Electrophysiological Quantification of Sleepiness and the Sleep Onset Period in Normal Sleepers and Narcoleptics

by

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Abstract

Traditionally, clinicians have quantified daytime sleepiness as the latency to intentional sleep onset on the Multiple Sleep Latency Test (Carskadon & Dement, 1978). Using this paradigm in excessively sleepy narcoleptics, Broughton and Aguirre (1987) reported that pressure for rapid eye movement (REM) sleep was increased relative to pressure for non-rapid eye movement (NREM) sleep. On the basis of these findings, Broughton and Aguirre (1987) proposed the existence of 'qualitatively' different states of sleepiness. However, the validity of this proposal is hindered because the statistics used to produce this finding were based on inflated degrees of freedom. Moreover, studies have questioned in general, the utility of the sleep latency measure of sleepiness due the confounding of sleepiness with the ability to fall asleep (Broughton, 1992), the near-zero latencies obtained in excessively sleepy populations (Sugarman & Walsh, 1989), and the failure of clinically effective pharmacological treatment to produce variations in latency to sleep onset (Hardge, Roth & Zorick, 1982). Furthermore, by relying on latency measures to distinguish normals and narcoleptics, differences within the microstructure of the transition from wakefulness to sleep onset have been largely ignored. An alternative to visually-scored sleep latency may be available in the technique of EEG power spectral analysis. Studies of experimentally-induced sleep deprivation in normals have shown increases in delta power during stages 2, 3, and 4 of recovery sleep (Borbely, Baumann, Brandeis, Strauch & Lehmann, 1981), and decreases in the ratio of eyes-closed to eyes-open alpha power during seated wakefulness on the Alpha Attenuation Test (Stampi, Stone & Michimori, 1993). Given these findings, research utilizing EEG power spectral analysis in clinical populations of excessively sleepy individuals appears warranted.

The present study is the first to quantify sleepiness and the sleep onset process in normal sleepers (n = 10) and excessively sleepy narcoleptics with cataplexy (n = 10) using EEG power spectral analysis in addition to visually-scored latency to sleep onset. Following nocturnal polysomnography, the Alpha Attenuation Test was administered at
0900, 1100, 1300, 1500 and 1700 hrs. The Multiple Sleep Latency Test paradigm was utilized to elicit opportunities for sleep onset at 1000, 1200, 1400, 1600 and 1800 hrs.

Power spectral analyses of wakefulness and stage 1 (stage-based strategy), and the chronological quartiles of the sleep onset period (time-based strategy) discriminated the sleep onset period in normal sleepers and narcoleptics according to the type of sleep to ensue. In particular, delta power was significantly enhanced within the sleep onset period of narcoleptic naps containing REM sleep and narcoleptic naps containing stage 2 sleep, in comparison to the sleep onset period of normal stage 1 and normal stage 2 naps. No spectral evidence was obtained to suggest that the narcoleptic sleep onset period prior to naps containing REM sleep differed qualitatively from the sleep onset period prior to NREM sleep. These findings have theoretical implications for the understanding of the microstructure of the process of normal and narcoleptic sleep onset, and the understanding of normal sleepiness and excessive sleepiness in narcoleptics during the intention to fall asleep. Furthermore, the Alpha Attenuation Test distinguished normal sleepers from excessively sleepy narcoleptics. Eyes-closed alpha power and the alpha attenuation coefficient were significantly reduced in narcoleptics compared to normals. Given the limitations of the sleep latency measure of sleepiness, future research may implicate the Alpha Attenuation Test as an alternative to the Multiple Sleep Latency Test in the accurate assessment of physiological sleepiness and the evaluation of pharmacological treatment efficacy.
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INTRODUCTION

The purpose of the present study was to objectively quantify sleepiness and the sleep onset process in normal sleepers and narcoleptics using computer-based EEG power spectral analysis in addition to visually-scored latency to sleep onset.

The Multiple Sleep Latency Test (Carskadon & Dement, 1978) is the traditional measure for assessing physiological sleepiness in normals and individuals with excessive daytime sleepiness. The Multiple Sleep Latency Test quantifies sleepiness as the mean latency to visually-scored intentional sleep onset across multiple daytime nap opportunities given under optimal sleep-inducing conditions. Using the Multiple Sleep Latency Test, Broughton and Aguirre (1987) have demonstrated that selective pressure for rapid eye movement (REM) sleep and non-rapid cyc movement (NREM) sleep exist in excessively sleepy individuals with narcolepsy. Mean latency to sleep onset was found to be shorter prior to naps containing REM sleep than naps consisting of just NREM sleep, suggesting that the selective pressure for REM sleep (i.e., REM sleepiness) was objectively greater than the selective pressure for NREM sleep (i.e., NREM sleepiness). Broughton (1982) proposed that these selective pressures for two types of sleep (i.e., REM sleep and NREM sleep) were reflective of qualitatively different states of sleepiness. However, questionable use of statistics limit the validity of Broughton and Aguirre's (1987) findings. Moreover, there are a number of problems associated with the use of the Multiple Sleep Latency Test.

The Multiple Sleep Latency Test is time consuming, and relies on the presence of a polysomnographer to sleep score on line. In addition, it is limited by a floor effect (or near zero latencies) in excessively sleepy populations (Sugarman & Walsh, 1989), the failure of these patients to show significant improvements in sleep onset latencies following subjectively effective pharmacological treatments (Hartse, Roth & Zorick, 1982), and the
confounding of sleepiness with the ability to fall asleep (Broughton, 1982). These shortcomings suggest the need for an alternative to visually-scored latency to sleep onset in the assessment of normals and excessively sleepy populations.

Computer-based EEG power spectral analysis provides a measure of the energy or power within EEG frequency bands per unit time, and may potentially offer an alternative to visually scored sleep latency testing as a means of quantifying fluctuations in sleepiness and alertness and the transition from wakefulness to sleep onset in normal sleepers and excessively sleepy individuals. The present study is unique in its purpose to investigate the utility of computer-based EEG power spectral analysis as an alternative to visually scored latency to sleep onset in the objective quantification of sleepiness and the sleep onset process in normal sleepers and narcoleptics.

Studies of shiftworkers (e.g., Torsvall & Akerstedt, 1983; 1987) and sleep deprived normals (e.g., Akerstedt, Torsvall & Gillberg, 1985) have documented variations in alpha power as a function of eyelid position as sleepiness and alertness wax and wane. As alertness decreases, alpha power decreases when eyes are closed, but increases when eyes are open. Based on these findings, the Alpha Attenuation Test (Michimori, Stampi & Stone, 1993) was recently developed. The Alpha Attenuation Test quantifies sleepiness as the ratio of mean eyes-closed to mean eyes-open alpha power in individuals seated in an illuminated room. This ratio is referred to as the alpha attenuation coefficient. As alertness decreases and sleepiness increases, the alpha attenuation coefficient decreases. The alpha attenuation coefficient has been found to correlate significantly with performance measures, subjective sleepiness estimates, and latency to sleep onset on the Multiple Sleep Latency Test in sleep deprived normals (Stampi, Stone & Michimori, 1993; Michimori et al., 1993). However, the Alpha Attenuation Test has yet to be investigated in a clinical population of excessively sleepy individuals, such as narcoleptics. This was an aim of the present study.
EEG power spectral analysis also provides a method of examining the microstructure of the process of sleep onset by tracking spectral changes in EEG activity throughout the entry into sleep. Few studies have investigated spectral changes occurring during the normal sleep onset process, and not one study has used EEG power spectral analysis to investigate the narcoleptic sleep onset process. Thus, the present study was unique in its aim to investigate EEG spectra across the traditional frequency bands (i.e., delta, theta, alpha, sigma and beta) during the transition from wakefulness to sleep in normals and narcoleptics. The Multiple Sleep Latency Test paradigm was used to elicit a number of daytime opportunities for intentional sleep onset. In addition to examining the normal sleep onset process and comparing it with the narcoleptic sleep onset process, the present study aimed to provide spectral evidence in support of Broughton's (1982) proposal that selective pressure for REM sleep and NREM sleep reflect the existence of qualitatively different states of sleepiness.

The introduction to this study is presented in four sections. In the first section, an overview of the features and theories of the pathophysiology of narcolepsy is provided. In the second section, the traditional methods of evaluating sleepiness are introduced; namely, subjective sleepiness measures, and the Multiple Sleep Latency Test. The utility of the Multiple Sleep Latency Test in the diagnosis of narcolepsy and the identification of qualitatively different states of sleepiness is discussed. This is followed by a summary of the limitations associated with the use of the Multiple Sleep Latency Test. EEG power spectral analysis is then presented as an alternative to the visual scoring of latency to sleep onset in the objective assessment of sleepiness. In particular, the recent development of the Alpha Attenuation Test is presented. In the third section, studies investigating spectral changes across the process of transition from wakefulness to sleep onset are introduced. Finally, in the fourth section the purpose, aims, hypotheses, and predictions of the present study are presented.
1. **OVERVIEW OF NARCOLEPSY**

The American Sleep Disorders Association (ASDA) (1990) defines narcolepsy as a sleep disorder of unknown etiology, characterized by excessive daytime sleepiness associated with cataplexy and other REM sleep phenomena, such as sleep paralysis and hypnagogenic hallucinations. It is currently estimated that narcolepsy affects 0.05% of the general population, or 50 individuals in every 100,000 (Hublin, Partinen, Kaprio, Koskenvuo & Guilleminault, 1994). However, the prevalence may vary depending on the population studied. Men and women are equally affected with narcolepsy. In Canada, narcolepsy occurs twice as often as multiple sclerosis, and half as often as Parkinson's disease (Hublin et al., 1994). Age of onset is usually during the second or third decade of life, with peak incidence around 14 years of age (ASDA, 1990).

**Essential features of narcolepsy**

*Excessive daytime sleepiness* is typically the first symptom to develop, and is characterized by frequent, brief episodes of voluntary naps, and by irresistible lapses into sleep (ASDA, 1990). Daytime sleep attacks are usually 10-20 minutes in duration. The narcoleptic will typically awaken feeling relatively refreshed from these sleep episodes, but within 1-3 hours, sleepiness returns. Narcoleptics do not sleep all day long: extreme and chronic drowsiness rather than excessive amounts of sleep are observed in narcolepsy. The quality of narcoleptic sleepiness has been likened to the sleepiness experienced by normal persons who are sleep deprived. For example, the sleepiness is most noticeable in boring or sedentary environments, and it may be temporarily relieved by movement or stimulation (Aldrich, 1992). However, narcoleptic sleepiness differs from the sleepiness associated with sleep deprivation in that no amount of nocturnal or daytime sleep will yield full alertness (Aldrich, 1992). With extreme effort, the narcoleptic may be able to temporarily avoid the pressure to sleep but ultimately, unless a nap is taken, inadvertent sleep attacks will occur (ASDA, 1990).
A history of cataplexy is a unique feature of narcolepsy. Cataplexy is a sudden loss of bilateral muscle tone, usually brought on by emotion or excitement (ASDA, 1990). Cataplexy may develop simultaneously or within a few months or years of the onset of excessive daytime sleepiness. Rarely does it precede the onset of excessive daytime sleepiness. Cataplexy is most commonly elicited by laughter; but anger, embarrassment, surprise, stress, fatigue, or heavy meals may also elicit it (Aldrich, 1990). During a cataplectic attack, consciousness is maintained, memory is not impaired, and respiration remains intact (ASDA, 1990). The loss of muscle tone varies in severity, and can range from a mild sensation of muscular weakness, such as a buckling of the knees, or a sagging of facial muscles, to a complete postural collapse and fall to the ground (ASDA, 1990). The duration of the attack is usually a few seconds or minutes. Frequency of occurrence varies between patients, from several attacks occurring daily, to less than once per month (ASDA, 1990). Cataplexy has been interpreted as representing a daytime triggering of the motor atonia or paralysis of REM sleep (Aldrich, 1992).

Associated features of narcolepsy

Sleep paralysis is a transient inability to move or speak during the onset of sleep or upon awakening from sleep (ASDA, 1990). Episodes usually last 1-10 minutes, during which time the person is fully aware of the condition, and can recall it afterward. Although the paralysis will resolve spontaneously, episodes may also be terminated by external stimulation, such as being touched or spoken to. Sleep paralysis is typically a frightening experience, and has been associated with the occurrence of sleep onset REM periods, or awakenings from REM sleep (ASDA, 1990). Approximately 10-50% of all narcoleptics experience sleep paralysis, however it is not unique to narcolepsy. Various studies of the incidence of sleep paralysis in normals have reported that 4.7-62.3 % of individuals surveyed reported experiencing at least one episode of sleep paralysis (e.g., Fukuda, 1994). However, it has been suggested that this wide range in reported incidence among normals may be due to cultural differences in the extent to which sleep paralysis is
known in folklore as a common occurrence rather than a medical condition (Fukuda, 1994).

**Hypnagogic hallucinations** are vivid perceptual experiences which occur during the transition between wakefulness and sleep onset (ASDA, 1990). They are usually visual in nature, and commonly occur in association with sleep paralysis in association with sleep onset REM periods or awakenings from REM sleep (ASDA, 1990). Hypnagogic hallucinations are experienced by 15-50% of all narcoleptics, but may also be experienced by those in the general population.

Excessive daytime sleepiness, cataplexy, sleep paralysis and hypnagogic hallucinations compose a classic group of symptoms referred to as the narcolepsy tetrad (Aldrich, 1992). However, there are other symptoms manifested by narcoleptics. For example, it has been estimated that 50-80% of narcoleptics experience **nocturnal sleep disruption** (ASDA, 1990). These disruptions include an increased number of awakenings, increased waketime after sleep onset, increased time spent in Stage 1, and REM sleep fragmentation (Montplaisir, 1976). Moreover, **lapses of memory** are reported by up to 50% of narcoleptics, and are most likely associated with impaired attention and concentration due to excessive daytime sleepiness rather than cortical dysfunction (Aldrich, 1992). Chronic excessive daytime sleepiness also may account for the reports of automatic or semipurposeful activity associated with amnesia in up to 80% of narcoleptics (Aldrich, 1992).

**Polysomnographic features of narcolepsy**

Premature occurrence of REM sleep (i.e., sleep onset REM period) in narcoleptics was first noted by Vogel in 1960, and was first associated descriptively to the narcoleptic symptom cluster by Rechtschaffen, Wolpert, Dement, Mitchell and Fisher in 1963 (Carskadon, 1986). The daytime polysomnography of narcoleptics typically reveals a reduced sleep onset latency of less than 10 minutes in addition to the occurrence of REM sleep within 10-20 minutes of sleep onset (i.e., sleep onset REM period) (ASDA, 1990).
Similarly, nocturnal polysomnography of narcoleptics typically reveals a reduced sleep onset latency and the occurrence of a sleep onset REM period in addition to nocturnal sleep disruption (ASDA, 1990).

**Treatment, course, and implications of narcolepsy**

Narcolepsy is a chronic, life-long condition, whose symptoms may be lessened, but not cured. Excessive daytime sleepiness may be somewhat alleviated with various classes of central nervous system stimulants (Aldrich, 1992). However, no drug has been found that can bring the narcoleptic to normal levels of alertness. Short daytime naps of 15-20 minute duration taken 3 times daily may assist in maintaining satisfactory levels of vigilance (Guilleminault, 1994). Although excessive daytime sleepiness typically remains at a constant level throughout the life of a narcoleptic, the frequency of incidents of cataplexy, sleep paralysis and hypnagogic hallucinations may decrease with increasing age (ASDA, 1990). Narcoleptics who experience severe cataplexy, sleep paralysis or hypnagogic hallucinations may be treated with tricyclic antidepressants, which have a side effect of reducing REM sleep, thereby reducing the frequency and severity of intrusions of REM sleep components (e.g., muscle atonia or vivid imagery) into wakefulness (Guilleminault, 1994). Nocturnal sleep disruption in narcolepsy usually worsens with increasing age at a rate faster than that observed in normals (Aldrich, 1992). Severely disturbed nocturnal sleep may be treated with sleeping pills (Aldrich, 1992).

Narcolepsy is a debilitating condition, having severe psychosocial and socioeconomic effects (Broughton & Broughton, 1994). During childhood and adolescence, narcoleptics commonly experience poor academic performance and embarrassment due to the occurrence of sleepiness or cataplexy (Broughton & Broughton, 1994). In adults, narcolepsy has been associated with loss of work hours, reduced productivity, increased work errors, and impaired social and family relations (Thorpy, 1992). Furthermore, narcolepsy is associated with an increased risk of accidents at home, while driving, and in the workplace (Thorpy, 1992; Broughton & Broughton, 1994).
Thus, narcolepsy impairs the quality of life, and negatively affects cognitive, intellectual and social functioning (Thorpy, 1992).

Theories of the pathophysiology of narcolepsy

Although the etiology of narcolepsy remains unknown, over the years a number of theories of pathophysiology have been proposed. There has been a tendency to explain the pathophysiology of narcolepsy as an essential dysfunction of REM sleep, because sleep onset REM periods, cataplexy, sleep paralysis and hypnagogic hallucinations are all considered to be abnormal manifestations of REM sleep (Guilleminault, Dement & Passouant, 1976). According to this theory, the neurophysiological system which typically regulates REM sleep is impaired in narcolepsy, producing abnormal sleep onset REM periods in addition to the symptoms of cataplexy, sleep paralysis and hypnagogic hallucinations. However, for several reasons, it is now debatable whether narcolepsy should be represented as a disease exclusively of REM sleep (Broughton, Valley, Aguirre, Roberts, Suwalski & Dunham, 1986). First, it is not at all clear how the proposed dysfunction of REM sleep would result in the primary symptom of excessive daytime sleepiness. Studies of daytime sleep in narcoleptics have demonstrated that approximately 50% of daytime naps and sleep attacks in narcolepsy consist of NREM sleep, and that nocturnal and daytime sleep latencies are abnormally short for sleep episodes beginning with NREM sleep and REM sleep (Broughton et al., 1986). This suggests that NREM and REM sleep mechanisms are equally involved in narcolepsy (Broughton et al., 1986).

A second reason that narcolepsy should not be seen as simply a disease of disordered REM sleep, is that excessive pressure for REM sleep and sleep onset REM periods is not uncommon in other situations. Sleep onset REM periods, for example, are known to occur in persons with endogenous depression, in normal individuals with irregular sleep/wake habits, during the recovery state after sleep deprivation, during ultrashort sleep schedules, during withdrawal from REM-suppressant drugs (Broughton et al., 1986), and during re-entry into nocturnal sleep following disrupted sleep (Fukuda,
Miyasita and Inugami, 1987). Moreover, sleep onset REM periods have also been observed in otherwise normal healthy individuals (Rosenthal et al., 1995). Similarly, hypnagogic hallucinations and sleep paralysis may also be seen in individuals without narcolepsy. Irregular sleep/wake schedules and drug withdrawal states can produce hypnagogic hallucinations. As well, isolated incidents and familial forms of sleep paralysis do exist (Broughton et al., 1986).

Because sleep onset REM periods, sleep paralysis and hypnagogic hallucinations are not unique to narcolepsy, Broughton et al. (1986) have questioned whether narcolepsy is best conceptualized as a disorder of REM sleep. Of all the REM sleep abnormalities, only cataplexy is truly diagnostic of narcolepsy, because cataplexy never occurs in non-narcoleptic individuals (Broughton et al., 1986). Researchers (e.g., Broughton et al., 1986; Aldrich, 1992) believe that cataplexy is best understood as a dissociation rather than a dysfunction of REM sleep. During the cataplectic attack, the motor atonia or paralysis appears without, or is dissociated from the other REM sleep components (Guilleminault, 1994). In other words, components of REM sleep intrude into wakefulness during cataplexy. Therefore, what is pathological in narcolepsy may not be the ability to enter into REM sleep directly from wakefulness, because non-narcoleptics can do this, too. Rather, it is now believed that what is uniquely pathological in narcolepsy is the presence of dissociated REM sleep during wakefulness in the form of cataplexy.

It has also been proposed that narcolepsy may represent a *chronobiological disorder*. Kripke (1976) proposed that a desynchronization of biological rhythms may contribute to the onset of daytime sleep attacks and nocturnal sleep disruption in narcolepsy. To date, there is little evidence to suggest that a circadian rhythm disruption is the primary factor leading to the onset of narcolepsy (Broughton & Mullington, 1994). However, a number of chronobiological disturbances may be associated with the symptomatology of narcolepsy (Broughton, 1989). Although total sleep time obtained by narcoleptics within a 24-hour period is equivalent to that obtained in normals, there is an
increase in the proportion of daytime to total sleep within a 24 hour period (Broughton, 1989). Narcoleptics tend to sleep more during the day, and less during the night compared to normals. This suggests that in narcolepsy there is a relative broadening of the circadian acrophase of sleep, or period of greatest probability that sleep will occur (Broughton, 1989).

The circadian acrophase of the nocturnal sleep stages in narcoleptics is also broadened relative to normals. In normals, the first REM period occurs approximately 60 minutes after sleep onset, and there is a progressive increase in the length of REM sleep episodes throughout the night. In normals, the probability of REM sleep occurrence is greatest during the last third of a night's sleep, and the REM sleep acrophase occurs approximately 7 1/2 hours after sleep onset, just at or after the usual time of awakening (Broughton, 1989a; Broughton, 1989b). By contrast, narcoleptics have nocturnal sleep onset REM periods, and they display a more even probability of REM sleep occurrence throughout the night. This suggests that the circadian REM sleep acrophase may be phase-advanced in narcoleptics (Broughton, 1989a). In normals, the slow wave sleep acrophase occurs within the first sleep cycle, specifically, approximately 1 hour after bedtime (Broughton, 1989b). In narcoleptics, this acrophase is also broadened, with slow wave sleep tending to be more evenly distributed throughout the night (Broughton, 1989a).

Although circadian rhythms appear to be somewhat disturbed in narcolepsy, the circasemidian and ultradian rhythms appear to be intact and even enhanced in narcoleptics. Circasemidian rhythms in normal adults represent a twice a day propensity for sleep, with a major sleep period typically occurring at night, and a second minor period of sleep or sleepiness occurring in the midafternoon (Broughton, 1989b). Nap studies and 24-hour continuous recordings of narcoleptic sleep patterns have shown a relative shortening of sleep onset latency during midafternoon naps (Broughton, 1989a; Broughton, 1989b),
suggesting that the secondary midafternoon increase in pressure for sleep (i.e., the circasemidian sleep propensity) is maintained in narcolepsy.

Ultradian rhythms in normal adults reflect a daytime rhythm of sleepiness and alertness that occurs with a periodicity approximately equivalent to the nocturnal NREM/REM cycle (90-120 min) (Broughton, 1989a; Broughton, 1989b). Kleitman (1963) called this ultradian rhythm the basic rest/activity cycle. Continuous EEG monitoring of narcoleptics has demonstrated that their daytime sleep episodes exhibit a spontaneous ultradian rhythmicity. For example, the probability of sleep onset REM period occurrence exhibits an ultradian increase every 90-100 minutes, with an overall circadian peak maximum in the morning (Broughton, 1989a; Broughton, 1989b).

To date, there is no evidence to suggest that the pathophysiology of narcolepsy represents a purely chronobiological disturbance. Although there is evidence for some degree of abnormality in circadian chronobiology in narcolepsy, it has not been shown to be diagnostic of or exclusive to narcolepsy. Thus, it may be concluded that despite mild chronobiological disruptions, the main features of normal circadian, circasemidian and ultradian distribution of sleep/wake patterns are maintained in narcolepsy (Broughton, 1989a).

Currently, a number of researchers have proposed that the underlying pathophysiology of narcolepsy represents a disturbance in the control of the boundaries between the normal states of sleep and wakefulness (Aldrich, 1992; Zorick, Roehrs, Wittig, Lamphere, Sicklesteel & Roth, 1986; Broughton et al., 1986). According to Broughton et al., (1986), "If it is true that the essential feature [of narcolepsy] is the existence of dissociated motor components of REM sleep in the form of cataplexy, it is equally apparent that dissociations of normal state boundaries and recombinations of state subcomponents are seen in myriad expressions of the disease...as though whatever neurochemical 'glues'...exist for sleep and wake state continuity and for their full reciprocity have somehow become dissolved" (p.213). For example, in narcolepsy, the
ability to sustain nocturnal sleep is impaired, as is the ability to maintain daytime wakefulness (Aldrich, 1992). The dissociated motor atonia components of REM sleep intrude into wakefulness as cataplexy and sleep paralysis (Broughton et al., 1986). Within sleep, episodes of REM sleep atonia intrude into NREM sleep, and NREM spindle activity may intrude into REM sleep (Broughton et al., 1986). Thus, what is pathophysiological in narcolepsy is not REM sleep per se, rather it is the inability to sustain a specific neural state (Zorick et al., 1986).

