orne, M. T., & Orne, E. C. (1985a). Assessing performance upon abrupt awakening from naps during quasi-continuous performance. *Behavior Research Methods, Instruments, & Computers*, 17, 37-45.
- Dinges, D. F., Orne, M. T., & Orne, E. C. (1985b). Sleep depth and other factors associated with performance upon abrupt awakening. *Sleep Research*, 14, 92.
- Dinges, D. F., & Powell, J. P. (1985). Microcomputer analysis of performance on a portable, simple visual RT task during sustained operation. *Behavior Research Methods, Instruments, & Computers*, 17, 652-655.
- Dinges, D. F., Orne, M. T., Whitehouse, W. G., & Orne, E. C. (1987). Temporal placement of a nap for alertness: Contributions of circadian phase and prior wakefulness. *Sleep*, 10(4), 313-29.

- Dinges, D. F., & Powell, J. W. (1988). Sleepiness is more than lapsing. *Sleep Research*, 17, 84.
- Dinges, D. F., Whitehouse, W. G., Orne, E. C., & Orne, M. T. (1988). The benefits of a nap during prolonged work and wakefulness. *Work & Stress*, 2(2), 139-153.
- Dinges, D. F. (1989). The nature of sleepiness: Causes, contexts and consequences. In A. Stunkard & A. Baum (Eds.), *Perspectives in Behavioural Medicine: Eating, Sleeping and Sex* (pp. 147-179) Hillsdale: Lawrence Earlbaum.
- Dinges, D. F., & Powell, J. W. (1989). Sleepiness impairs optimum response capability. *Sleep Research*(18), 366.
- Dinges, D. F. (1990). Are you awake? Cognitive performance and reverie during hypnopomic state. In R. Bootzin, J. Kihlstrom & D. Schacter (Eds.), *Sleep and Cognition* (pp. 159-175). Washington: American Psychological Association.
- Dinges, D. F. (1991). *What management should know about fatigue in corporate aviation safety*. Flight Safety Foundation 36th Corporate Air Safety Seminar, White Plains, NY: Flight Safety Foundation.
- Dinges, D. F., & Kribbs, N. B. (1991). Performing while sleepy: Effects of experimentally-induced sleepiness. In T. H. Monk (Ed.), *Sleep, Sleepiness, and Performance* (pp. 97-128): John Wiley and Sons Ltd.
- Dinges, D. (1992a). Probing the limits of functional capability: The effects of sleep loss on short-duration tasks. In R. Ogilvie (Ed.), *Sleep, Arousal, and Performance* (pp. 176-187). Boston: Birkhauser.
- Dinges, D. F. (1992). Adult napping and its effects on ability to function. In C. Stampi (Ed.), *Why We Nap: Evolution, Chronobiology, and Functions of Ultrashort Sleep* (pp. 118-134). Boston, MA: Birkhauser.
- Dinges, D. F., Douglas, S. D., Zaugg, L., Campbell, D. E., Mcmann, J. M., Whitehouse, W. G., Orne, E. C., Kapoor, S. C., Icaza, E., & Orne, M. T. (1994). Leukocytosis and natural killer cell function parallel neurobehavioural fatigue induced by 64 h of sleep deprivation. *The Journal of Clinical Investigation*, 93, 1930-1939.

- Dinges, D. F., Pack, F., Williams, K., Gillen, K. A., Powell, J. W., Ott, G. E., Aptowicz, C., & Pack, A. I. (1997). Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4-5 hours per night. *Sleep*, 20(4), 267-277.
- Dinges, D. F., Mallis, M. M., Maislin, G., & Powell, J. W. (1998). *Evaluation of Techniques for Ocular Measurement as an Index of Fatigue and as the Basis for Alertness Management*. (DOT HS 808 762) Washington, DC: National Highway Traffic Safety Administration.
- Dinges, D. F., Maislin, G., Kuo, A., Carlin, M. M., Powell, J. W., Van Dongen, H. P. A., & Mullington, J. M. (1999). Chronic sleep restriction: neurobehavioural effects of 4hr, 6hr, and 8hr TIB. *Sleep*, 22(Suppl. 1), S115-S116.
- Doran, S. M., Van Dongen, H. P. A., & Dinges, D. F. (2001). Sustained attention performance during sleep deprivation: Evidence of state instability. *Archives Italiennes de Biologie*, 139, 253-267.
- Drummond, S. P. A., Brown, G. A., Stricker, J. L., Buxton, R. C., Wong, E. C., & Gillin, J. C. (1999). Sleep deprivation-induced reduction in cortical functioning response to serial subtraction. *NeuroReport*, 10, 3745-3748.
- Drummond, S. P. A., Brown, G. A., Gillin, J. C., Stricker, J. L., Wong, E. C., & Buxton, R. C. (2000). Altered brain response to verbal learning following sleep deprivation. *Nature*, 403, 655-657.
- Englund, C. E., Ryman, D. H., Naitoh, P., & Hodgdon, J. A. (1985). Cognitive performance during successive sustained physical work episodes. *Behavior Research Methods, Instruments, & Computers*, 17, 75-85.
- Evans, F. J., & Orne, M. T. (1975). *Recovery from fatigue*. (NTIS No. A100347). Fort Detrick, Frederick, MD: U. S. Army Medical Research and Development Command.
- Feinberg, I., Fein, G., & Floyd, T. C. (1980). EEG patterns during and following extended sleep in young adults. *Electroencephalography and Clinical Neurophysiology*, 50, 467-476.

- Ferrara, M., & Gennaro, L. D. (2001). How much sleep do we need? *Sleep Medicine Reviews*, 5(2), 155-179.
- Fisch, B. J. (1991). *Speblmann's EEG Primer*. Amsterdam: Elsevier.
- Fischgold, H., Schwartz, B. A., & Dreyfuss-Brisac, C. (1959). Indicateur de l'état de présence et tracés électroencéphalographiques dans le sommeil nembutalique. *Electroencephalography & Clinical Neurophysiology*, 11, 23-33.
- Folkard, S., & Condon, R. (1984). Night shift paralysis in air traffic controllers. *Ergonomics*, 30, 1353-1363.
- Folkard, S., & Åkerstedt, A. (1992). A three-process model of the regulation of alertness-sleepiness. In R. J. Broughton & R. D. Ogilvie (Eds.), *Sleep, Arousal, and Performance* (pp. 11-26). Boston: Birkhäuser.
- Fruhstorfer, H., Langanke, P., Meinzer, K., Peter, J. H., & Pfaff, U. (1977). Neurophysiological vigilance indicators and operational analysis of a train monitoring device: A laboratory and field study. In R. R. Mackie (Ed.), *Vigilance* (147-162). New York: Plenum Press.
- Gagnon, P., De Koninck, J., & Broughton, R. (1985). Reappearance of electroencephalogram slow waves in extended sleep with delayed bedtime. *Sleep*, 8, 118-128.
- Gander, P. H., Nguyen, D., Rosekind, M. R., & Connell, L. J. (1993). Age, circadian rhythms, and sleep loss in flight crews. *Aviation, Space, & Environmental Medicine*, 64, 189-195.
- Gander, P. H., Barnes, R. M., Gregory, K. B., Graeber, R. C., Connell, L. J., & Rosekind, M. R. (1998a). Flight Crew Fatigue III: North Sea Helicopter Air Transport Operations. *Aviation, Space, and Environmental medicine*, 69(9), B16 - B25.
- Gander, P. H., Barnes, R. M., Gregory, K. B., Graeber, R. C., Connell, L. J., & Rosekind, M. R. (1998b). Flight crew fatigue III: North sea helicopter air transport operations. *Aviation, Space, & Environmental Medicine*, 69(9), B16 - B25.

- Gander, P. H., Gregory, K. B., Connell, L. J., Graeber, R. C., Miller, D. L., & Rosekind, M. R. (1998c). Flight crew fatigue IV: Overnight cargo operations. *Aviation, Space, & Environmental Medicine*, 69(9), B26 - B36.
- Gander, P. H., Gregory, K. B., Graeber, R. C., Connell, L. J., Miller, D. L., & Rosekind, M. R. (1998d). Flight crew fatigue II: Short-haul fixed-wing air transport operations. *Aviation, Space, & Environmental Medicine*, 69(9), B8 - B15.
- Gander, P. H., Gregory, K. B., Miller, D. L., Graeber, R. C., Connell, L. J., & Rosekind, M. R. (1998e). Flight crew fatigue V: Long-haul air transport operations. *Aviation, Space, & Environmental Medicine*, 69(9), B37 - B48.
- Gander, P. H., Rosekind, M. R., & Gregory, K. B. (1998f). Flight crew fatigue VI: A synthesis. *Aviation, Space, & Environmental Medicine*, 69(9), B49 - B60.
- Garland, D. J., & Hopkin, V. D. (1994). Controlling automation in future air traffic control: The impact on situational awareness. In R. D. Gilson, D. J. Garland & J. M. Koonce (Eds.), *Situational Awareness in Complex Systems* (pp. 179-197). Prescott, Az: Embry-Riddle University Press.
- Gillberg, M. (1984). The effects of two alternative timings of a one-hour nap on early morning performance. *Biological Psychology*, 19(1), 45-54.
- Gillberg, M., & Åkerstedt, T. (1994). Sleep restriction and SWS-suppression: Effects on daytime alertness and night-time recovery. *Journal of Sleep Research*, 3, 144-151.
- Gillberg, M., Kecklund, G., & Åkerstedt, T. (1996). Sleepiness and performance of professional drivers in a truck simulator: Comparisons between day and night driving. *Journal of Sleep Research*, 5, 12-5.
- Grandjean, E. P., Wotzka, G., Schaad, R., & Gilgen, A. (1971). Fatigue and stress in air traffic controllers. *Ergonomics*, 14, 159-165.
- Hamelin, P. (1987). Lorry drivers' time habits in work and their involvement in traffic accidents. *Ergonomics*(30), 1323-1333.

- Harma, M., Knauth, P., & Ilmarinen, J. (1989). Daytime napping and its effects on alertness and short-term memory performance in shiftworkers. *International Archives of Occupational & Environmental Health*, 61(5), 341-5.
- Harma, M., Suvanto, S., Popkin, S., Pulli, K., Mulder, M., & Hirvonen, K. (1998). A dose-response study of total sleep time and the ability to maintain wakefulness. *Journal of Sleep Research*, 7(3), 167-174.
- Harris, C. D. (1991). Recording Montage. In J. W. Shepard, Jr. (Ed.), *Atlas of Sleep Medicine* (pp. 1-18). Armonk, N.Y.: Futura Publishing Company, Inc.
- Harris, W. (1977). Fatigue, circadian rhythms and truck accidents. In R. R. Mackie (Ed.), *Vigilance* (pp. 133-147). New York: Plenum Press.
- Harrison, Y., & Horne, J. A. (1995). Should we be taking more sleep? *Sleep*, 18(10), 901-907.
- Harrison, Y., & Horne, J. A. (1996a). Occurrence of "microsleeps" during daytime sleep onset in normal subjects. *Electroencephalography & Clinical Neurophysiology*, 98(5), 411-6.
- Harrison, Y., & Horne, J. A. (1996b). Long-term extension to sleep - Are we chronically sleep deprived? *Psychophysiology*, 33, 22-30.
- Harrison, Y., & Horne, J. A. (1997). Sleep deprivation affects speech. *Sleep*, 20, 871-878.
- Harrison, Y., & Horne, J. A. (1998). Sleep loss affects risk-taking. *Journal of Sleep Research*, 7(Suppl. 2), 113.
- Harrison, Y., & Horne, J. A. (2000). The impact of sleep deprivation on decision making: A review. *Journal of Experimental Psychology: Applied*, 6(3), 236-249.
- Hartley, L. (1974). A comparison of continuous and distributed reduced sleep schedules. *Quantitative Journal of Experimental Psychology*, 26, 8-14.
- Hartman, E., Baekeland, F., Zwillig, G., & Hoy, P. (1971). Sleep need: How much and what kind? *American Journal of Psychiatry*, 127, 1001-1008.

- Haslam, D. R. (1981). The military performance of soldiers in continuous operations: Exercises early call I and II. In L. C. Johnson, D. I. Tepas, W. P. Colquhoun & M. J. Colligan (Eds.), *Biological Rhythms and Shift Work* (pp. 435-458). New York: Spectrum.
- Haslam, D. R. (1982). Sleep loss, recovery sleep, and military performance. *Ergonomics*, 25, 163-178.
- Heitmann, A., Stampi, C., & Anandan, S. (1995). The Alpha Attenuation Test: Its First Application for Objective Assessment of Alertness. *Sleep Research*, 24, 100.
- Herscovitch, J., & Broughton, R. (1981a). Performance deficits following short-term partial sleep deprivation and subsequent recovery oversleeping. *Canadian Journal of Psychology*, 35, 309-322.
- Herscovitch, J., & Broughton, R. (1981b). Sensitivity of the Stanford Sleepiness Scale to the effects of cumulative partial sleep deprivation and recovery oversleeping. *Sleep*, 4, 83-92.
- Heslegrave, R. J., & Angus, R. G. (1985). The effects of task duration and work-session location on performance degradation induced by sleep loss and sustained cognitive work. *Behavior Research Methods, Instruments, & Computers*, 17(6), 592-603.
- Heslegrave, R. J., Rhodes, W., Szlapetis, I., Ujimoto, V., Hahn, K., & Moldofsky, H. (1996). Impact of shiftwork and overtime on air traffic controllers: Subjective estimates of performance deficits as a function of shift, chronobiological typology, and age. *Sleep Research*, 25, 183.
- Higuchi, S., Liu, Y., Yuasa, T., Maeda, A., & Motohashi, Y. (2001). Diurnal variations in alpha power density and subjective sleepiness while performing repeated vigilance tasks. *Clinical Neurophysiology*, 112(6), 997-1000.
- Hildebrandt, G., Rohmert, W., & Rutenfraz, J. (1974). 12 & 24 h rhythms in error frequency of locomotive drivers and the influence of tiredness. *International Journal of Chronobiology*, 2, 175-180.
- Hopkin, V. D. (1988). Air traffic control. In E. L. Wiener & D. C. Nagel (Eds.), *Human Factors in Aviation* (pp. 639-663). San Diego, CA: Academic Press.

- Hori, T., Hayashi, M., & Morikawa, T. (1994). Topographical EEG changes and the hypnagogic experience. In J. R. Harsh & R. D. Ogilvie (Eds.), *Sleep Onset: Normal and Abnormal Processes* (237-253). Washington, DC: American Psychological Association.
- Horne, J. (1991). Dimensions to Sleepiness. In T. H. Monk (Ed.), *Sleep, Sleepiness and Performance* (pp. 169-196). Chichester: John Wiley & Sons.
- Horne, J. A. (1993). Human sleep, sleep deprivation and behaviour: Implications for the prefrontal cortex and psychiatric disorder. *British Journal of Psychiatry*, 162, 413-419.
- Horne, J. A., & Reyner, L. A. (1996). Counteracting driver sleepiness: Effects of napping, caffeine, and placebo. *Psychophysiology*, 33(3), 306-9.
- Horvath, M., Frantik, E., Kopriva, K., & Meissner, J. (1976). EEG theta activity increase coinciding with performance decrement in a monotonous task. *Activitas Nervosa Superior (Prague)*, 18, 207-210.
- Jean-Louis, G., Kripke, D. F., Ancoli-Israel, S., Klauber, M. R., & Sepulveda, R. S. (2000). Sleep duration, illumination and activity patterns in a population sample: Effects of gender and ethnicity. *Biological Psychiatry*, 47(10), 921-927.
- Jewett, M. E., Kronauer, R. E., & Czeisler, C. A. (1994). Phase/amplitude resetting of the human circadian pacemaker via bright light: A further analysis. *Journal of Biological Rhythms*, 9, 295-314.
- Jewett, M. E., Dijk, D. J., Kronauer, R. E., & Dinges, D. F. (1999a). Dose-response relationship between sleep duration and human psychomotor vigilance and subjective alertness. *Sleep*, 22(2), 171-9.
- Jewett, M. E., Wyatt, J. K., Ritz-De Cecco, A., Khalsa, S. B., Dijk, D. J., & Czeisler, C. A. (1999b). Time course of sleep inertia dissipation in human performance and alertness. *Journal of Sleep Research*, 8(1), 1-8.
- Jones, B., & Kenward, M. G. (1989). *Design and Analysis of Cross-Over Trials*. London: Chapman and Hall.

- Jones, B. E. (2000). Basic mechanisms of sleep-wake states. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and Practice of Sleep Medicine*. (3rd ed., pp. 134-154). Philadelphia: W. B. Saunders.
- Jung, T. P., Makeig, S., Stensmo, M., & Sejnowski, T. J. (1997). Estimating alertness from the EEG power spectrum. *IEEE Transactions on Biomedical Engineering*, 44(1), 60-9.
- Kecklund, G., & Åkerstedt, T. (1993). Sleepiness in long distance truck driving: an ambulatory EEG study of night driving. *Ergonomics*, 36(9), 1007-1017.
- Kecklund, G., Akerstedt, T., & Lowden, A. (1997). Morning work: Effects of early rising on sleep and alertness. *Sleep*, 20(3), 215-223.
- Klein, K. E., Moore, R. Y., & Reppent, S. M. (1991). *Suprachiasmatic Nucleus: The Mind's Clock*. New York: Oxford University Press.
- Kleinbaum, D. G., Kupper, L. L., Muller, K. E., & Nizam, A. (1998). *Applied Regression Analysis and Other Multivariable Methods*. Pacific Grove: Duxbury Press.
- Knauth, P., & Rutenfranz, J. (1972). Untersuchungen zum problem des schlafverhaltens bei experimenteller schichtarbeit. *Internationales Archiv für Arbeitsmedizin*, 30, 1-22.
- Knauth, P., Landau, K., Dröge, C., Schwitteck, M., Widynski, M., & Rutenfranz, J. (1980). Duration of sleep depending on the type of shift work. *International Archives of Occupational and Environmental Health*, 46, 167-177.
- Koenig, R. L. (1995). Research suggests that some rotating work shift schedules do not harm air traffic controllers' sleep patterns. *Flight Safety Foundation. Airport Operations*, 21(3), 1-6.
- Kogi, K. (1981). Comparison of resting conditions between various shift rotation systems for industrial workers. In A. Reinberg, N. Vieux & P. Andlauer (Eds.), *Night and Shift Work - Biological and Social Aspects* (pp. 155-160). Oxford: Pergamon Press.
- Kribbs, N. B., & Dinges, D. F. (1994). Vigilance decrement and sleepiness. In R. D. Ogilvie & J. R. Harsh (Eds.), *Sleep Onset: Normal and Abnormal Processes* (pp. 113-125). Washington, DC: American Psychological Association.