2. METHODS OF ASSESSING SLEEPINESS

Subjective sleepiness

Two of the most commonly used self-rating scales or introspective measures of sleepiness are the Stanford Sleepiness Scale, developed by Hoddes and colleagues at Stanford University in the early 1970's (Hoddes, Dement & Zarcone, 1971; Hoddes, Zarcone, Smythe, Phillips & Dement, 1973), and the Visual Analogue Sleepiness Scale (Folstein & Luria, 1973). The Stanford Sleepiness Scale is a seven point Lykert Scale; individuals rate their present level of alertness according to one of 7 statements ranging from 'wide awake' (1) to 'cannot stay awake' (7) (Hoddes et al., 1973). The Visual Analogue Sleepiness Scale is a horizontal line approximately 100 mm in length, labeled 'very alert' on the left, and 'very sleepy' on the right. Individuals indicate their present state of sleepiness by drawing a vertical mark at the appropriate point on the line. The Stanford Sleepiness Scale and Visual Analogue Sleepiness Scale were designed to quantify perceptions of introspective or subjective sleepiness and alertness (Carskadon & Dement, 1982). Indeed, initial validation studies for the Stanford Sleepiness Scale indicated that sleepiness ratings correlated with performance measures prior to and throughout a sleep deprivation paradigm, and that Stanford Sleepiness Scale values indicated greater sleepiness during sleep loss compared to baseline (Hoddes et al., 1973). However, subjective sleepiness ratings may be unreliable in excessive sleepy patient populations, as
patients may tend to underestimate the presence of sleepiness due to a loss of reference for what feeling alert is really like (Mitler, Gujavarty, Sampson & Browman, 1982). It is not uncommon for excessively sleepy patients to fall asleep despite the fact that they had just rated themselves as fully alert (Thorpy, 1992). Thus, there was a need for reliable and valid, non-introspective (i.e., objective) measures of sleepiness.

**Multiple Sleep Latency Test**

Polygraphic monitoring in conjunction with 'on-line' visual sleep stage scoring during multiple daytime nap opportunities provided researchers with one method of obtaining objective measures of alertness which were independent of the patient's estimates of subjective sleepiness (Hartse, Roth & Zorick, 1982). Measurement of sleepiness using sleep latency tests originated with the development of the Multiple Sleep Latency Test (Carskadon & Dement, 1977a). In the early 1970's, a group of researchers led by Carskadon and Dement at Stanford University, reasoned that sleepiness or tendency for sleep onset could be measured at any moment as the speed of falling asleep at that moment. One of the first attempts to objectively quantify normal variations in sleep tendency involved placing normal young adults on a 90-minute schedule consisting of 30 minutes of sleep opportunity followed by 60 minutes of enforced wakefulness (Carskadon & Dement, 1975; Carskadon & Dement, 1977a). Participants were given 16 opportunities throughout a 24-hour period to fall asleep while lying down with their eyes closed in a darkened room. The 90-minute schedule was maintained for approximately six 24-hour days. Repeated measures of sleep latency were readily obtained, thereby allowing Carskadon and Dement to conclude that the approach of multiple sleep latency testing was feasible. Subsequent modifications to the Multiple Sleep Latency Test involved increasing the testing interval from 90 minutes to 2 hours, awakening individuals following a clear indication of Stage 1 (which was assumed to reflect tendency for sleep onset), and decreasing the time in bed to a maximum of 20 minutes per nap opportunity (Carskadon & Dement, 1982).
Given that the setting of the Multiple Sleep Latency Test is intended to promote sleep onset (i.e., it is administered in a quiet, darkened room, and participants are told to close their eyes and try to fall asleep), the Multiple Sleep Latency Test was designed to measure physiological sleep tendency in the absence of alerting factors. That is, the Multiple Sleep Latency Test purportedly assesses the underlying or latent tendency for sleep to occur (Carskadon & Dement, 1982). This latent sleep tendency must be contrasted with what Carskadon and Dement (1982) referred to as manifest sleep tendency, which is not measured by the Multiple Sleep Latency Test. Carskadon and Dement (1982) likened manifest sleep tendency to introspective or behavioral indices of sleepiness or alertness, in that it may change on a moment-to-moment basis depending of the presence or absence of stimuli such as light, noise, activity level, motivation. A physiologically sleepy person may be able to temporarily relieve manifest sleepiness by engaging in physical activity or movement. Carskadon and Dement (1982) proposed that by reducing impinging stimuli, the Multiple Sleep Latency Test environment is able to unmask the latent physiological sleep tendency. According to Carskadon and Dement (1982), a truly alert person will neither feel sleepy nor appear to be sleepy when placed in the low-stimulus environment of the Multiple Sleep Latency Test.

In 1978, the Stanford group evaluated physiological sleep tendency in narcoleptics and normals by measuring sleep onset latency according to the Multiple Sleep Latency Test paradigm (Richardson, Carskadon, Flagg, Van den Hoed, Dement & Mitler, 1978; Mitler et al., 1979). In study 1, participants were given 6 nap opportunities every 2 hours, beginning at either 0800 or 0930 hr. Participants were instructed to lie quietly and to try to fall asleep while lying in a darkened, quiet room. Each nap opportunity was terminated after either 1 minute of NREM or REM sleep, or after 20 minutes of wakefulness. In study 2, participants were given 5 nap opportunities every 2 hours, beginning at 1000 hr, and were allowed a maximum of 10 minutes of sleep on each nap. The Multiple Sleep Latency Test was insensitive to differences between 6 nap- and 5 nap opportunities, or to
minor variations in the timing of the onset of nap opportunities. The protocol of study 2 was adopted as the clinical version of the Multiple Sleep Latency Test. Narcoleptics consistently fell asleep faster than the control participants. Mean latency to sleep onset across naps ranged from 1.5-3.4 minutes for narcoleptics, and from 7.4-14.9 minutes for controls. Narcoleptics had on average, 3.7 sleep onset REM periods, with actual occurrences ranging from 2 to 5. Time of test did not affect the likelihood of a REM sleep episode. By contrast, not one sleep onset REM period was observed in the controls. It was concluded that multiple daytime sleep latency testing could provide a means for differentiating narcoleptics from normal controls in the assessment of physiological sleep tendency and the documentation of sleep onset REM periods.

Since the initial study in 1978, the Multiple Sleep Latency Test has become the traditional diagnostic tool in evaluating narcolepsy. In an individual complaining of excessive daytime sleepiness and cataplexy, the presence of two or more sleep onset REM periods in conjunction with a mean latency to sleep onset of 5 minutes or less is considered to be diagnostic of narcolepsy (ASDA, 1990). Researchers have concluded that the Multiple Sleep Latency Test appears to be a sensitive and reliable test of sleepiness in narcolepsy. More than 80% of narcoleptics have mean sleep onset latencies of less than 5 minutes, and demonstrate 2 or more sleep onset REM periods (Van den Hoed et al., 1981; Amira, Joamson & Logowitz, 1985; Thorpy, 1992). Mean sleep onset latency in narcoleptics has been shown not to vary significantly over a one-month interval (Scrima, Hartman, Johnson, Thomas & Hiller, 1990). Furthermore, one study showed that 13 of 14 narcoleptics had two or more sleep onset REM periods during initial testing and re-testing, and that REM latency during initial testing correlated significantly with REM latency during re-testing (Rosenthal et al., 1992). Similarly, the test-retest reliability for mean latency to sleep onset on the Multiple Sleep Latency Test in normals has been found to be .97 over a 4-14 month test-retest interval (Zwyghuizen-Doorenbos, Roehrs, Schaefer & Roth, 1988).
Given that the Multiple Sleep Latency Test provides an objective evaluation of daytime sleepiness, there should be variations in sleep onset latencies as individuals report varying degrees of daytime sleepiness (Hartse, Roth & Zorick, 1982). The Multiple Sleep Latency Test has been validated as a tool for detecting varying degrees of sleepiness in paradigms that experimentally increase sleepiness, such as sleep deprivation studies (e.g., Carskadon & Dement, 1977b; 1979), and in paradigms that decrease sleepiness, such as sleep extension or caffeine ingestion studies (e.g., Thorpy, 1992).

**Qualitatively different states of sleepiness**

The Multiple Sleep Latency Test was developed to measure the functional consequence of sleepiness; that is, falling asleep (Thorpy, 1992). Studies by Broughton and his colleagues (1982, 1987) utilizing the Multiple Sleep Latency Test have provided evidence to suggest that sleepiness may be more than a unitary state, varying only in magnitude or degree.

Broughton (1982) proposed that rather than being a unitary state or phenomenon varying only in magnitude or degree, sleepiness may consist of qualitatively different states. Accordingly, Broughton provided evidence for this proposal by noting the different effects of undersleeping versus oversleeping on subjective state and performance abilities. Whereas sleep loss tends to lead to irritability and over-reactivity, sleep extension or satiation may cause a feeling of thick-headedness and hyporesponsiveness. Furthermore, Broughton (1982) proposed that there are three basic mammalian states, wakefulness, REM sleep, and NREM sleep, which are unique and show qualitative differences. Furthermore, Broughton suggested that qualitatively different types of sleepiness exist based upon the predominant involvement of these individual states of wakefulness and sleep. Thus, selective pressure for REM sleep would produce REM sleepiness, selective pressure for NREM sleep would produce NREM sleepiness, and impairment of the reticulo-cortical mechanisms sustaining wakefulness would produce de-arousal sleepiness. Patients with narcolepsy spontaneously exhibit both REM and NREM
sleep at sleep onset, thus allowing for the investigation of REM and NREM sleepiness prior to REM and NREM naps elicited by the Multiple Sleep Onset Test. Broughton and colleagues have succeeded in documenting the existence of states of REM sleepiness and NREM sleepiness in narcoleptics.

Broughton and Aguirre (1987) compared sleep onset latencies on the Multiple Sleep Latency Test and subjective estimates of sleepiness in order to investigate REM sleepiness and NREM sleepiness immediately prior to naps containing REM sleep and those consisting of NREM sleep alone. Participants consisted of twelve patients (mean age 46.3 y) with narcolepsy-cataplexy who were untreated or withdrawn from antidepressants for at least 3 weeks, and from stimulants for at least one week prior to testing. Twelve sex- and age-matched controls were also tested. The Multiple Sleep Latency Test was performed on two consecutive days, with nap opportunities occurring at 1000, 1200, 1400, 1600 and 1800 hrs. The Stanford Sleepiness Scale was administered just prior to, and 3 minutes following each nap on the Multiple Sleep Latency Test. Each nap was terminated after 20 minutes if sleep did not occur, or after 10 consecutive minutes of sleep. Sleep onset latency was defined as the interval between the beginning of each test (lights-out), and the occurrence of 6 consecutive 30 second epochs (i.e., 2 minutes) of any of Stage 1, Stage 2 or REM sleep. Broughton and Aguirre (1987) reported that during REM sleepiness compared to NREM sleepiness, narcoleptics were both subjectively and objectively sleepier, having significantly greater scores on the Stanford Sleepiness Scale, and significantly shorter mean latencies to subsequent sleep onset, providing evidence for qualitatively different states of sleepiness.

However, it must be mentioned that Broughton and Aguirre (1987) based their conclusions on questionable statistical analyses. They analyzed scores on the Stanford Sleepiness Scale and sleep latency for REM naps and NREM naps using paired t-tests, which erroneously treated the data as if there were 50 narcoleptics rather than 10 (i.e., 10 participants x 5 nap opportunities), thereby inflating the degrees of freedom and the
statistical power of the t-test. In any case, it was reported that narcoleptics fell asleep almost twice as rapidly into REM-containing naps (mean stage 1 latency = 1.8 min), as they did into NREM-only naps (mean stage 1 latency = 3.3 min).

Broughton (1992) reported that these findings (Broughton & Aguirre, 1987) were replicated by Burton (1988, cited in Broughton, 1992). However, other studies have either found non-significant differences between REM and NREM sleepiness, or found results opposite to those obtained by Broughton & Aguirre (1987). Zorick et al. (1986) investigated latency to sleep onset on the Multiple Sleep Latency Test for naps containing REM sleep, and naps consisting of only NREM sleep in 103 unmedicated patients with narcolepsy (mean age 43.7 y). No mention was made of whether the presence of cataplexy was a pre-requisite for the diagnosis of narcolepsy. The Multiple Sleep Latency Test was performed on one day only, with nap opportunities occurring at 1000, 1200, 1400 and 1600 hrs. Each nap was terminated after 20 minutes if sleep did not occur, or after 15 consecutive minutes of sleep. Zorick et al. also evaluated objective sleepiness using the erroneous t-test method mentioned above (i.e., the four nap opportunities from each narcoleptic were treated as between-subjects rather than repeated-measures data). However, although the latency to stage 1 sleep in naps containing REM sleep was found to be shorter than the latency to naps consisting of just NREM sleep (2.3 minutes versus 3.2 minutes), this difference was not statistically significant. Thus, no evidence for qualitatively different states of sleepiness (i.e., REM sleepiness versus NREM sleepiness) in narcoleptics was found.

Billiard (1976) investigated sleep onset latencies to naps containing REM sleep and naps consisting only of NREM sleep in 10 unmedicated patients with narcolepsy-cataplexy (mean age 42.7 y). Patients were given 15-minute nap opportunities at 0930, 1215, 1430, 1645, and 1900 hrs. Contrary to the results of Broughton & Aguirre (1987), NREM sleepiness was found to be objectively greater than REM sleepiness. Latency to stage 1 was shorter for naps consisting of NREM sleep (1.6 min) compared to naps
containing REM sleep (2.2 min), however, no statistical analysis was reported for this effect.

One of the aims of the present study was to attempt to provide evidence in support of Broughton and Aguirre's (1987) findings that latency to sleep onset on the Multiple Sleep Latency Test was shorter prior to naps containing REM sleep compared to naps containing just NREM sleep. Moreover, the present study utilized a novel approach in the investigation of qualitatively different states of sleepiness by comparing EEG power spectra during the sleep onset period of REM and NREM naps in narcoleptics with normal NREM sleep onsets.

Limitations of the Multiple Sleep Latency Test

Despite the widespread use of the Multiple Sleep Latency Test, a number of limitations hinder its utility. Because the Multiple Sleep Latency Test was developed to measure the functional consequence of sleepiness; that is, falling asleep (Thorpy, 1992), latency to sleep onset on the Multiple Sleep Latency Test should be related to subjective estimates of sleepiness. Initially, studies suggested a high correlation between the Stanford Sleepiness Scale and latency to sleep onset on the Multiple Sleep Latency Test in normals (Carskadon & Dement, 1975; 1979; 1981). However, discrepancies soon began to appear; reduced or non-significant relationships were noted in normals, insomniacs and apneics. Indeed, physiological sleepiness as measured by the Multiple Sleep Latency Test cannot be reliably introspected by most patients (Carskadon, 1986). Studies of the effects of stimulant medications in the treatment of narcolepsy have failed to demonstrate a reduction in sleepiness at clinically effective doses (Van den Hoed et al., 1981). In this vein, Hartse, Roth and Zorick (1982) studied the objective effects of stimulant treatment in 5 narcoleptics who had reported subjective improvement in their daytime alertness following treatment with stimulants. They found that the pre-treatment latencies on the Multiple Sleep Latency Test did not differ from the post-treatment latencies, suggesting that pharmacological treatment may not change the propensity for sleep as measured by
the Multiple Sleep Latency Test. This is a significant liability to the Multiple Sleep Latency Test, if one considers that the goal of pharmacological treatment is to decrease a patient's level of sleepiness, and increase alertness or the ability to sustain wakefulness (Hartse et al., 1982). It has been suggested that stimulant treatment may affect factors relating manifest sleepiness without influencing physiological sleep tendency (Hartse et al., 1982).

Carskadon and Dement (1982) concede that in narcoleptics who tend to fall asleep almost instantaneously on the Multiple Sleep Latency Test, there may be subtle variations in the patients' level of sleepiness/alertness and the ability to resist sleep that are not evident on the Multiple Sleep Latency Test. There are limits to the sensitivity of the Multiple Sleep Latency Test in assessing excessively sleepy patients (Sugarman & Walsh, 1989). Sugarman and Walsh (1989) propose that regardless of the underlying physiological level of sleep tendency, as sleep latency on the Multiple Sleep Latency Test approaches zero there is a limit to the rate at which an individual can fall asleep. This 'floor effect' hinders the ability to discriminate between various levels of sleepiness once extremely short sleep onset latencies begin to occur on the Multiple Sleep Latency Test (Sugarman & Walsh, 1989). Thus, attempts to pharmacologically reduce sleepiness in narcoleptics may not be reflected in latency to sleep onset on the Multiple Sleep Latency Test, despite subjective reports of improvement (Sugarman & Walsh, 1989).

Despite arguments to the contrary, it does appear that latency to sleep onset on the Multiple Sleep Latency Test is influenced by factors other than the physiological state of sleepiness (Thorpy, 1992). Although persons experiencing pathological levels of sleepiness are likely to fall asleep when given the opportunity on the Multiple Sleep Latency Test, their tendency toward sleep onset will be affected by their present psychological or behavioral state (Thorpy, 1992). A severely sleepy individual may present little evidence of sleepiness on the Multiple Sleep Latency Test if he/she is mentally stimulated by psychological, behavioral, or medicinal means (Thorpy, 1992).
This discrepancy between the underlying physiological or latent drive for sleep, and the overt manifest level of sleepiness may limit the reliability of the Multiple Sleep Latency Test in some patients at some times (Thorpy, 1992).

The Multiple Sleep Latency Test is promised on the belief that sleepy individuals fall asleep more quickly than alert individuals (Hartse et al., 1982). However, the validity of the Multiple Sleep Latency Test may be questioned due to the confounding of sleepiness with the learned ability to fall asleep, or 'sleepability' (Broughton, 1992). It is not unusual for non-complaining normals to fall asleep almost instantaneously on the Multiple Sleep Latency Test, because they have learned or developed the ability to fall asleep 'as soon as their head hits the pillow' (Hartse et al., 1982, Lavie & Scherson, 1981 cited in Broughton, 1992).

Thus, the validity of the Multiple Sleep Latency Test may be questioned due to the confounding of sleepiness with sleepability, the floor effect (or near zero latencies) in clinical populations, and the failure of patients to show significant improvement in Multiple Sleep Latency Test latencies following subjectively effective pharmacological treatment. Furthermore, the Multiple Sleep Latency Test is time consuming. Also, it relies on the presence of a polysomnographer to score the test on line, adding a subjective component into a so-called objective measure of sleepiness. These shortcomings suggest the need for alternate means of detecting pathological sleepiness.

One of the aims of the present study was to investigate the utility of a measure based on EEG power spectral analysis as an alternative to visually-scored latency to sleep onset (i.e., the Multiple Sleep Latency Test) in the objective assessment of daytime sleepiness in normals and excessively sleepy individuals, such as narcoleptics.

**EEG power spectral analysis**

Loomis, Harvey and Hobart (1936; 1937) were one of the first group of researchers to report EEG descriptions of the process of falling asleep. They demonstrated that in relaxed normals lying with their eyes closed, the EEG is dominated
by regular rhythms or trains of waves having a frequency of 8-12 cycles per second. This alpha activity corresponded with the participants' ability to respond to environmental stimuli. As alpha activity began to break up, the participants stopped responding to stimuli. Other studies (e.g., Rodin, Luby & Gottlieb, 1962; Naitoh, Kales, Kollar, Smith & Jacobson, 1969) using multiple scheduled sessions with eyes-closed have shown that the percent alpha time remaining after eye closure decreases as fatigue, sleep deprivation, and performance deterioration increase. Therefore, in relaxed, eyes-closed conditions, the presence of alpha activity signals alertness, and the absence of alpha activity signals sleepiness. In contrast, if alpha activity is present when the eyes are open, this signals a decrement in alertness, (i.e., an increase in drowsiness or sleepiness) (Akerstedt, Torsvall & Gillberg, 1987).

Computer-based EEG analysis has provided a more precise alternative to the visual analysis of EEG bands. The technique of EEG power spectra analysis divides the EEG pattern into its component frequencies by applying the fast Fourier transformation (FFT) (Akerstedt, Torsvall & Gillberg, 1987). The resulting spectrum is then integrated across selected frequency bands (Akerstedt et al., 1987). When the resulting values are squared, a measure is obtained which reflects the energy or power in the band (Akerstedt et al., 1987). The advantage of power spectral analysis over visual analysis of the EEG is its ability to quantify background EEG activity much faster and with greater amounts of information than is possible by visual analysis alone (Akerstedt et al., 1987). The output provides a continuous scale of EEG activity which may offer a potential alternative to sleep latency testing as a means of quantifying fluctuations in sleepiness and alertness in normal individuals as well as pathologically sleepy patients.

To illustrate, Akerstedt and colleagues have investigated spectral changes in EEG under conditions of experimentally induced sleepiness, and in field studies of shiftworkers. Torsvall and Akerstedt (1983; 1987) continuously recorded EEG of 11 train drivers during 4.5-hour night and day trips along the same route. The drivers were divided into
two groups: those who reported severe sleepiness on the night shift, and those who did not. During the day shift, EEG alpha power did not differ between the groups. During the night shift, alpha power was significantly increased above the daytime levels for the self-rated sleepy group. Moreover, the sleepy group had significantly greater alpha power than the alert group during the night shift. Similar results were reported for theta power. Subjective sleepiness ratings, which were given every 30 minutes, paralleled the EEG data, and were significantly correlated intraindividually during the night shift but not the day shift. It was concluded that night shift work led to increased subjective sleepiness, the degree of which was reflected in spectral changes in the EEG, particularly within the alpha and theta bands. The finding of increased theta or alpha may thus be interpreted as signifying an increase in sleepiness.

Akerstedt et al. (1985) deprived normal sleepers of sleep for one night. Every two hours between 2300 and 1100 hr, participants rated their current level of alertness/sleepiness before sitting in a chair and focusing on a spot on the wall for 5 minutes, and then closing their eyes for 2 minutes. Spectral analysis of the EEG showed that during subjectively rated alertness, alpha power was low when eyes were open, but significantly increased when eyes were closed. By contrast, during maximum subjectively rated sleepiness, alpha power was high when eyes were open, but significantly decreased when eyes were closed. Theta power also increased during sleepiness, but regardless of eye-lid position. The authors report that alpha power and theta power were significantly correlated with the subjective ratings of sleepiness, but do not mention whether eye-lid position was open, closed or both. Thus, studies by Akerstedt and colleagues have demonstrated that the spectral analysis of the EEG produces data that discriminate between variations in subjective reports of alertness and sleepiness.

Akerstedt and colleagues have also demonstrated that FFT analysis of EEG provides an objective and quantitative indication of the degree of sleepiness (Torsvall & Akerstedt, 1985). Torsvall and Akerstedt (1988) investigated spectral EEG parameters in
normal individuals in which sleepiness was induced by exposure to a monotonous 45
minute visual vigilance test at night. Participants sat in front of a test display and a video
camera, and were instructed to watch the display and press a hand-held switch whenever
the signal appeared. The authors selected a signal that was easily detectable so that any
omission would be due to a true absence (i.e., to extreme behavioral sleepiness), rather
than to a brief lapse of attention. Examination of the spectral content of the EEG just
prior to hits, misses, and dozing off episodes showed that the greatest levels of alpha,
theta and delta power occurred just prior to dozing off episodes, and the lowest levels
occurred just before hits. Thus, the intrusion of alpha, theta and delta activity into the
beta activity of active wakefulness may be interpreted as indicating sleepiness (Torsvall &
Akerstedt, 1988).

**Alpha Attenuation Test**

To sum, studies (Akerstedt et al., 1985; 1987) have demonstrated that as
individuals move from alertness towards sleepiness, the EEG power in the alpha frequency
range decreases when eyes are closed, but increases when eyes are open. Based on these
findings, the Alpha Attenuation Test (Stampi, Stone & Michimori, 1993; Michimori,
Stampi & Stone, 1993; Stampi, Michimori & Aguirre, 1994) was developed as a new
method of quantifying variations in physiological sleepiness.

During the original version of the Alpha Attenuation Test (Michimori et al., 1993),
individuals were polysomnographically recorded while seated in an illuminated room.
Individuals were asked to repeatedly vary their eyelid position from open to closed every
two minutes for a total of 12 minutes. The EEG (O1-A2) was subjected to spectral
analysis to obtain the mean power spectrum for the alpha band (8-12 Hz) for the eyes-
closed and eyes-open conditions. The ratio of eyes-closed to eyes-open EEG alpha power
is referred to as the alpha attenuation coefficient. The authors proposed that fluctuations
in alertness levels would be mirrored in the alpha attenuation coefficient, such that the
lower the alpha attenuation coefficient, the lower the alertness level (Stampi et al., 1994).
Accordingly, four normals were deprived of sleep for 36 hours, from 0900 to 1900 hr the following day, during which time they underwent a number of tests at two hour intervals. Each test session consisted of two subjective sleepiness scales, one Alpha Attenuation Test, one nap opportunity from the Multiple Sleep Latency Test, and four performance measures from the Walter Reed Performance Assessment Battery. Alpha attenuation coefficients were correlated intraindividually with latency to sleep onset on the Multiple Sleep Latency Test, and with mean reaction time for correct responses for the four performance measures. The alpha attenuation coefficient correlated significantly with three of the four performance measures in two of the four participants (Michimori et al., 1993). The alpha attenuation coefficient correlated significantly with latency to sleep onset on the Multiple Sleep Latency Test in all 4 subjects, and these correlations were greater than the correlations between the Multiple Sleep Latency Test and any of the subjective sleepiness or performance assessment measures, although the authors do not report the actual results (Stampi et al., 1993).

Modifications to the Alpha Attenuation Test were made in a subsequent study in which ten normals underwent one night of sleep deprivation (Stampi et al., 1994; Stampi, Michimori & Aguirre, 1995). Every two hours from 1700 to 0900 hr, participants performed 3 versions of the Alpha Attenuation Test, followed by a nap opportunity on the Multiple Sleep Latency Test. The Visual Analogue Sleepiness Scale was administered before each test, and a 5-minute interval separated each Alpha Attenuation Test and Multiple Sleep Latency Test. For the Alpha Attenuation Test, participants were instructed to vary their eyelid position from closed to open every 30 seconds for a total of 5 minutes (version 1), every 60 seconds for a total of 6 minutes (version 2), and every 2 minutes for a total of 12 minutes (original version). Alpha attenuation coefficients for each Alpha Attenuation Test version and latency to stage 1 were standardized using z-scores, and then correlated. For each version of the Alpha Attenuation Test, the alpha attenuation coefficient correlated significantly with sleep latency and with subjective sleepiness.
However, correlations were highest for the 12-minute version of the Alpha Attenuation Test, and lowest for the 5-minute version. Although the 12-minute version of the Alpha Attenuation Test produced the highest correlation with sleep latency, it also produced the greatest decrement in subjective sleepiness. Thus, the authors concluded that the effectiveness of the Alpha Attenuation Test in assessing physiological sleepiness without artificially promoting subjective sleepiness is maintained by making the test as short as 6 minutes, and by utilizing a one-minute alternation between eyelid position (Stampi et al., 1995).

The Alpha Attenuation Test has also been tested in shiftworkers working the nightshift (Heitmann, Stampi & Anandan, 1995). Seven shiftworkers working the nightshift (1700-0500 hr) were administered the 6-minute Alpha Attenuation Test, Visual Analogue Sleepiness Scale, Stanford Sleepiness Scale and performance measures four times per night for a total of six nights. Alpha attenuation coefficients were correlated with the Visual Analogue Sleepiness Scale and Stanford Sleepiness Scale intrindividually. Correlations were lowest for participants who produced extremely 'low' or extremely 'high' alpha attenuation coefficients, and were highest for participants who produced 'medium'-level alpha attenuation coefficients, suggesting that the Alpha Attenuation Test may not reliably reflect subjective levels of sleepiness in persons producing extremely high or low ratios of eyes-closed to eyes-open alpha power. However, this does not necessarily preclude the Alpha Attenuation Test as an accurate assessment of physiological sleepiness. Subjective sleepiness measures assess manifest sleepiness, which may change on a moment-to-moment basis depending on the presence or absence of alerting stimuli (Carskadon & Dement, 1982). It is not uncommon for subjective measures to be unrelated to objective measures (e.g., the Alpha Attenuation Test) which assess latent or physiological sleepiness (Thorpy, 1992; Mitler et al., 1982).

Thus, it may be concluded that the Alpha Attenuation Test is a quick and practical alternative to the Multiple Sleep Latency Test for the objective assessment of
physiological sleep tendency in sleep deprived normals and shiftworkers. The Alpha Attenuation Test eliminates the subjective aspect of sleep stage scoring and the confound of sleepability with sleepiness, and it is not as likely to artificially promote sleepiness. (Stampi et al., 1993; Stampi et al., 1994; Stampi et al., 1995; Heitmann et al., 1995).

Freedman (1986) utilized eyes-open versus eyes-closed conditions in his investigation of EEG power spectra in persons with sleep onset insomnia. Prior to bedtime, participants sat with their eyes open in an armchair for 15 minutes, and then laid down in bed with their eyes closed for an unspecified duration. Eyes-open and eyes-closed EEG power at 9 Hz (i.e., alpha activity) were significantly reduced in insomniacs compared to controls. The author made no attempt to interpret these findings. The mean eyes-closed alpha power was greater than eyes-open alpha power, but the significance level was not reported. Insomniacs also showed significantly greater beta power during the eyes-open (26-30 Hz) and eyes-closed (21 Hz) conditions. Moreover, eyes-closed delta power (1 Hz) was increased in the insomniacs versus controls. However, the Alpha Attenuation Test has yet to be validated in excessively sleepy individuals such as narcoleptics.

One of the aims of the present study was to investigate the utility of the Alpha Attenuation Test, a measure based on EEG power spectral analysis, as an alternative to visually-scored latency to sleep onset in the objective assessment of daytime sleepiness in excessively sleepy narcoleptics and normal sleepers.

It is of interest to consider the effects of increasing sleepiness on EEG power within frequency bands other than alpha. Physiological sleepiness may be manipulated experimentally by increasing or decreasing the amount of wakefulness prior to sleep. The effects of sleep deprivation on EEG spectra within the sleep stages have been studied in normals, with the intent to investigate the homeostatic regulation of NREM sleep (e.g., Borbely, Baumann, Brandeis, Strauch & Lehmann, 1981; Akerstedt & Gillberg, 1986; Brunner, Dijk & Borbely, 1993). Borbely et al. (1981) compared EEG power and the
percentage of time spent in sleep stages during baseline sleep and recovery sleep following one night of sleep deprivation in normals. The amount of time spent in stages 3 and 4, but not 2 or REM, was increased relative to baseline during the recovery sleep. By contrast, EEG power in the delta and theta frequency bands was found to be higher during stages 2, 3, 4 and REM during the recovery sleep, whereas beta power was reduced during stage 2, and alpha power was reduced during REM sleep. During the baseline and recovery nights, there was a progressive reduction of delta and theta EEG power within stages 2, 3 and 4 of successive NREM/REM sleep cycles. Thus, it appears that experimentally increased physiological sleepiness may be better reflected by EEG power within the sleep stages than by the percentage of time spent in the stages.

Borbely et al. (1981) concluded that the prevalence of delta power in the EEG of nocturnal sleep following sleep deprivation was indicative of the intensity of the sleep process. They proposed that delta power was related to an endogenous sleep enhancing factor which accumulates in the brain during the usual waking period, particularly during extended sleep deprivation, and is eliminated or inactivated during sleep. What is of relevance to the present study is whether a clinical population of excessively sleepy individuals (such as narcoleptics) would exhibit EEG power spectra similar to those observed in the recovery sleep of sleep deprived normals. In order to investigate this hypothesis in narcoleptics, researchers must quantify sleep EEG by spectral analysis, rather than limiting their analyses to visually scored sleep stages. Few studies have done this.

Bisset, Tafti, Nobile and Billiard (1994) investigated EEG power in the delta frequency range during sleep in narcoleptics and controls under baseline conditions, and conditions designed to increase prior wakefulness via sleep deprivation. Their purpose was to investigate the homeostatic regulation of sleep in narcoleptics. Nonetheless, it is noteworthy that following both 16 hours and 24 hours of sleep deprivation, delta power during subsequent sleep was significantly higher in narcoleptics compared to normals.
controls, and both groups exhibited a decrease in delta power throughout the sleep episode. The authors conclude that the homeostatic function of NREM sleep (i.e., the progressive reduction of delta power for successive NREM/REM cycles), is maintained in narcolepsy. However, for the purpose of the present study, it may be concluded that the increased physiological sleepiness inherent to narcolepsy was reflected in the enhanced delta power which was present during the sleep of narcoleptics compared to normals.

Rather than focusing on EEG power spectra during nocturnal sleep stages, the present study aimed to investigate changes in EEG spectra throughout the transition from wakefulness to sleep onset in normals sleepers and excessively sleepy individuals with narcolepsy.

3. **ASSESSING THE SLEEP ONSET PERIOD**

There is much debate about the precise point at which sleepiness ends, and sleep begins. The physiological onset of sleep has traditionally been characterized by decreases in fast, low voltage EEG beta activity, decreases in the regularity and frequency of EEG alpha activity, and increases in slow delta and theta EEG activities (Pivik, 1991). However, behavioral changes occurring throughout the transition into sleep are gradual, desynchronized, and vary widely across individuals (Rechtschaffen, 1994; Ogilvie, 1993). Responsivity to auditory stimuli decreases gradually (rather than abruptly) throughout the transition to sleep, responsiveness to visual stimuli diminishes faster than responsiveness to auditory stimuli, and the onset of these behavioral changes fluctuates widely between individuals (Rechtschaffen, 1994; Ogilvie, Wilkinson & Allison, 1989). Thus, it is unlikely that an exact or precise point of behavioral or physiological sleep onset can be identified. Indeed, Kleitman (1963) concluded that it is impossible to identify either electrophysiologically or behaviorally, whether a person is "fully asleep at a particular moment". Moreover, he proposed that "definite sleep is not a level value" to be descended to at the "moment" of sleep onset (Kleitman, 1963, p. 80). Rather, the
transition from wakefulness to sleep "involves a succession [i.e., a 'wavelike alternation']
of intermediate states, part wakefulness and part sleep in varying proportions" (Kleitman,
1963, p. 71). Stage 1 sleep [defined by Rechtschaffen & Kales (1968) as the decline in the
amount of EEG alpha activity to less than 50 % of a 30-second epoch] is now commonly
viewed as the transition period between wakefulness and sleep, because individuals are
still responsive to behavioral stimuli (Ogilvie, 1993). It is not until the onset of stage 2
sleep (with spindles and K complexes) that individuals become much less responsive to
stimuli.

Spectral changes occurring in the EEG during the sleep onset period have been
investigated in normal sleepers by Ogilvie and colleagues (Ogilvie et al., 1989; Ogilvie,
Participants were instructed to attempt to fall asleep while responding to a faint auditory
tone. FFT power data were collected on the 5-second period prior to the tone onset, and
were sorted into one of five bins on the basis of whether and how quickly the participant
responded to the tone. Comparing the longest response time with the shortest response
time, theta power increased, and alpha and beta power decreased. Significant increases in
power were found across all frequency bands (delta, theta, alpha, sigma and beta) at the
transition into sleep (defined once the participant failed to respond to the tone). It was
concluded that computer-based FFT analyses of 5-second EEG epochs detect transient
fluctuations in arousal during the transition to sleep that remain unseen in visual scoring of
30-second EEG epochs. Ogilvie et al. (1989, 1991) identified the sleep onset period as
consisting of "the lengthening of response times and intermittent response failure", and
defined sleep onset as "behavioral response cessation coupled with sharp increases in EEG
synchronization".

Badia, Wright and Wauquier (1994) investigated spectral fluctuations of EEG
during the transition from wakefulness to sleep in normal sleepers. Participants who
demonstrated a 'smooth transition' into sleep, having at least three continuous 30-seconds
of wakefulness followed by three continuous 30-second epochs of stage 1 sleep were
selected for analysis. Spectral EEG changes across sleep onset period (i.e., the three
epochs of wakefulness and the three epochs of stage 1) were investigated for the
broadbands 3-7 Hz (delta-theta), 8-12 Hz (alpha), and 13-25 Hz (sigma-beta) using 30-
second epochs, and for the single-hertz EEG changes (i.e., from 3-4 Hz to 24-25 Hz)
using both 30- and 5-second epochs. Power within the 8-12 Hz broadband decreased,
whereas power within the 3-7 Hz broadband increased during the last epoch of
wakefulness prior to stage 1. No changes were found in the 13-25 Hz broadband across
the sleep onset period. Single-hertz analyses of spectral changes occurring within the 30-
and 5-second epochs across the transition into sleep revealed significant increases in the 3-
4 Hz delta band and the 4-5 Hz theta band, and decreases in the 10-11 Hz alpha band.
Other studies of spectral changes in the EEG during sleep onset in normal sleepers have
shown decreases in beta activity (Merica, Fortune & Gaillard, 1991).

Hori (1985) investigated the oscillatory nature or unsteadiness of EEG activity
during the transition from wakefulness to sleep in normal young adults. Mean power and
the coefficient of variation (i.e., standard deviation / mean) were determined for 1 minute
segments from 10 minutes before to 30 minutes after the onset of visually scored stage 1
sleep. Mean delta and theta power increased rapidly after the onset of stage 1. The
coefficient of variation for delta, theta and alpha frequency bands increased significantly
just before or immediately after the onset of stage 1, and continued to increase
significantly for about 10 minutes (Hori, 1985). Thus, an unsteadiness or oscillation of
EEG activity was shown to be characteristic of the sleep onset period, as indicated by the
increased coefficients of variation (Hori, 1985).

A search of the literature revealed one abstract that compared EEG power spectra
during sleep onset REM periods with those occurring during nocturnal periods of REM
sleep in three narcoleptics (Kamei, Ishizuka, Usui, Hukuzawa & Kariya, 1991). Sleep
onset REM periods and REM sleep did not differ in the amount of delta, theta, alpha or beta power.

A systematic quantitative investigation of the EEG of normal sleepers and narcoleptics during the transition from wakefulness to the onset of NREM sleep and REM sleep has yet to be carried out. This was an aim of the present study.

4. PRESENT STUDY: PURPOSE, AIMS, HYPOTHESES AND PREDICTIONS

It has been demonstrated that narcolepsy is a disorder of excessive daytime sleepiness, characterized by extremely fast latencies to sleep onset on the Multiple Sleep Latency Test. However, it has been noted that although the Multiple Sleep Latency Test is the traditional measure used in the diagnosis of narcolepsy, its validity is lessened because of a number of limitations. Moreover, there is some evidence for qualitatively different states of sleepiness, such that sleepiness prior to REM onsets may be greater than that prior to NREM onsets, resulting in shorter latencies to stage 1 sleep (Broughton & Aguirre, 1987). However, questionable statistics (erroneous use of t-tests) limit the interpretability of these findings. The technique of computer-based EEG power spectral analysis has been introduced as an alternative to the traditional visual scoring of sleep latency in the assessment of physiological sleep tendency. Studies of sleep deprived normals, shiftworkers and insomniacs have shown that sleepiness may be quantified by assessing spectral changes in eyes-closed versus eyes-open EEG alpha power during seated wakefulness (Stampi et al., 1993), and by comparing EEG power within the delta, theta, alpha and beta frequency bands during nocturnal stages of sleep (Borbely et al., 1981). Furthermore, the technique of EEG power spectral analysis has enabled the examination of the microstructure of the process of normal sleep onset (Ogilvie et al., 1991; Badia et al., 1994; Hori, 1985).

The present study is unique in its purpose to objectively quantify sleepiness and the sleep onset process in normal sleepers and narcoleptics using computer-based EEG power
spectral analysis in addition to the traditional measure of visually scored sleep stages.

Specific aims of the present study were as follows:

(I) To further examine the electrophysiological nature of the normal sleep onset process by quantifying EEG via spectral analysis.

(II) To distinguish the narcoleptic sleep onset process from the normal sleep onset process by quantifying EEG via spectral analysis.

(III) To attempt to resolve the issue of qualitatively different states of sleepiness using EEG power spectral analysis of the sleep onset period in addition to visually-scored latency to sleep onset.

(IV) To investigate the utility of the Alpha Attenuation Test, a measure based on EEG power spectral analysis, as an alternative to the Multiple Sleep Latency Test in the objective assessment of the excessive daytime sleepiness associated with narcolepsy.

The first three aims were pursued using the Multiple Sleep Latency Test paradigm to elicit multiple daytime nap opportunities under conditions which were optimal for the onset of sleep. Computer-based EEG power spectral analysis was performed according to two strategies:

Stage-based strategy: EEG power spectral analysis was performed on the visually-scored stages of wakefulness and stage 1 sleep elicited by the Multiple Sleep Latency Test. In this way, EEG power spectra during the waking intent to fall asleep were compared with those of stage 1 sleep. However, this type of analysis requires that EEG epochs be sorted by sleep/wake stage, regardless of whether they occurred early or late in a particular nap opportunity. Any chronological changes which might exist during the transition into sleep are thereby ignored.

Time-based strategy: Thus, because individuals may fluctuate between wakefulness and stage 1 throughout the entry into sleep, EEG power spectral analysis was performed on successive 5-second EEG epochs throughout the sleep onset period. Then,
the sleep onset period was divided into quartiles based on the length of the sleep onset period, and mean power spectra within each quartile were calculated. The oscillatory nature of the process of sleep onset was also investigated by examining fluctuations in arousal level within each quartile. Changes in the direction or 'slope' of EEG power values within each frequency band were examined. Thus, the chronology of the process of sleep entry was investigated by comparing EEG power spectra and the proportion of reversals in power values (or 'slope changes') across each quartile of the sleep onset period.

The normal process of entry into sleep (*Aim I*) was investigated by comparing normal control naps containing NREM stage 2 with naps containing just stage 1 sleep according to the stage-based and time-based strategies outlined above. The narcoleptic sleep onset process was compared with the normal sleep onset process (*Aim II*) by first investigating differences in the general process of sleep entry in normals and narcoleptics, making no attempt to differentiate the ensuing type of sleep (i.e., NREM or REM). To investigate the presence of qualitatively different states of sleepiness (*Aim III*), the sleep onset process was compared between narcoleptic naps containing REM sleep and narcoleptic naps containing NREM sleep. Power spectral differences in the process of sleep onset were then investigated by comparing normal control naps containing NREM (stage 2) sleep with narcoleptic naps containing REM sleep and narcoleptic naps containing just NREM (stage 2) sleep. Because the EEG of REM sleep and stage 1 are nearly electrophysiologically identical (Rechtschaffen & Kales, 1968), it was of interest to compare the sleep onset process for normal control naps containing just stage 1 and narcoleptic naps containing REM sleep. For completeness, the sleep onset process was also compared between the normal control naps containing just stage 1 and the narcoleptic naps containing NREM sleep.

The fourth aim was pursued by using the Alpha Attenuation Test to obtain multiple assessments of spectral changes in EEG alpha power as a function of variations in eyelid position during seated wakefulness.
Hypotheses

Given that sleepiness reflects sleep tendency, it has been operationalized as the latency to intentional sleep onset (Carskadon & Dement, 1978). Research using the Multiple Sleep Latency Test (e.g., Richardson et al., 1978; Mitler et al., 1979) has substantiated the narcoleptic's complaint of excessive daytime sleepiness: Narcoleptics fall asleep faster than normals do when placed in an environment conducive to sleep onset. Moreover, the latency to sleep onset tends to be shorter prior to narcoleptic sleep onset REM periods than narcoleptic NREM onsets, suggesting that sleepiness may differ qualitatively in narcoleptics, reflecting either selective pressure for REM sleep or pressure for NREM sleep (Broughton & Aguirre, 1987).

On the assumption that sleepiness can vary in quantity and quality, it was hypothesized that physiological sleep tendency is greater in narcoleptics than normals, and that within narcoleptics, the pressure for REM sleep is qualitatively different than the pressure for NREM sleep. Moreover, it was further hypothesized that these quantitative and qualitative differences in narcoleptic and normal sleepiness and sleep onset processes may be assessed using computerized analyses of EEG power spectra in addition to the traditional visual sleep stage scoring techniques.

More specifically, the following predictions were made:

Sleep latency predictions

On the basis of previous studies of narcoleptics and normals (e.g., Richardson et al., 1978; Mitler et al., 1979), it was predicted that narcoleptics would have shorter latencies to sleep onset than normals on the Multiple Sleep Latency Test. Furthermore, on the basis of Broughton and Aguirre's (1987) proposal of qualitatively different states of sleepiness, it was predicted that selective pressures for REM sleep and NREM sleep would be evident in the narcoleptic nap opportunities elicited by the Multiple Sleep Latency Test. It was expected that within the narcoleptics, latency to sleep onset would
be shorter prior to naps containing REM sleep compared to naps containing just NREM sleep.

**EEG power spectra predictions**

Although the present study is the first to investigate spectral differences across the sleep onset period of narcoleptics and normals, studies of recovery sleep in sleep deprived normals (e.g., Borbely et al., 1981) and sleep deprived narcoleptics (e.g., Besset et al., 1994) have demonstrated the sensitivity of EEG power spectra to experimentally-induced increases in sleepiness. Given that sleepiness may be quantified by the tendency to enter a sleep-like state, it was expected that EEG power spectra during wakefulness and stage 1 sleep, and across the quartiles of the sleep onset process would differ between excessively sleepy narcoleptics and normal sleepers, and would uniquely document the selective pressures for REM and NREM sleep proposed by Broughton (1982).

It was expected that EEG power spectra obtained from the stage-based and time-based analyses of the sleep onset period would differ among normal naps containing NREM stage 2 sleep, normal naps containing only stage 1 sleep, narcoleptic NREM stage 2 naps and narcoleptic REM naps. It was predicted that compared to normal controls, narcoleptics would exhibit greater delta and theta EEG power and less alpha, sigma and beta EEG power during wakefulness and stage 1, and across the quartiles of the sleep onset period. More specifically, it was predicted that compared to normal naps containing only stage 1 sleep, EEG power during wakefulness and stage 1, and the quartiles of the sleep onset period for normal naps containing NREM stage 2 would be greater within the delta, theta and sigma frequencies, and lower within the alpha and beta frequencies. It was also predicted that compared to narcoleptic naps containing NREM sleep, EEG power during wakefulness and stage 1, and the quartiles of the sleep onset period for narcoleptic naps containing REM sleep would be greater within the delta and theta frequencies, and lower within the alpha, sigma and beta frequencies.
The present study uniquely investigated the smoothness of the sleep entry process in normals and narcoleptics by comparing the proportion of changes in the 'slope' or direction of the power value within each frequency band across the quartiles of the sleep onset period. On the basis of studies demonstrating a rapid entry into sleep in narcoleptics (e.g., Richardson et al., 1978), it was expected that compared to normal sleepers, EEG power spectra in narcoleptics would demonstrate a smoother transition to sleep onset, having fewer changes in direction. It was predicted that the proportion of slope changes in the delta, theta, alpha, sigma and beta frequencies across the quartiles of the sleep onset period would be smaller for narcoleptics compared to normals. More specifically, it was predicted that compared to normal naps containing only stage 1 sleep, the proportion of slope changes across the quartiles of the sleep onset period for normal naps containing NREM stage 2 would be smaller within the delta, theta alpha, sigma and beta frequencies. It was also predicted that compared to narcoleptic naps containing NREM sleep, the proportion of slope changes across the quartiles of the sleep onset period for narcoleptic naps containing REM sleep would be smaller within the delta, theta alpha, sigma and beta frequencies.

**Alpha Attenuation Test predictions**

The present study is the first to investigate the utility of the Alpha Attenuation Test in distinguishing narcoleptics from normals. However, on the basis of EEG power spectra studies of sleep deprived normals and shiftworkers (e.g., Michimori et al., 1993; Heitmann et al., 1995; Akerstedt et al., 1985; Torsvall & Akerstedt, 1983; 1987), it was predicted that the ratio of eyes-closed to eyes-open alpha power (i.e., the alpha attenuation coefficient) would be lower in narcoleptics than normal controls, and that compared to controls, narcoleptics would demonstrate greater eyes-open alpha power and less eyes-closed alpha power. It was also predicted that the alpha attenuation coefficient would correlate with estimates of subjective sleepiness.
Method

PARTICIPANTS

Five female and 5 male patients diagnosed with narcolepsy-cataplexy, and 10 normal sleepers matched for age and gender were studied. Narcoleptics ranged in age from 29 to 62 years \(M = 44.3, \ SD = 11.9 \text{ years}\), and normal controls ranged from 28 to 56 years \(M = 42.7, \ SD = 10.6 \text{ years}\). All participants gave their written informed consent (see Appendix A-1), and received an honorarium for their participation in the study.

Narcoleptic participants were recruited by referral from Dr. Colin Shapiro of the Department of Psychiatry, The Toronto Hospital (Western Division). Normal controls were recruited from posters placed at The Toronto Hospital (Western Division), and by word-of-mouth. A screening process (see Appendix B-1 for questionnaire) ensured that narcoleptics met the American Sleep Disorders Association's diagnosis criteria for narcolepsy, and that normal controls did indeed report normal sleep habits.

On the screening questionnaire (see Appendix B-1), all narcoleptics complained of daytime sleepiness, and reported taking naps daily or several times weekly. Normals reported that they were generally alert during the day. Four normals refrained from daytime napping, five reported taking naps twice a week or less, and one reported napping daily or almost daily. All narcoleptics had a history of cataplexy, six reported experiencing sleep paralysis and hypnagogic hallucinations at least once a month, and two reported incidents of hypnagogic hallucinations that occurred 1-5 times during their lifetime. No normals reported a history of cataplexy, although two normals reported experiencing incidents of sleep paralysis and four reported incidents of hypnagogic hallucinations 1-5 times during their lifetime. All narcoleptics reported nocturnal awakenings. Five narcoleptics reported awakening 1-3 times, 3 reported awakening 4-6 times, and 2 reported awakening more than 6 times during the night. The normal controls were self-reported normal sleepers, having normal nocturnal sleep (6-8 h of sleep per night) and no
sleep-related complaints. Four normals reported no nocturnal awakenings, and six reported awakening 1-3 times during the night. Participants were also given a Sleep Diary (see Appendix B-2), in which they recorded their sleep and wake patterns for the 7 days preceding testing. The self-reported mean duration of nocturnal sleep ranged from 4.7 to 10.8 h for the narcoleptics (M = 7.9 h, SD = 2.1 h), and from 6.2 to 8.6 h for the normal controls (M = 7.4 h, SD = 0.9 h). Narcoleptics and normals did not differ with respect to the self-reported mean duration of nocturnal sleep.