- Kuhlo, W., & Lehmann, D. (1964). Das Einschlafen und seine neurophysiologischen korrelate. *Archiv für Psychiatrie und Nervenkrankheiten*, 205, 687-716.
- Kushida, C. A., Chang, A., Gadkary, C., Guilleminault, C., Carrillo, O., & Dement, W. C. (2001). Comparison of actigraphic, polysomnographic, and subjective assessment of sleep parameters in sleep disordered patients. *Sleep Medicine*, 2(5), 389-396.
- Lafrance, C., & Dumont, M. (2000). Diurnal variations in the waking EEG: Comparisons with sleep latencies and subjective alertness. *Journal of Sleep Research*, 9, 243 - 248.
- Landolt, H., Dijk, D.-J., Gaus, S. E., & Borbély, A. A. (1995). Caffeine reduces low-frequency delta activity in the human sleep EEG. *Neuropsychopharmacology*, 12, 229-238.
- Landolt, H., Dijk, D.-J., Achermann, P., & Borbély, A. A. (1996). Effect of age of the sleep EEG: Slow wave activity and spindle frequency activity in young and middle-aged men. *Brain Research*, 738, 205-212.
- Landström, U., & Lindholm Häggqvist, S. P. L. (1988). Low frequency noise in lorries and correlated effects in drivers. *Journal of Low Frequency Noise Vibration*, 7, 104-109.
- Landström, U., Englund, K., Nordström, B., & Åström, A. (1994). *Field studies of a waking sound system*. (Report 28). Sweden: Arbetsmiljö Institute.
- Lauber, J., & Kayton, P. (1988). Keynote Address: Sleepiness, Circadian Dysrhythmia, and Fatigue in Transportation System Transport. *Sleep*, 11, 503-512.
- Lavie, P., & Zvuluni, A. (1992). The 24-hour sleep propensity function: Experimental bases for somnology. *Psychophysiology*, 29(566-575).
- Liberson, W. T., & Liberson, C. W. (1965). EEG records, reaction times, eye movements, respiration, and mental content during drowsiness. *Recent Advances in Biological Psychiatry*, 8, 295-302.

- Lille, F., & Cheliout, F. (1982). Variations in diurnal and nocturnal waking state in air traffic controllers. *European Journal of Applied Physiology & Occupational Physiology*, 49(3), 319-28.
- Lisper, H.-O., & Kjellberg, A. (1972). Effects of 24-hours of sleep deprivation on rate of decrement in a 10-minute auditory reaction time task. *Journal of Experimental Psychology*, 96(2), 287-290.
- Littell, R. C., Henry, P. R., & Ammerman, C. B. (1998). Statistical analysis of repeated measures data using SAS procedures. *Journal of Animal Science*, 76, 1216-1231.
- Littell, R. C., Pendergast, J., & Natarajan, R. (2000). Modelling covariance structure in the analysis of repeated measures data. *Statistics in Medicine*, 2000(19), 1793-1819.
- Loomis, A. L., Harvey, E. N., & Hobart, G. A. (1937). Cerebral states during sleep, as studied by human brain potentials. *Journal of Experimental Psychology*, 21, 127-144.
- Luna, T. D., French, J., Mitcha, J. L., & Neville, K. J. (1992). *Forward Rapid Rotation Shiftwork in USAF Air Traffic Controllers: Sleep, Activity, Fatigue and Mood Analyses*. (AL/CF-TR-1994-0156). Brooks Air Force Base, Texas: Crews Systems Directorate.
- Luna, T. D. (1997). Air traffic controller shiftwork: What are the implications for aviation safety? A review. *Aviation, Space, & Environmental Medicine*, 68(1), 69-79.
- Luna, T. D., French, J., & Mitcha, J. L. (1997). A study of USAF air traffic controller shiftwork: Sleep, fatigue, activity, and mood analyses. *Aviation, Space, & Environmental Medicine*, 68(1), 18-23.
- Mackworth, J. F. (1968). Vigilance, arousal, and habituation. *Psychological Review*, 75, 308-322.
- Mackworth, J. F. (1969). *Vigilance and Habituation*. Maryland: Penguin.
- Makeig, S., & Inlow, M. (1993). Lapses in alertness: Coherence of fluctuations in performance and EEG spectrum. *Electroencephalography and Clinical Neurophysiology*, 86, 23-35.

- Makeig, S., & Jung, T.-P. (1995). Changes in alertness are a principal component of variance in the EEG spectrum [On-line] Available: <http://www.cnl.salk.edu/~jung/pca95/pca95.html>.
- Makeig, S., & Jung, T.-P. (1996). Tonic, phasic, and transient EEG correlates of auditory awareness in drowsiness. *Cognitive Brain Research*, 4, 15-25.
- Makeig, S., Jung, T.-P., & Sejnowski, T. J. (2000). Awareness during drowsiness: Dynamics and electrophysiological correlates. *Canadian Journal of Experimental Psychology*, 54(4), 266-273.
- Matsumoto, K., & Morita, Y. (1987). Effects of nighttime nap and age on sleep patterns of shift workers. *Sleep*, 10(6), 580-9.
- Matsumoto, K., & Harada, M. (1994). The effect of night-time naps on recovery from fatigue following night work. *Ergonomics*, 37(5), 899-907.
- Maulsby, R. L., Kellaway, P., Graham, M., Frost, J. D. J., Proler, M. L., Low, M. D., & North, R. R. (1968). *The normative electroencephalographic data reference library*. (Contract NAS 9-1200). National Aeronautics and Space Administration.
- Melton, C. E., & Bartanowicz, R. (1986). *Biological Rhythms and Rotating Shiftwork: Some Considerations for Air Traffic Controllers and Managers*. (DOT/FAA/AM-86-2). U.S. Federal Aviation Administration (FAA).
- Millar, K. (1992). Some chronic problems in the methodology of performance tasks in applied clinical settings. In R. Ogilvie (Ed.), *Sleep, Arousal, and Performance* (pp. 131-153). Boston: Birkhauser.
- Millar, M. A. (2001). *Hours of work, sleep loss, and performance of anaesthetists: Implications for safety in medical practice*. Unpublished doctoral dissertation, University of Otago, Wellington.
- Minors, D. S., & Waterhouse, J. M. (1987). The role of naps in alleviating sleepiness during an irregular sleep-wake schedule. *Ergonomics*, 30(9), 1261-1273.

- Minors, D. S., Waterhouse, J. M., & Åkerstedt, T. (1994). The effect of the timing, quality and quantity of sleep upon depression (masking) of body temperature on an irregular sleep/wake schedule. *Journal of Sleep Research*, 3, 45-51.
- Mirsky, A. F., & Cardon, P. V. (1962). A comparison of behavioural and physiological changes accompanying sleep deprivation and chlorpromazine administration in man. *Electroencephalography & Clinical Neurophysiology*, 14, 1-10.
- Mollard, R., Bougrine, S., Cabon, P., Cointot, B., & Martel, A. (1997). *Effects of different work schedules on sleep habits and fatigue for air traffic controllers*. XIII International Symposium on Night and Shiftwork, Majvik, Finland: Scientific Committee on Night and Shiftwork, International Commission on Occupational Health.
- Molloy, R., & Parasuraman, R. (1996). Monitoring an automated system for a single failure: Vigilance and task complexity effects. *Human Factors*, 38(2), 311-322.
- Monk, T. H., Buysse, D. J., Reynolds, C. F., 3rd, Berga, S. L., Jarrett, D. B., Begley, A. E., & Kupfer, D. J. (1997). Circadian rhythms in human performance and mood under constant conditions. *Journal of Sleep Research*, 6(1), 9-18.
- Moore, R. Y., & Eichler, V. B. (1972). Loss of circadian adrenal corticosterone rhythm following suprachiasmatic lesions in the rat. *Brain Research*, 42, 201-206.
- Mullaney, D. J., Kripke, D. F., Fleck, P. A., & Johnson, L. C. (1983). Sleep loss and nap effects on sustained continuous performance. *Psychophysiology*, 20(6), 643-51.
- Murray, E. J. (1965). *Sleep, Dreams, and Arousal*. New York: Appleton-Century-Crofts.
- Muzet, A., Nicolas, A., Tassi, P., Dewasmes, G., & Bonneau, A. (1995). Implementation of napping in industry and the problem of sleep inertia. *Journal of Sleep Research*, 4(Suppl. 2), 67-69.
- Nagel, D. C. (1988). Human error in aviation operations. In E. L. Wiener & D. C. Nagel (Eds.), *Human Factors in Aviation* (263-303). San Deigo: Academic Press.
- Naitoh, P. (1981). Circadian cycles and restorative power of naps. In L. C. Johnson, D. Tepas, W. Colquhoun & M. J. Colligan (Eds.), *Biological Rhythms, Sleep and Shift Work* (553-580). New York: Spectrum.

- Naitoh, P., Englund, C. E., & Ryman, D. (1982). Restorative power of naps in designing continuous work schedules. *Journal of Human Ergology*, 11(Suppl.), 259-278.
- Naitoh, P., & Angus, R. G. (1989). Napping and human functioning during prolonged work. In D. F. Dinges & R. J. Broughton (Eds.), *Sleep and Alertness: Chronobiological, Behavioral, and Medical Aspects of Napping* (pp. 221-246). New York: Raven Press.
- Naitoh, P. (1992). Minimal Sleep to Maintain Performance: The Search for Sleep Quantum in Sustained Operations. In C. Stampi (Ed.), *Why We Nap: Evolution, Chronobiology, and Functions of Ultrashort Sleep* (199-216). Boston, MA: Birkhauser.
- Naitoh, P., Kelly, T., & Babkoff, H. (1993). Sleep inertia: Best time not to wake up? *Chronobiology International*, 10(2), 109-18.
- Nicholson, A. N., Pascoe, P. A., Roehrs, T., Roth, T., Spencer, M. B., Stone, B. M., & Zorick, F. (1985). Sustained performance with short evening and morning sleeps. *Aviation, Space, & Environmental Medicine*, 56, 105-114.
- Niedermeyer, E. (1999). The Normal EEG of the Waking Adult. In E. Niedermeyer & F. H. Lopes da Silva (Eds.), *Electroencephalography: Basic Principles, Clinical Applications and Related Fields*. (4th ed., pp. 149-173). Baltimore: Williams & Wilkins.
- Ogilvie, R., Simons, I., Kuderain, R., Macdonald, T., & Rustenburg, J. (1991). Behavioural, event-related potential, and EEG/FFT changes at sleep onset. *Psychophysiology*, 28, 54-64.
- Ogilvie, R. D., Wilkinson, R. T., & Allison, S. (1989). The detection of sleep onset: Behavioural, physiological, and subjective convergence. *Sleep*, 12, 458-474.
- Ogilvie, R. D., & Simmons, S. (1992). Falling asleep and waking up: A comparison of EEG spectra. In R. Broughton & R. D. Ogilvie (Eds.), *Sleep, Arousal, and Performance* (pp. 71-87). Boston: Birkhäuser.
- O'Hanlon, J. F., & Beatty, J. (1977). Concurrence of electroencephalographic and performance changes during a simulated radar watch and some implications for

- the arousal theory of vigilance. In R. R. Mackie (Ed.), *Vigilance* (pp. 1189-1202). New York: Plenum Press.
- O'Hanlon, J. F., & Kelley, G. R. (1977). Comparison of performance and physiological changes between drivers who perform well and poorly during prolonged vehicular operation. In R. R. Mackie (Ed.), *Vigilance* (87-110). New York: Plenum Press.
- Petiau, C., Harrison, Y., Delfiore, G., Degueldre, C., Luxen, A., Franck, G., Horne, J. A., & Maquet, P. (1998). Modification of fronto-temporal connectivity during a verb generation task after a 30 hour total sleep deprivation: A PET study. *Journal of Sleep Research*, 2(Suppl. 2), 208.
- Pilcher, J. J., & Huffcutt, A. I. (1996). Effects of sleep deprivation on performance: a meta-analysis. *Sleep*, 19(4), 318-26.
- Pittendrigh, C. S. (1981). Circadian systems: Entrainment. In J. Aschoff (Ed.), *Handbook of Behavioural Biology: Biological Rhythms* (pp. 95-124). New York: Plenum Press.
- Pivik, R. T. (1991). The several qualities of sleepiness: Psychophysiological considerations. In T. H. Monk (Ed.), *Sleep, Sleepiness, and Performance* (pp. 3-37): John Wiley and Sons Ltd.
- Porcu, S., Bellatreccia, A., Ferrara, M., & Casagrande, M. (1997). Performance, ability to stay awake, and tendency to fall asleep during the night after a diurnal sleep with temazepam or placebo. *Sleep*, 20(7), 535-41.
- Powell, J. W. (1999). *PVT-192 and Analysis Software Reference Manual*. Philadelphia: Unit for Experimental Psychiatry, University of Pennsylvania.
- Price, W. J., & Holley, D. C. (1990). Shiftwork and safety in aviation. *Occupational Medicine: State of the Art Reviews* (343-377). Philadelphia: Hanley & Belfus, Inc.
- Rajaratnam, S. M. W., & Arendt, J. (2001). Health in a 24-h society. *The Lancet*, 358, 999-1005.
- Reason, J. (1997). *Managing the Risks of Organizational Accidents*. Aldershot: Ashgate Publishing Ltd.

- Reason, J. (2000). Human error: Models and management. *BMJ*, 320(7237), 768-770.
- Rechtschaffen, A., & Kales, A., (1968). *A Manual of Standardised Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects*. Los Angeles, Brain Information Service/Brain Research Institute, UCLA.
- Redding, R. E. (1992). *Analysis of operational errors and workload in air traffic control*. Human Factors Society 36th Annual Meeting, Atlanta, GA: Human Factors Society.
- Reilly, E. L. (1999). EEG Recording and Operation of the Apparatus. In E. Niedermeyer & F. H. Lopes da Silva (Eds.), *Electroencephalography: Basic Principles, Clinical Applications, and Related Fields*. (4th ed., pp. 122-142). Baltimore: Williams & Wilkins.
- Reyner, L. A., & Horne, J. A. (1997). Suppression of sleepiness in drivers: Combination of caffeine with a short nap. *Psychophysiology*, 34(6), 721-5.
- Rhodes, W., Szlapatis, I., Hahn, K., Heslegrave, R., & Ujimoto, K. (1994). *A Study of the Impact of Shiftwork and Overtime on Air Traffic Controllers. Phase I: Determining Appropriate Research Tools and Issues* (TP 12257E). Montreal: Transport Canada.
- Rhodes, W., Heslegrave, R. J., Ujimoto, V., Hahn, K., Zanon, S., Marino, A., Côté, K., Szlapetis, I., & Pearl, S. (1996). *Impact of Shiftwork and Overtime on Air Traffic Controllers. Phase II: Analysis of Shift Schedule Effects on Sleep, Performance, Physiology and Social Activities*. (TP 12816E). Montreal: Transport Canada.
- Ribak, J., Ashkenzai, I. E., Klepfish, A., Avgar, D., Tall, J., Kallnar, B., & Noyman, Y. (1983). Diurnal rhythmicity and Air Force flight accidents due to pilot error. *Aviation, Space, & Environmental Medicine*, 54, 1096-1099.
- Roehrs, T., Timms, V., Zwyghuizen-Doorenbos, A., & Roth, T. (1989). Sleep extension in sleepy and alert normals. *Sleep*, 12(5), 449-57.
- Roehrs, T., Shore, E., Papineau, K., Rosenthal, L., & Roth, T. (1996). A two-week sleep extension in sleepy normals. *Sleep*, 19(7), 576-582.

- Roehrs, T., Carskadon, M. A., Dement, W. C., & Roth, T. (2000). Daytime sleepiness and alertness. In H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and Practice of Sleep Medicine*. (3rd ed., pp. 43-52). Philadelphia: W. B. Saunders Company.
- Rogers, A. S., Spencer, M. B., Stone, B. M., & Nicholson, A. N. (1989). The influence of a 1 h nap on performance overnight. *Ergonomics*, 32(10), 1193-205.
- Rosa, R. R. (1993). Napping at home and alertness on the job in rotating shift workers. *Sleep*, 16(8), 727-35.
- Rosekind, M. R., & Schwartz, G. E. (1988a). The perception of sleep and wakefulness. I: Accuracy and certainty of subjective judgements. *Sleep Research*, 17, 89.
- Rosekind, M. R., & Schwartz, G. E. (1988b). The perception of sleep and wakefulness. II: Information used for discrimination and effect of personality style. *Sleep Research*, 17, 131.
- Rosekind, M. R., Dinges, D. F., Connell, L. J., Rountree, M. S., Spinweber, C. L., & Gillen, K. A. (1994). *Crew Factors in Flight Operations IX: Effects of Planned Cockpit Rest on Crew Performance and Alertness in Long-Haul Operations*. (TM 108839). Moffett Field, California: National Aeronautics and Space Administration.
- Rosenthal, L., Roehrs, T. A., Rosen, A., & Roth, T. (1993). Level of sleepiness and total sleep time following various time in bed conditions. *Sleep*, 16(3), 226-32.
- Roske-Hofstand, R. (1995). *Raising awareness for fatigue among air traffic controllers*. Eighth International Symposium on Aviation Psychology, Ohio State University, Columbus, Ohio: Ohio State University.
- Rowland, L., Thorne, D., Balkin, T., Sing, H., Wesensten, N., Redmond, D., Johnson, D., Anderson, A., Cephus, R., Hall, S., Thomas, M., Powell, J., Dinges, D., & Belenky, G. (1997). The effects of four different sleep-wake cycles on psychomotor vigilance. *Sleep Research*, 26, 627.
- Sadeh, A., Hauri, P. J., Kripke, D. F., & Lavie, P. (1995). The role of actigraphy in the evaluation of sleep disorders. *Sleep*, 18(4), 288-302.