Morningness-eveningness was assessed with the Circadian Rhythm Questionnaire (Horne & Ostberg, 1967; see Appendix B-3), which places individuals along a morningness-eveningness scale, on the basis of whether they report being more alert and active in the morning or evening. The scores obtained on the Circadian Rhythm Questionnaire for the narcoleptics ranged from 36.0 to 68.0 (M = 52.7, SD = 10.4), and for the normals ranged from 50.0 to 80.0 (M = 60.1, SD = 8.4). Narcoleptics and normals did not differ in their degree of morningness-eveningness [t(18) = 1.74, p = .098]. Six narcoleptics and four normals were classified as neither morning nor evening, two narcoleptics and five normals were classified as moderate morning types, two narcoleptics were classified as moderate evening types, and one normal was classified as a definite morning person.

Symptoms related to depression (which may elicit sleep onset REM periods) were assessed with the Beck Depression Inventory (Beck, 1967; see Appendix B-4), which measures the intensity of depression with 21 statements reflecting "symptom-attitude categories" of depression (Shaver & Brennan, 1991). Participants rank each statement to reflect the degree to which it is experienced (from neutral [0] to maximal severity [3]). Thus, scores can range from 0 to 63 (Shaver & Brennan, 1991). The scores obtained on the Beck Depression Inventory for the narcoleptics ranged from 1.0 to 25.0 (M = 8.6, SD = 7.47), and for the normals ranged from 1.0 to 16.0 (M = 4.5, SD = 5.0). Narcoleptics and normals did not differ with respect to the number of depression-related symptoms they
reported \( t(18) = 1.44, p = .166 \). However, it should be noted that the mean of 8.6 for the narcoleptics is classified as reflecting mild depression, whereas the mean of 4.5 for the normals is classified as reflecting no depression or minimal depression (Shaver & Brennan, 1991).

Prior (but not during) testing, nine of the ten narcoleptics were taking medications for their symptoms. Six narcoleptics were taking central nervous system stimulants (five took Ritalin, one took Dexedrine) for their excessive daytime sleepiness, three were taking sleeping pills (Immovane) to improve their nocturnal sleep, and two were taking REM sleep suppressants (Clomipramine) to reduce their cataplexy. Two of the narcoleptics taking Ritalin were withdrawn for two days prior to testing, and the seven remaining medicated narcoleptics were withdrawn from their medications for 7 days prior to testing. All participants agreed to refrain from alcohol, caffeine, and sleep-related medications during the 24 hours preceding testing.

**TESTING PROCEDURE AND APPARATUS**

Procedure

Participants reported to the Sleep Research Unit at 2130 hr for orientation and electrode application. Following administration of the Presleep Questionnaire* (see Appendix C-1), participants retired for the night at 2300 hr, and slept undisturbed while polysomnographically recorded until 0800 hr. Upon awakening, participants were administered the Postsleep Questionnaire* (see Appendix C-2). The Alpha Attenuation Test (Stampi et al., 1993) was administered at 0900, 1100, 1300, 1500 and 1700 hrs. The Multiple Sleep Latency Test (Carskadon & Dement, 1978) was administered at 1000, 1200, 1400, 1600 and 1800 hrs. The Stanford Sleepiness Scale (Hoddes et al., 1972; see

*NOTE: The Presleep Questionnaire and Postsleep Questionnaire were administered as part of the standard nocturnal polysomnography protocol at the Sleep Research Unit, Toronto Hospital (Western Division). However, for the purposes of the present study, neither these questionnaires were statistically investigated
Appendix D-1), and the Visual Analogue Sleepiness Scale (Folstein & Luria, 1973; see Appendix D-1) were administered every half hour, including immediately prior to and following each Multiple Sleep Latency Test and Alpha Attenuation Test session. Oral temperature* was also recorded each time subjective sleepiness was assessed. Participants were not allowed to ingest coffee, tea, caffeinated pop, or alcohol during the study. Breakfast, lunch and snacks were provided by the Sleep Research Unit, or by the subject.

**Polysomnographic Recordings**

Electrodes were positioned according to the standard placements suggested by Rechtschaffen and Kales (1968), thereby enabling the monitoring of EEG, electromyogram (EMG) and electrooculogram (EOG) activity. EEG sites at Cz (central) and O2 (right occipital) were measured according to the International 10-20 system (Jasper, 1968). The EEG electrodes were affixed to the scalp using collodion and gauze. Electrodes were taped to the left and right outer canthi for the recording of horizontal eye movements (EOG), and two electrodes were taped under the chin for recording of submental muscle activity (EMG). Two electrodes were taped on the left and right mastoid behind each ear. EEG and EOG electrodes were referenced to the left mastoid electrode. The right mastoid electrode served as a ground.

A 16-channel Nihon Kohden Neurofax polygraph was used to amplify the polysomnographic recordings. Time constants were set at 0.3 for EEG and EOG recordings, and at 0.03 for EMG recordings. High frequency filters were set at 30 Hz for EEG and EOG, and at 70 Hz for EMG. Recording sensitivity was 7 μV/mm for EEG and EOG, and 1-3 μV/mm for EMG. Paper speed was set at 10 mm/second.

Polysomnographic data from the polygraph were acquired on paper and on computer using the software program Microcomputer Quantitative Electrophysiology (MQE) (Imaging Research Inc., under continued development at Brock University). Digitized

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* Note: For the purposes of the present study, oral temperature measures were not statistically investigated.
recordings of nocturnal sleep, nap opportunities on the Multiple Sleep Latency Test and the eyes-open and eyes-closed conditions of the Alpha Attenuation Test were saved on optical disk. Nocturnal sleep and nap opportunities on the Multiple Sleep Latency Test were scored on paper using 30-sec epochs, according to the standard criteria outlined by Rechtschaffen and Kales (1968).

**Multiple Sleep Latency Test**

The Multiple Sleep Latency Test (Carskadon & Dement, 1978) was administered according to the guidelines for clinical use outlined in the Report From the American Sleep Disorders Association (Thorpy, 1992). Following the nocturnal polysomnography, nap opportunities were given at 1000, 1200, 1400, 1600 and 1800 hrs. At the start of each session of the Multiple Sleep Latency Test, participants were instructed to try to fall asleep while lying quietly with their eyes closed in a darkened room. During the Multiple Sleep Latency Test session, EEG, EOG and EMG were recorded on paper and computer according to the parameters outlined above, and were scored in 30 second epochs using the criteria of Rechtschaffen and Kales (1968). Sleep onset was defined as the first 3 consecutive minutes of Stage 1, 2 or REM sleep. Each Multiple Sleep Latency Test session was terminated either 15 minutes following the initial onset of sleep, or after 20 minutes in bed with no sleep.

**Alpha Attenuation Test**

The 8-minute version of the Alpha Attenuation Test (Stampi et al., 1993) was administered at 0900, 1100, 1300, 1500 and 1700 hrs. Participants were polysomnographically recorded while seated in an illuminated room within three feet of a wall upon which an 'X' made of black tape had been placed at eye-level. Participants were instructed to sit quietly with their eyes open and focus on the black tape on the wall. Following one minute of artifact-free recording with eyes open, participants were instructed to sit quietly with their eyes closed for one minute. Each eyes-open and eyes-closed session was repeated three more times so that a total of eight one-minute
samples of artifact-free EEG were obtained. Power spectral analyses of eyes-open and eyes-closed EEG at 02 were calculated using Fast Fourier Transformations (FFT) on 10-second epochs within the alpha frequency band (8-12 Hz). The ratio of mean eyes-closed to mean eyes-open alpha power at (i.e., the alpha attenuation coefficient) was calculated. A two-factor 2x5 (group by session) ANOVA was performed on the alpha attenuation coefficient, and a three-factor 2x2x5 (group by eyelid position by Alpha Attenuation Test session) ANOVA was performed on mean alpha power.

Subjective Sleepiness Measures

Subjective sleepiness was assessed using the Stanford Sleepiness Scale (Hoddes et al., 1972; see Appendix D-1), and the Visual Analogue Sleepiness Scale (Folstein & Luria, 1973; see Appendix D-1). The Stanford Sleepiness Scale consists of seven statements describing progressive changes in subjective sleepiness, ranging from "feeling active and vital; alert; wide awake" (1), to "almost in reverie; sleep onset soon; lost struggle to remain awake" (7). Participants were asked to choose the statement which best represented their present state of sleepiness. The Visual Analogue Sleepiness Scale is a horizontal line approximately 100 mm in length, labeled "Very Alert" on the left, and "Very Sleepy" on the right. Participants were asked to draw a vertical mark on the line at the point corresponding to their present state of sleepiness. Scores were obtained by measuring the distance of the mark from the left end of the line; higher scores being associated with more intense feelings of sleepiness. The Stanford Sleepiness Scale and Visual Analogue Sleepiness Scale were administered prior and following each Multiple Sleep Latency Test and Alpha Attenuation Test session, and at half-hour intervals between Multiple Sleep Latency Test and Alpha Attenuation Test testing. Pre- and post- scores on the Stanford Sleepiness Scale and Visual Analogue Sleepiness Scale were correlated intraindividually with latency to stage 1 on the Multiple Sleep Latency Test and with alpha attenuation coefficients. A series of t-tests compared mean pre- and post- scores on the
Stanford Sleepiness Scale and Visual Analogue Sleepiness Scale between narcoleptics and normal controls.

**POWER SPECTRAL ANALYSIS OF THE SLEEP ONSET PERIOD**

The sleep onset period elicited by the Multiple Sleep Latency Test was quantified using power spectral analysis. Two strategies were used to evaluate the process of falling asleep: a stage-based investigation, and a time-based investigation. Because the type of sleep obtained on each nap varied within participants and groups, the first nap to occur containing just NREM stages 1 and 2, and the first nap to occur containing REM sleep were selected for analysis for each narcoleptic participant, and the first nap to occur containing NREM stages 1 and 2, and the first nap to occur containing just NREM stage 1 were selected for analysis for each normal control participant.

**Stage-based Strategy of Power Spectral Analysis of Sleep Onset Period**

The first set of analyses involved computerized power spectral analysis of EEG frequency bands during wakefulness, and stages 1, 2 and REM. Each Multiple Sleep Latency Test sleep onset period was analyzed by scoring 5-second epochs beginning at lights out and continuing until the first 2 minutes of stage 2 or REM sleep were reached, or until 20 minutes had passed from lights out. EEG data were sorted into one of four windows; wakefulness, stage 1, stage 2 or stage REM. Power spectral analyses of EEG were calculated within each window for delta (0.3-4 Hz), theta (4-8 Hz), sigma (12-15 Hz) and beta (15-30 Hz) frequencies at Cz and alpha (8-12 Hz) at O2.

**Time-based Strategy of Power Spectral Analysis of Sleep Onset Period**

The second set of computerized power spectral analyses investigated spectral changes during the sleep onset period, without sorting epochs into stages, thereby maintaining the chronology of the transition from wakefulness into sleep. FFT for delta (0.3-4 Hz), theta (4-8 Hz), sigma (12-15 Hz) and beta (15-30 Hz) frequencies at Cz and alpha (8-12 Hz) at O2 were calculated using 5-second epochs beginning at lights out and
continuing until the first 2 minutes of stage 2 or REM sleep were reached, or until 20
minutes had passed from lights out.

The power data for each frequency band obtained from each nap opportunity were
passed separately through the Arouse software program (R. D. Ogilvie & J. Ogilvie, under
continued development at Brock University). The length of the sleep onset period was
divided into quartiles, with equal number of power values within each quartile. The mean
power within each quartile was calculated. Using 50% of the overall standard deviation
of power as a 'normative window', the number of changes in slope direction greater than
this value was calculated within each quartile as a measure of jitter or random variation in
power levels. The proportion of 'slope changes' occurring within each quartile was
calculated as a function of the total number of power values within each quartile.

STATISTICAL ANALYSES OF POWER DATA FROM THE SLEEP ONSET PERIOD

To determine if there were any differences between narcoleptics and normals in
overall power at Cz or O2 during wakefulness or stage 1, four three-factor 2x5x5 (group
by frequency bin by Multiple Sleep Latency Test session) ANOVAs were performed.
Spectral differences between narcoleptics and normal controls were investigated during
wakefulness and stage 1 of the sleep onset period.

Spectral differences across wakefulness and stage 1, and across the quartiles of the
sleep onset period were compared for narcoleptic sleep onsets leading to REM sleep,
narcoleptic sleep onsets leading to NREM sleep (stage 2), normal control sleep onsets
leading to NREM sleep (stage 2), and normal control sleep onsets leading to just NREM
stage 1. Specifically, the following analyses were carried out: (a) The sleep onset process
in general (i.e., collapsing over naptype) was compared among narcoleptics and normals.
(b) Next, the normal sleep onset process was analyzed in greater detail, with comparisons
among normal naps containing NREM stage 2 and normal NREM naps containing just
stage 1. (c) Then, the narcoleptic sleep onset process was analyzed more specifically.
Narcoleptic REM naps were compared with narcoleptic NREM naps. Following this, the different types of narcoleptic and normal naps were compared in greater detail, with analyses made between (d) narcoleptic stage 2 naps and normal stage 2 naps, (e) narcoleptic stage 2 naps and normal stage 1 naps, (f) narcoleptic REM naps and normal stage 2 naps, and (g) narcoleptic REM naps and normal stage 1 naps.

To investigate EEG power spectra during wakefulness and stage 1, mixed two-factor 2x2 (group or naptype by stage) ANOVAs were performed. Main effects of group or naptype, main effects of stage, and group by stage interactions were examined for mean delta, theta, sigma and beta power at Cz and alpha power at O2. To investigate EEG power spectra and the proportion of slope changes across the quartiles of the sleep onset period, mixed two-factor 2x4 (group or naptype by quartile) ANOVAs were performed. Main effects of group or naptype, main effects of quartile, and group by quartile interactions were examined for mean power and mean proportion of slope changes within delta, theta, sigma and beta bands at Cz and alpha at O2. In the presence of a significant main effect of group and the absence of a significant interaction, t-tests were used to investigate hypotheses related to group differences at wakefulness and stage 1, and at each quartile of the sleep onset period. Alpha level was maintained at the $p = .05$ level.
Results

The results are presented in the following manner: In section 1, visually-scored latencies and sleep stage percentages for the nocturnal polysomnography carried out the night prior to testing are presented for narcoleptics and normals. In section 2, the visually-scored data from the Multiple Sleep Latency Test are presented. Following a summary of the distribution of the type of sleep obtained by narcoleptics and normals at each nap opportunity, intergroup comparisons of latency to sleep onset and sleep stage percentages are presented. In addition, latency to sleep onset for narcoleptic REM and NREM naps is investigated in order to document the presence of qualitatively different states of sleepiness.

In sections 3 and 4, findings from the EEG power spectral analyses of the sleep onset period of selected nap opportunities elicited by the Multiple Sleep Latency Test are presented. Section 3 consists of findings from the stage-based (wakefulness and stage 1) strategy, whereas the findings from time-based (chronological quartiles) strategy are dealt with in section 4. The format of presentation in both of these sections is as follows: (a) The sleep onset process in general (i.e., with no specification of the ensuing type of sleep) is compared among narcoleptics and normal sleepers. (b) Next, the normal sleep onset process is investigated in greater detail, with comparisons being made among normal naps containing stage 2 sleep, and normal naps containing only stage 1 sleep. (c) Then, the narcoleptic sleep onset process is investigated in greater detail, with comparisons being made between narcoleptic naps containing stage 2 sleep, and narcoleptic naps containing REM sleep. Following this, the different types of narcoleptic and normal naps are investigated in greater detail, with comparisons being made among (d) narcoleptic stage 2 naps and normal stage 2 naps, (e) narcoleptic stage 2 naps and normal stage 1 naps, (f) narcoleptic REM naps and normal stage 2 naps, and (g) narcoleptic REM naps and normal stage 1 naps.
In section 5, the findings from the Alpha Attenuation Test are presented. Data from the eyes-closed and eyes-open testing conditions, along with the alpha attenuation coefficient and its correlation with latency to stage 1 on the Multiple Sleep Latency Test are presented. In section 6, findings detailing the relationship between the subjective sleepiness scales and the Alpha Attenuation Test and the Multiple Sleep Latency Test are presented.

1. **NOCTURNAL SLEEP**

Table 1 shows mean latencies to sleep onset, sleep stage percentages, total sleep time, and sleep efficiency for the nocturnal sleep of narcoleptics and normals. Narcoleptics had a shorter mean latency to stage 1 (7.2 min) compared to normals (17.2 min) \( t(18) = 2.29, p = .034 \), but latency to stage 2 sleep did not differ between narcoleptics (24.6 min) and normals (23.4 min). Seven of ten narcoleptics, but none of the normals had a sleep onset REM period during their nocturnal sleep onset. Mean latency to REM sleep from sleep onset (stage 1) was 26.4 min for narcoleptics, and 86.1 min for normals \( t(18) = 4.24, p = .001 \). Narcoleptics spent a greater percentage of their sleep time in stage 1 (20.2%) compared to normals (11.2%) \( t(18) = 2.37, p = .029 \). However, percentage of time spent in stage 2, stage 3, stage 4, and REM sleep did not differ between the groups. Total sleep time and sleep efficiency also did not differ between groups.

2. **VISUALLY-SCORED DATA FROM THE MULTIPLE SLEEP LATENCY TEST**

Distribution of naps

Table 2 presents the distribution of NREM and REM naps occurring at each Multiple Sleep Latency Test session for narcoleptics and normals.

* Narcoleptics: Narcoleptics experienced a total of 26 REM-containing naps and 24 NREM-only naps. Eleven of the REM naps contained stages 1, 2 and REM, and fifteen
contained just stage 1 and REM. Twenty-three of the 24 NREM naps contained both stage 1 and stage 2 sleep. A total of six REM naps occurred in the 1200 h and 1400 h naps, five occurred in the 1000 h and 1600 h naps, and four occurred in the 1800 h nap. Two narcoleptics had REM sleep on all five naps, five other narcoleptics had two or more REM containing naps, and three had only one nap containing REM sleep. Of these three narcoleptics, one had a sleep onset REM period during the nocturnal sleep.

**Normals**: Normal controls experienced a total of 44 NREM naps, and 6 naps with no sleep onset. Thirty of the 44 NREM naps contained stage 1 and stage 2 sleep, whereas 14 contained only stage 1 sleep. One of the normal control participants (RK) had two naps which contained REM sleep, one at 1400 h and the other at 1600 h.

**Multiple Sleep Latency Test intergroup comparisons**

Mixed two-factor 2x5 (group by Multiple Sleep Latency Test session) ANOVAs were performed to investigate main effects of group and time of day on the latency to stage 1 sleep, the percentage of time spent in sleep stages, and the total sleep time and wake time after sleep onset*. Significant group effects were found for stage 1 latency, total sleep time and total wake time after sleep onset.

As predicted, mean latency to stage 1 sleep was significantly shorter in the narcoleptics (3.5 min) compared to the normal sleepers (10.4 min) [F(1,18) = 15.11, p = .001] (see Table 3 and Figure 1). Narcoleptics spent significantly more time asleep (15.2 min) than the normals (12.2 min) [F(1,18) = 6.53, p = .02], and significantly less time awake after sleep onset (0.2 min) compared to normals (1.5 min) [F(1,18) = 14.53, p = .002] (see Table 3 and Figure 2). However, these main effects were mediated by an interaction with time of day [F(4,72) = 2.96, p = .025 for total sleep time, and F(4,72) = 4.24, p = .005 for total wake time after sleep onset]. While narcoleptics did not vary

*NOTE: Latency to stage 2 and REM sleep percentage were not investigated due to the fact that 8 narcoleptics and 6 normals had at least one nap opportunity that contained no stage 2 sleep, and 9 of the 10 normals had just NREM sleep.*
across Multiple Sleep Latency Test sessions in the amount of sleep time or wake time after
sleep onset, normals slept longer and had less wake time after sleep onset at the 1400 h
nap (see Figure 2). No main effects of time of day were found.

**REM versus NREM sleepiness in narcoleptics**

The presence of qualitatively different states of sleepiness in the narcoleptics prior
to naps containing REM sleep and naps containing just NREM sleep was investigated.
For each narcoleptic, the mean latency to stage 1 sleep prior to REM naps, and prior to
NREM naps was calculated. Contrary to predictions, a paired t-test showed no significant
difference between mean latency to sleep onset for REM naps (3.2 min) and NREM naps
(4.3 min).

3. **STAGE-BASED POWER SPECTRAL ANALYSES OF THE SLEEP ONSET PERIOD**

In this section, the process of transition from wakefulness to sleep onset is
quantified in normals and narcoleptics using the stage-based FFT strategy; EEG power
spectral analysis was performed on visually-scored wakefulness and stage 1 sleep obtained
from selected nap opportunities on the Multiple Sleep Latency Test.

To determine whether normals and narcoleptics differed in mean EEG power at Cz
or O2 during wakefulness or stage 1, four three-factor 2x5x5 (group by frequency band by
Multiple Sleep Latency Test session) ANOVAs were performed. Main effects of group
were investigated. Collapsing across frequency bands and Multiple Sleep Latency Test
session, mean EEG power at Cz and O2 during wakefulness did not differ between
narcoleptics and normals. Nor did mean EEG power at Cz during stage 1 differ between
narcoleptics and normals. However, mean EEG power at O2 during stage 1 was
significantly higher in narcoleptics (9.1 μV²) compared to normals (7.6 μV²) [F(1,18) =
5.4, p = .032].

The process of falling asleep during the selected nap opportunities on the Multiple
Sleep Latency Test was investigated in normals and narcoleptics by analyzing the spectral
changes occurring in the EEG during visually-scored wakefulness and stage 1 of the sleep onset period. Dependent measures included mean power values for delta, theta, sigma and beta frequency bands at Cz and for alpha at O2. Table 2 displays the distribution of REM, NREM (stage 1) and NREM (stages 1 and 2) naps selected for analysis in narcoleptics and normals.

(a) Normal versus narcoleptic sleep onset process

The normal sleep onset process was compared with the narcoleptic sleep onset process by first comparing EEG power spectra during sleep onset, without specifying the type of sleep to ensue. Focusing on the sleep onset process in general, irrespective of how the naps were classified, mixed two-factor 2x2 (group by stage) ANOVAs were conducted on power spectra during wakefulness and stage 1 in narcoleptics and normal controls. The results are presented in Table 4 and Figures 3-5. Significant main effects of group were found for delta and sigma power, and a trend was found for mean theta power. There were no significant interactions between group and stage for any of the frequency bands.

Collapsing across stage, mean delta power was significantly higher in narcoleptics (5.6 \(\mu V^2\)) than normals (4.1 \(\mu V^2\)) \([F(1,32) = 11.24, p = .002]\) (see Figure 3). Specifically, as predicted, mean delta power was significantly higher in narcoleptics than normals during wakefulness \([t(32) = 2.83, p = .008]\) and stage 1 \([t(32) = 3.03, p = .005]\).

Collapsing across stage, a trend was observed for mean theta power to be higher in narcoleptics (3.0 \(\mu V^2\)) than normals (2.3 \(\mu V^2\)) \([F(1,32) = 3.79, p = .060]\) (see Figure 4). Collapsing across stage, (as predicted) mean sigma power significantly lower in narcoleptics (0.5 \(\mu V^2\)) compared to normals (0.7 \(\mu V^2\)) \([F(1,32) = 4.88, p = .035]\) (see Figure 5), although during wakefulness and stage 1, this effect did not quite reach significance \([t(32) = 1.91, p = .065; t(32) = 1.90, p = .066\) respectively].
Significant main effects of stage* were found for mean delta, alpha, sigma and beta power. Collapsing across nap types, mean delta power increased from wakefulness to stage 1, and mean alpha, sigma and beta power decreased from wakefulness to stage 1. Theta power did not differ between wakefulness and stage 1.

(b) Normal NREM (stage 2) naps versus normal NREM (stage 1) naps

To further investigate the microstructure of the normal transition from wakefulness to sleep, power spectra during wakefulness and stage 1 were compared between normal control naps containing stage 2 and normal control naps containing just stage 1 sleep using mixed two-factor 2x2 (group by stage) ANOVAs. The results are presented in Table 5. A significant main effect of nap type was found for delta power. Collapsing across stage, (as predicted) mean delta power was higher for normal naps containing stage 2 sleep (4.4 μV²) compared to normal naps containing no stage 2 sleep (3.6 μV²) [F(1,14) = 4.94, p = .043] (see Figure 6). No significant nap type by stage interactions were found; however there was a trend for nap type to interact with stage for delta power [F(1,14) = 4.53, p = .052]. The increase in delta power from wakefulness to stage 1 was greater for normal naps containing stage 2 than for normal control naps containing just stage 1.

(c) Narcoleptic REM naps versus narcoleptic NREM (stage 2) naps

To investigate differences in the microstructure of the transition from wakefulness to REM sleep and the transition from wakefulness to NREM stage 2 sleep in narcoleptics, and to provide evidence for qualitatively different states of sleepiness, power spectra during wakefulness and stage 1 were compared between narcoleptic naps containing REM sleep and narcoleptic naps containing NREM sleep using mixed two-factor 2x2 (nap type by stage) ANOVAs. Table 5 displays these results. Contrary to predictions, no significant main effects of nap type or nap type by stage interactions were found.

*NOTE: Identical main effects of stage were obtained in each of the following stage FFT analyses. For brevity, these results will not be restated.
(d) Narcoleptic NREM (stage 2) naps versus normal NREM (stage 2) naps

To investigate differences between narcoleptics and normals in the microstructure of the transition from wakefulness to NREM stage 2 sleep, power spectra during wakefulness and stage 1 were compared between narcoleptic naps containing NREM sleep (stage 2) and normal naps containing NREM sleep (stage 2) using mixed two-factor 2x2 (group by stage) ANOVAs. The results are presented in Table 5. A significant main effect of group was found for delta power. No significant group by stage interactions were observed.

Collapsing across wakefulness and stage 1, (as predicted) narcoleptic naps containing NREM stages 1 and 2 had significantly higher mean delta power (5.7 \( \mu V^2 \)) compared to normal naps containing NREM stages 1 and 2 (4.4 \( \mu V^2 \)) \([F(1,16) = 5.42, p = .033]\) (see Figure 6). Specifically, mean delta power was significantly higher in narcoleptics during wakefulness \([t(16) = 2.32, p = .034]\) but not stage 1 \([t(16) = 1.87, p = .079]\).

(e) Narcoleptic NREM (stage 2) naps versus normal NREM (stage 1) naps

To investigate differences in the microstructure of the narcoleptic transition from wakefulness to NREM stage 2 sleep and the normal transition to stage 1 sleep, power spectra during wakefulness and stage 1 were compared between narcoleptic naps containing NREM sleep (stage 2) and normal naps containing just NREM sleep stage 1 using mixed two-factor 2x2 (group by stage) ANOVAs. The results are presented in Table 5. Main effects of group were found for delta power, and a trend was observed for theta power.

Collapsing across stage, (as predicted) mean delta power was significantly higher in the narcoleptic naps containing NREM stages 1 and 2 (5.7 \( \mu V^2 \)) compared to normal naps containing just stage 1 (3.6 \( \mu V^2 \)) \([F(1,12) = 8.28, p = .014]\) (see Figure 6). Specifically, delta power was higher during wakefulness \([t(12) = 2.26, p = .043]\) and stage 1 \([t(12) = 2.83, p = .015]\) for narcoleptic NREM naps. Moreover, a significant group by
stage interaction \[ F(1,12) = 5.04, p = .044 \] was present such that in the narcoleptic NREM naps containing stage 2, a greater increase in delta power from wakefulness to stage 1 was present compared to the normal naps containing just stage 1.

Collapsing across stage, (as predicted) there was a trend for mean theta power to be higher in the narcoleptic naps containing NREM stages 1 and 2 (3.2 μV²) compared to normal naps containing just stage 1 (1.9 μV²) \[ F(1,12) = 3.47, p = .087 \] (see Figure 7).

(f) Narcoleptic REM naps versus normal NREM (stage 2) naps

To investigate differences in the microstructure of the narcoleptic transition from wakefulness to REM sleep and the normal transition to NREM stage 2 sleep, power spectra during wakefulness and stage 1 were compared between narcoleptic naps containing REM sleep, and normal naps containing NREM sleep stage 2 using mixed two-factor 2x2 (group by stage) ANOVAs. Table 5 presents these results. Trends for main effects of group were found for delta and sigma power. No significant group by stage interactions were found.

Collapsing across stage, (as predicted) delta power tended to be higher in the narcoleptic REM naps (5.5 μV²) compared to the normal NREM naps containing stages 1 and 2 (4.4 μV²) \[ F(1,18) = 3.84, p = .066 \] (see Figure 6). Collapsing across stage, (as predicted) ) sigma power tended to be lower in the narcoleptic REM naps (0.5 μV²) compared to the normal NREM naps containing stages 1 and 2 (0.6 μV²) \[ F(1,18) = 3.27, p = .087 \] (see Figure 8).

(g) Narcoleptic REM naps versus normal NREM (stage 1) naps

To investigate differences in the microstructure of the narcoleptic transition from wakefulness to REM sleep and the normal transition to stage 1 sleep, power spectra during wakefulness and stage 1 were compared between narcoleptic naps containing REM sleep, and normal naps containing just NREM sleep stage 1 using mixed two-factor 2x2 (group by stage) ANOVAs. The results are presented in Table 5. Significant main effects
of group were found for delta, theta and sigma power. No significant group by stage interactions were found.

Collapsing across stage, (as predicted) mean delta power was significantly higher in the narcoleptic REM naps (5.5 µV²) compared to the normal NREM naps containing just stage 1 (3.6 µV²) \( F(1,14) = 6.44, p = .024 \) (see Figure 6). Specifically, delta power in the narcoleptic REM naps was significantly higher during stage 1 \( t(14) = 2.79, p = .014 \), but not during wakefulness \( t(14) = 1.65, p = .120 \).

Collapsing across stage, (as predicted) mean theta power was significantly higher in the narcoleptic REM naps (2.8 µV²) compared to the normal NREM naps containing just stage 1 (1.9 µV²) \( F(1,14) = 5.48, p = .035 \) (see Figure 7). Specifically, theta power in the narcoleptic REM naps was significantly higher during wakefulness \( t(14) = 2.32, p = .036 \), but not stage 1 \( t(14) = 1.77, p = .098 \).

Collapsing across stage, (as predicted) mean sigma power was significantly lower in the narcoleptic REM naps (0.48 µV²) compared to the normal NREM naps containing just stage 1 (0.67 µV²) \( F(1,14) = 7.12, p = .018 \) (see Figure 8). Specifically, sigma power in the narcoleptic REM naps was significantly lower during stage 1 \( t(14) = 2.79, p = .014 \), but not during wakefulness \( t(14) = 2.02, p = .063 \).

4. TIME-BASED POWER SPECTRAL ANALYSES OF THE SLEEP ONSET PERIOD

In this section, the process of transition from wakefulness to sleep onset is quantified in normals and narcoleptics using the time-based FFT strategy; EEG power spectral analysis was performed on successive quartiles (consisting of equal units of time) across the chronological development of the sleep onset period of selected nap opportunities on the Multiple Sleep Latency Test. Dependent measures for each quartile of the sleep onset period included mean power values and the proportion of mean number of fluctuations in power values (i.e., slope changes) for delta, theta, sigma and beta frequency bands at Cz and for alpha at O2. Table 2 displays the distribution of REM,
NREM (stage 1) and NREM (stages 1 and 2) naps selected for analysis in narcoleptics and normals.

(a) Normal versus narcoleptic sleep onset process

The normal sleep onset process was compared with the narcoleptic sleep onset process by first comparing EEG power spectra during sleep onset, without specifying the type of sleep to ensue. Focusing on the sleep onset process in general, irrespective of how the naps were classified, mixed two-factor 2x2 (group by quartile) ANOVAs were conducted on power spectra and fluctuations in slope during the quartiles of the sleep onset period in narcoleptics and normals for the selected naps outlined in Table 2. The results are presented in Table 6. Significant main effects of group were found for mean delta and alpha power, and a trend was observed for mean theta power.

Collapsing across quartiles, (as predicted) mean delta power was significantly higher in narcoleptics (7.6 μV^2) compared to normals (4.9 μV^2) [F(1,32) = 19.08, p = .001] (see Figure 9). Specifically, delta power was greater in narcoleptics across each quartile of the sleep onset period [t(32) = 3.12, p = .004; t(32) = 4.84, p = .001; t(32) = 4.17, p = .001; t(32) = 2.66, p = .001].

Collapsing across quartiles, (as predicted) mean theta power tended to be higher in the narcoleptics (2.7 μV^2) compared to normals (2.1 μV^2) [F(1,32) = 3.50, p = .071] (see Figure 10). Moreover, a significant group by quartile interaction was found for mean theta power [F(3,96) = 4.79, p = .004]. The greatest difference between narcoleptics and normals in mean theta power occurred during the first quartile of the sleep onset period. Across the second, third and fourth quartiles, this difference was reduced.

Collapsing across quartiles, (as predicted) mean alpha power was significantly lower in narcoleptics (1.6 μV^2) compared to normals (2.6 μV^2) [F(1,32) = 7.51, p = .010] (see Figure 11). Specifically, alpha power tended to be lower in the narcoleptics in the first quartile [t(32) = 1.86, p = .072], and was significantly lower in narcoleptics across
the second, third and fourth quartiles of the sleep onset period \[ t(32) = 2.30, p = .028; t(32) = 2.65, p = .012; t(32) = 2.90, p = .007 \].

Contrary to predictions, no main effects of group were observed for the mean proportion of slope changes in delta, theta, alpha, sigma or beta power. There was a trend for a group by quartile interaction in mean proportion of slope changes in theta power \[ F(3,96) = 2.42, p = .071 \] (see Figure 12). Mean number of slope changes in theta power across the quartiles of the sleep onset period tended to remain constant in narcoleptics and increase in normals.

Significant main effects of chronological quartile* were found for mean delta, alpha, sigma and beta power, and for the mean proportion of slope changes in delta, theta, alpha and sigma power. Collapsing across group, mean power and mean proportion of slope changes in power across the four quartiles of the sleep onset period increased within the delta and sigma frequency bands, and decreased for the alpha band. Mean proportion of slope changes in theta power increased, and mean beta power decreased across the quartiles of the sleep onset period.

(b) Normal NREM (stage 2) naps versus normal NREM (stage 1) naps

To further investigate the chronological microstructure of the normal transition from wakefulness to sleep, power spectra and fluctuations in slope during the quartiles of the sleep onset period were compared between normal control naps containing stage 2 and normal control naps containing just stage 1 sleep using mixed two-factor 2x4 (naptype by quartile) ANOVAs. These results are presented in Tables 7 and 8. Significant main effects of naptype were found for delta and alpha power.

Collapsing across quartiles, (as predicted) mean delta power was significantly higher during the normal naps containing stage 2 sleep \( (5.6 \mu V^2) \) compared to the normal naps containing just stage 1 \( (3.8 \mu V^2) \) \[ F(1,14) = 20.38, p = .001 \] (see Figure 13). This

* NOTE: Identical main effects of quartile were obtained in each of the following time-based FFT analyses. For brevity, these results will not be restated.
effect was mediated by a significant naptype by quartile interaction $[F(3,42) = 12.79, \ p = .001]$. During the first quartile of the sleep onset period, delta power did not differ between normal naps containing stage 2 and normal naps containing just stage 1. However, by the third and fourth quartile, delta power had increased for the normal naps containing stage 2, but remained at a constant level for the naps containing just stage 1.

Collapsing across quartiles, (as predicted) mean alpha power was significantly lower during the normal naps containing stage 2 sleep (2.1 $\mu V^2$) compared to the normal naps containing just stage 1 (3.5 $\mu V^2$) $[F(1,14) = 4.73, \ p = .047]$ (see Figure 14). This effect was mediated by a significant naptype by quartile interaction $[F(3,42) = 7.32, \ p = .001]$. During the first quartile of the sleep onset period, mean alpha power did not differ between normal naps containing stage 2 and normal naps containing just stage 1. However, mean alpha power decreased across the quartiles of the sleep onset period for stage 2 naps, whereas alpha power remained at a constant high level for stage 1 naps. A significant nap type by quartile interaction was found for mean sigma power $[F(3,42) = 5.42, \ p = .003]$ (see Figure 15). Across the first three quartiles of the sleep onset period, mean sigma power was higher for normal naps containing just stage 1. However, by the fourth quartile, a cross over occurred, and mean sigma power was higher for the normal naps containing stage 2.

Significant main effects of naptype were found for mean proportion of slope changes in delta, alpha, sigma and beta power. Collapsing across quartiles, (as predicted) mean proportion of delta slope changes was significantly lower during the normal naps containing stage 2 sleep (0.35) compared to the normal naps containing just stage 1 (0.44) $[F(1,14) = 10.15, \ p = .007]$ (see Figure 16). This effect was mediated by a significant naptype by quartile interaction $[F(3,42) = 3.60, \ p = .021]$. During the first and second quartiles of the sleep onset period a higher proportion of delta slope changes were present for the normal stage 1 naps, whereas during the third and fourth quartiles, the normal
stage 1 naps and normal stage 2 naps did not differ in the proportion of delta slope changes.

Collapsing across quartiles, (as predicted) the mean proportion of alpha slope changes was significantly lower during the normal naps containing stage 2 sleep (0.29) compared to the normal naps containing just stage 1 (0.46) \[F(1,14) = 18.33, p = .001\] (see Figure 17). This effect was mediated by a significant naptype by quartile interaction \[F(3,42) = 8.45, p = .001\]. During the first quartile of the sleep onset period, the proportion of alpha slope changes did not differ between normal stage 2 naps and normal stage 1 naps. However, whereas the proportion of alpha slope changes decreased across quartiles for normal stage 2 naps, the proportion of alpha slope changes increased across quartiles for normal stage 1 naps.

Collapsing across quartiles, (as predicted) the mean proportion of sigma slope changes was significantly lower during the normal naps containing stage 2 sleep (0.39) compared to the normal naps containing just stage 1 (0.47) \[F(1,14) = 5.62, p = .033\] (see Figure 18). Specifically, the proportion of sigma slope changes was significantly lower during normal naps containing stage 2 for the first \[t(14) = 2.97, p = .010\] and second \[t(14) = 2.25, p = .041\] quartiles, but not the third and fourth quartiles of the sleep onset period.

Collapsing across quartiles, (as predicted) the mean proportion of beta slope changes was significantly lower during the normal naps containing stage 2 sleep (0.39) compared to the normal naps containing just stage 1 (0.47) \[F(1,14) = 4.76, p = .047\] (see Figure 19). Specifically, the proportion of beta slope changes was significantly lower during normal naps containing stage 2 for the fourth quartile \[t(14) = 2.96, p = .010\], but not the first three quartiles of the sleep onset period.

c) Narcoleptic REM naps versus narcoleptic NREM (stage 2) naps

To investigate differences in the microstructure of the transition from wakefulness to REM sleep and the transition from wakefulness to NREM stage 2 sleep in narcoleptics,
and to provide evidence for qualitatively different states of sleepiness, power spectra and fluctuations in slope during the quartiles of the sleep onset period were compared between narcoleptic naps containing REM sleep and narcoleptic naps containing NREM sleep using mixed two-factor 2x4 (naptype by quartile) ANOVAs. These results are presented in Tables 7 and 8.

Contrary to predictions, no significant main effects of naptype were found for mean power within the delta, theta, alpha, sigma or beta bands. However, significant naptype by quartile interactions were found for mean delta power \([F(3, 48) = 3.90, p = .014]\) (see Figure 13) and mean sigma power \([F(3, 48) = 5.71, p = .002]\) (see Figure 15). For both the narcoleptic REM naps and the narcoleptic NREM naps, mean delta power increased across the quartiles of the sleep onset period. However, during the first quartile delta power did not differ between the REM and NREM naps, whereas by the third and fourth quartile delta power was higher for the NREM naps than the REM naps. Mean sigma power remained at a constant level across the quartiles of the sleep onset period of narcoleptic REM naps. During the first quartile, sigma power did not differ between narcoleptic NREM and REM naps. However, by the fourth quartile, mean sigma power was higher for the narcoleptic NREM naps.

Contrary to predictions, no significant main effects of naptype were found for mean proportion of slope changes within the delta, theta, alpha, sigma or beta bands. A significant naptype by quartile interaction was found for mean proportion of slope changes in theta power \([F(3, 48) = 3.99, p = .013]\) (see Figure 20). During the first quartile of the sleep onset period, the proportion of theta slope changes did not differ between narcoleptic REM and NREM naps. However, by the fourth quartile, the proportion of theta slope changes was higher for narcoleptic NREM naps than narcoleptic REM naps.

(d) Narcoleptic NREM (stage 2) naps versus normal NREM (stage 2) naps

To investigate differences between narcoleptics and normals in the microstructure of the transition from wakefulness to NREM stage 2 sleep, power spectra and fluctuations
in slope during the quartiles of the sleep onset period were compared between narcoleptic
naps containing NREM sleep (stage 2) and normal naps containing NREM sleep (stage 2)
using mixed two-factor 2x4 (naptype by quartile) ANOVAs. These results are presented
in Tables 7 and 8. A significant main effect of group was found for delta power. No
group by quartile interactions were found.

Collapsing across quartiles, (as predicted) mean delta power was significantly
higher during narcoleptic naps consisting of NREM sleep stages 1 and 2 (8.2 \mu V^2)
compared to normal naps containing NREM sleep stages 1 and 2 (5.6 \mu V^2) \[F(1,16) = 9.36, p = .007\] (see Figure 13). Specifically, delta power was greater in narcoleptics
across each quartile of the sleep onset period \[t(16) = 2.31, p = .035; t(16) = 2.86, p =
.011; t(16) = 2.89, p = .011; t(16) = 2.33, p = .033\].

Contrary to predictions, no significant main effects of group or group by quartile
interactions were found for mean proportion of slope changes within any frequency band.

(e) Narcoleptic NREM (stage 2) naps versus normal NREM (stage 1) naps

To investigate differences in the microstructure of the narcoleptic transition from
wakefulness to NREM stage 2 sleep and the normal transition to stage 1 sleep, power
spectra and fluctuations in slope during the quartiles of the sleep onset period were
compared between narcoleptic naps containing NREM sleep (stage 2) and normal naps
containing just NREM stage 1 using mixed two-factor 2x4 (naptype by quartile)
ANOVAs. These results are presented in Tables 7 and 8. Significant main effects of
group were found for delta and alpha power, and a trend was observed for theta power.
Significant group by quartile interactions were found for delta, alpha and sigma power.

Collapsing across quartiles, (as predicted) mean delta power was significantly
higher during narcoleptic naps consisting of NREM sleep stages 1 and 2 (8.2 \mu V^2)
compared to normal naps containing just NREM stage 1 (3.8 \mu V^2) \[F(1,12) = 16.90, p =
.001\] (see Figure 13). This effect was mediated by a significant group by quartile
interaction \[F(3,36) = 11.54, p = .001\]. In the narcoleptic NREM naps mean delta power
increased across the quartiles of the sleep onset period, whereas in the normal control naps containing just stage 1, mean delta power remained at a constant level.

Collapsing across quartiles, (as predicted) mean alpha power was significantly lower during narcoleptic NREM naps (1.6 μV²) compared to normal naps containing just stage 1 (3.5 μV²) [F(1,12) = 9.10, p = .011] (see Figure 14). Moreover, this effect was mediated by a significant group by quartile interaction [F(3,36) = 6.30, p = .002]. In the narcoleptic NREM naps mean alpha power decreased across the quartiles of the sleep onset period, whereas in the normal naps containing just stage 1, mean alpha power remained at a constant level.

Collapsing across quartiles, (as predicted) mean theta power tended to be higher during narcoleptic NREM naps (2.8 μV²) compared to normal naps containing just stage 1 (1.8 μV²) [F(1,12) = 4.61, p = .053] (see Figure 21). Sigma power was mediated by a group by quartile interaction [F(3,36) = 3.83, p = .018] (see Figure 15). During the first three quartiles of the sleep onset period, mean sigma power was lower for narcoleptic NREM naps compared to normal naps containing just stage 1. However a cross-over occurred such that during the fourth quartile sigma power was higher in the narcoleptic NREM naps.

Significant main effects of group were found for the mean proportion of slope changes within the delta and alpha frequency bands. Collapsing across quartiles, (as predicted) the mean proportion of slope changes in delta power was significantly lower for narcoleptic NREM naps (0.33) than for normal naps containing just stage 1 (0.44) [F(1,12) = 5.51, p = .037] (see Figure 16). Moreover, this effect was mediated by a significant group by quartile interaction [F(3,36) = 3.64, p = .022]. The mean proportion of delta slope changes increased in narcoleptic NREM naps and tended to remain at a constant level in the normals such that, during the first and second quartiles but not the third and fourth quartiles, the mean proportion of delta slope changes was lower in narcoleptic NREM naps.
Collapsing across quartiles, (as predicted) the mean proportion of slope changes in alpha power was significantly lower for narcoleptic NREM naps (0.29) than for normal naps containing just stage 1 (0.46) \([E(1,12) = 13.00, p = .004]\) (see Figure 17). Specifically, the mean proportion of alpha slope changes did not differ between narcoleptics and normals across the first three quartiles, but was significantly lower in the narcoleptics during the fourth quartile of the sleep onset period \([t(12) = 5.11, p = .001]\).

(f) Narcoleptic REM naps versus normal NREM (stage 2) naps

To investigate differences in the microstructure of the narcoleptic transition from wakefulness to REM sleep and the normal transition to NREM stage 2 sleep, power spectra and fluctuations in slope during the quartiles of the sleep onset period were compared between narcoleptic naps containing REM sleep and normal naps containing NREM sleep stages 1 and 2 using mixed two-factor 2x4 (naptype by quartile) ANOVAs. These results are presented in Tables 7 and 8. Significant group main effects were found for mean delta power, and a trend was observed for mean sigma power. Significant naptype by quartile interactions for mean delta, theta and sigma power.

Collapsing across quartiles, (as predicted) mean delta power was significantly higher during the narcoleptic REM naps (7.2 \(\mu V^2\)) compared to the normal naps containing NREM stages 1 and 2 (5.6 \(\mu V^2\)) \([E(1,18) = 5.36, p = .029]\) (see Figure 13). This effect was mediated by a group by quartile interaction \([E(3,54) = 3.27, p = .028]\). During the first three quartiles of the sleep onset period, delta power was higher for narcoleptic REM naps than normal stage 2 naps. However, by the fourth quartile of the sleep onset period, delta power did not differ between the narcoleptic REM naps and normal stage 2 naps.

Collapsing across quartiles, (as predicted) mean sigma power tended to be lower in narcoleptic REM naps (0.4 \(\mu V^2\)) compared to normal naps containing stages 1 and 2 (0.6 \(\mu V^2\)) \([E(1,18) = 3.97, p = .062]\) (see Figure 15). This effect was mediated by a group by quartile interaction \([E(3,54) = 3.83, p = .015]\). Whereas mean sigma power remained at a
constant level across all four quartiles of the sleep onset period in narcoleptic REM naps, in the normal stage 2 naps mean sigma power remained at a constant level across the first three quartiles, and then increased in the fourth quartile of the sleep onset period.

There was a significant nap type by quartile interaction for mean theta power \( [F(3,54) = 5.33, p = .003] \) (see Figure 21). For both narcoleptic REM naps and normal stage 2 naps, mean theta power tended to decrease across the quartiles of the sleep onset period. However, during the first quartile mean theta power did not differ between narcoleptic REM naps and normal stage 2 naps, whereas by the fourth quartile mean theta power tended to be lower in narcoleptic REM naps than normal stage 2 naps.

Contrary to predictions, no significant main effects of group were found for mean proportion of slope changes in delta, theta, alpha, sigma or beta power. A significant group by quartile interaction was found for mean proportion of slope changes in theta power \( [F(3,54) = 3.95, p = .013] \) (see Figure 21 for this complex interaction).

(g) Narcoleptic REM naps versus normal NREM (stage 1) naps

To investigate differences in the microstructure of the narcoleptic transition from wakefulness to REM sleep and the normal transition to stage 1 sleep, power spectra and fluctuations in slope during the quartiles of the sleep onset period were compared between narcoleptic naps containing REM sleep and normal naps containing just NREM sleep stage 1 using mixed two-factor 2x4 (nap type by quartile) ANOVAs. These results are presented in Tables 7 and 8. Significant main effects of group were found for mean delta, theta, alpha and sigma power.

Collapsing across quartiles, (as predicted) mean delta power was significantly higher during the narcoleptic REM naps (7.2 \( \mu \text{V}^2 \)) compared to the normal naps containing just NREM stage 1 (3.8 \( \mu \text{V}^2 \)) \( [F(1,14) = 16.68, p = .001] \) (see Figure 13). Specifically, mean delta power was higher for narcoleptic REM naps across all four quartiles of the sleep onset period \( [t(14) = 1.98, p = .067; t(14) = 3.71, p = .002; t(14) = 5.25, p = .001; t(14) = 2.93, p = .011] \).
Collapsing across quartiles, (as predicted) mean theta power was significantly higher during the narcoleptic REM naps (2.6 $\mu$V$^2$) compared to the normal naps containing just NREM stage 1 (1.8 $\mu$V$^2$) [$F(1,14) = 4.82$, $p = .046$] (see Figure 21). This effect was mediated by a significant group by quartile interaction [$F(3,42) = 3.47$, $p = .024$]. Mean theta power tended to decrease across the quartiles of the sleep onset period in narcoleptic REM naps, but increase in normal stage 1 naps.

Collapsing across quartiles, (as predicted) mean alpha power was significantly lower during the narcoleptic REM naps (1.7 $\mu$V$^2$) compared to the normal naps containing just NREM stage 1 (3.5 $\mu$V$^2$) [$F(1,14) = 9.09$, $p = .009$] (see Figure 14). This effect was mediated by a significant group by quartile interaction [$F(3,42) = 4.72$, $p = .006$]. Mean alpha power deceased across the quartiles of the sleep onset period in narcoleptic REM naps, but remained at a constant level in normal stage 1 naps.