- Saito, Y., & Sasaki, T. (1996). The effect of length of nocturnal nap on fatigue feelings during subsequent early morning hours. *Journal of Science of Labour*, 72(1), 15-23.
- Salame, P., Otzenberger, H., Ehrhart, J., Dewasmes, G., Nicolas, A., Tassi, P., Libert, J.-P., & Muzet, A. (1995). Effects of sleep inertia on cognitive performance following a 1-hour nap. *Work & Stress*, 9(4), 528-539.
- Saldivar, J. T., Hoffman, S. M., & Melton, C. E. (1977). *Sleep in air traffic controllers*. (FAA-AM-77-5). Oklahoma city: Federal Aviation Administration, Office of Aviation Medicine.
- Sallinen, M., Harma, M., Akerstedt, T., Rosa, R., & Lillqvist, O. (1998). Promoting alertness with a short nap during a night shift. *Journal of Sleep Research*, 7, 240-247.
- Santamaria, J., & Chiappa, K. H. (1987). The EEG of drowsiness in normal adults. *Journal of Clinical Neurophysiology*, 4, 327-382.
- Schein, E. H. (1957). The effects of sleep deprivation on performance in a simulated communication task. *Journal of Applied Psychology*, 41, 247-252.
- Schroeder, D., Rosa, R., & Witt, L. (1998). Some effects of 8- vs. 10-hour work schedules on the test performance/alertness of air traffic control specialists. *International Journal of Industrial Ergonomics*, 21, 307-321.
- Schroeder, D. J., & Nye, L. G. (1993). An examination of the workload conditions associated with operational errors/deviations at air route traffic control centres. In M. D. Rodgers (Ed.), *An Examination of the Operational Error Database for Air Traffic Control Centres* (pp. 1-9). Washington, DC: Federal Aviation Administration.
- Schroeder, D. J., Rosa, R. R., & Witt, L. A. (1995). *Effects of 8- vs. 10-hour work schedules on the performance/alertness of air traffic control specialists*. (DOT/FAA/AM-95-32). Washington, DC: Federal Aviation Administration, Office of Aviation Medicine.
- Schweitzer, P. K., Muehlback, M. J., & Walsh, J. K. (1992). Countermeasures for night work performance deficits: The effect of napping or caffeine on continuous performance at night. *Work & Stress*, 6(4), 355-365.

- Silver, R., Lesauter, J., Tresco, P. A., & Lehman, M. N. (1996). A diffusible coupling signal from transplanted suprachiasmatic nucleus controlling circadian locomotor rhythms. *Nature*, 382(6594), 810-813.
- Simons, M., & Valk, P. J. L. (1997). *Effects of a controlled rest on the flight deck on crew performance and alertness*. (NLRGC 1997-B3). Netherlands Aerospace Medical Centre.
- Smith, R. C., Melton, C. E., & McKenzie, J. M. (1971). *Affect adjective check list assessment of mood variations in air traffic controllers*. (FAA-AM-71-21). Oklahoma City, Oklahoma: FAA Civil Aeromedical Institute.
- Spencer, M. B., Rogers, A. S., Birch, C. L., & Belyavin, A. J. (2000). A diary study of fatigue in air traffic controllers during a period of high workload. In S. Hornberger, P. Knauth, G. Costa & S. Folkard (Eds.), *Shiftwork in the 21st Century: Challenges for Research and Practice* (pp. 251-256) Frankfurt: Peter Lang.
- Stampi, C., Stone, P., & Michimori (1993). The Alpha Attenuation Test: A New Method for Assessing Sleepiness and its Relation to the MSLT. *Sleep Research*, 22, 115.
- Stephan, F. K., & Zucker, I. (1972). Circadian rhythms in drinking behaviour and locomotor activity of rats are eliminated by hypothalamic lesions. *Proceedings of the National Academy of Sciences USA*, 69(6), 1538-1586.
- Steriade, M., Gloor, P., Llinas, R. R., & Lopes Da Silva, F. H. (1990). Basic mechanisms of cerebral rhythmic activities. *Electroencephalography & Clinical Neurophysiology*, 76, 481-508.
- Steriade, M., McCormick, E. J., & Sejnowski, T. J. (1993). Thalamo-cortical oscillations in the sleeping and aroused brain. *Science*, 262, 679-685.
- Steriade, M. (2000). Brain electrical activity and sensory processing during waking and sleep states. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and Practice of Sleep Medicine*. (3rd ed., pp. 93-111). Philadelphia: W. B. Saunders Company.
- Strogatz, S. H. (1986). *The Mathematical Structure of the Human Sleep Wake Cycle*. New York: Springer-Verlag.

- Sugerman, J. L., & Walsh, J. K. (1989). Physiological sleep tendency and ability to maintain alertness at night. *Sleep*, 12(2), 106-12.
- Tabachnick, B. G., & Fidell, L. S. (1996). *Using Multivariate Statistics*. New York: HarperCollins College Publishers.
- Taillard, J., Philip, P., & Bioulac, B. (1999). Morningness/eveningness and the need for sleep. *Journal of Sleep Research*, 8(291-295).
- Tassi, P., Nicolas, A., Dewasmes, G., Eschenlauer, R., Ehrhart, J., Salame, P., Muzet, A., & Libert, J. P. (1992). Effects of noise on sleep inertia as a function of circadian placement of a one-hour nap. *Perceptual & Motor Skills*, 75(1), 291-302.
- Taub, J. M., Globus, G., Phoebus, E., & Drury, R. (1971). Extended sleep and performance. *Nature*, 233, 142-143.
- Taub, J. M., & Berger, R. J. (1973). Performance and mood following variations in the length and timing of sleep. *Psychophysiology*, 10, 559-570.
- Taub, J. M. (1980). Effects of ad lib extended-delayed sleep on sensorimotor performance, memory and sleepiness in the young adult. *Physiology & Behavior*, 25, 77-87.
- Tepas, D. I., & Carvalhais, A. B. (1990). Sleep patterns of shiftworkers. *Occupational Medicine*, 5(2), 199-208.
- Thackery, R. I., & Touchstone, R. M. (1981). *Age-related differences in complex monitoring performance*. (FAA-AM-81-12). Oklahoma City: FAA Civil Aeromedical Institute.
- Thomas, M., Sing, H., Belenkey, G., Holcomb, H., Mayberg, H., Dannals, R., Wagner, H., Thorne, D., Popp, K., Rowland, L., Welsh, A., Balwinski, S., & Redmond, D. (2000). Neural basis of alertness and cognitive performance impairments during sleepiness. I. Effects of sleep deprivation on waking human regional brain activity. *Journal of Sleep Research*, 9, 335-352.
- Tilley, A. J., & Wilkinson, R. T. (1984). The effects of a restricted sleep regime on the composition of sleep and on performance. *Psychophysiology*, 34, 406-412.

- Torsvall, L., & Åkerstedt, T. (1987). Sleepiness on the job: Continuously measured EEG changes in train drivers. *Electroencephalography and Clinical Neurophysiology*, 66, 502-511.
- Torsvall, L., & Åkerstedt, T. (1988). Extreme sleepiness: Quantification of EOG and spectral EEG parameters. *International Journal of Neuroscience*, 38(3-4), 435-41.
- Turek, F. W., & Zee, P. C. (1999). Introduction to sleep and circadian rhythms. In F. W. Turek & P. C. Zee (Eds.), *Regulation of Sleep and Circadian Rhythms* (1-17). New York: Marcel Dekker, Inc.
- Van Cauter, E., Sturis, J., & Byrne, M. M. (1993). Preliminary studies on the intermediate phase-shifting effects of light and exercise on the human circadian clock. *Journal of Biological Rhythms*, 8, S99-S108.
- Van Cauter, E., & Turek, F. W. (1995). Endocrine and other biological rhythms. In L. J. DeGroot (Ed.), *Endocrinology* (2487-2548). Philadelphia: W. B. Saunders Company.
- Van Dongen, H. P. A., & Dinges, D. F. (2000). Circadian rhythms in fatigue, alertness, and performance. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and Practice of Sleep Medicine* (391-399). Philadelphia: W. B. Saunders Company.
- Van Dongen, H. P. A., Price, N. J., Mullington, J. M., Szuba, M. P., Kapoor, S. C., & Dinges, D. F. (2001). Caffeine eliminates psychomotor vigilance deficits from sleep inertia. *Sleep*, 24(7), 813-819.
- Virkkala, J., Pihl, S., Harma, M., & Muller, K. (2000). *Comparison of Alpha Attenuation Test with Maintenance of Wakefulness Test*. 15th Congress of the European Sleep Research, Istanbul.
- Volani, C., Devoto, A., & Lucidi, F. (1996). Individual traits of subjects able to extend their sleep beyond 10 hours. *Journal of Sleep Research*, 5(Suppl. 1), 244.
- Webb, R. K., & Agnew, H. W. (1974). The effects of chronic limitation of sleep length. *Psychophysiology*, 11, 265-274.

- Webb, R. K., & Agnew, H. W. (1975a). The effects on subsequent sleep of an acute restriction in sleep length. *Psychophysiology*, 12, 367-370.
- Webb, R. K. (1986). Enhanced slow sleep in extended sleep. *Electroencephalography & Clinical Neurophysiology*, 58, 27-30.
- Webb, W. B., & Agnew, H. W. (1964). Reaction time and serial response efficiency on arousal from sleep. *Perceptual & Motor Skills*, 18, 783-784.
- Webb, W. B., & Agnew, H. W. (1965). Sleep: Effects of a restricted regime. *Science*, 150, 1745-1747.
- Webb, W. B., & Agnew, H. W. (1975b). Are we chronically sleep deprived? *Bulletin of the Psychonomic Society*, 6, 47-48.
- Wesensten, N. J., Balkin, T. J., & Belenkey, G. (1999). Does sleep fragmentation impact recuperation? A review and reanalysis. *Journal of Sleep Research*, 8(4), 237-245.
- Wilkinson, R. T., Edwards, R. S., & Haines, E. (1966). Performance following a night of reduced sleep. *Psychonomic Science*, 5(12), 471-472.
- Wilkinson, R. T. (1968). Sleep deprivation: Performance tests for partial and selective sleep deprivation. In R. Abt (Ed.), *Progress in Clinical Psychology: Dreams and Dreaming* (28-43). New York: Grune & Stratton.
- Wilkinson, R. T., & Stretton, M. (1971). Performance after awakening at different times of the night. *Psychonomic Science*, 23, 283-285.
- Wilkinson, R. T., & Houghton, D. (1982). Field test of arousal: A portable reaction timer with data storage. *Human Factors*, 24, 487-493.
- Williams, H. L., Lubin, A., & Goodnow, J. J. (1959). Impaired performance with acute sleep loss. *Psychological Monographs: General and Applied*, 73, 1-26.
- Williams, H. L., Granda, A. M., Jones, R. C., Lubin, A., & Armington, J. C. (1962). EEG frequency and finger pulse volume as predictors of reaction time during sleep loss. *Electroencephalography & Clinical Neurophysiology*, 14, 64-70.

-
- Wojtczak-Jaroszowa, J., & Pawłowska-Skyba, K. (1967). Praca nocna i zmianowa: I. Dobowe wahania sprawności a wydajność pracy [Work at night and shiftwork: I. Day and night oscillations of working capacity and the work efficiency.] *Medycyna Pracy*, 18, 1-10.
- Wright, K. P. J., Badia, P., & Wauquier, A. (1995). Topographical and temporal patterns of brain activity during the transition from wakefulness to sleep. *Sleep*, 18(10), 880-889.
- Wright, N., & McGown, A. (2001). Vigilance on the civil flight deck: Incidence of sleepiness and sleep during long-haul flights and associated changes in physiological parameters. *Ergonomics*, 44(1), 82-106.
- Wyatt, J. K., Dijk, D.-J., Ronda, Jewett, M. E., Powell, J. W., Dinges, D. F., & Czeisler, C. A. (1997). Interaction of circadian and sleep/wake homeostatic-processes modulate psychomotor vigilance test (PVT) performance. *Sleep Research*(26), 759.
- Zulley, J., & Campbell, S. (1985). Napping behavior during "spontaneous internal desynchronisation": Sleep remains in synchrony with temperature. *Human Neurobiology*, 4, 123-126.

APPENDIX A SLEEP DIARY AND ACTIWATCH PROTOCOL

The small watch sized object you are about to put on your wrist is an actiwatch. It is designed to sense movement through a small accelerometer. The recorded movements are downloaded to a PC at a later date.

The data from the actiwatch are analysed in conjunction with the information from the sleep diary to determine sleep length and sleep quality.

Information about wearing the actiwatch:

- Wear the watch on your non-dominant wrist (the hand you don't write with).
- It is important that you do not change wrists as this may significantly change the information that we get from the watch.
- Place the watch on your wrist with the event marker (small indentation on the face) closest to your thumb.
- Wear the watch with the face on the outside of your wrist. It should be attached reasonably firmly so that it does not move about on your wrist. If it does move about tighten the strap slightly.
- The watch is water resistant, not waterproof, so we prefer that it does not get completely covered in water. This means you may take it off to have a shower, but it is important that you put it back on again.
- If you take the watch off for any reason (to have a shower, take a swim etc) then please note in your sleep diary the times you took the watch off and put it back on.
- It is not uncommon for people to take the actiwatch off and forget to put it back on again after having a shower. If this happens, put the watch back on as soon as you remember. Please note in the sleep diary the time when you put the watch back on.
- Please note that we can not tell what you are doing from the actiwatch data. We can only tell whether you are moving or not. For example there is no difference

in what we see when you are brushing your teeth, getting a glass of water etc. compared to having sex.

- On the face is a small indentation, which is an event marker. If you push this a small mark will appear on the data output. It does not stop or start the watch. The watch will keep going the entire time you are wearing it.
- We would like you to push the event marker when you start trying to sleep and again when you stop trying to sleep. Please do this whenever you intend to sleep for **10 minutes or longer**.

Information about filling out the sleep diary:

- The sleep diary is set out so that each line represents 24 hours, from midnight to midnight on one day.
- Please write the date for each day beside the start of the line.
- We are interested in **any** sleep that is 10 minutes or longer. It does not matter whether this is during the day or during the night.
- The information that is essential to us are the times that you **begin trying to sleep**, times when you think you went to sleep, think you woke up and when you **finish trying to sleep** after any sleep that is **10 minutes or longer**.
- Please place a mark on the line at each of these times and then write underneath what the line relates to (i.e. began trying to sleep), with the time in hours and minutes. There are abbreviations listed on the sleep diary for each of these events. When writing in the sleep diary we prefer that you use the 24 hour clock.
- **BGN** is the time when you begin trying to sleep. Some people may get into bed and read etc, but we do not need to know this, we only need to know when you begin trying to go to sleep. **FSH** is when you wake up and are no longer trying to sleep. At this time you may either get out bed or begin to read etc, but you are no longer trying to sleep.

- **BGN** and **FSH** are the times we would like you to push the event marker on the actiwatch.
- We would also like you to record the times when you think you went to sleep (**SL**) and when you woke up (**WU**), but we realise these are harder to judge. You do not need to push the event marker for these events. Sometimes the time you begin trying to sleep and sleep start, and finish trying to sleep and sleep end may be very close together.
- If you wake up during your sleep to get a drink, go to the toilet etc, you do not need to write anything in the sleep diary. If you get up for **more than 10 minutes** then please treat any later sleep as a new sleep period.

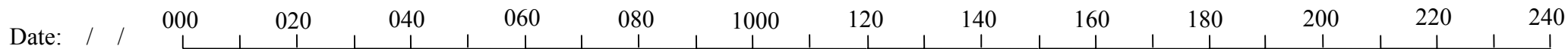
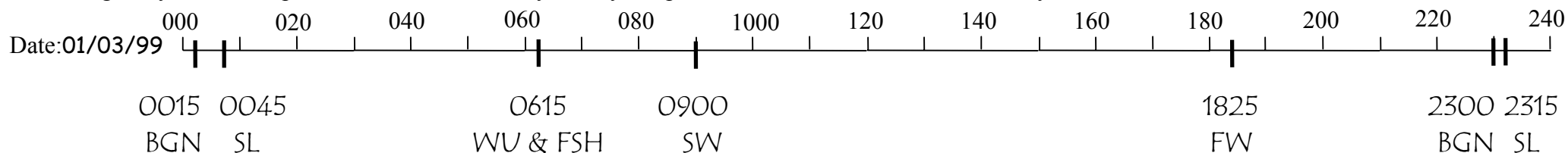
APPENDIX B SLEEP DIARY

Each line represents 24 hours from midnight one day to midnight the following day

Please enter on each line:

1. when you begin trying to sleep (**BGN**), think you went to sleep (**SL**), woke up (**WU**), finished trying to sleep (**FSH**) for any sleep **10 minutes or longer** (24 hour clock, including minutes)
2. times you had a shower (SH), and started work (SW) and finished work (FW) for Airways (24 hour clock, including minutes)
3. please **push the event marker only** when you begin trying to sleep and finish trying to sleep.

This line gives you an example how to fill out the diary. Carry sleep time over to the next line if necessary.



ID #:_____

For enquiries contact Leigh Signal (04) 385 5999 ext. 6217 or 021 120 4853

Each line represents 24 hours from midnight one day to midnight the following day

Please enter on each line:

- 1. when you begin trying to sleep (BGN), think you went to sleep (SL), woke up (WU), finished trying to sleep (FSH) for any sleep 10 minutes or longer (24 hour clock, including minutes)
- 2. times you had a shower (SH), and started work (SW) and finished work (FW) for Airways (24 hour clock, including minutes)
- 3. please push the event marker on the activity monitor only when you begin trying to sleep and finish trying to sleep.

Date: / / 000 020 040 060 080 1000 120 140 160 180 200 220 240

Date: / / 000 020 040 060 080 1000 120 140 160 180 200 220 240

Date: / / 000 020 040 060 080 1000 120 140 160 180 200 220 240

Date: / / 000 020 040 060 080 1000 120 140 160 180 200 220 240

APPENDIX C PVT (PSYCHOMOTOR VIGILANCE TEST) PROTOCOL

The test you are being asked to perform measures your ability to continuously monitor and respond to a stimulus. Please perform this test in a quiet place. It is important that there are no distractions, good lighting, and that you can sit comfortably in a chair. You can either place the PVT on a desk or table, or you can hold it with both hands. Most people prefer to hold the test with both hands. The first time you do the test it will be a demonstration to allow you to practice. This will not count in the napping study.

Components of the test

1. Sleepiness scale

Each time you do the test in the napping study Leigh will turn the test on for you. You will first see a scale asking you to rate how sleepy you currently feel. Indicate how sleepy you feel **right now** by using the **left** button to move the cursor along the scale closer to “yes” or “no” as you choose. Press the right button to register your choice.

2. Responding

During the test, as soon as you see the red numbers appear in the top window, press and release the button using the hand you write with. You may use your thumb or a finger, but use the same finger for all the tests once you have decided. The numbers in the display show how fast you responded each time. The smaller the number, the better you did. **Always try to do your best and get the lowest possible number you can.**

3. Possible mistakes

- If you press the button too early (before the numbers appear) you will see the error message “FS”.
- If you press the button on the wrong side of the machine (the one closest to the hand you don’t write with), you will see the message “ERR”.
- If you forget to release the button, after a short time the test will remind you.

4. Finishing the test

When the test is complete, you will be asked again to rate how sleepy you feel at that time. Again use the left button to move the cursor and the right to register your choice. When you have finished, do not switch the PVT off. Leigh will do this for you.

APPENDIX D ALPHA ATTENUATION TEST PROCEDURE

The participant should be seated in a chair with an upright back and placed 2 m from the wall. Their point of focus is a cross, located at eye level, and measuring 15cm by 15cm.

Participants are given the standard instructions, and then the test begins. The test is done on-line to ensure each phase of the test provides 2 minutes of artefact free recording.

Timing of the two-minute periods is taken off the computer with each change recorded by the insertion of the appropriate event marker.

Prior to the commencement of the first AAT a bio-calibration is completed. The bio-calibration involves focusing on the cross, then being asked to:

- Keep your head still
- Look to the left then back to the cross
- Look to the right then back to the cross
- Look up and then back to the cross
- Look down and then back to the cross
- Look straight ahead at the cross then blink rapidly
- Keep looking straight ahead and raise your eyebrows
- Finally, relax your jaw and now clench your teeth

Standard Instructions:

The test I am about to ask you to do is designed to measure your brain activity while you are relaxed.

During the test I would like you to sit quietly and relax, but remain awake. Please focus on the black cross on the wall in front of you. I'll ask you to do this for 2 minutes. Then I will ask you to close your eyes for a further 2 minutes.

This procedure will then be repeated, so that the whole test takes a total of 8 minutes. Do you have any questions about the test?

We will now start, so please focus on the cross.

At 2 minutes: Please close your eyes now.

At 4 minutes: Please open your eyes and focus on the cross.

At 6 minutes: Please close your eyes again.

At 8 minutes: The test is now complete.

APPENDIX E CRITERIA FOR EEG ARTEFACT

The following features were deemed to indicate artefact in the EEG channel:

1. High frequency components (> 20 Hz), with high amplitude ($> 50 \mu\text{V}$).
2. Abrupt baseline movement.
3. Spiking (intermittent, non-rhythmic high frequency waves).
4. Amplitude of any baseline movement exceeding that of surrounding faster activity.
5. Lack of rhythmicity and smooth tracing in any frequency component.

Exceptions included:

6. Artefact that appeared to be exclusively of cardiac origin was accepted in this study. This was because such artefact occurred throughout many recordings, and the characteristics of the accepted data were not being analysed in this study.
7. Baseline movement below 1 Hz was accepted as artefact free if the amplitude did not exceed twice that of the overlying activity.
8. If it was unclear as to whether an area was artefactual or merely cerebral, it was accepted if it occupied $< 5 \%$ of the epoch.

All decisions on these features were conservative, with the approach being to reject an epoch if uncertainty existed.

APPENDIX F DETAILS AND STRUCTURE OF LABVIEW PROGRAMME USED TO VIEW AND ANALYSE EEG DATA

This Appendix provides detail on the manipulation of the EEG data recorded by the *Embla*TM in order to view and analyse it in the desired manner. Adrian Ruthe, Leigh Signal and Alexander Garden developed the programme specifically for use in this, and one other, study.

LabVIEW uses a graphical programming language called “G”. Within it, a block, or wiring diagram, is created which is subsequently compiled into machine code. Each *LabVIEW* programme is called a virtual instrument (VI) and has a front panel, wiring diagram and icon.

The front panel of the *LabVIEW* VI used in the present study to view and score the EEG data can be seen in Figure F -1. The various waveform graphs, indicators and controls seen on this front panel have been explained in Chapter 2.

Figure F-2 is the left side of a wiring diagram that joins with Figure F-3, Figure F-4, Figure F-5, and Figure F-6 to create the main VI. Figure F-3, Figure F-4, Figure F-5, and Figure F-6 display four different cases, or levels of operations, that are called in sequence within this programme. They are like a deck of cards that sit on top of each other.

When any *LabVIEW* VI is run it begins by actioning functions on the left side of VI and works across to the right. In the present study the main programme starts by calling another, or sub-VI, whose function is to open an *Embla*TM file. The structure of this VI can be seen in Figure F-7. Within this VI there are two further sub-VIs. The first of these can be seen in Figure F-8. This sub-VI searches through an entire binary *Embla*TM file and extracts the header data, information on the structure of the binary information, and a “cluster” of information called the record details that contains all the components of the file. The output of this sub-VI is fed into a second sub-VI, whose structure can be seen in Figure F-9. This sub-VI takes the record details and feeds it into several further identical sub-VIs (see Figure F-10 for wiring information), each of which is searching the entire “cluster” of raw *Embla*TM information for certain labels. These labels identify each of the components in an *Embla*TM file. The sub-VI then outputs the particular component of information that was sought. The sub-VI seen in Figure F-9 then changes the format of some the components of information and outputs all of them back to the

sub-VI seen in Figure F-7. From here each component of information is available to the main VI.

The loop structure on the far left of the main VI is another deck of cards that carries out a sequence of actions in a particular order (only the top layer is shown in Figure F-2). In this instance the actions are to ask for a user id, the location for the results, and then the number of *Embla*TM files to be opened (as per the above process). Each *Embla*TM file is a channel of data.

The function of the area adjacent to this loop structure is to set up the scrollbar, which controls the movement of the EEG trace as it is viewed. The scrollbar control can be seen beneath the large waveform graph on the front panel.

In Figure F-3, Figure F-4, Figure F-5, and Figure F-6 the same large outer loop structure can be seen. It allows the programme to continue to operate until the “exit” button is pushed. The controls that sit between it and the slightly smaller inner loop are to indicate the index value of a particular epoch and to initiate further actions when the “accept” or “reject” button for each epoch is selected.

The inner loop is another structure like a deck of cards. Figure F-3 is the default layer and is an idle mode. It also controls how many channels of data can be seen at once and the colour of each.

Figure F-4 is the next layer and its function is to pass the epoch of data read from the *Embla*TM file to the two waveform graphs on the front panel. Each epoch of data is also passed to a sub-VI (seen in Figure F-12) that applies a Hanning window to the data and then performs the Fast Fourier Transform. The power spectral information is then plotted and presented in another waveform graph on the front panel.

In the layer seen in Figure F-5, the power spectral data, time information, and whether the epoch was accepted or rejected, is passed to a sub-VI. This sub-VI, seen in Figure F-11, gathers all this information, as well as additional information on the *Embla*TM file that was opened, and writes it to an output file.

The final layer, seen in Figure F-6, is called when the “exit” button is pressed. It checks to see if all the epochs of data in the *Embla*TM file have been viewed, and if not produces a warning message.

The final small loop seen on the far right of Figure F-3 to Figure F-6 closes all the *Embla*[™] files that were opened.

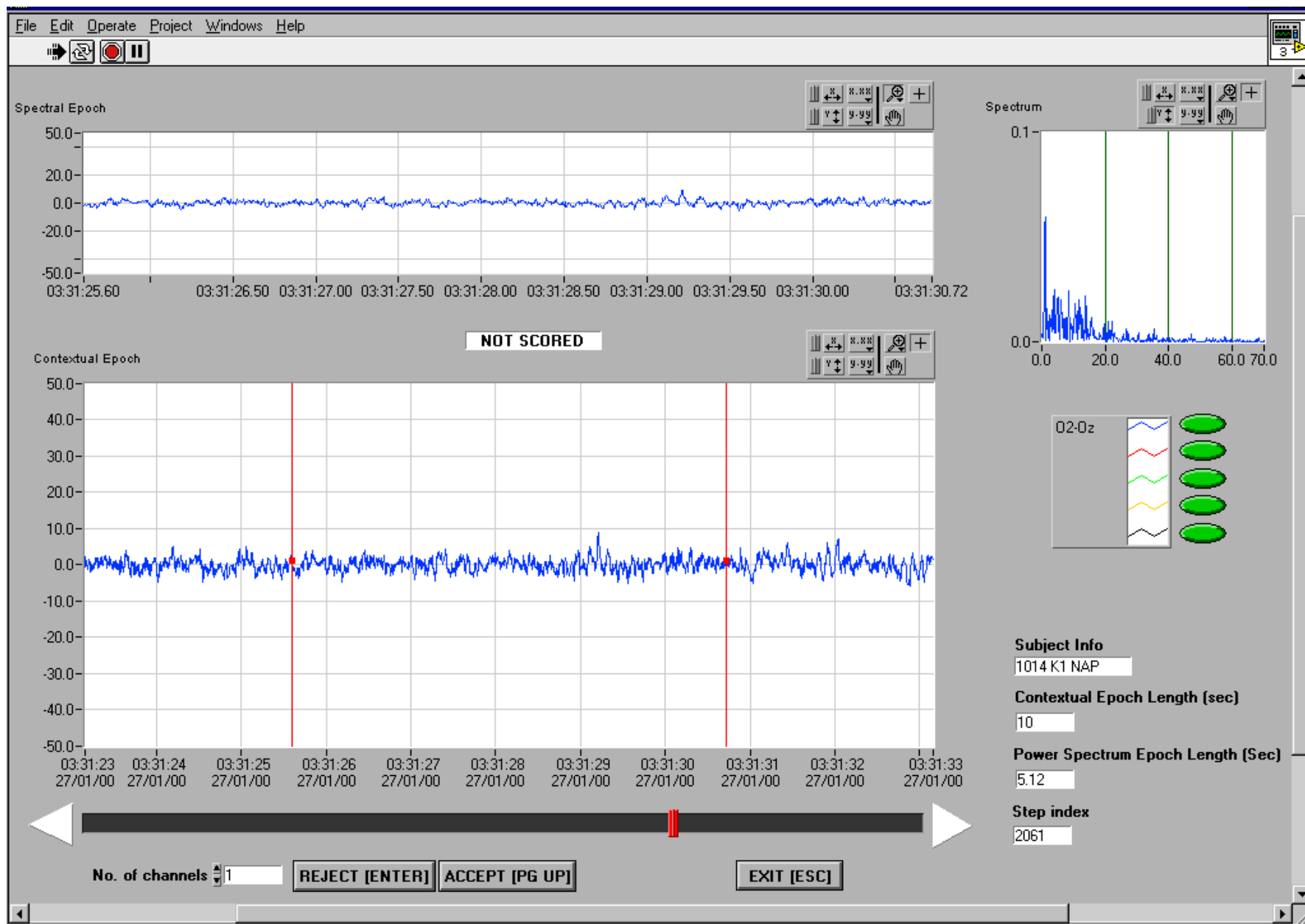


Figure F -1: Front Panel of LabVIEW VI

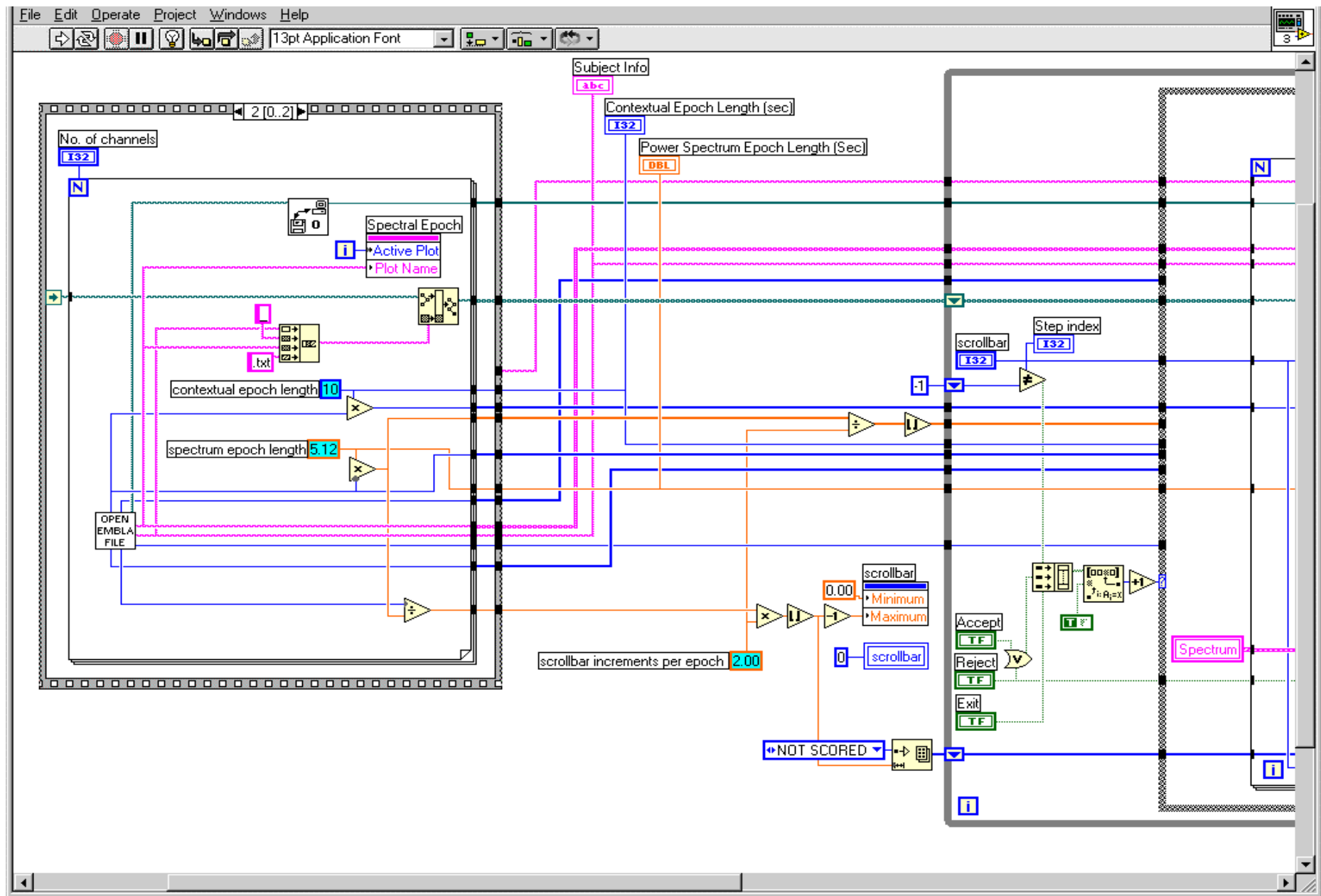


Figure F-2: Wiring Diagram of Main LabVIEW VI (Left Side)

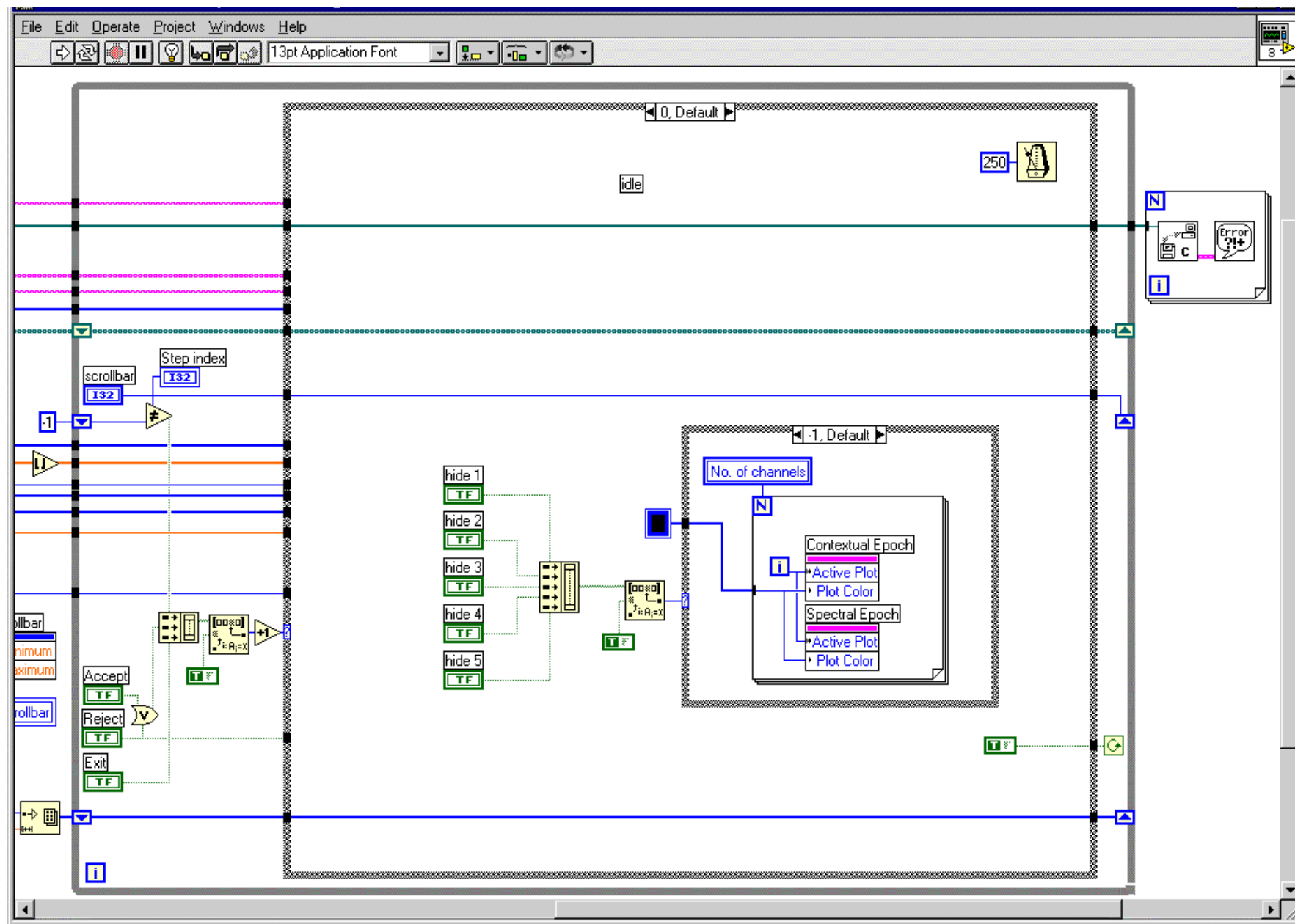


Figure F-3: Wiring Diagram of Main LabVIEW VI (Right Side and Default Case)