Collapsing across quartiles, (as predicted) mean sigma power was significantly lower during the narcoleptic REM naps (0.4 $\mu$V$^2$) compared to the normal naps containing just NREM stage 1 (0.7 $\mu$V$^2$) [$F(1,14) = 5.44$, $p = .035$] (see Figure 15). Specifically, mean sigma power was significantly lower in the narcoleptic REM naps for the first [$t(14) = 2.44$, $p = .028$], and second [$t(14) = 3.28$, $p = .005$] quartiles, but not the third or fourth quartiles of the sleep onset period.

Trends for main effects of group were observed for mean proportion of slope changes in alpha and beta power. Collapsing across quartiles, (as predicted) the mean proportion of slope changes in alpha power tended to be lower during the narcoleptic REM naps (0.35) compared to the normal naps containing just NREM stage 1 (0.46) [$F(1,14) = 4.51$, $p = .052$] (see Figure 17). This effect was mediated by a significant group by quartile interaction [$F(3,42) = 4.45$, $p = .008$]. During the first quartile of the sleep onset period, mean proportion of alpha slope changes did not differ between narcoleptic REM naps and normal stage 1 naps. However, across the second, third and
fourth quartiles, the proportion of alpha slope changes decreased for narcoleptic REM naps, and increased for normal stage 1 naps.

Collapsing across quartiles, (as predicted) the mean proportion of slope changes in beta power tended to be lower during the narcoleptic REM naps (0.42) compared to the normal naps containing just NREM stage 1 (0.47) \( [F(1, 14) = 3.66, p = .077] \) (see Figure 19). There was a significant naptype by quartile interaction for mean proportion of slope changes in theta power \( [F(3, 42) = 3.55, p = .022] \) (see Figure 20). During the first quartile of the sleep onset period, mean proportion of theta slope changes did not differ between narcoleptic REM naps and normal stage 1 naps. However, by the fourth quartile, mean proportion of theta slope changes was lower for the narcoleptic REM naps than the normal stage 1 naps.

5. **ALPHA ATTENUATION TEST**

Table 9 presents mean alpha power at O2 for narcoleptics and normals during the eyes-open and eyes-closed conditions of the Alpha Attenuation Test, and the ratio of mean eyes-closed to mean eyes-open alpha power (i.e., alpha attenuation coefficient) as a function of group and time of testing.

Mean eyes-closed alpha power and eyes-open alpha power were investigated in narcoleptics and normals using a mixed three-factor 2x2x5 (group by eyelid position by Alpha Attenuation Test session) ANOVA. There was a significant group by eyelid position interaction \( [F(1, 18) = 6.52, p = .020] \) (see Figure 22). As predicted, during the eyes-closed condition, mean alpha power was significantly lower in the narcoleptics \( (2.4 \mu V^2) \) than the normals \( (3.9 \mu V^2) \) \( [t(18) = 2.5, p = .022] \). However, contrary to predictions, during the eyes-open condition, mean alpha power did not differ between the groups \( (1.7 \mu V^2 \text{ in both narcoleptics and normals}) \). The main effect of group (i.e., collapsing across eyelid position and session) did not reach significance; however, there was a trend for mean alpha power to be lower in narcoleptics \( (2.0 \mu V^2) \) compared to
normals (2.8 $\mu V^2$) [$F(1,18) = 4.01, p = .060$]. There was a significant main effect for eyelid position [$F(1,18) = 19.76, p = .001$], such that collapsing across group and session, mean eyes-closed alpha power (3.2 $\mu V^2$) was greater than mean eyes-open alpha power (1.7 $\mu V^2$). No session main effect or group by session interaction was found for mean alpha power.

The ratio of eyes-closed to eyes-open mean alpha power (i.e., alpha attenuation coefficient) was subjected to a mixed two-factor 2x5 (group by session) ANOVA. Significant main effects of group [$F(1,18) = 4.97, p = .039$] and session [$F(4,72) = 3.28, p = .016$] were found (see Figure 23). Collapsing across session, (as predicted) the mean alpha attenuation coefficient was lower for the narcoleptics (1.4) compared to the normals (2.5). Collapsing across group, the mean alpha attenuation coefficient was lowest at the 1500 h session. No group by session interaction was found.

Comparing the Multiple Sleep Latency Test with the Alpha Attenuation Test

Latency to stage 1 on the Multiple Sleep Latency Test and alpha attenuation coefficients were correlated intraindividually. Eight of the ten narcoleptics had positive correlations (i.e., lower alpha attenuation coefficient associated with shorter latency to stage 1), which ranged in magnitude from .09 to .71 ($M = .43, SD = .21$). By contrast, six of the ten normals had positive correlations, which ranged in magnitude from .06 to .95 ($M = .43, SD = .34$). However, none of the ten narcoleptics, and one of the ten normals demonstrated a significant correlation between latency to sleep onset and the alpha attenuation coefficient.

6. SUBJECTIVE SLEEPINESS MEASURES

To compare differences in subjective sleepiness between normals and narcoleptics, mean scores on the Stanford Sleepiness Scale and Visual Analogue Sleepiness Scale were calculated within each participant, and t-tests were used to assess group differences. Mean Stanford Sleepiness Scale scores tended to be higher for narcoleptics (3.1) than
normals (2.4) [t(18) = 2.04, p = .056]. Similarly, mean Visual Analogue Sleepiness Scale scores tended to be higher for narcoleptics (39.0) than normals (25.1) [t(18) = 1.98, p = .063].

Relationship of subjective sleepiness measures with the Alpha Attenuation Test

Scores on the Stanford Sleepiness Scale and Visual Analogue Sleepiness Scale administered prior to and following each Alpha Attenuation Test session were correlated intrindividually with the alpha attenuation coefficient. One of the ten narcoleptics demonstrated a significant correlation between the alpha attenuation coefficient and the Visual Analogue Sleepiness Scale administered prior to testing, and another narcoleptic demonstrated a significant correlation between the alpha attenuation coefficient and the Stanford Sleepiness Scale administered after testing. Two of the ten normal controls demonstrated a significant correlation between post-Alpha Attenuation Test scores on the Visual Analogue Sleepiness Scale and the alpha attenuation coefficient.

Relationship of subjective sleepiness measures with the Multiple Sleep Latency Test

Scores obtained on the Stanford Sleepiness Scale and Visual Analogue Sleepiness Scale prior to and following each Multiple Sleep Latency Test were correlated intrindividually with latency to stage 1 sleep. One of the ten narcoleptics demonstrated a significant correlation between post-Multiple Sleep Latency Test Visual Analogue Sleepiness Scale and latency to stage 1. Two of the ten normals demonstrated a significant correlation between post-Multiple Sleep Latency Test Stanford Sleepiness Scale and latency to stage 1.
Discussion

The purpose of the present study was to objectively quantify sleepiness and the sleep onset process in normals and narcoleptics using computer-based EEG power spectral analysis in addition to the traditional measure of visually scored sleep stages. The present study is one of the first to conduct power spectral analysis of the EEG in both normals and narcoleptics during the transition from wakefulness to sleep (i.e., on the Multiple Sleep Latency Test) and during seated wakefulness (i.e., the Alpha Attenuation Test). Specific aims were as follows:

(I) To further examine the electrophysiological nature of the normal sleep onset process by quantifying EEG via spectral analysis.

(II) To distinguish the narcoleptic sleep onset process from the normal sleep onset process by quantifying EEG via spectral analysis.

(III) To attempt to resolve the issue of qualitatively different states of sleepiness using EEG power spectral analysis of the sleep onset period in addition to visually-scored latency to sleep onset.

(IV) To investigate the utility of the Alpha Attenuation Test, a measure based on EEG power spectral analysis, as an alternative to the Multiple Sleep Latency Test in the objective assessment of the excessive daytime sleepiness associated with narcolepsy.

Evidence was obtained in support of the hypothesis that quantitative and qualitative differences in narcoleptic and normal sleepiness and sleep onset processes may be assessed using computerized analyses of EEG power spectra.

Following an overview of the findings for visual stage scoring and power spectral analysis of the sleep onset period elicited by the Multiple Sleep Latency Test, the Alpha Attenuation Test results will be presented. This, in turn, will be followed by a discussion of the theoretical and practical implications of the present study.
MULTIPLE SLEEP LATENCY TEST

Visually-Scored Latency to Sleep Onset

As predicted, mean latency to sleep onset on Multiple Sleep Latency Test was significantly shorter for narcoleptics than normals, indicating that narcoleptics were physiologically sleepier than normals. This finding has been widely demonstrated (e.g., Richardson et al., 1978). It was also predicted that within narcoleptics, the pressure for REM sleep would be greater than the pressure for NREM sleep, resulting in shorter sleep onset latencies for REM naps compared to NREM naps. Mean latency to stage 1 for REM naps and for NREM was determined within each narcoleptic, and then compared using a paired t-test. Although mean latency to stage 1 sleep for narcoleptics was shorter on average for naps containing REM sleep (3.2 min) than for naps containing just NREM sleep (4.3 min), contrary to predictions, this difference was not significant.

However, when the individual latencies obtained from each narcoleptic nap opportunity were analyzed using a between-groups t-test as per Broughton and Aguirre (1987)*, mean latency to stage 1 for REM-containing naps (2.4 min) was significantly shorter than latency to stage 1 for NREM-only naps (4.8 min) [t(48) = 2.64, p = .011]. Statistical considerations aside, these findings replicate the direction of difference reported by Broughton and Aguirre (1987), and are consistent with their finding that REM sleepiness was objectively greater than NREM sleepiness, thus supporting their proposal that REM and NREM sleepiness reflect qualitatively different states of sleepiness. However, although the present study replicated Broughton and Aguirre's (1987) findings using inflated degrees of freedom, it must be noted that the effect size is very small. Indeed, further confirmation of these findings involving a larger sample of narcoleptics

* NOTE: Broughton & Aguirre (1987) erroneously treated their data as if there were 10 participants x 5 sessions = 50 narcoleptics, rather than the actual 10 narcoleptics, thereby inflating the degrees of freedom for their t-test.
appears warranted before the existence of qualitatively different states of sleepiness using
latency to sleep onset as an index of physiological sleepiness can be verified.

**EEG Power Spectral Analysis of the Sleep Onset Process**

The electrophysiological nature of the sleep onset process was investigated by
performing EEG power spectral analysis on the nap opportunities elicited by the Multiple
Sleep Latency Test in normals and narcoleptics. The data were first analyzed according to
a visually-scored stage-based strategy (wakefulness and stage 1). Then, because
individuals may fluctuate between wakefulness and stage 1 throughout the entry into
sleep, the sleep onset period was divided into quartiles based on the length of the sleep
onset period. This strategy (four time-based quartiles) enabled the chronological
development of the sleep onset process to be studied. The oscillatory nature of the
process of sleep onset was investigated by examining fluctuations in arousal level (i.e., the
proportion of slope changes in each frequency band) within each quartile of the sleep
onset period.

**Normal sleep onset process**

The present study provided information on the normal process of entry into sleep
by comparing normal naps containing NREM stage 2 with naps containing just stage 1
sleep. As predicted, both the stage-based and time-based analyses demonstrated that
mean delta power was higher for normal stage 2 naps than normal stage 1 naps (see
Figures 6 and 13). Moreover, interaction effects revealed that during wakefulness and the
first two quartiles of the sleep onset period, mean delta power did not differ between
normal stage 2 naps and normal stage 1 naps. During stage 1 and the third and fourth
quartiles, delta power was higher for normal stage 2 naps than the normal stage 1 naps.
The fact that early on in the sleep onset process delta power did not differ between normal
stage 1 naps and normal stage 2 naps suggests that physiological sleepiness did not differ
at the onset of the nap opportunity. However, an increase in delta power occurred later in
the sleep onset period of naps which progressed to include the development of stage 2
sleep. This finding is consistent with those of Badia et al. (1994) and Ogilvie et al. (1989; 1991; 1992), and suggests that delta power is sensitive to variations within the microstructure of the normal sleep onset process according to whether the development of stage 2 sleep is imminent.

The time-based analyses provided additional information about changes in alpha power across the sleep onset period. As predicted, mean alpha power was lower for normal stage 2 naps than normal stage 1 naps (see Figure 14). Moreover, interaction effects revealed that mean alpha power remained at a consistently high level across quartiles for the normal stage 1 naps, suggesting that alertness/sleepiness levels did not vary much throughout this time. During the first quartile of the sleep onset period, mean alpha power did not differ between stage 1 and stage 2 naps, suggesting that initial alertness/sleepiness levels following lights out were identical and could not predict whether stage 2 would develop. The decrease in mean alpha power across the quartiles of the sleep onset period for stage 2 naps indicate that sleepiness increased as the entry into sleep stage 2 unfolded, and is consistent with findings from Badia et al. (1994). These finding suggest that alpha power is sensitive to variations within the microstructure of the normal sleep onset process according to whether the development of stage 2 sleep is imminent.

In examining the oscillatory nature of the normal process of entry into sleep for stage 2 naps and stage 1 naps, significant variations in slope were observed for delta, alpha, sigma and beta frequency bands (see Figures 16-19). As predicted, for each of these frequency bands, the mean proportion of slope changes in EEG power occurring across the normal sleep onset period was lower for the naps containing stage 2 sleep than for the naps which contained only stage 1. These findings suggest the oscillation of EEG activity within the microstructure of the normal sleep onset process varied depending on whether the development of stage 2 sleep was imminent. 'Smoother' entries into sleep were observed for naps in which stage 2 developed.
Main effects of stage and quartile (i.e., collapsing across naptype) were also found for mean delta, alpha, sigma and beta power. Mean delta power increased whereas mean alpha and beta power decreased from wakefulness to stage 1 and across the quartiles of the sleep onset period. These findings substantiate those of Ogilvie et al. (1989; 1991; 1992), who observed decreases in alpha and beta power during the progression towards the onset of sleep when the longest reaction times to auditory stimuli were compared with the shortest reaction times. Also supported are the findings of Hori (1985) and Badia et al. (1994), who observed increases in delta power across the sleep onset period. Interestingly, the stage-based strategy showed an increase in mean sigma power from wakefulness to stage 1, whereas the time-based strategy showed that sigma power tended to decrease across the quartiles of the sleep onset period. Contrary to Hori (1985), mean theta power did not differ between wakefulness and stage 1 or across the quartiles of the sleep onset period. Furthermore, the mean proportion of slope changes for delta, theta and sigma increased, whereas the mean proportion of slope changes for alpha decreased across the quartiles of the sleep onset period. These findings differ slightly from those observed by Hori (1985), who reported increases in the coefficient of variation (i.e., ratio of standard deviation to mean) for delta, theta and alpha just before or immediately after the onset of stage 1.

**Narcoleptic versus normal sleep onset process**

Differences between normals and narcoleptics in the general process of sleep entry were first investigated by comparing narcoleptic naps with normal control naps, making no attempt to differentiate the type of sleep (NREM or REM) which ensued. To investigate the presence of qualitatively different states of sleepiness, differences between the process

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*NOTE: The stage-based and time-based analyses provided main effects of stage or quartile by averaging across naptype or group. No matter whether normal stage 2 naps were compared with normal stage 1 naps, or normal naps were compared with narcoleptic REM naps or narcoleptic NREM naps, the same significant findings were found, suggesting that the main effects for spectral changes across wakefulness and stage 1, and across quartiles of the sleep onset period were consistent and inherent to the process of sleep onset in general both for normals and narcoleptics.*
of sleep onset were then compared for narcoleptic naps containing REM sleep and narcoleptic naps containing NREM sleep. To further investigate differences in the normal entry into sleep and the narcoleptic entry into REM sleep versus NREM (stage 2) sleep, normal stage 2 naps and normal stage 1 naps were compared with narcoleptic REM naps and narcoleptic stage 2 naps. It was predicted that compared to normals, narcoleptics would exhibit greater delta and theta EEG power and less alpha, sigma and beta EEG power during wakefulness and stage 1, and during the quartiles of the sleep onset period. It was also predicted that compared to narcoleptic naps containing NREM sleep and normal control naps containing NREM sleep, EEG power for the narcoleptic naps containing REM sleep would be greater in the delta and theta frequency bands, and lower in the alpha, sigma and beta frequencies during wakefulness and stage 1, and across the quartiles of the sleep onset period.

*Delta power:* Perhaps the most robust finding was the enhancement of delta power during the narcoleptic transition from wakefulness to sleep. As predicted, both the stage-based and time-based analyses demonstrated that mean delta power was higher for narcoleptics than normals during wakefulness and stage 1 and across quartiles, regardless of whether the narcoleptic versus the normal sleep onset in general was compared, or whether the narcoleptic NREM naps or REM naps were compared with the normal stage 2 naps or normal stage 1 naps (see Figures 3, 6, 9 and 13). These findings represent the first documentation of spectral differences in delta power within the sleep onset period of narcoleptics and normal sleepers.

Elevated delta power has been noted in the nocturnal sleep of narcoleptics following 16 h and 24 h without sleep (Besset et al., 1994), in the recovery sleep of sleep deprived normals (Borbely et al., 1981) and in wakefulness prior to sleep onset in insomniacs (Freeman, 1986). However, it must be stressed that in the present study, no evidence was found to suggest that the narcoleptics were sleep deprived. Mean nocturnal sleep length did not differ significantly between normals and narcoleptics for either the
sleep diary completed for 7 days prior testing, or the nocturnal polysomnography carried out immediately preceding daytime testing. Nor did the nocturnal polysomnography yield significant differences between narcoleptics and normals in sleep efficiency, or percent stage 2, 3 4 and REM differ in narcoleptics and normals. The only differences found between narcoleptics and normal sleepers during the nocturnal polysomnography was that latency to stage 1 and REM sleep was shorter and percentage of time spent in stage 1 was higher for narcoleptics. Thus, there is no evidence to suggest that the enhanced delta power observed during the daytime process of sleep entry in narcoleptics could be accounted for by the effects of experimentally-induced nocturnal sleep deprivation.

The process of entering sleep was compressed in narcoleptics, as indicated by the significantly shorter mean latency to sleep onset observed in narcoleptics on the Multiple Sleep Latency Test compared to normals. The compressed sleep onset period in the presence of enhanced delta power that was observed in narcoleptics mimics the effects of sleep deprivation in normals. However, it is proposed that the increased delta power observed throughout the sleep onset period in the narcoleptics may indicate the presence of an enhanced physiological need for sleep in the absence of sleep deprivation, and when considered in light of the rapid sleep onset data, may reflect a fundamental difference in the process of entering sleep in narcoleptics compared to normals. According to Borbely et al. (1981), the prevalence of delta power in the EEG of nocturnal sleep in normals following sleep deprivation is indicative of the intensity of the sleep process: Delta power is related to an endogenous sleep enhancing factor which accumulates in the brain during the usual waking period, particularly during extended sleep deprivation, and is eliminated or inactivated during sleep. The present findings of enhanced delta power in the absence of sleep deprivation in narcoleptics may suggest the presence of an over-accumulation of Borbely's proposed endogenous sleep enhancing factor. Moreover, given that delta power is mediated by the incidence and amplitude of synchronized EEG rhythms induced by the co-activation of large groups of cortical pyramidal cells with connections to thalamic and
hypothalamic basal forebrain systems (Steriade & McCarley, 1990), the enhanced delta power observed in the narcoleptics may also reflect an over-activation within this neuronal system.

*Theta power:* Support was obtained for the prediction that theta power would be higher during the sleep onset period of narcoleptics compared to normals. Theta power during wakefulness and stage 1, and across the quartiles of the sleep onset period was significantly higher for narcoleptic REM naps compared to normal stage 1 naps, and tended to be higher for narcoleptic NREM (stage 2) naps compared to normal stage 1 naps. When narcoleptic REM naps were compared with normal NREM (stage 2) naps, a group by quartile interaction was observed, such that during the first three quartiles but not the final quartile of the sleep onset period, theta power was higher for narcoleptic REM naps.

These findings are consistent with studies showing that elevated theta power is present in sleepy normals working a nightshift, and in normals deprived of sleep (Torsvall & Akerstedt, 1987; Akerstedt et al., 1987). It is proposed that the elevated theta power present during the sleep onset period of narcoleptics compared to that of normals is in keeping with the narcoleptic complaint of excessive daytime sleepiness, and reflects a fundamental difference in the process of sleep entry. Contrary to predictions, using either the stage-based or time-based strategies, theta power did not differ between the sleep onset period of narcoleptic REM naps and narcoleptic NREM naps.

*Alpha power:* The stage-based analyses failed to produce significant differences between narcoleptic and normal sleep onset process. However, as predicted, alpha power was lower for narcoleptics during the quartiles of the sleep onset period when the sleep onset process in general was compared in narcoleptics and normals, irrespective of selected nap type. Furthermore, alpha power was lower during the quartiles of the sleep onset period when narcoleptic NREM (stage 2) naps were compared with normal naps containing just NREM (stage 1), and when narcoleptic REM naps were compared with
normal naps containing just NREM (stage 1). These findings also support that hypothesis that physiological sleepiness is greater in narcoleptics than normals. The decreased (eyes-closed) alpha power observed throughout the sleep onset period for the narcoleptics supports the proposal that a greater degree of physiological sleepiness was present in the narcoleptics. These findings are consistent with studies of sleep deprived normals that have demonstrated that the presence of decreased (eyes-closed) alpha power during wakefulness is indicative of increased physiological sleepiness (Torsvall & Akerstedt, 1988; Stumpf et al., 1993). Contrary to predictions, the stage-based and time-based strategies failed to show that alpha power differed during the sleep onset period of narcoleptic REM naps and narcoleptic NREM naps.

**Sigma power**: As predicted, the stage-based analyses found that sigma power was significantly lower in narcoleptics during wakefulness and stage 1 compared to normals, collapsing over naptype. Furthermore, sigma power was significantly lower during wakefulness and stage 1, and during the quartiles of the sleep onset period when narcoleptic REM naps were compared with normal stage 1 naps, and sigma power tended to be lower when narcoleptic REM naps were compared with normal NREM (stage 2) naps. When narcoleptic NREM (stage 2) naps were compared with normal naps containing just NREM stage 1, a significant interaction was observed: during the first three quartiles of the sleep onset period sigma power was lower for narcoleptics, but during the fourth quartile sigma power was higher for narcoleptics.

The lower levels of sigma power observed in narcoleptics may reflect a decreased level of lower frequency beta power, which may be viewed as being indicative of a lower level of cortical arousal (Freedman, 1986). A significant interaction was observed when narcoleptic REM naps were compared with narcoleptic NREM naps. During REM naps, sigma power remained at a constant level across the quartiles of the sleep onset period, whereas during NREM naps, sigma power increased from the third to fourth quartile,
presumably reflecting the imminent onset of stage 2 sleep and sleep spindles (sigma frequency).

**Beta power:** Contrary to predictions, beta power did not differ between the narcoleptic and normal sleep onset process. Nor were differences were between narcoleptic REM and NREM naps.

**Slope changes:** The oscillatory nature of the process of entry into sleep was compared in narcoleptics and normals by examining fluctuations in arousal level, operationalized as the proportion of changes in the direction or slope of EEG power bands within each quartile of the sleep onset period. It was predicted that narcoleptics would display a smoother entry into sleep, as indicated by a smaller proportion of slope changes within the frequency bands. Contrary to predictions, no significant differences were observed for any of the frequency bands when the sleep onset process in general was compared between narcoleptics and normals irrespective of the naptype. Nor did the oscillatory nature of the sleep onset process differ when narcoleptic (NREM) stage 2 naps were compared with normal stage 2 naps. However, as predicted, a significantly lower proportion of slope changes in delta power and alpha power was observed for narcoleptic stage 2 naps compared to normal stage 1 naps. Also, as predicted, there tended to be a lower proportion of slope changes in alpha and beta power for narcoleptic REM naps compared to normal stage 2 naps. In general, these findings tend to suggest that spectral variation within the delta, alpha and beta bands in narcoleptics reflected a smoother transition into sleep than that observed in normals.

**Spectral evidence for qualitatively different states of sleepiness**

Contrary to predictions, neither the stage-based nor the time-based spectral analyses provided evidence in support of Broughton and Aguirre’s (1987) findings that within narcoleptics, the pressure for REM sleep was greater than the pressure for NREM sleep. In the present study, mean delta, theta, alpha, sigma and beta power did not differ
significantly between the sleep onset periods of narcoleptic REM naps and narcoleptic NREM naps.

However, the present study provided the first evidence to suggest that spectral differences exist within the sleep onset period of narcoleptic naps containing REM sleep and normal naps containing NREM sleep. Although the EEG of REM sleep and stage 1 sleep appears nearly indistinguishable when it is assessed using visually-based sleep scoring techniques, EEG power spectral analysis using stage-based and/or time-based strategies revealed enhanced delta and theta power, and decreased alpha and sigma power throughout the sleep onset period of narcoleptic REM naps compared to normal stage 1 naps. In addition, delta power was higher during the sleep onset period of narcoleptic REM naps compared to normal stage 2 naps. These findings are the first to suggest the existence of physiological differences within the microstructure of the narcoleptic sleep onset during REM naps and the normal process of sleep onset during stage 1 naps and stage 2 naps.

Comparison of stage-based strategy versus time-based strategy

In general, the stage-based and time-based power spectral analysis strategies produced similar findings for spectral differences between normals and narcoleptics in mean power. However, a greater number of significant effects were obtained using the time-based strategy. This may be due to the fundamental difference between the two strategies in the manner by which the microstructure of the sleep onset process was analyzed. The stage-based strategy performed power spectral analysis on the visually scored stages of wakefulness and stage 1. This strategy was unable to track the moment to moment fluctuations between sleep and wakefulness that occur throughout the entry into sleep (Kleitman, 1963). The time-based strategy enabled the chronological examination of the sleep onset process by dividing the sleep onset period into quartiles which each contained an equal numbers of epochs. Rather than analyzing power spectra during two stages (wakefulness and stage 1) collapsed across time, the time-based strategy
analyzed power spectra at four successive points during the progression towards sleep. In this way, the oscillatory nature of the process of sleep entry was better assessed. Furthermore, the time-based strategy enabled the analysis of the proportion of changes occurring in slope or direction of EEG power within each quartile. Thus, the time-based strategy of power spectral analysis was better able to examine the microstructure of the process of sleep onset in the present study. Moreover, the time-based strategy successfully freed the researcher of the circularity involved in visually scoring sleep stages using EEG criteria, and then evaluating EEG via spectral analysis to look for spectral differences between the stages.

Limitation of Multiple Sleep Latency Test data selection process

Because the type of sleep to occur (i.e., REM, stage 2, or stage 1) on each nap opportunity varied within and between subjects, the decision was made to select for analysis the first NREM nap and REM nap to occur in narcoleptics, and the first stage 2 nap and stage 1 nap to occur in normals. It is possible that results contrary to those presently reported may have been obtained had a different system of nap selection (e.g., a random selection) been implemented. However, the method was chosen because it increased the likelihood that all variations in sleep stages (i.e., REM sleep, stage 1 and stage 2) would be obtained, but perhaps at the cost of eliminating data from later on in the testing period.

ALPHA ATTENUATION TEST

The present study was the first to demonstrate that the Alpha Attenuation Test can be used to distinguish a clinical population of excessively sleepy individuals, such as narcoleptics, from normals. As predicted, the ratio of mean eyes-closed to mean eyes-open alpha power (i.e., the alpha attenuation coefficient) was significantly smaller for narcoleptics than normals, suggesting that increased physiological sleepiness was associated with lower alpha attenuation coefficients. These findings are consistent with
those of Stampi et al. (1993), who observed a decrease in alpha attenuation coefficients in normals throughout 32 hours of sleep deprivation. Studies of experimentally-sleep deprived normals and shiftworkers (e.g., Akerstedt et al., 1985) have demonstrated that during maximal sleepiness, alpha power was lower during eyes-closed conditions than during eyes-open conditions, and that during maximal alertness, alpha power was higher during eyes-closed conditions than during eyes open conditions. In the present study, it was predicted that narcoleptics would demonstrate lower mean eyes-closed alpha power, and higher mean eyes-open alpha power than normals. However, it was found that mean eyes-closed alpha power was significantly reduced in narcoleptics compared to normals, whereas mean eyes-open alpha power did not differ between narcoleptics and normals. It appears that when the eyes were open, the illumination and the task of focusing on a target on the wall may have acted as alerting stimuli for the narcoleptics, enabling the suppression of the latent physiological sleepiness that was observed once they closed their eyes. Thus, the significantly reduced alpha attenuation coefficient in narcoleptics was mediated almost entirely by the eyes-closed condition of the Alpha Attenuation Test. The decreased alpha attenuation coefficient and increased eyes-closed alpha power in narcolepsy mimics effects of shiftwork and experimentally-induced sleep deprivation in normals. However, in the absence of sleep deprivation in the narcoleptics, some neurophysiological mechanism is producing an elevation in the physiological need for sleep in narcoleptics.

The Alpha Attenuation Test versus the Multiple Sleep Latency Test

The alpha attenuation coefficient correlated with latency to stage 1 on the Multiple Sleep Latency Test in only one of 20 subjects, suggesting that physiological sleepiness measured by the Alpha Attenuation Test was not related to that measured by the Multiple Sleep Latency Test. This result is contrary to the findings of Michimori et al. (1993), who demonstrated that the alpha attenuation coefficient correlated with latency to sleep onset on the Multiple Sleep Latency Test in three out of four sleep deprived normal subjects.
However, whereas Michimori et al. (1993) measured latency to sleep onset within minutes of measuring eyes-open and eyes-closed alpha power, in the present study more than 45 minutes separated the two measurements. It is proposed that the Alpha Attenuation Test and Multiple Sleep Latency Test produced relatively independent measures of sleepiness because they were scheduled to begin on alternate hours throughout the day. These tests were not scheduled to run consecutively so as to avoid both sleepiness priming and sleep inertia effects. It was desirable that subjects not become sleepy and fall asleep faster on the Multiple Sleep Latency Test because they had just finished sitting quietly while staring at the wall or closing their eyes, and vice versa.

Subjective sleepiness ratings obtained just prior to or following four of the Alpha Attenuation Test sessions did not to differ significantly between narcoleptics and normals. However, sleepiness ratings obtained just prior to or following three of the Multiple Sleep Latency Test sessions were significantly higher for narcoleptics than normals. This discrepancy may be due in part to the environment in which the sleepiness ratings were obtained. For the Alpha Attenuation Test, sleepiness ratings were obtained while subjects were seated in a chair. By contrast, for the Multiple Sleep Latency Test, sleepiness ratings were obtained while subjects were lying down in bed, in anticipation of or just following a nap opportunity. Thus, in situations which promote sleepiness (i.e., lying down for Multiple Sleep Latency Test versus sitting in a chair for Alpha Attenuation Test), subjective sleepiness ratings were higher for narcoleptics than normals. What is noteworthy is that the Alpha Attenuation Test successfully differentiated the narcoleptics and normals in the absence of group differences in subjective sleepiness ratings.

One of the normal subjects in the present study (RK), presents an interesting case study demonstrating the merit of the Alpha Attenuation Test in the situation of false-positive Multiple Sleep Latency Test results. RK was a normal sleeper (slept on average 6.9 hours each night the week before testing, and 8.7 hours the night before the Multiple Sleep Latency Test), who reported no need to nap during the day. He had no history of
excessive daytime sleepiness, cataplexy, or sleep paralysis, but reported having unusual visual or auditory experiences (i.e., hypnagogic hallucinations) 1-5 times during his lifetime. He was not taking any medications, and nor did he demonstrate symptoms of depression (he scored 2 out of 63 on the Beck Depression Inventory). RK experienced two sleep onset REM periods* on the Multiple Sleep Latency Test, and had a mean latency to stage 1 sleep of 3.6 minutes, suggesting that he met the diagnostic criteria for narcolepsy. However, RK subjectively rated himself as alert throughout the day (mean Stanford Sleepiness Scale = 1.4, mean Visual Analogue Sleepiness Scale = 10.2). Moreover, his Alpha Attenuation Test results gave no indication of excessive sleepiness. On the contrary, they suggested an above average degree of physiological alertness. RK's mean alpha attenuation coefficient (3.1) was higher than the average alpha attenuation coefficient for normals (2.5), as was his mean eyes-closed alpha power (5.9 $\mu V^2$ versus 3.9 $\mu V^2$), and his mean eyes-open alpha power (1.9 $\mu V^2$) was comparable with that obtained in narcoleptics and normals (1.7 $\mu V^2$). Thus, the Alpha Attenuation Test confirmed the presence of a normal level of physiological alertness in a subject matching the Multiple Sleep Latency Test criteria for the diagnosis of narcolepsy.

The implications of the Alpha Attenuation Test in the assessment of daytime sleepiness cannot be understated. It is quick and simple to administer, and is free of the serious limitations associated with the assessment of sleepiness via the Multiple Sleep Latency Test (namely, a floor effect in excessively sleepy populations, the confounding of sleepiness with the ability to fall asleep, and the reliance on the presence of a polysomnographer to sleep score the EEG record 'on line' during each nap opportunity). Although the Multiple Sleep Latency Test is perhaps the best measure for documenting the occurrence of sleep onset REM periods (Mitler et al., 1978), it clearly has the potential

*NOTE: The occurrence of sleep onset REM periods in otherwise normal sleepers has been documented by Rosenthal et al. (1995), who reported that 15% (i.e., 11 of 73) of their drug-free normal sleepers (asymptomatic for narcolepsy) experienced 2 or more sleep onset REM periods on the Multiple Sleep Latency Test.
to produce misleading evaluations of physiological sleepiness (due to the sleepability confound and floor effects).

The present study has documented the ability of the Alpha Attenuation Test to confirm the presence of physiological alertness in a normal subject matching the diagnostic criteria for narcolepsy on the Multiple Sleep Latency Test. Future research is needed in order to test the ability of the Alpha Attenuation Test to assess sleepiness in clinical populations other than narcoleptics. For example, it is of interest whether the Alpha Attenuation Test would identify increased physiological sleepiness in sleep-onset insomniacs (who would have trouble falling asleep on the Multiple Sleep Latency Test), and in other patients complaining of excessive daytime sleepiness (e.g., central and obstructive apneics).

Future research may demonstrate the implications of the Alpha Attenuation Test in the evaluation of pharmacological treatment efficacy for excessive daytime sleepiness. Given that studies of subjectively effective stimulant medications in narcoleptics have failed to demonstrate a reduction in sleepiness on the Multiple Sleep Latency Test, it would be advantageous for future studies to investigate the ability of the Alpha Attenuation Test to detect variations in sleepiness/alertness following clinically effective pharmacological treatment.

The inclusion of the Alpha Attenuation Test in the clinical assessment of patients with sleep-related complaints appears warranted. The Alpha Attenuation Test may easily be accommodated into the Multiple Sleep Latency paradigm by scheduling these two tests to occur on alternate hours throughout the day, as was done in the present study.

Limitations of the Alpha Attenuation Test

The Alpha Attenuation Test may be limited in its ability to assess sleepiness in high alpha producers. Indeed, Stampi et al. (1995) reported in their studies of sleep deprived normals that in individuals producing extremely high alpha attenuation coefficients (labeled 'high alpha producers'), the alpha attenuation coefficient tended not to correlate with
latency to sleep onset on the Multiple Sleep Latency Test. In this study, the Multiple Sleep Latency was administered 5 minutes following the administration of the Alpha Attenuation Test. The 'high alpha producers' demonstrated a high level of alertness on the Alpha Attenuation Test (i.e., high alpha attenuation coefficient), however this degree of alertness apparently was not related to latency to sleep onset on the Multiple Sleep Latency Test. That is, presumably, these individuals were able to fall asleep within the 20-minute nap opportunity. This may reflect more on the sleepability confound of the Multiple Sleep Latency Test rather than on a limitation of the Alpha Attenuation Test.

However, Heitmann et al. (1995) reported in their study of shiftworkers working a nightshift that the alpha attenuation coefficient correlated best with subjective sleepiness measures in individuals producing 'medium'-level alpha attenuation coefficients. This suggests that the Alpha Attenuation Test may be less sensitive to subjective sleepiness in individuals who demonstrate extreme levels of alertness or sleepiness on their alpha attenuation coefficients.

However, in the present study, subjective sleepiness measures administered prior to and following each Alpha Attenuation Test correlated with the alpha attenuation coefficient in just four participants (two narcoleptics and two normals), and no pattern was apparent for persons having higher or lower levels of alpha attenuation coefficients. It is proposed that the subjective sleepiness measures tend to reflect manifest sleepiness, which is influenced by moment-to-moment fluctuations in alerting stimuli within the environment, whereas the alpha attenuation coefficient reflects the underlying or latent physiological sleepiness, and thus the lack of correlation among these measures in some individuals may not present a serious consequence.

**CONCLUSIONS**

The present study is the first to investigate EEG power spectra during sleepiness and the sleep onset period in both normal sleepers and excessively sleepy narcoleptics.
Traditionally, EEG studies of narcoleptics have focused on visually-scored sleep stages and latency to sleep onset in order to differentiate physiological sleepiness in narcoleptics and normals and provide evidence for selective pressures for REM sleep and NREM sleep in narcoleptics. However, the present study provided unique evidence in support of the hypothesis that quantitative and qualitative differences in narcoleptic and normal sleepiness and sleep onset processes may be assessed using computerized analyses of EEG power spectra.

Two strategies of spectral analysis, one stage-based, the other time-based, provided a novel perspective on the current understanding of the microstructure of the sleep onset process in normals and narcoleptics. Analyses of the electrophysiological changes occurring during the transition from wakefulness to stage 1, and across the chronological quartiles of the sleep onset period demonstrated that the technique of EEG power spectral analysis can successfully discriminate sleep onset periods in normals and narcoleptics according to the type of sleep to ensue. In particular, delta power varied in magnitude according to whether a normal sleep onset period progressed into stage 2 sleep, or consisted only of stage 1 sleep. Furthermore, although no spectral evidence was obtained to suggest that the narcoleptic sleep onset period prior to naps containing REM sleep differed qualitatively from the sleep onset period prior to NREM sleep, differences among the EEG power spectra of the narcoleptic sleep onset period and the normal sleep onset period were obtained. Variations in delta power within the sleep onset period were robust when the narcoleptic REM and NREM (stage 2) naps were compared with normal stage 1 and normal stage 2 naps.

Enhanced delta power during the sleep onset period of narcoleptic REM and NREM naps, compared to normal stage 2 and stage 1 naps is a novel finding. It is, however, consistent with past studies of normals (e.g., Borbely et al., 1981) and narcoleptics (e.g., Basset et al., 1994), which have associated increases in delta power during recovery sleep stages 2, 3 and 4 with increased sleepiness elicited by
experimentally-induced sleep deprivation. However, the present study is the first to document spectral differences among normal sleepers and narcoleptics within the sleep onset period of daytime nap opportunities in the absence of experimentally-induced sleep deprivation. These findings have theoretical implications for the understanding of the microstructure of the processes of both normal and narcoleptic sleep onset. Given that sleepiness may be quantified as the tendency to enter a sleep-like state (i.e., the sleep onset period) (Torsvall & Akerstedt, 1987), the present findings also have theoretical implications for the understanding of normal sleepiness and excessive sleepiness in narcoleptics during the intention to fall asleep in the optimal sleep-inducing environment of the Multiple Sleep Latency Test.

Furthermore, the present study is also the first to demonstrate the usefulness of the Alpha Attenuation Test, a measure based on EEG power spectral analysis of alpha during eyes-open and eyes-closed conditions, in the differentiation of normal sleepers and excessively sleepy narcoleptics during seated wakefulness in an illuminated room. Decreased eyes-closed alpha power, and reduced alpha attenuation coefficients were observed in narcoleptics compared to normals. In light of the sleepability confound, floor effects and false-positive results associated with the use of the Multiple Sleep Latency Test, the present findings may implicate the Alpha Attenuation Test as an alternative to the Multiple Sleep Latency Test in the accurate assessment of physiological sleepiness and the evaluation of pharmacological treatment efficacy.

To conclude, the present study is the first to demonstrate that normal sleepers can be distinguished from excessively sleepy narcoleptics on the basis of EEG power spectral analysis of delta power throughout the sleep onset period and alpha power during seated wakefulness.
References


Table 1
Mean latencies and sleep stage percentages for nocturnal sleep in narcoleptics and normals

<table>
<thead>
<tr>
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<th>Mean (SD)</th>
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<tr>
<td></td>
<td>Narcoleptics</td>
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<tr>
<td>Stage 1 latency (min)</td>
<td>7.2 (7.9)</td>
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<tr>
<td>Stage 2 latency (min)</td>
<td>24.6 (12.8)</td>
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<tr>
<td>REM latency (min) (from Stage 1)</td>
<td>26.4 (34.8)</td>
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<tr>
<td>% Stage 1</td>
<td>20.2 (11.2)</td>
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<tr>
<td>% Stage 2</td>
<td>50.3 (8.6)</td>
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<tr>
<td>% Stage 3</td>
<td>6.6 (2.9)</td>
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<td>% Stage 4</td>
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<tr>
<td>% REM sleep</td>
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<tr>
<td>Total sleep time (min)</td>
<td>439.8 (58.8)</td>
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<td>Sleep Efficiency (time asleep/time in bed)</td>
<td>86.0 (9.8)</td>
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Table 2
Number and type of nap selected* for power spectral analysis (stage-based and time-based) from the total number of naps available at each Multiple Sleep Latency Test session for narcoleptics and normals

<table>
<thead>
<tr>
<th>Multiple Sleep Latency Test Session (h)</th>
<th>1000</th>
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<tr>
<td><strong>Narcoleptics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>REM</td>
<td>5/5</td>
<td>2/6</td>
<td>2/6</td>
<td>1/5</td>
<td>0/4</td>
</tr>
<tr>
<td>NREM (stage 2)</td>
<td>4/4</td>
<td>3/4</td>
<td>1/4</td>
<td>0/5</td>
<td>0/6</td>
</tr>
<tr>
<td>NREM (stage 1)</td>
<td>0/1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Normals</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>REM</td>
<td>0</td>
<td>0</td>
<td>0/1</td>
<td>0/1</td>
<td>0</td>
</tr>
<tr>
<td>NREM (stage 2)</td>
<td>6/6</td>
<td>1/7</td>
<td>2/9</td>
<td>0/5</td>
<td>0/3</td>
</tr>
<tr>
<td>NREM (stage 1)</td>
<td>2/2</td>
<td>1/2</td>
<td>0/0</td>
<td>1/4</td>
<td>2/6</td>
</tr>
</tbody>
</table>

*Note:
Because the type of sleep obtained on each nap varied within subjects and groups, the first nap to occur containing just NREM stages 1 and 2, and the first nap to occur containing REM sleep were selected for analysis for each narcoleptic subject. For control subjects, the first nap to occur containing NREM stages 1 and 2, and the first nap to occur containing just NREM stage 1 were selected.
Table 3
Mean latency and sleep stage percentages for the Multiple Sleep Latency Test in narcoleptics and normals

<table>
<thead>
<tr>
<th></th>
<th>Mean (SD)</th>
<th>Multiple Sleep Latency Test Session (h)</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1000</td>
<td>1200</td>
</tr>
<tr>
<td><strong>Stage 1 latency (min)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptics</td>
<td>5.0 (5.3)</td>
<td>3.0 (3.0)</td>
<td>2.8 (2.0)</td>
</tr>
<tr>
<td>Normals</td>
<td>11.1 (7.2)</td>
<td>10.6 (6.3)</td>
<td>8.1 (4.7)</td>
</tr>
<tr>
<td><strong>% Stage 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptics</td>
<td>36.0 (27.0)</td>
<td>34.9 (23.4)</td>
<td>35.3 (20.4)</td>
</tr>
<tr>
<td>Normals</td>
<td>20.5 (19.0)</td>
<td>32.4 (24.0)</td>
<td>32.1 (22.9)</td>
</tr>
<tr>
<td><strong>% Stage 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptics</td>
<td>39.2 (38.1)</td>
<td>37.0 (32.9)</td>
<td>48.5 (27.1)</td>
</tr>
<tr>
<td>Normals</td>
<td>48.0 (35.0)</td>
<td>59.8 (30.3)</td>
<td>56.8 (25.2)</td>
</tr>
<tr>
<td><strong>% Stage 3</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptics</td>
<td>0</td>
<td>4.3 (10.6)</td>
<td>3.3 (7.2)</td>
</tr>
<tr>
<td>Normals</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>% Stage 4</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptics</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Normals</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>% REM sleep over all naps</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptics</td>
<td>24.5 (31.1)</td>
<td>24.4 (28.3)</td>
<td>12.9 (14.7)</td>
</tr>
<tr>
<td>Normals</td>
<td>0</td>
<td>0</td>
<td>1.1 (3.5)</td>
</tr>
<tr>
<td><strong>% REM sleep on REM-containing naps</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptics</td>
<td>49.0 (26.0)</td>
<td>48.9 (17.5)</td>
<td>25.8 (8.3)</td>
</tr>
<tr>
<td>Normals</td>
<td>0</td>
<td>0</td>
<td>11.1 (RK)</td>
</tr>
<tr>
<td><strong>Total sleep (min)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptics</td>
<td>15.3 (1.0)</td>
<td>15.1 (0.4)</td>
<td>15.1 (0.2)</td>
</tr>
<tr>
<td>Normals</td>
<td>10.8 (7.5)</td>
<td>14.2 (4.8)</td>
<td>15.9 (1.2)</td>
</tr>
<tr>
<td><strong>Total wake after sleep onset (min)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptics</td>
<td>0.4 (0.5)</td>
<td>0.1 (0.2)</td>
<td>0.1 (0.2)</td>
</tr>
<tr>
<td>Normals</td>
<td>0.5 (0.8)</td>
<td>1.2 (1.7)</td>
<td>0.3 (0.4)</td>
</tr>
</tbody>
</table>

**Note:**
* Significant group effect.  + Significant session effect.  ^ Significant group by session interaction.  [p < .05]
The normal subject RK experienced two sleep onset REM periods.
Table 4
Mean power in delta, theta, sigma and beta bands at Cz and alpha at O2 during wakefulness and stage 1 in narcoleptics and normals, collapsing over nap type

<table>
<thead>
<tr>
<th></th>
<th>Mean Power (μV²) (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Wakefulness</td>
</tr>
<tr>
<td><strong>Cz Delta</strong></td>
<td></td>
</tr>
<tr>
<td>Narcoleptics</td>
<td>4.3 (1.4)</td>
</tr>
<tr>
<td>Normals</td>
<td>3.2 (0.8)</td>
</tr>
<tr>
<td><strong>Cz Theta</strong></td>
<td></td>
</tr>
<tr>
<td>Narcoleptics</td>
<td>3.1 (1.5)</td>
</tr>
<tr>
<td>Normals</td>
<td>2.2 (1.2)</td>
</tr>
<tr>
<td><strong>O2 Alpha</strong></td>
<td></td>
</tr>
<tr>
<td>Narcoleptics</td>
<td>3.3 (1.3)</td>
</tr>
<tr>
<td>Normals</td>
<td>3.9 (1.7)</td>
</tr>
<tr>
<td><strong>Cz Sigma</strong></td>
<td></td>
</tr>
<tr>
<td>Narcoleptics</td>
<td>0.6 (0.2)</td>
</tr>
<tr>
<td>Normals</td>
<td>0.7 (0.2)</td>
</tr>
<tr>
<td><strong>Cz Beta</strong></td>
<td></td>
</tr>
<tr>
<td>Narcoleptics</td>
<td>1.2 (0.5)</td>
</tr>
<tr>
<td>Normals</td>
<td>1.3 (0.6)</td>
</tr>
</tbody>
</table>

*Note:
* Significant main effects of group (i.e., collapsing across stage) were found for mean delta power [F(1,32) = 11.24, p = .002] and mean sigma power [F(1,32) = 4.88, p = .035].
Table 5
Mean power for delta, theta, sigma and beta at Cz and alpha at O2 during wakefulness and stage 1 in selected NREM (stage 2), NREM (stage 1) and REM naps in narcoleptics and normals

<table>
<thead>
<tr>
<th></th>
<th>Mean Power (μV²) (SD)</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stage</td>
<td>Wakefulness</td>
<td>Stage 1</td>
</tr>
<tr>
<td><strong>Cz Delta</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptic REM</td>
<td>4.4 (1.7)</td>
<td>6.6 (2.1)</td>
<td>5.5~</td>
</tr>
<tr>
<td>Narcoleptic NREM (Stage 2)</td>
<td>4.3 (1.0)</td>
<td>7.0 (2.4)</td>
<td>5.7^ #</td>
</tr>
<tr>
<td>Normal NREM (Stage 2)</td>
<td>3.2 (0.9)</td>
<td>5.5 (0.9)</td>
<td>4.4</td>
</tr>
<tr>
<td>Normal NREM (Stage 1)</td>
<td>3.1 (0.7)</td>
<td>4.0 (1.2)</td>
<td>3.6&gt;</td>
</tr>
<tr>
<td><strong>Cz Theta</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptic REM</td>
<td>2.9 (1.1)</td>
<td>2.8 (0.9)</td>
<td>2.8~</td>
</tr>
<tr>
<td>Narcoleptic NREM (Stage 2)</td>
<td>3.3 (2.0)</td>
<td>3.0 (1.2)</td>
<td>3.2</td>
</tr>
<tr>
<td>Normal NREM (Stage 2)</td>
<td>2.4 (1.4)</td>
<td>2.5 (1.0)</td>
<td>2.5</td>
</tr>
<tr>
<td>Normal NREM (Stage 1)</td>
<td>1.8 (0.4)</td>
<td>2.1 (0.6)</td>
<td>1.9</td>
</tr>
<tr>
<td><strong>O2 Alpha</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptic REM</td>
<td>3.5 (1.4)</td>
<td>1.4 (0.5)</td>
<td>2.5</td>
</tr>
<tr>
<td>Narcoleptic NREM (Stage 2)</td>
<td>3.0 (1.2)</td>
<td>1.4 (0.5)</td>
<td>2.2</td>
</tr>
<tr>
<td>Normal NREM (Stage 2)</td>
<td>4.0 (1.8)</td>
<td>1.4 (0.5)</td>
<td>2.7</td>
</tr>
<tr>
<td>Normal NREM (Stage 1)</td>
<td>3.8 (1.7)</td>
<td>2.0 (0.9)</td>
<td>2.9</td>
</tr>
<tr>
<td><strong>Cz Sigma</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptic REM</td>
<td>0.5 (0.2)</td>
<td>0.4 (0.1)</td>
<td>0.5~</td>
</tr>
<tr>
<td>Narcoleptic NREM (Stage 2)</td>
<td>0.6 (0.2)</td>
<td>0.5 (0.2)</td>
<td>0.5</td>
</tr>
<tr>
<td>Normal NREM (Stage 2)</td>
<td>0.7 (0.2)</td>
<td>0.5 (0.1)</td>
<td>0.6</td>
</tr>
<tr>
<td>Normal NREM (Stage 1)</td>
<td>0.7 (0.2)</td>
<td>0.6 (0.1)</td>
<td>0.6</td>
</tr>
<tr>
<td><strong>Cz Beta</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptic REM</td>
<td>1.5 (0.4)</td>
<td>0.9 (0.4)</td>
<td>1.2</td>
</tr>
<tr>
<td>Narcoleptic NREM (Stage 2)</td>
<td>1.4 (0.5)</td>
<td>0.9 (0.3)</td>
<td>1.1</td>
</tr>
<tr>
<td>Normal NREM (Stage 2)</td>
<td>1.3 (0.6)</td>
<td>0.9 (0.2)</td>
<td>1.1</td>
</tr>
<tr>
<td>Normal NREM (Stage 1)</td>
<td>1.3 (0.6)</td>
<td>1.0 (0.3)</td>
<td>1.1</td>
</tr>
</tbody>
</table>