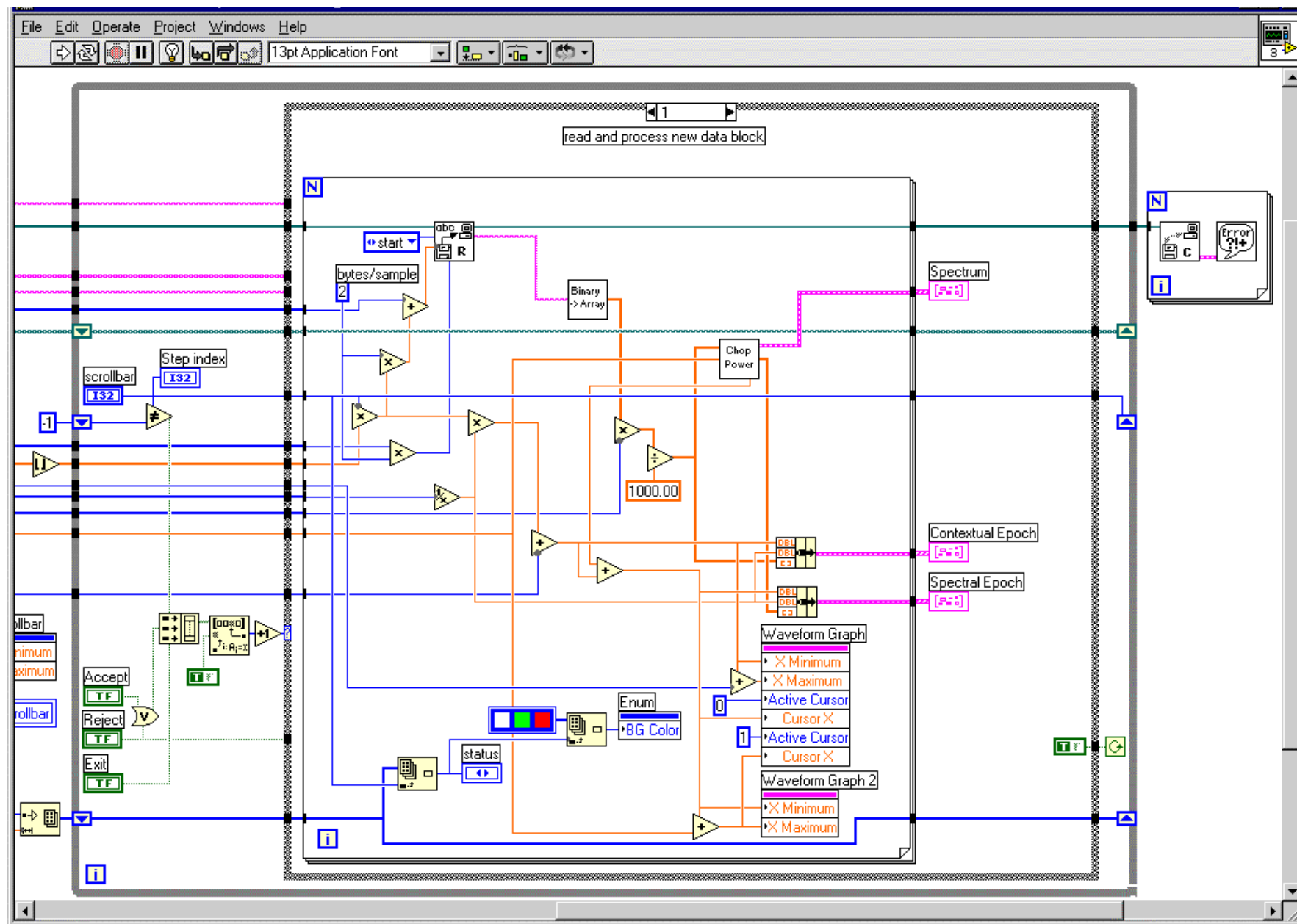


Figure F-4: Wiring Diagram of Main LabVIEW VI (Right Side and 1st Case)

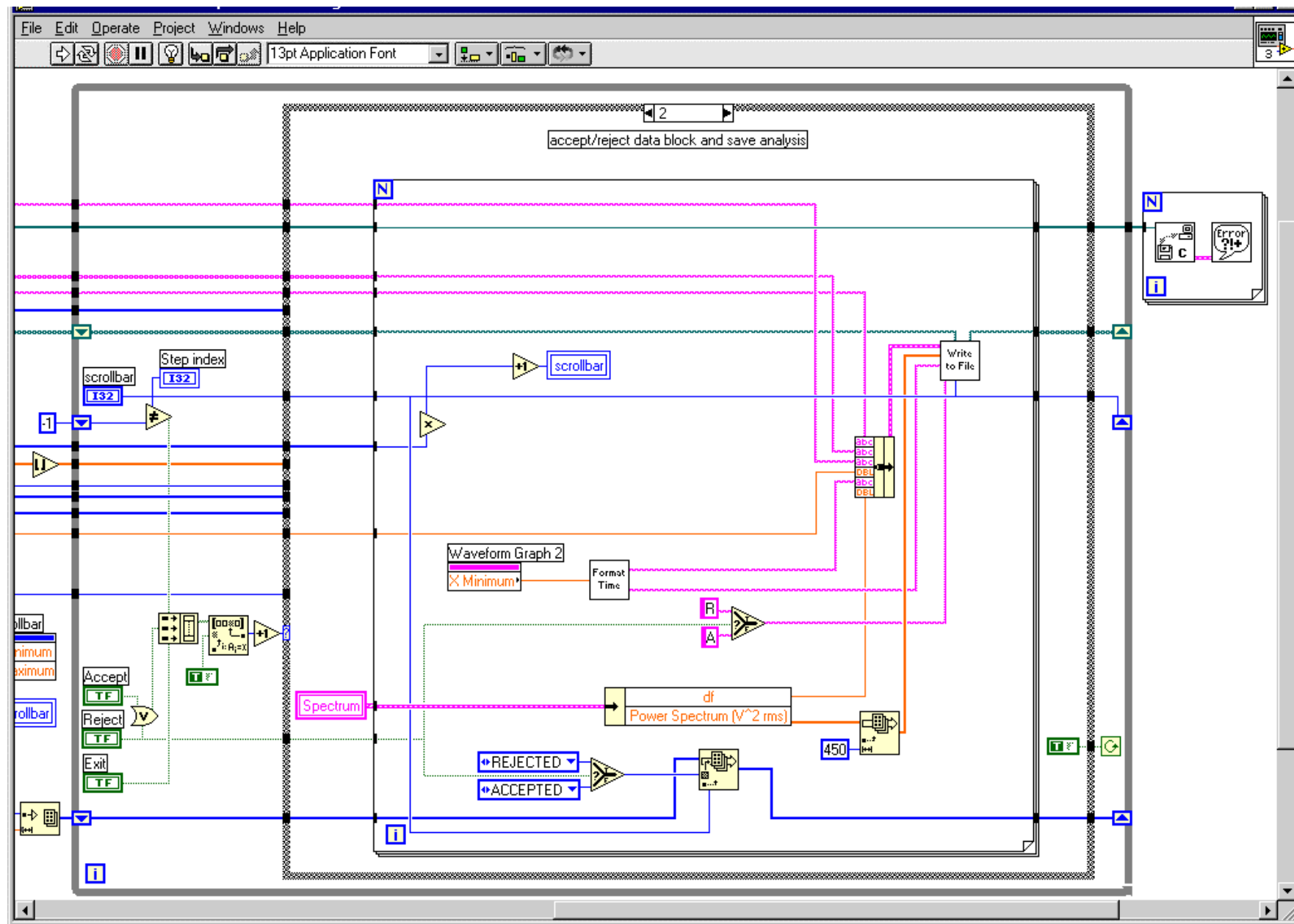


Figure F-5: Wiring Diagram of Main LabVIEW VI (Right Side and 2nd Case)

Figure F-6: Wiring Diagram of Main LabVIEW VI (Right Side and 3rd Case)

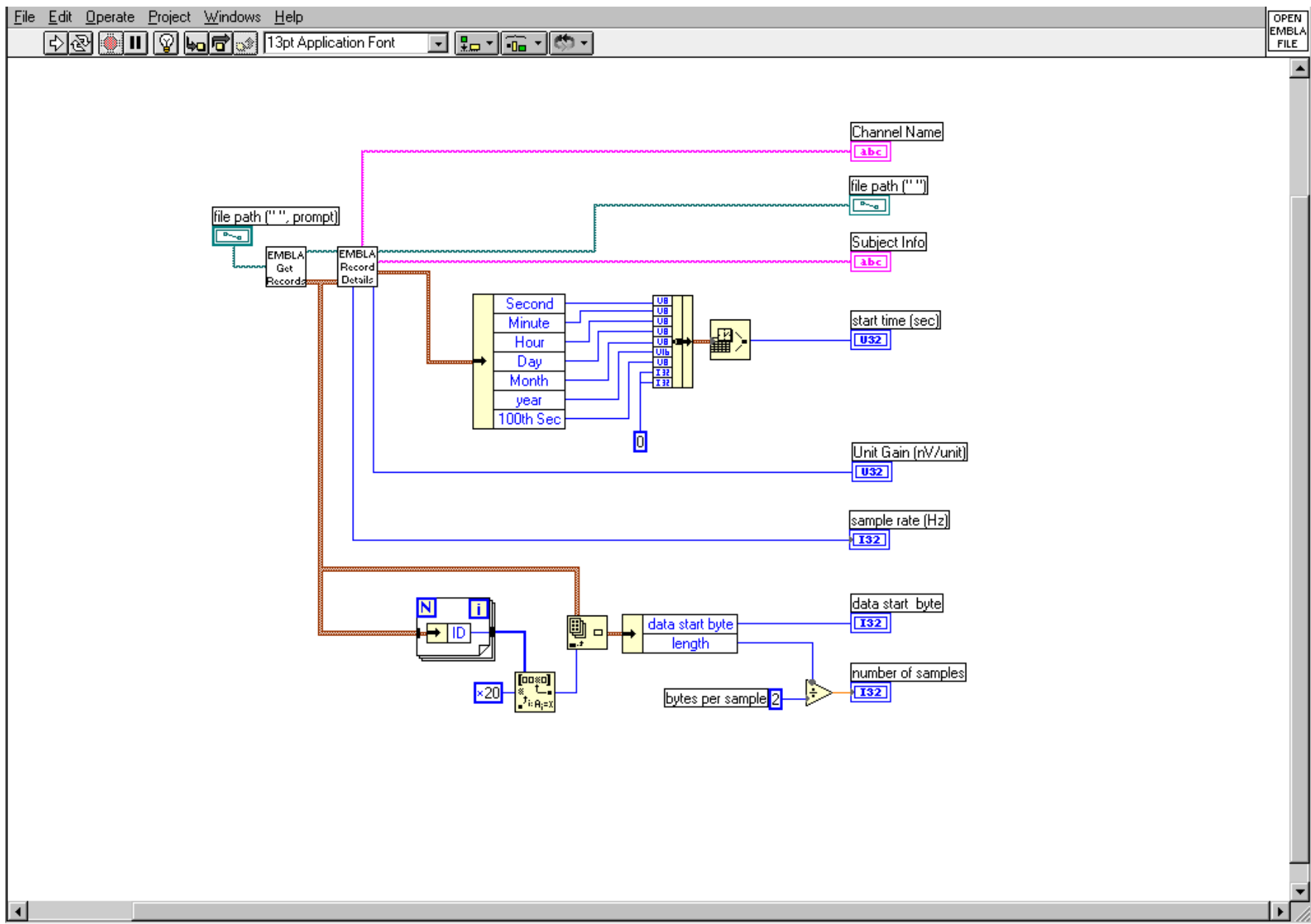


Figure F-7: Wiring Diagram of "Open Embla File" Sub-VI

Figure F-8: Wiring Diagram of “Get Embla Records” Sub-VI

Figure F-9: Wiring Diagram of “Get Embla Record Details” Sub-VI

Figure F-10: Wiring Diagram of "Find Record Data" Sub-VI

Figure F-11: Wiring Diagram of “Write to File” Sub-VI

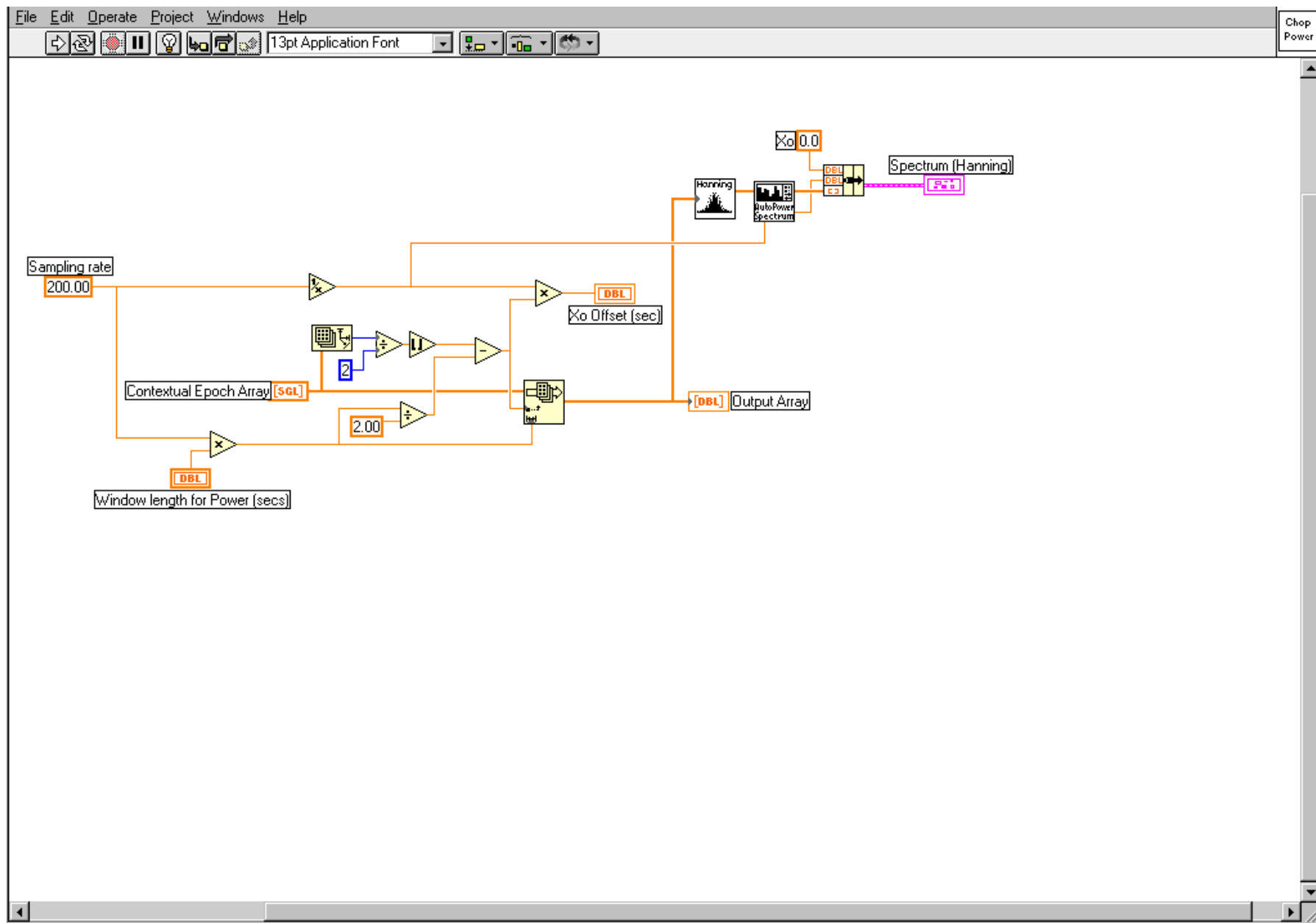


Figure F-12: Wiring Diagram of "Power Spectral" Sub VI

APPENDIX G OBJECTIVELY DEFINING SLOW ROLLING EYE MOVEMENTS

There are varied but relatively sparse definitions of what constitutes an SEM. Simple definitions include involuntary, slow, movements of the eye (Carskadon & Dement, 2000), and slow rolling, predominantly horizontal eye movements that signify a loss of oculomotor control (Cajochen, Khalsa, Wyatt, Czeisler & Dijk, 1999). Slightly more objective descriptions include binocularly asymmetrical, lateral movements of 3-4 seconds duration (Aserinsky & Kleitman, 1953), movements with a frequency of 0.2-0.6 Hz, amplitude of 20-60°, with no vertical component (Kuhlo & Lehmann, 1964), and greater than 1 second, rolling excursions of the EOG, with an amplitude of at least 100 μ V (Åkerstedt & Gillberg, 1990).

Kuhlo and Lehmann (1964) report that SEMs increase in amplitude, becoming more regularly sinusoidal with increasing drowsiness and that these changes were closely related to changes seen in the EEG. Mulsby et al. (1968) define SEMs as 0.25 Hz, pendular eye movements that were seen in all participants they studied, and for 50 % of these individuals they were the first signs of drowsiness.

A study by Wright and McGown (2001) is one of the few where SEMs have been described in a group of individuals who were working and trying to remain awake. Their study of civil airline pilots investigated the frequency of periods of increased sleepiness on the flight deck. They describe early SEMs as having relatively high amplitude and a frequency of 0.5-1.0 Hz, normally coupled with alpha activity in the EEG. Slower eye movements of approximately 0.1-0.3 Hz, with lower amplitude, were seen with theta activity in the EEG, and equated to the characteristics of stage 1 sleep as defined by Rechtschaffen and Kales (1968).

Santamaria and Chiappa (1987) state that first SEMs are of small amplitude, while later SEMs produce a medium to high excursion of the EOG trace and have a velocity less than 0.5 per second. These larger SEMs were more likely to be associated with periods of drowsiness in the EEG than those of smaller amplitude.

Because there are no published standard criteria for what constitutes an SEM, the information in the existing literature was assessed, and the following criteria were developed and used in the present study for viewing and defining a SEM. It is important to note that nearly all of the studies, which discuss the characteristics and occurrence of

SEMs, have focussed on individuals who have their eyes closed and are trying to fall asleep. In contrast, the present study was conducted with participants who were awake and at work, therefore they were generally attempting to keep their eyes open.

During the EOG recordings outside the nap opportunity and testing sessions, there is no way of knowing whether participants in the study had their eyes open or closed at a particular point in time. It is presumed that SEMs can only occur when the eyes are closed, but this is not explicit in the literature. There is a possibility that SEMs might differ in appearance in individuals who are trying to keep their eyes open. For example a blink may occur very close in time to an SEM, altering its appearance if an individual was alternating between having their eyes open and eyes closed. In addition, because the recordings in the present study are of individuals awake at work, various forms of artefact were frequently seen in the EOG tracings, which may also alter or obscure SEMs.

To be certain that a rolling movement in the EOG trace was actually an SEM, a conservative approach was taken to scoring these eye movements. This may have led to some of the smaller amplitude, less well defined SEMs, often reported during the earlier stages of drowsiness (Santamaria & Chiappa, 1987), being excluded.

SEMs were scored at the same time as the ambulatory data were scored for sleep. This resulted in the EOG traces being viewed using the gain, filter settings, and epoch length applied in the sleep scoring template. These have been previously discussed but include:

- Viewing at an amplitude of 100 $\mu\text{V}/\text{cm}$. This was to allow identification of both SEMs and any possible REMs if REM sleep was entered. Santamaria and Chiappa (1987) used a gain of 20 $\mu\text{V}/\text{cm}$, which would have resulted in a much larger deflection in the EOG trace with a SEM, but it in the present study it was considered that a gain of 100 $\mu\text{V}/\text{cm}$ allowed the identification of SEMs. SEMs had been clearly seen at this gain during the earlier scoring of the napping opportunities.
- A low pass filter of 30 Hz was applied to the EOG trace. SEMs have a frequency well below 30 Hz and this filter was applied throughout the study to EOG traces to remove high frequency noise not of interest. In addition, all EOG data had been bandpass filtered at 0.5 – 90 Hz when recorded.

- EOG traces were viewed in 30-second epochs.

Further criteria applied in an attempt to objective define a SEM included:

- Amplitude: SEMs must have a minimum amplitude of 10 $\mu\text{V}/\text{cm}$. This was applied to ensure small movements of the baseline could not be interpreted as SEMs. A maximum amplitude was not defined, but generally SEMs did not exceed 80 μV . Previous studies in which the amplitude of SEMs was mentioned have either scored SEMs regardless of their amplitude (Cajochen et al., 1999), or required them to be at least 100 μV (Åkerstedt & Gillberg, 1990). This later criteria is thought to be excessive and would result in very few or no SEMs being scored.
- SEMs must have a period greater than 1.5 seconds, but less than or equal to 5 seconds, between points where the EOG trace crosses the baseline. This results in a maximum frequency of 0.67 Hz and a minimum of 0.2 Hz for an SEM, which is similar to that reported by Aserinsky & Kleitman (1953) and the upper frequency reported by Åkerstedt et al. (1990). Åkerstedt et al. (1987) report a minimum frequency of 0.1 Hz, which is extremely slow and would result in a SEM of 10 seconds duration. To exclude labelling possible baseline drifts as SEMs an upper period of 5 seconds was used.
- An SEM could not lead into and/or out of a rapid eye movement (defined as a sharp wave). On many occasions a rolling type wave is created preceding or following a rapid eye movement, such as a blink. A number of examples of this are shown below in Figure G-5 and Figure G-7.
- SEMs were required to be a relatively smooth movement of the trace. Sharp peaks preceding or following the rolling movement, as discussed above, were not scored as SEMs. Slight peaks that occur at the maximum excursion of the EOG trace from baseline were accepted as SEMs, if all other criteria were met. Slightly sharper, faster eye movements, which were more difficult to interpret, were categorised as SEMs if there was clear alpha activity in the EEG traces and/or alpha could be seen in the EOG traces.
- The EOG channels show movements that are approximately 180° out of phase with each other. Because of the location of the EOG electrodes the EOG

channels have opposite polarity from each other. This is normal practice and is done to discriminate any type of eye movement from artefact. However, the SEMs are not always perfectly 180° out of phase from each other. Occasionally, one eye lags slightly behind the other, or produces a slightly different rolling movement.

The following figures illustrate examples of various commonly seen eye movements which fall both within and outside the criteria stipulated above.

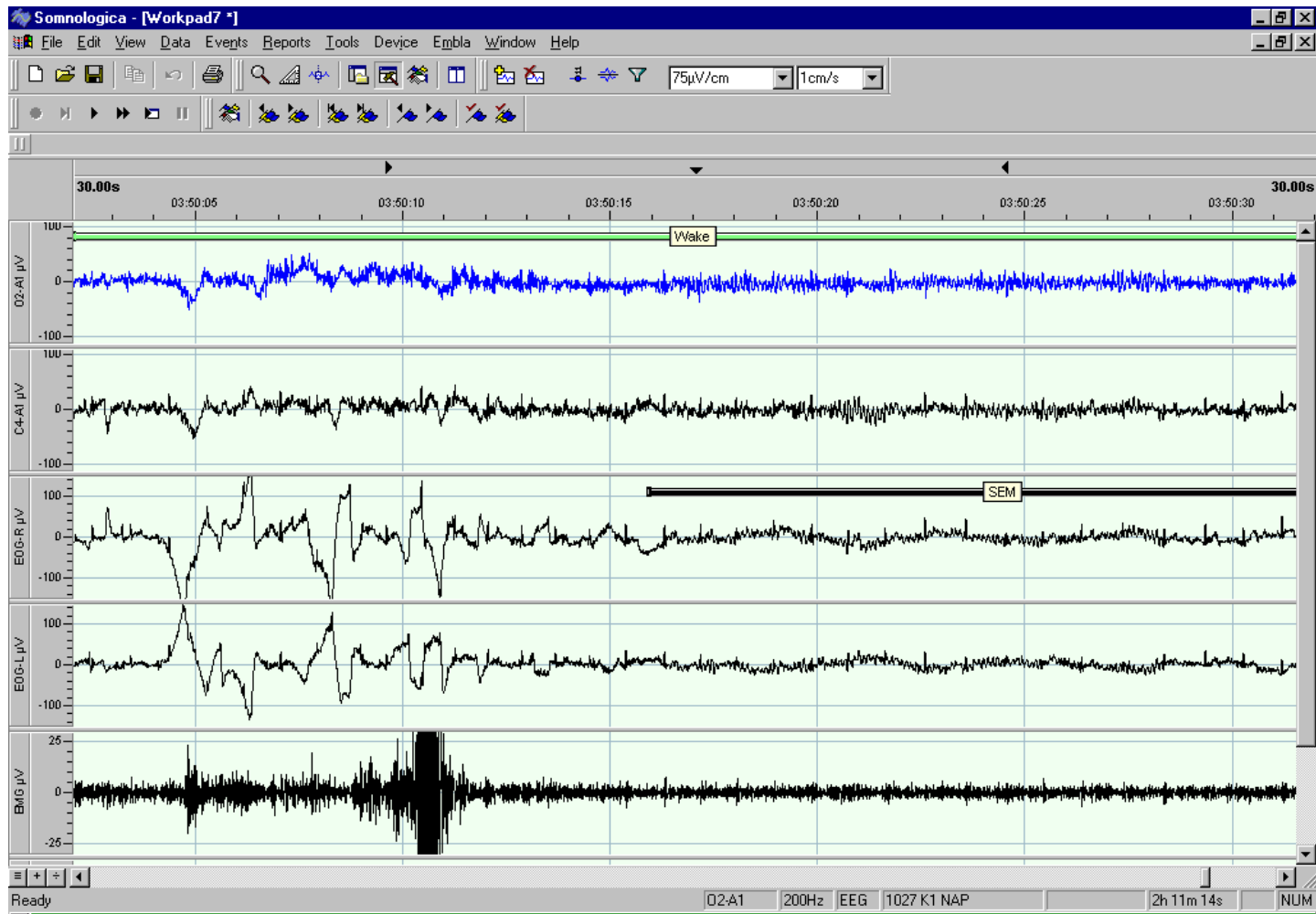


Figure G-1: Large Rapid Eye Movements Leading to Smaller Rapid Eye Movements Then Clear SEMs

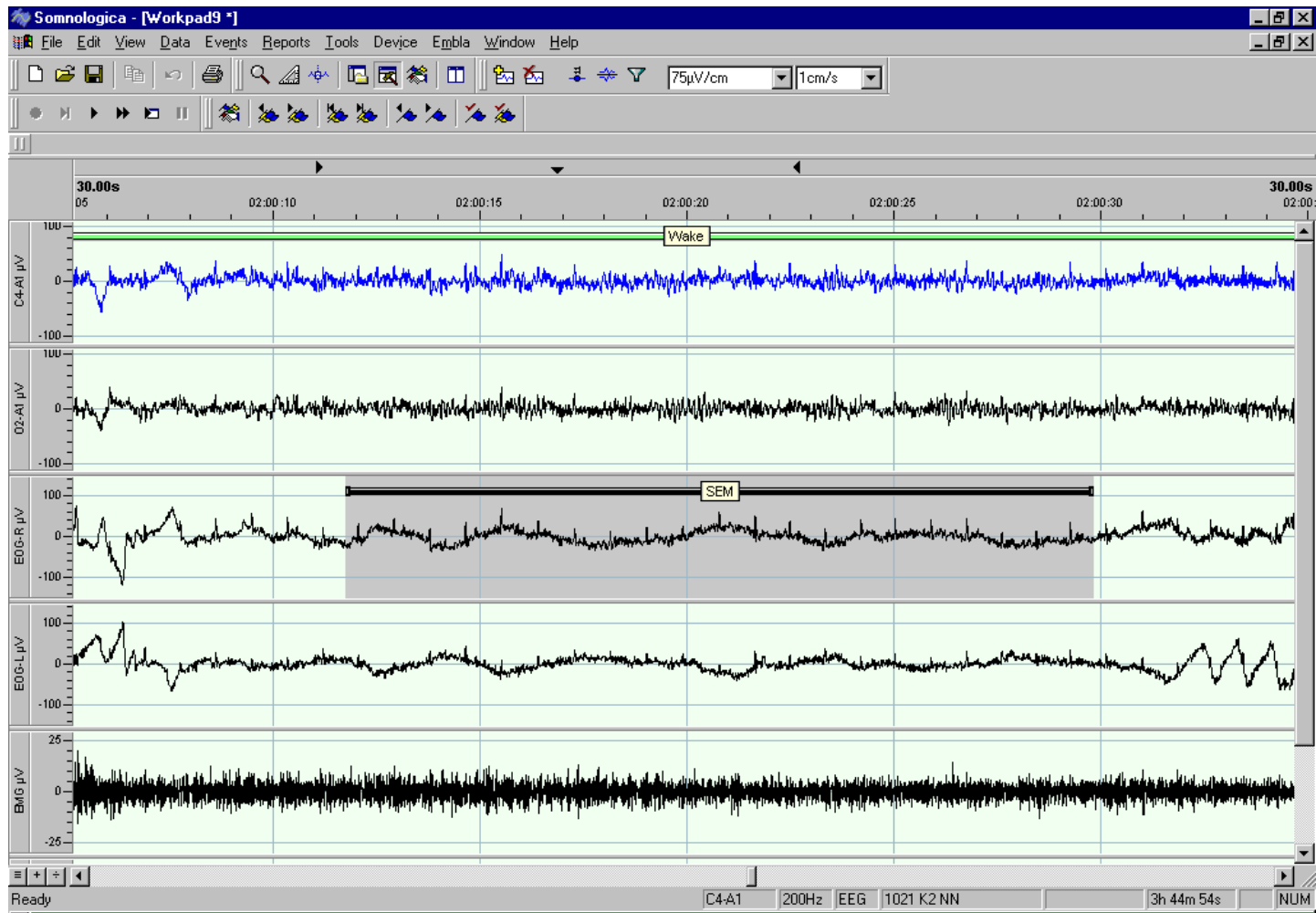


Figure G-2: Clear Run of SEMs (Not Always Binocularly Symmetrical)

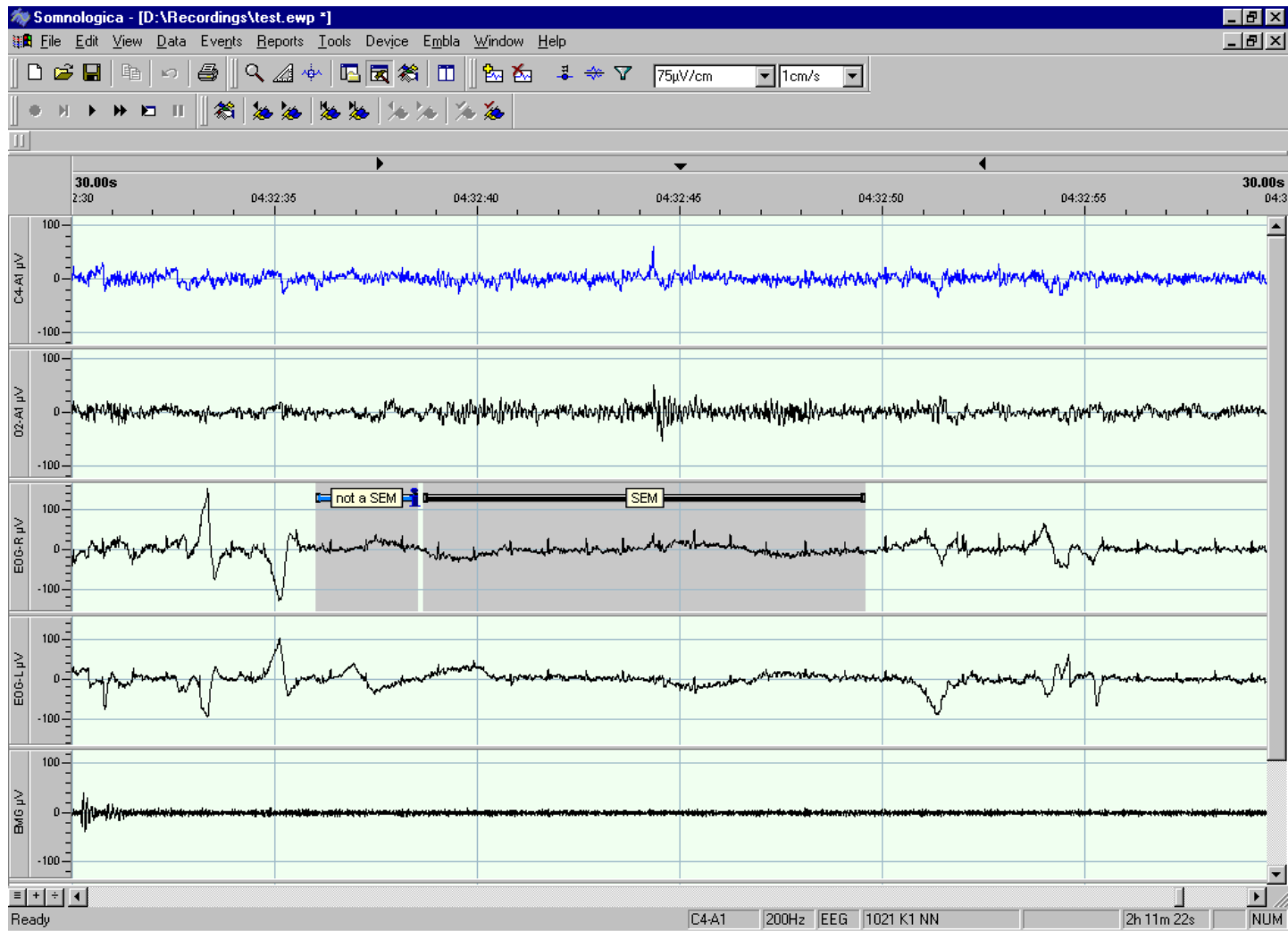


Figure G-3: Initial Rolling Movement Not an SEM Due to Sharp Positive Peak in Left EOG Trace

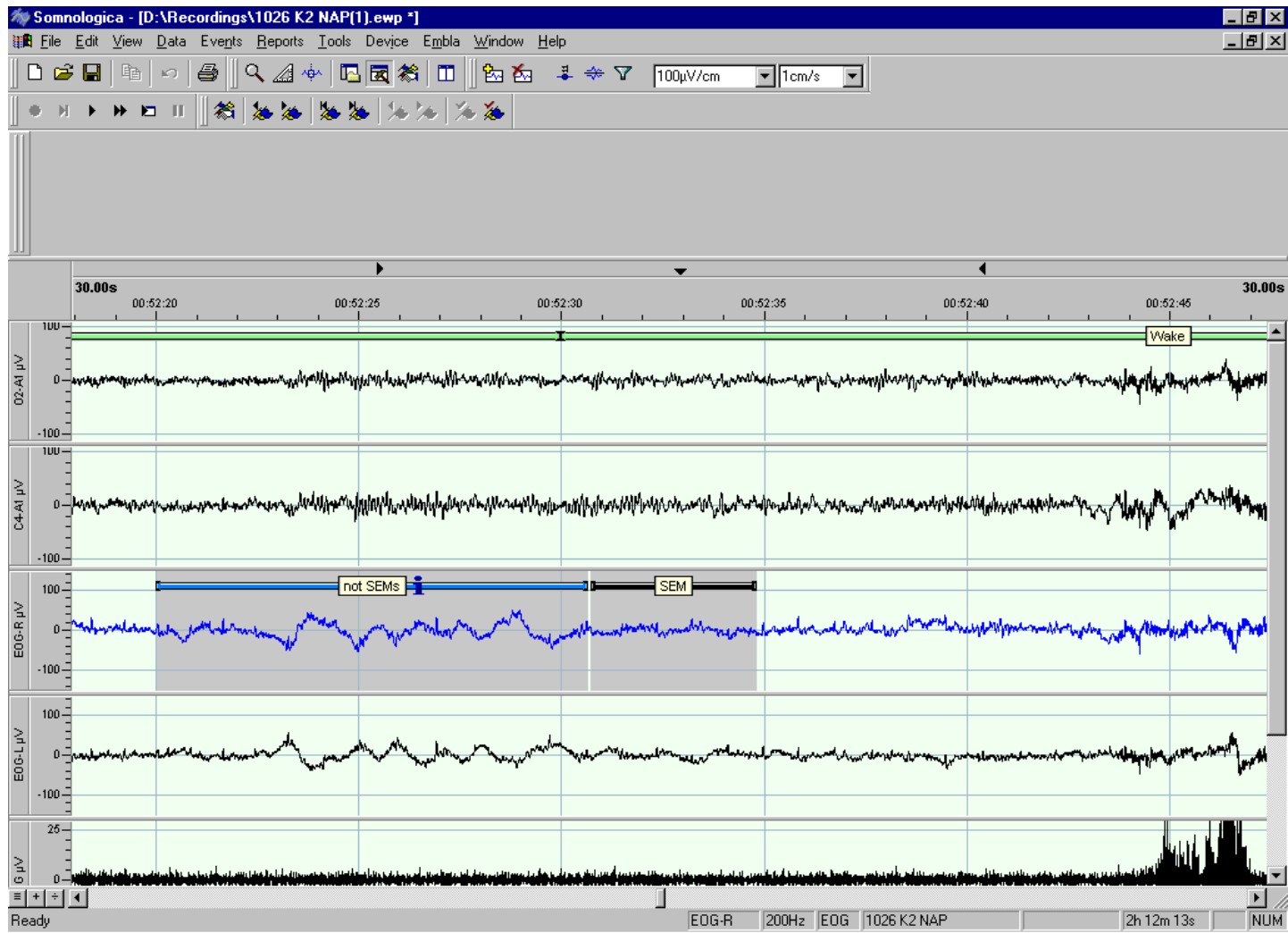


Figure G-4: Initial Rolling Movements Not Scored as SEMs Due to Sharp Peaks Between Waves

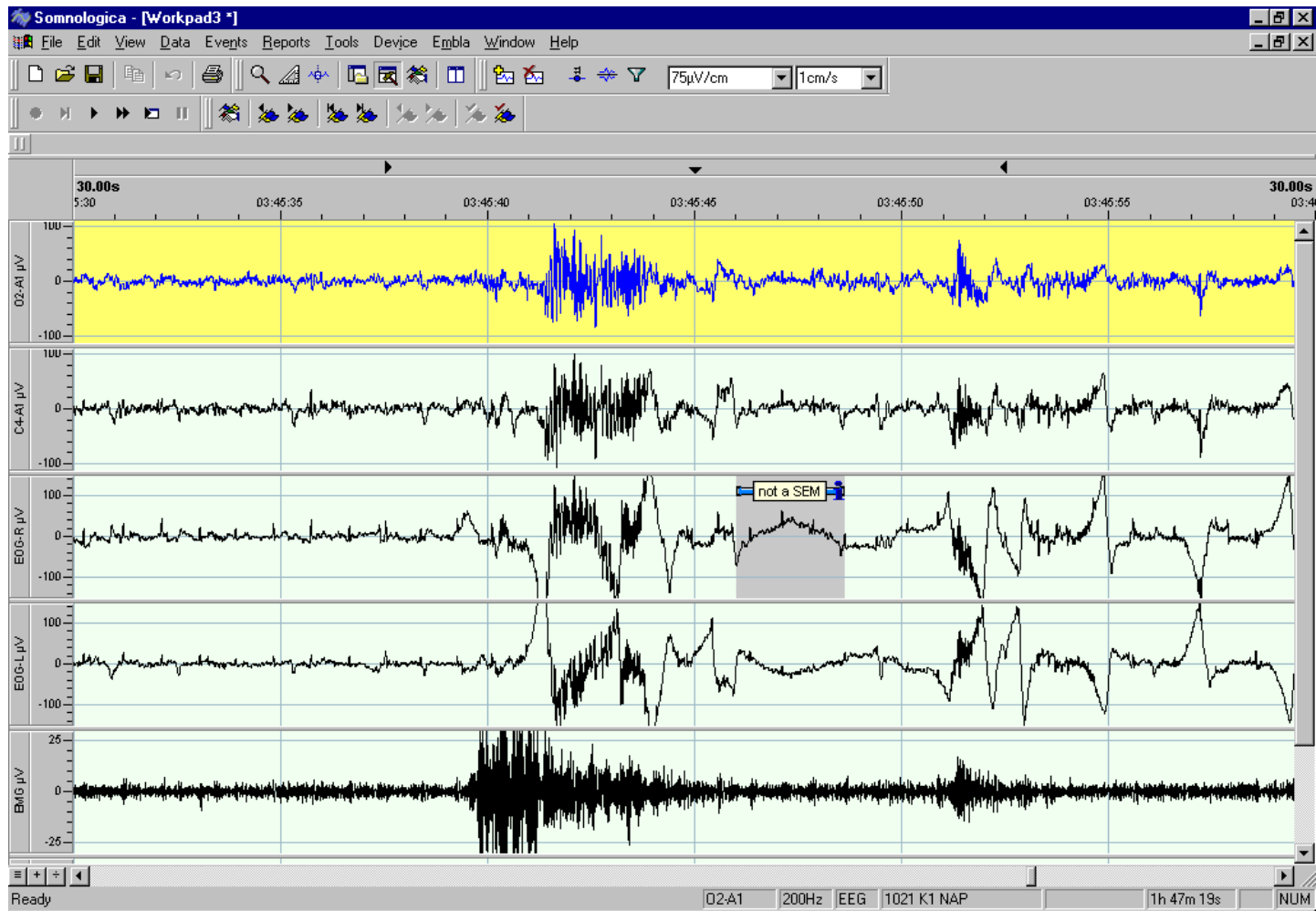


Figure G-5: A Rapid Eye Movement Prior to a Rolling Movement (Which is Not Considered an SEM)

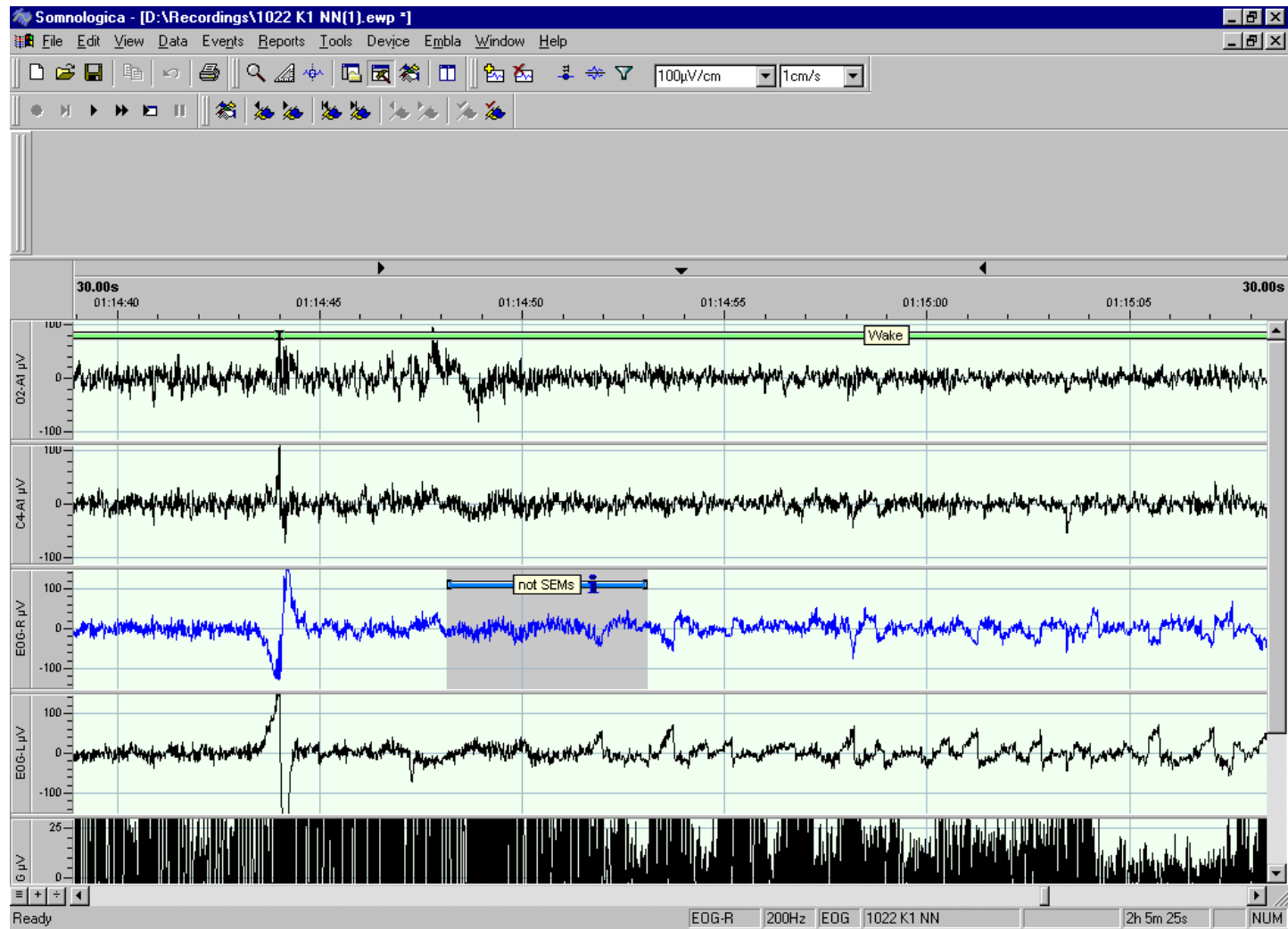


Figure G-6: Small Rapid Eye Movements Preceding and Following a Rolling Type Movement (Which Are Not SEMs)

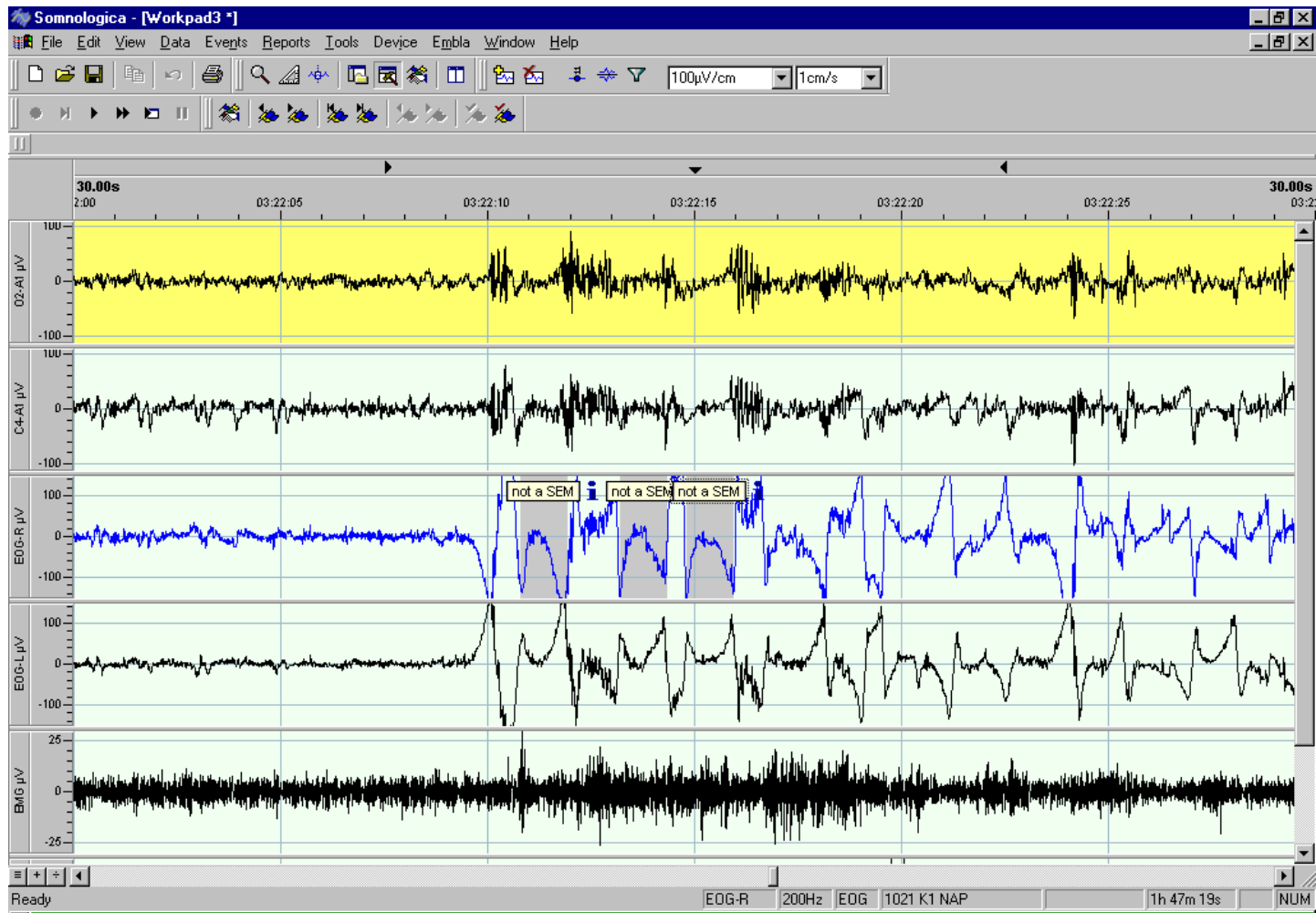


Figure G-7: Series of Rapid Eye Movements Resulting in Rolling Type Movements (Which Are Not SEMs)



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APPENDIX H PRE-STUDY QUESTIONNAIRE

1. Age: _____ years

2. Gender (*please circle*) Female Male ☐

3. How long have you been an air traffic controller?
_____ years

4. What do you do normally to prepare for a K1 night shift?

5. Do you normally nap on a K1 night shift? (*place a cross on the line*) ☐ ☐

Never |-----| Always

6. How many caffeine containing drinks would you normally consume over a K1 night shift?
_____ drinks

7. At what times would you normally have something to eat during a K1 night shift?

_____ hours

_____ hours

_____ hours

8. Do you normally nap on a K2 night shift? (*place a cross on the line*)

☐ ☐



9. What do you normally do to prepare for a K2 night shift?

☐ ☐

10. How many caffeine containing drinks would you normally consume over a K2 night shift?

☐ ☐

_____ drinks

11. At what times would you normally have something to eat during a K2 night shift?

_____ hours

☐ ☐ ☐ ☐

_____ hours

☐ ☐ ☐ ☐

_____ hours

☐ ☐ ☐ ☐

12. Do you currently suffer from a diagnosed sleep disorder? (*please circle one*)

☐

Yes No

13. How many hours of sleep do you normally need (on an undisturbed night) to feel fully rested

☐ ☐ ☐

_____ . _____ hours

14. Do you write with your right or left hand? _____
(this is for setting up the performance test)

☐



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APPENDIX I POST NIGHT SHIFT QUESTIONNAIRE

Night shift: _____ ☐

Nap or no nap: _____ ☐

1. Was your pre-shift preparation any different from normal (*please circle one*) ☐

Yes No

2. If Yes, how was your pre-shift preparation different from normal. ☐ ☐

3. How long, in total, do you think you slept for during the napping opportunity? ☐ ☐ ☐

_____ minutes

4. Was this night shift any different from a normal shift of this type? (*please circle one*) ☐

Yes No

5. If Yes, in what way was this shift different. ☐ ☐

6. How would you rate the workload over the night shift? (*place a cross on the line*) ☐ ☐

|-----|

Very low Very high

7. How many caffeine containing drinks did you consume over this night shift? ☐ ☐

_____ drinks

8. When did you have something to eat during this night shift?

_____ hours

_____ hours

_____ hours

If you had a nap on this night shift, please answer the following questions

9. How difficult did you find it to fall asleep? (*place a cross on the line*)

|-----|
 Not at all Very
 difficult difficult

10. How would you rate the quality of the sleep during your nap? (*place a cross on the line*)

|-----|
 Very poor Very good

11. To what extent did you awaken feeling refreshed from your nap? (*place a cross on the line*)

|-----|
 Not at all Completely
 refreshed refreshed

12. How would you rate the depth of sleep you gained during your nap?
(*place a cross on the line*)

|-----|
 Light Deep

13. Do you feel that the nap was helpful in managing your fatigue during the remainder of the night shift? (*place a cross on the line*)

|-----|
 Very unhelpful Very helpful

14. Where did you take your nap?



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APPENDIX J INFORMATION SHEET

Naps, Alertness, and Performance

You are invited to take part in a study to determine the effects of a pre-planned nap during the night shift.

Study aims

- To determine if naps during the night shift have any effect on the alertness and performance of air traffic controllers.
- To determine the sleep patterns of air traffic controllers in the days leading up to the night shift and immediately afterwards.

What are the outcomes of the study?

- You will receive a summary of the findings of the study and have access to a copy of the final report.
- This is an opportunity for you to learn about the effect of fatigue on performance and alertness and to learn about the use of strategies to counter fatigue on the night shift.
- Information directly applicable to Christchurch Controllers about the usefulness of napping on the night shift.

What is involved if you decide to participate?

- If you decide to be in the study you will be asked to:
- Wear a small watch-sized activity monitor during four shift cycles and complete an accompanying sleep diary.
- Wear a set of electrodes placed on your head on four night shifts. These are connected to a small recording device.
- Complete short reaction time tests on the night shifts you are wearing the electrodes.
- Take a 40 minute nap on two of the night shifts, and remain awake on the other two night shifts.

Important points

- This study has not been commissioned by the Airways Corporation of New Zealand and the company has no means of influencing the study findings. Airways has provided a small portion (approximately 10 %) of the total cost of the study.
- You are free to withdraw from the study at any time and to ask for the hard copy of your personal data.
- There is no way that you will be able to be identified in any reports on the study.
- After the night shifts without naps, taxi cabs will be available to get you home if you wish and then back to work later in the day to collect your car.

What do I do now?

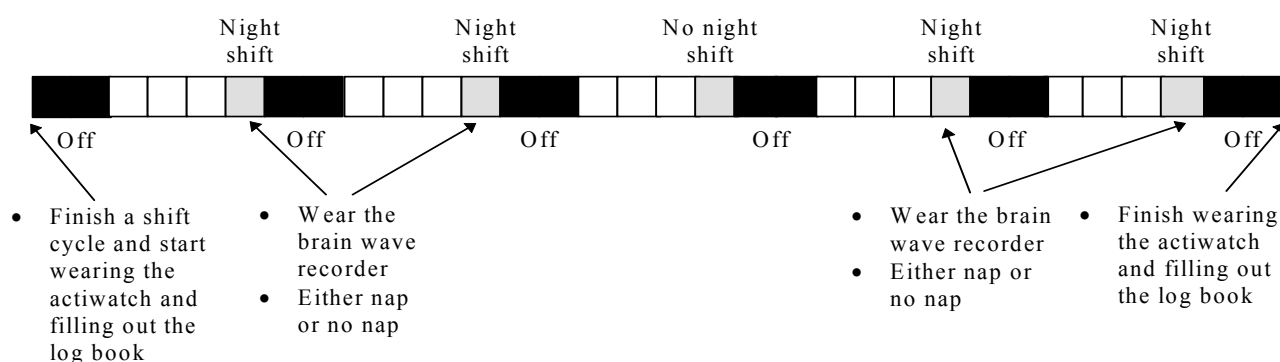
If you choose to participate after reading this information sheet then please complete the **first** attached form and return it to Sue Brash. If you choose not to participate in the study then please complete the **second** attached form and also return it to Sue Brash (the reasons for this are outlined on the form). If Sue has not received a form from you a month from now then she will contact you to ask you whether you wish to participate in the study or not. Only the names of those who agree to participate will be forwarded to the research team, who will then contact each person and arrange a time to begin their involvement in the study.

The following information explains what will happen during the study.

At the end of one shift cycle and before your days off you will be contacted by Leigh Signal and given an activity monitor and sleep diary. The activity monitor is the same size as a watch and is worn on your non-dominant wrist (the one you don't write with). The sleep diary is filled out by you, to help us know when you have gone to sleep and woken up again. Together, the activity monitor and sleep diary give information on when you are sleeping, how long you are sleeping and the quality of each sleep period. The diagram below shows how long you will wear the activity monitor and when you will wear the brain wave recorder. The study will include two night shifts which start at 2220 hours (K1) and two night shifts that start at the later time of 2345 hours (K2).

Before a study night we will ask you to come in approximately 30 minutes early.

Airways has agreed to pay for this additional half hour of your time. Leigh Signal will connect the recording equipment which involves having eight small electrodes will attached to your head. Four are placed on the top of your head, two are placed near your eyes, and two under your chin. The skin area where the electrodes are attached will be cleaned in order to allow a clear, good quality signal but no hair is removed. Attaching and wearing the electrodes is painless and most people forget that they have them on. Each electrode has a thin wire attached to it that plugs into a small recording box about the size of a pound of butter. The recording box has a shoulder bag which means you can easily walk around with the electrodes and recorder. The recording box measures brain activity, eye movements, and muscle tension which will tell us how alert you are.



After the recorder is attached and before starting work we will ask you to complete a short reaction time test. This test will be repeated once during the night and again at the end of the shift. At the end of the night shift we will take the electrodes off, which will take about 5-10 minutes. The cream used to hold the electrodes on is completely water soluble, but you will most likely want to wash your hair when you get home.

On two night shifts you will be asked to try and sleep for 40 minutes during a scheduled break. On another two night shifts you will be asked to remain awake. If you are at all concerned about feeling tired at the end of the night shifts without a nap then we will give you taxi chits to get you home and then back to work to pick up your car later in the day.

Your brain wave recording, activity monitor recording, logbook data and results of the performance tests will not have your name on them. Instead they will have a code number and all data will be stored in a secure cabinet at the Wellington School of Medicine. No material which could personally identify you will be used in any reports on the study.

Risks and benefits

There are no personal risks involved in wearing the brain wave recorder or activity monitor. If your brain wave recording suggests that you have a sleep problem or there are any other abnormalities in the brain wave recording we will tell you and suggest where you can go for further evaluation and treatment. The activity monitor will not detect any disorders.

By participating in the study you will learn more about the causes of fatigue and effective strategies you can use to overcome fatigue when working night shifts.

It is a requirement of the Ethics Committee that you are informed that in the unlikely event of a physical injury as a result of your participation in this study, you will be covered by the accident compensation legislation with its limitations. If you have any questions about ACC please feel free to ask the researcher before you agree to take part in this study.

Participation

All air traffic controllers in Christchurch Area have been asked to participate in the study. Your participation is entirely voluntary. If you do agree to take part you are free to withdraw from the study at any time without having to give a reason and this will in no way affect your employment. You can choose to stop the recording carried out on night shifts at any time and if you wish, the data disk will be given to you.

If you have any queries or concerns regarding your rights as a participant in this study, you may wish to contact a Health and Disability Services Consumer Advocate, telephone 377 7501 or 0800 377 766.

Thank you for taking the time to consider being involved in the study. Leigh Signal or Philippa Gander would be happy to answer any questions you have about this study.

Researchers:

Leigh Signal
Research Training Fellow
Sleep/Wake Research Centre
Wellington School of Medicine
ph: 025 247 2786 or (04) 386 4933
(you are able to call these numbers collect)

Assoc. Prof. Philippa Gander
Director
Sleep/Wake Research Centre
Wellington School of Medicine
ph: (04) 4899 2880



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Please return this form if **you agree** to being contacted by Leigh Signal from the Sleep/Wake Research Centre regarding participation in the napping study.

Name: _____

Home address: _____

Home phone: _____

Cell phone: _____

Email address: _____

Please note that filling in this form does not mean that you consent to participate in the study. It only allows us to contact you to discuss the study further. You are still free to decide not to participate in the study at any time.

Please return this completed form to Sue Brash by the 30th July



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Please return this form if **you do not wish** to be contacted by Leigh Signal from the Sleep/Wake Research Centre regarding participation in the napping study.

Name: _____

The researchers would like to know why you have chosen not to participate in this study. This is so that they can be sure that those who choose to participate are not different in some way from those who choose not to participate. Such differences can possibly affect the usefulness of the study findings.

Sue Brash will remove the top section with your name before showing this response to the researchers.

I do not wish to participate in this napping study because: _____

Please return this completed form to Sue Brash by the 30th July



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APPENDIX K CONSENT FORM

Naps, Alertness and Performance

- I have read and I understand the information sheet dated 8 July 1999 for volunteers taking part in the study designed to investigate the effects of napping on night shifts. I have had the opportunity to discuss this study. I am satisfied with the answers I have been given.
- I understand that participation in this study is voluntary and that I may withdraw from the study at any time and this will in no way affect my employment.
- I understand that participation in this study is confidential and that no material, which could identify me, will be used in any reports on this study.
- I have had sufficient time to consider whether to take part.
- I know whom to contact if I have any questions about the study.

I (full name) _____ hereby consent to take part in this study.

Date _____

Signature _____

Researchers:	Leigh Signal	Philippa Gander
Contact phone number:	(04) 385 5999 ext. 6217 025 247 2786	(04) 385 5999 ext. 6051

Project explained by: _____

Project role: _____

Signature: _____ Date: _____

APPENDIX L ACTIGRAPHY PROTOCOL

For a sleep started after midnight, the date was checked to ensure that it was for the correct 24 hour period.

Bedtime/Sleep Start Guidelines

1. If the event marker was pressed, the logbook time matched, and actigraphy confirmed bedtime: The event marker was used for bedtime.
2. If there was no event marker, but a logbook time was given and matched the actigraphy data: The logbook time was used to determine bedtime.

Generally, auto-calculate in the *Actiware*TM software was used for sleep start. This was due to evidence suggesting that individuals are poor at judging the length of time it takes to go to sleep and normally over-estimate this time frame.

Get Up Time/Sleep End Guidelines

1. If the event marker was pressed, matched the logbook time, and the actigraphy data confirmed get up time: The event marker was used for get up time.
2. If the event marker was pressed, but there was no time recorded in the logbook, and the event marker did not closely match actigraphy: Actigraphy was used to determine get up time (as the event marker may be forgotten and pressed retrospectively)
3. If the event marker was pressed, but the logbook time does not match, and the event marker does not closely match actigraphy - Actigraphy was used to determine get up time (as event marker may be forgotten and pressed after getting up and logbook may also be completed retrospectively)
4. If the event marker was pressed, logbook time does not match, but event marker and actigraphy match: The event marker was used for get up time.
5. If the event marker was pressed, but matched wake up label on sleep log and actigraphy showed very little movement until “finished sleeping” label in logbook: The event marker was used as wakeup, given that the individual was sufficiently awake to press the marker. The actigraphy and logbook time were used for get up time.

6. If there was no event marker, and logbook and actigraphy did not match: Actigraphy was used for get up time (as logbook may be completed retrospectively)

Sleep shorter than 60 minutes

Actiware software cannot be used to calculate variables for periods of sleep shorter than 60 minutes. This is because the accuracy of the algorithm is reduced for such short time frames. In these instances the event marker was still pressed on the watch and the logbook completed, therefore bed time, get up time, and time in bed can be determined with combined reference to the event markers and labels in the sleep log. A variable was created in the final database to identify whether a sleep period was calculated based on full actigraphy (A) or limited actigraphy and sleep log data (or possibly sleep log data on its own) (L).

APPENDIX M BASELINE SLEEP

Before examining sleep loss it is necessary to determine how much sleep individuals require. Ideally, baseline sleep episodes would not be restricted by work or other demands, nor include sleep obtained to recover from a sleep debt. In addition, waking should be spontaneous. However, a range of factors including work patterns and family commitments often impinge on this, resulting in individuals obtaining less than their baseline sleep need.

If the amount of sleep being obtained is less than the baseline sleep requirement the consequence is a sleep debt. If, over a period of days, less sleep than the baseline value is obtained the sleep debt accumulates. This has been likened to a bank balance becoming further and further in “the red”. However, theory suggests that the reverse, or a sleep “credit”, is not possible to obtain. Instead, an individual can only clear their sleep debt by having two full nights of recovery sleep (Bonnet & Arand, 1994).

Because there is an enormous amount of individual variability in the amount of sleep required to feel fully rested, it is not suitable to apply a population average to a particular individual and expect it to represent their baseline sleep need (Ferrara & Gennaro, 2001). Therefore, baseline sleep requirements were calculated on an individual basis.

Calculating Baseline Sleep Requirements

It is important that sleep episodes included in the calculation of baseline sleep need do not contain recovery sleep. Carskadon and Dement (1981; 1982) have demonstrated that in recovering from two nights of 4 hours sleep, or seven nights of 5 hours sleep, levels of day time sleepiness return to baseline levels after one night of unrestricted sleep.

However, when sleep was prohibited for 48 hours, full recovery was only achieved after the second night of recovery sleep (Carskadon & Dement, 1982). Dinges et al (1997) have also demonstrated that two full nights of unrestricted sleep were required for performance to return to a baseline level after seven consecutive nights of sleep restriction.

Based on these findings, the first main night sleep episode following a working day was excluded when calculating baseline sleep requirements. The second night following the working week was included but was compared to the other non-working nights before baseline calculations were made. From the remaining data, four possible episodes could

be considered for calculating individual baseline sleep need. These are the first and second days of data collected (pre-cycle day 1 and 2), since data collection commenced the day after the end of a shift cycle; i.e. after one night of recovery sleep. The eighth and ninth days of data collection (post-cycle days 1 and 2) also met this requirement, as they are again at the end of a shift cycle and follow a night of recovery sleep.

To ensure sleep episodes included in the baseline calculations were as “unrestricted” as possible, any sleep that was followed by a work period commencing before midday on the following day was excluded from the calculation. This could occur on rostered days off if an additional shift was worked. In addition, only actigraphically calculated sleep episodes were utilised, to maximise the reliability of the data.

Baseline values for TIB, assumed sleep, and actual sleep were calculated, which involved summing all sleep that occurred between midday on one day and midday on the following day to give a total TIB or sleep length for a 24 hour period. The inclusion of all sleep over a 24 hour period was considered important, as not all individuals are necessarily able to obtain their sleep in one main sleep episode (Aschoff, 1994; Åkerstedt, 1998; Ferrara & Gennaro, 2001). Dividing 24 hour periods at the midday point is somewhat arbitrary, but this point in time was chosen so that main night sleep episodes did not have to be split between two consecutive days.

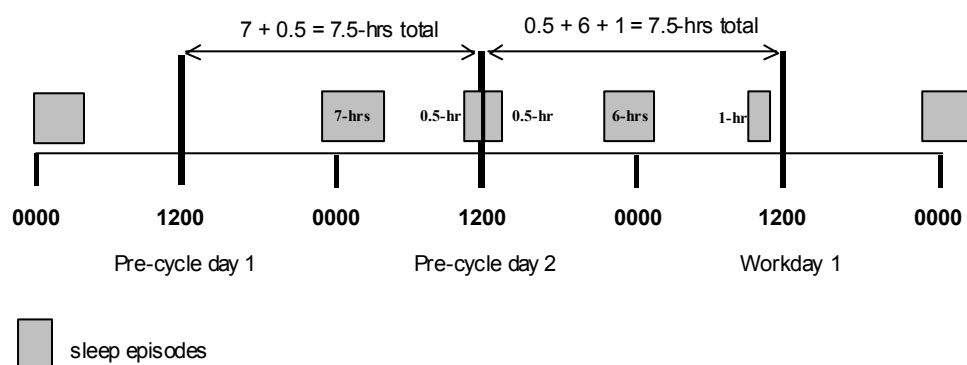


Figure M-1: Example Calculation of Baseline Sleep Requirement.

When a sleep episode crossed midday, the sleep was split across two 24 hour periods (see Figure M-1). Identical processes were applied to assumed sleep length. However, for

actual sleep this approach was not available, as there was no actigraphically calculated value for the amount of actual sleep contained in part of a sleep episode. To estimate this value, the difference in assumed sleep between either sleep start and midday or midday and sleep end was determined and multiplied by the actual sleep percentage for the entire sleep episode to give an indication of actual sleep length.

Baseline Sleep - Findings

Prior to further analyses the values for each 24 hour period, as calculated above, were utilised in a mixed model ANOVA. This was to establish whether there were any differences between the four 24 hour periods deemed suitable for the calculation of baseline sleep (pre-cycle days 1 and 2, and post-cycle days 1 and 2 in each study period) and if there were any differences related to the experimental condition. The factors included in this model are outlined in Table M-1.

Table M-1: Dependent and Independent Variables for Analyses of Baseline Sleep.

Dependent Variables	Independent Variables ⁵¹
<i>Baseline sleep</i>	
TIB	Shift cycle Nap
Assumed sleep	Baseline day Shift cycle x Nap
Actual sleep	Baseline day x Shift cycle Baseline day x Nap Shift cycle x Nap x Baseline day

None of the three mixed model ANOVAs identified any significant differences between fixed factors. This resulted in all available baseline episodes for each individual being averaged to provide an overall baseline value for TIB, assumed sleep, and actual sleep. On average, twelve 24 hour periods of data were available for each person for this calculation (range 7-16).

The mean baseline TIB value was 8.86 hours ($SD = 0.65$, $Range = 7.67-10.08$, $N = 27$) and mean baseline assumed sleep was 8.51 hours ($SD = 0.61$, $Range = 7.45-9.79$, $N = 27$).

The distribution of baseline assumed sleep is plotted in Figure M-2 below along with the

⁵¹ Shift cycle= shift cycle worked (includes either a K1 or K2 night shift)

Nap = napping condition on night shift (nap opportunity provided or not)

Baseline day = day within the study period (1 to 8)

distribution of baseline actual sleep (Figure M-3), which had a mean value of 7.51 hours ($SD = 0.45$, $Range = 6.63-8.54$, $N = 27$).

In comparison, controllers reported a sleep requirement of 8.0 hours ($SD = 0.9$, $Range = 6.0-9.5$, $N = 28$) in response to a pre-study questionnaire item asking how many hours of sleep were normally required to feel fully rested (Figure M-4).

All baseline variables, including self-reported sleep need, were normally distributed. Paired t tests were conducted to determine if self-reported sleep need differed from actigraphically calculated baseline values. In all tests significant differences were found, with baseline TIB and assumed sleep values being 0.87 hours and 0.52 hours greater respectively than self-reported sleep need, ($t_{(26)} = 5.23$, $p < .001$ and $t_{(26)} = 3.12$, $p = .004$ respectively), while baseline actual sleep was 0.48 hours less than self-reported sleep need ($t_{(26)} = -2.93$, $p = .007$).

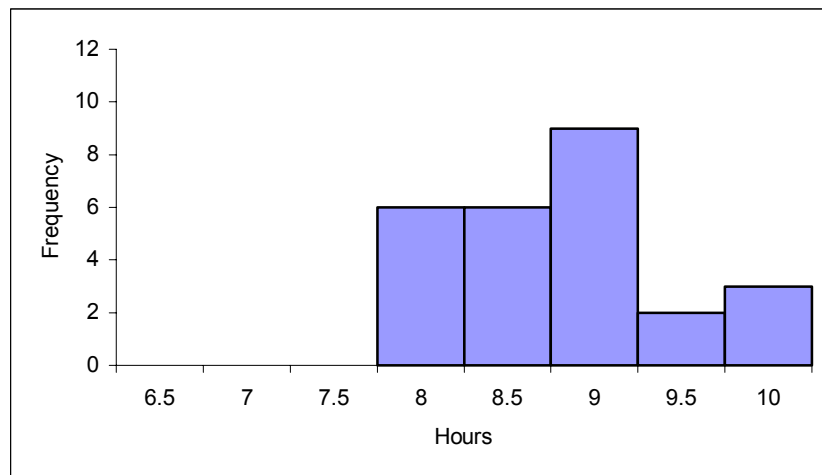


Figure M-2: Distribution of Baseline Assumed Sleep Values

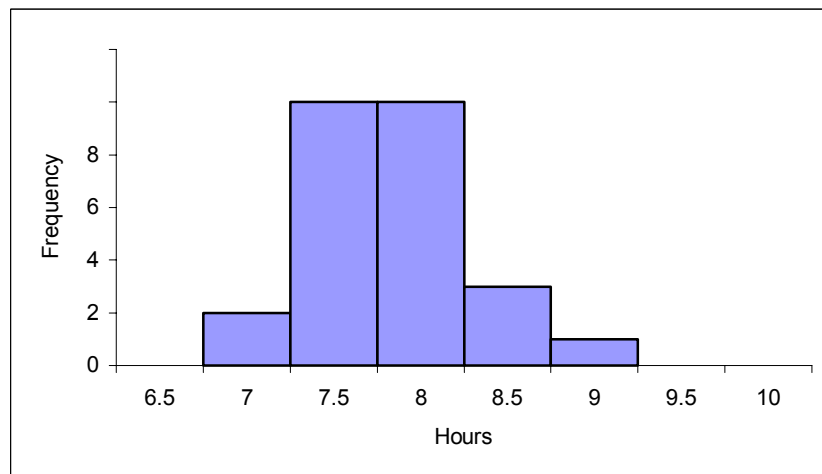


Figure M-3: Distribution of Baseline Actual Sleep Values

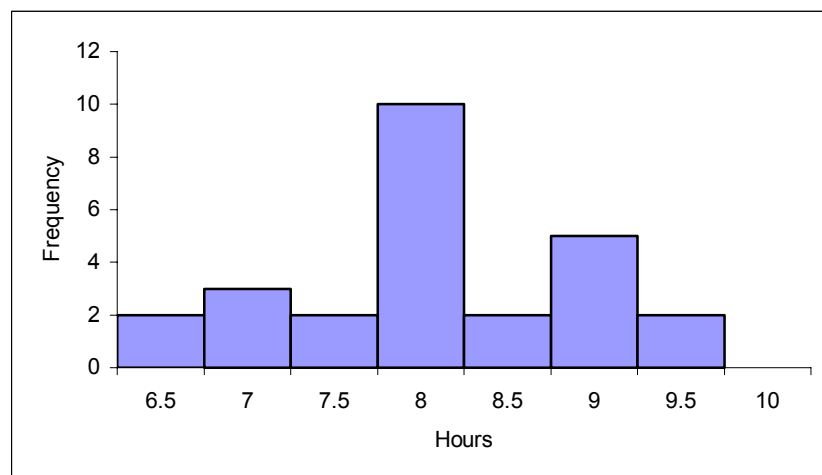


Figure M-4: Distribution of Subjective Baseline Sleep Requirement