**Note:**
Significant main effects of group (i.e., collapsing across stage) were as follows: (p < .05)
~ narcoleptic REM naps vs. control NREM (stage 1) naps
^ narcoleptic NREM naps vs. control NREM (stage 2) naps
# narcoleptic NREM naps vs. control NREM (stage 1) naps
> control NREM (stage 1) vs. control NREM (stage 2) naps
Table 6
Mean power and proportion of slope changes in delta, theta, sigma and beta bands at Cz and alpha bands at O2 throughout the chronological quartiles of the sleep onset period in selected NREM (stage 2), NREM (stage 1) and REM naps in narcoleptics and normals, collapsing over naptype

<table>
<thead>
<tr>
<th></th>
<th>Mean Power (μV²) (SD)</th>
<th>Mean Proportion of Slope Changes (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Quartile 1 2 3 4</td>
<td>Quartile 1 2 3 4</td>
</tr>
<tr>
<td><strong>Cz Delta</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptic</td>
<td>5.5 (2.2) 7.1 (2.0)</td>
<td>.27 (.12) .32 (.12) .43 (.10) .45 (.15)</td>
</tr>
<tr>
<td>Normal</td>
<td>3.7 (0.8) 4.3 (1.1)</td>
<td>.29 (.14) .36 (.12) .39 (.09) .50 (.08)</td>
</tr>
<tr>
<td><strong>Cz Theta</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptic</td>
<td>3.0 (1.4) 2.5 (1.0)</td>
<td>.44 (.15) .42 (.08) .46 (.11) .46 (.12)</td>
</tr>
<tr>
<td>Normal</td>
<td>1.9 (1.0) 2.1 (1.0)</td>
<td>.39 (.13) .44 (.08) .42 (.07) .52 (.09)</td>
</tr>
<tr>
<td><strong>O2 Alpha</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptic</td>
<td>2.7 (1.1) 1.6 (1.0)</td>
<td>.41 (.15) .34 (.15) .31 (.18) .25 (.15)</td>
</tr>
<tr>
<td>Normal</td>
<td>3.6 (1.6) 2.6 (1.6)</td>
<td>.42 (.08) .36 (0.9) .32 (.16) .32 (.18)</td>
</tr>
<tr>
<td><strong>Cz Sigma</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptic</td>
<td>0.5 (0.2) 0.4 (0.2)</td>
<td>.35 (.16) .44 (.13) .39 (.14) .44 (.11)</td>
</tr>
<tr>
<td>Normal</td>
<td>0.6 (0.1) 0.6 (0.1)</td>
<td>.36 (.09) .41 (.09) .42 (.11) .47 (.10)</td>
</tr>
<tr>
<td><strong>Cz Beta</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptic</td>
<td>1.1 (0.4) 0.9 (0.4)</td>
<td>.40 (.12) .45 (.10) .40 (.14) .42 (.16)</td>
</tr>
<tr>
<td>Normal</td>
<td>1.2 (0.4) 1.1 (0.4)</td>
<td>.41 (.06) .42 (.10) .43 (.14) .43 (.12)</td>
</tr>
</tbody>
</table>

* Significant main effects of group (i.e., collapsing across quartiles) were found for mean delta power \([F(1,32) = 19.08, p = .001]\) and mean alpha power \([F(1,32) = 7.51, p = .010]\).
Table 7
Mean power in delta, theta, sigma and beta bands at Cz and alpha bands at O2 throughout the chronological quartiles of the sleep onset period in selected NREM (stages 1 and 2), NREM (stage 1) and REM naps in narcoleptics and normals

<table>
<thead>
<tr>
<th></th>
<th>Mean Power (μV²) (SD)</th>
<th></th>
<th></th>
<th></th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Quartile</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td><strong>Cz Delta</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptic REM</td>
<td>5.5 (2.3)</td>
<td>7.4 (2.0)</td>
<td>7.8 (1.2)</td>
<td>8.1 (3.3)</td>
<td>7.2+ ~</td>
</tr>
<tr>
<td>Narcoleptic NREM (Stage 2)</td>
<td>5.6 (2.3)</td>
<td>6.7 (2.1)</td>
<td>9.8 (3.6)</td>
<td>10.6 (3.3)</td>
<td>8.2^ #</td>
</tr>
<tr>
<td>Normal NREM (Stage 2)</td>
<td>3.8 (1.0)</td>
<td>4.5 (1.0)</td>
<td>6.1 (1.7)</td>
<td>8.0 (1.2)</td>
<td>5.6</td>
</tr>
<tr>
<td>Normal NREM (Stage 1)</td>
<td>3.6 (2.0)</td>
<td>4.0 (1.2)</td>
<td>3.6 (0.7)</td>
<td>4.0 (1.1)</td>
<td>3.8&gt;</td>
</tr>
<tr>
<td><strong>Cz Theta</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptic REM</td>
<td>3.0 (1.3)</td>
<td>2.4 (0.8)</td>
<td>2.6 (0.6)</td>
<td>2.3 (0.7)</td>
<td>2.6~</td>
</tr>
<tr>
<td>Narcoleptic NREM (Stage 2)</td>
<td>3.0 (1.6)</td>
<td>2.7 (1.3)</td>
<td>2.7 (0.9)</td>
<td>2.9 (0.8)</td>
<td>2.8</td>
</tr>
<tr>
<td>Normal NREM (Stage 2)</td>
<td>2.1 (1.3)</td>
<td>2.2 (1.2)</td>
<td>2.4 (0.9)</td>
<td>2.6 (0.7)</td>
<td>2.3</td>
</tr>
<tr>
<td>Normal NREM (Stage 1)</td>
<td>1.6 (0.2)</td>
<td>2.0 (0.7)</td>
<td>1.8 (0.4)</td>
<td>2.0 (0.7)</td>
<td>1.8</td>
</tr>
<tr>
<td><strong>O2 Alpha</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptic REM</td>
<td>2.8 (1.2)</td>
<td>1.6 (1.2)</td>
<td>1.3 (0.7)</td>
<td>1.0 (0.4)</td>
<td>1.7~</td>
</tr>
<tr>
<td>Narcoleptic NREM (Stage 2)</td>
<td>2.7 (0.9)</td>
<td>1.5 (0.8)</td>
<td>1.1 (0.4)</td>
<td>1.1 (0.3)</td>
<td>1.6#</td>
</tr>
<tr>
<td>Normal NREM (Stage 2)</td>
<td>3.6 (1.6)</td>
<td>2.1 (1.0)</td>
<td>1.5 (0.9)</td>
<td>1.2 (0.5)</td>
<td>2.1</td>
</tr>
<tr>
<td>Normal NREM (Stage 1)</td>
<td>3.6 (1.8)</td>
<td>3.5 (2.0)</td>
<td>3.2 (1.4)</td>
<td>3.5 (1.6)</td>
<td>3.5&gt;</td>
</tr>
<tr>
<td><strong>Cz Sigma</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptic REM</td>
<td>0.5 (0.2)</td>
<td>0.4 (0.1)</td>
<td>0.4 (0.3)</td>
<td>0.4 (0.2)</td>
<td>0.4~</td>
</tr>
<tr>
<td>Narcoleptic NREM (Stage 2)</td>
<td>0.5 (0.1)</td>
<td>0.5 (0.2)</td>
<td>0.6 (0.2)</td>
<td>0.9 (0.5)</td>
<td>0.6</td>
</tr>
<tr>
<td>Normal NREM (Stage 2)</td>
<td>0.6 (0.1)</td>
<td>0.5 (0.1)</td>
<td>0.5 (0.1)</td>
<td>0.7 (0.2)</td>
<td>0.6</td>
</tr>
<tr>
<td>Normal NREM (Stage 1)</td>
<td>0.7 (0.1)</td>
<td>0.6 (0.1)</td>
<td>0.7 (0.3)</td>
<td>0.6 (0.2)</td>
<td>0.7</td>
</tr>
<tr>
<td><strong>Cz Beta</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Narcoleptic REM</td>
<td>1.0 (0.4)</td>
<td>0.8 (0.4)</td>
<td>0.8 (0.4)</td>
<td>0.7 (0.4)</td>
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<tr>
<td>Narcoleptic NREM (Stage 2)</td>
<td>1.2 (0.4)</td>
<td>1.0 (0.5)</td>
<td>0.8 (0.3)</td>
<td>0.7 (0.3)</td>
<td>0.9</td>
</tr>
<tr>
<td>Normal NREM (Stage 2)</td>
<td>1.2 (0.5)</td>
<td>1.0 (0.3)</td>
<td>0.9 (0.3)</td>
<td>0.7 (0.2)</td>
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<tr>
<td>Normal NREM (Stage 1)</td>
<td>1.2 (0.4)</td>
<td>1.2 (0.4)</td>
<td>1.2 (0.4)</td>
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<td>1.2</td>
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</table>

**Note:**
Significant main effects of group (i.e., collapsing across quartiles) were as follows: (p < .05)
~ narcoleptic REM naps vs. control NREM (stage 1) naps
^ narcoleptic NREM naps vs. control NREM (stage 2) naps
# narcoleptic NREM naps vs. control NREM (stage 1) naps
> control NREM (stage 1) vs. control NREM (stage 2) naps
Table 8
Mean proportion of slope changes in delta, theta, sigma and beta bands at Cz and alpha bands at O2 throughout the chronological quartiles of the sleep onset period in selected NREM (stages 1 and 2), NREM (stage 1) and REM naps in narcoleptics and normals

<table>
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<tr>
<th></th>
<th>Mean Proportion of Slope Changes (SD)</th>
<th>Quartile</th>
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<th>2</th>
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<th>4</th>
<th>Mean</th>
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<tr>
<td><strong>Cz Delta</strong></td>
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<tr>
<td>Narcoleptic REM</td>
<td>.30 (.13)</td>
<td>.36 (.11)</td>
<td>.46 (.10)</td>
<td>.44 (.16)</td>
<td>.39</td>
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<tr>
<td>Narcoleptic NREM (Stage 2)</td>
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<td>.26 (.11)</td>
<td>.39 (.11)</td>
<td>.47 (.15)</td>
<td>.33#</td>
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<tr>
<td>Normal NREM (Stage 2)</td>
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<td>.30 (.09)</td>
<td>.38 (.07)</td>
<td>.50 (.08)</td>
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<tr>
<td>Normal NREM (Stage 1)</td>
<td>.40 (.09)</td>
<td>.46 (.07)</td>
<td>.41 (.12)</td>
<td>.49 (.08)</td>
<td>.44&gt;</td>
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<tr>
<td><strong>Cz Theta</strong></td>
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<td>Narcoleptic REM</td>
<td>.43 (.17)</td>
<td>.41 (.09)</td>
<td>.50 (.11)</td>
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<td>.41 (.11)</td>
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<tr>
<td>Normal NREM (Stage 2)</td>
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<td>.41 (.09)</td>
<td>.41 (.06)</td>
<td>.51 (.09)</td>
<td>.43</td>
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</tr>
<tr>
<td>Normal NREM (Stage 1)</td>
<td>.41 (.08)</td>
<td>.48 (.06)</td>
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<td><strong>O2 Alpha</strong></td>
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<td>Narcoleptic REM</td>
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<td>.35 (.14)</td>
<td>.33 (.16)</td>
<td>.28 (.17)</td>
<td>.35</td>
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<td>.29 (.21)</td>
<td>.20 (.12)</td>
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<td>.23 (.15)</td>
<td>.21 (.14)</td>
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<tr>
<td>Normal NREM (Stage 1)</td>
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<td>.42 (.05)</td>
<td>.47 (.05)</td>
<td>.51 (.06)</td>
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<td>Narcoleptic REM</td>
<td>.40 (.15)</td>
<td>.46 (.15)</td>
<td>.41 (.17)</td>
<td>.44 (.12)</td>
<td>.43</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcoleptic NREM (Stage 2)</td>
<td>.28 (.15)</td>
<td>.42 (.12)</td>
<td>.37 (.11)</td>
<td>.44 (.09)</td>
<td>.38</td>
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<tr>
<td>Normal NREM (Stage 2)</td>
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<td>.39 (.11)</td>
<td>.46 (.11)</td>
<td>.39</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal NREM (Stage 1)</td>
<td>.43 (.05)</td>
<td>.47 (.09)</td>
<td>.48 (.09)</td>
<td>.50 (.08)</td>
<td>.47&gt;</td>
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<td><strong>Cz Beta</strong></td>
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<tr>
<td>Narcoleptic REM</td>
<td>.39 (.40)</td>
<td>.44 (.09)</td>
<td>.42 (.14)</td>
<td>.43 (.06)</td>
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<td></td>
<td></td>
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<tr>
<td>Narcoleptic NREM (Stage 2)</td>
<td>.40 (.10)</td>
<td>.46 (.11)</td>
<td>.37 (.16)</td>
<td>.41 (.24)</td>
<td>.41</td>
<td></td>
<td></td>
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<tr>
<td>Normal NREM (Stage 2)</td>
<td>.40 (.06)</td>
<td>.40 (.12)</td>
<td>.41 (.11)</td>
<td>.37 (.11)</td>
<td>.39</td>
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<tr>
<td>Normal NREM (Stage 1)</td>
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<td>.45 (.06)</td>
<td>.52 (.07)</td>
<td>.47&gt;</td>
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Note:
Significant main effects of group (i.e., collapsing across quartiles) were as follows: (p < .05)
# narcoleptic NREM naps vs. control NREM (stage 1) naps
> control NREM (stage 1) vs. control NREM (stage 2) naps
Table 9
Mean for eyes-open and eyes-closed alpha power at O2 and ratio of mean eyes-closed to eyes-open alpha power (alpha attenuation coefficient) at each Alpha Attenuation Test session in narcoleptics and normals

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<tr>
<th></th>
<th>Mean (SD)</th>
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<td>Alpha Attenuation Test Session (h)</td>
<td>0900</td>
<td>1100</td>
<td>1300</td>
<td>1500</td>
<td>1700</td>
<td>Mean</td>
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<tr>
<td><strong>Eyes-closed alpha power (μV²)</strong></td>
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<tr>
<td>Narcoleptics</td>
<td>2.3 (0.7)</td>
<td>2.3 (0.9)</td>
<td>2.2 (1.1)</td>
<td>2.4 (0.9)</td>
<td>2.6 (1.3)</td>
<td>2.4*</td>
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<tr>
<td>Normals</td>
<td>3.8 (1.8)</td>
<td>3.8 (1.7)</td>
<td>4.0 (1.9)</td>
<td>3.9 (1.9)</td>
<td>3.9 (1.8)</td>
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</tr>
<tr>
<td><strong>Eyes-open alpha power (μV²)</strong></td>
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<tr>
<td>Narcoleptics</td>
<td>1.7 (0.7)</td>
<td>1.7 (0.6)</td>
<td>1.5 (0.5)</td>
<td>1.9 (0.6)</td>
<td>1.9 (0.8)</td>
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<td>Normals</td>
<td>1.5 (06)</td>
<td>1.7 (0.9)</td>
<td>1.8 (0.8)</td>
<td>1.9 (0.8)</td>
<td>1.8 (0.8)</td>
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<tr>
<td><strong>Alpha attenuation coefficient</strong></td>
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<tr>
<td>Narcoleptics</td>
<td>1.6 (0.8)</td>
<td>1.4 (0.7)</td>
<td>1.5 (0.7)</td>
<td>1.3 (0.5)</td>
<td>1.4 (0.6)</td>
<td>1.4*</td>
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<tr>
<td>Normals</td>
<td>2.9 (1.6)</td>
<td>2.6 (1.8)</td>
<td>2.4 (1.2)</td>
<td>2.1 (1.0)</td>
<td>2.3 (1.2)</td>
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</tbody>
</table>

**Note:**
* Significant main effects of group (i.e., collapsing across testing session) were found for mean eyes-closed alpha power \(F(1, 18) = 6.25, p = .022\) and the mean alpha attenuation coefficient \(F(1, 18) = 4.97, p = .039\).
Table 10
Mean for eyes-open and eyes-closed alpha power at O2 within the 9-10 Hz, 10-11 Hz and 11-12 Hz bands for narcoleptics and normals collapsed across Alpha Attenuation Test session

<table>
<thead>
<tr>
<th>Mean Power (μV^2) (SD)</th>
<th>Eyes-closed</th>
<th>Eyes-open</th>
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<td>Alpha Attenuation Test Condition</td>
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<tr>
<td>8-9 Hz Band</td>
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<tr>
<td>Narcoleptics</td>
<td>0.80 (0.1)</td>
<td>0.61 (0.1)</td>
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<tr>
<td>Normals</td>
<td>1.01 (0.1)</td>
<td>0.49 (0.1)</td>
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<tr>
<td>9-10 Hz Band</td>
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<tr>
<td>Narcoleptics</td>
<td>0.80 (0.1)</td>
<td>0.53 (0.1)</td>
</tr>
<tr>
<td>Normals</td>
<td>1.32 (0.1)</td>
<td>0.48 (0.1)</td>
</tr>
<tr>
<td>10-11 Hz Band</td>
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</tr>
<tr>
<td>Narcoleptics</td>
<td>0.51 (0.1)</td>
<td>0.39 (0.1)</td>
</tr>
<tr>
<td>Normals</td>
<td>0.99 (0.1)</td>
<td>0.47 (0.1)</td>
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<tr>
<td>11-12 Hz Band</td>
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<tr>
<td>Narcoleptics</td>
<td>0.25 (0.1)</td>
<td>0.24 (0.1)</td>
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<tr>
<td>Normals</td>
<td>0.57 (0.1)</td>
<td>0.33 (0.1)</td>
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</table>
AS A FUNCTION OF TIME OF DAY
LATENCY TO STAGE 1 ON MSLT IN NARCOTIC AS AND NORMALS

Figure 1.
Figure 2:

Narcoleptics and Normals as a Function of Time of Day

Total Sleep Time and Wake Time After Sleep Onset for Narcoleptics ———— Sleep Time for Normals ———— Wake Time After Sleep Onset for Narcoleptics ———— Wake Time for Normals

TIME (hr)

TIME (min)
Figure 3: Delta power during wakefulness and stage 1 for selected naps (collapsing over naptypes) in normals and normals who were significantly different from normals.
STAGE

Wakefulness

Stage 1

THETA POWER (squared µV)

(Normal) vs. (Narcoleptic)

(Collapsing over NAPs)

IN NARCOLEPTICS AND NORMALS

THETA POWER DURING WAKEFULNESS AND STAGE 1 FOR SELECTED NAPS

Figure 4:
Figure 5: Sigma Power during waking wakefulness and stage 1 for selected NAPS (collapsing over naphyplex) in narcoleptics and normals.
**Figure 6:** Delta Power During Wakefulness and Stage 1 for Narcolicptic NREM and REM NAPS, and Normal NREM NAPS.
NARCOLEPTIC NREM AND REM NAPS AND NORMAL NREM NAPS

THETA POWER DURING WAKEFULNESS AND STAGE 1 FOR STAGE 1

Figure 7:

STAGE

Wakenlessness

Stage 1

0

0.5

1

1.5

2

2.5

3

3.5

4

THETA POWER (squared µV)

Narcoleptic (REM)

Narcoleptic (stage 2)

Normal (stage 1)

Normal (stage 2)
Sigma Power during wakefulness and stage 1 for narcotic NREM and REM NAPS, and normal NREM NAPS.
Figure 6: Delta power across quartiles of sleep onset period (collapsing over nap type) for selected naps in narcoleptics and normals.
QUARTILES OF SLEEP ONSET PERIOD

(Collapsing over NAP Type)

For selected NAPS in Narcoleptic and Normals

Theta Power Across Quartiles of Sleep Onset Period
Figure 1.1: Alpha power across quartiles of sleep onset period (collapsing over NAP type) for selected naps in narcoleptic and normals. Quartiles of Quartile 4 Quartile 3 Quartile 2 Quartile 1

Alpha power (squared \(\mu\)V)
Quartiles of Sleep Onset Period

Proportion of Theta Slope Changes across Quartiles of Sleep Onset Period for Selected NAPS in Narcoleptic and Normals (Collapsing over NAP Type)
Figure 13: Delta power across quartiles of sleep onset period for narcoleptic NREM and REM naps, and normal NREM naps.
Guttiles of Sleep Onset Period

Quartile 1
Quartile 2
Quartile 3
Quartile 4

ALPHA POWER (squared µV)

Narcoleptic NREM and REM NAPS, and Normal NREM NAPS

Alpha power across quartiles of sleep onset period for Narcoleptic NREM and REM NAPS, and Normal NREM NAPS.
Fig 15: Sigma power across quartiles of sleep onset period for narcoleptic REM and REM naps, and normal REM naps.
Deltage slope changes across quartiles of sleep onset

Figure 16:
Quartiles of Sleep Onset Period

PROPORTION OF ALPHA SLOPE CHANGES

PERIOD FOR SELECTED NARCOLEPTIC AND NORMAL NAPS

ALPHA SLOPE CHANGES ACROSS QUARTILES OF SLEEP ONSET

Figure 7
Figure 16: Graph showing the proportion of sigma slope changes across quartiles of sleep onset period for selected narcoleptic and normal naps.
Quartiles of Sleep Onset Period

Proportion of Beta Slope Changes

Period for Selected Narcoleptic and Normal Naps

Beta Slope Changes Across Quartiles of Sleep Onset
Quartiles of Sleep Onset Period

Proportion of Theta Slope Changes

Period for Selected Narcoleptic and Normal NAPS

Theta Slope Changes Across Quartiles of Sleep Onset
Figure 21: Theta power across quartiles of sleep onset period for NARCOLEPTIC NREM AND REM NAPS, AND NORMAL NREM NAPS.
NARCOLEPTICS AND NORMALS AS A FUNCTION OF TIME OF DAY
MEAN EYES-OPEN AND EYES-CLOSED ALPHA POWER IN

Figure 22:
Normals as a Function of Time of Day

Alpha Attenuation Coefficient (AAC) in Narcotics and Narcoleptics

Figure 23:
Appendices
I, _______________________, agree to spend twenty-three hours in the Sleep Research Unit at the Toronto Hospital (Western Division) (416-369-5723). I have been fully oriented to the procedure and understand the following points:

(1) I understand that I will be required to complete a screening questionnaire, a depression inventory, and a morningness-eveningness questionnaire prior to participation.

(2) I agree to keep a log of my sleep and wake times, to maintain a regular bedtime and rise-time, and to periodically rate my level of daytime sleepiness throughout the 7 days leading up to my stay in the Sleep Research Unit.

(3) I agree to refrain from using non-prescription drugs, including alcohol and caffeine, for the 24-hour period prior to my stay in the Sleep Research Unit, and I agree to refrain from using sleep-related prescription drugs for a duration of 1 week prior to my stay in the Sleep Research Unit.

(4) I understand that I will be required to report to the Sleep Research Unit at 9:30 p.m. (date to be specified), for a 21-hour period.

(5) I have been informed that electrodes will be placed on my head, by my eyes, behind my ears, and under my chin. I have been informed that data from the electrodes will be recorded on tape, paper and computer.

(6) I have been told that I will be required to undergo the following procedure while in the Sleep Research Unit:

(a) At approximately 11 p.m., I will retire to bed for the night. I will be awakened at 8 a.m. the next morning.

(b) I will be asked to take naps at 10 a.m., 12 p.m., 2 p.m., 4 p.m., and 6 p.m..

(c) At the start of each nap I will be asked to lie down with my eyes closed in a darkened room, and try to fall asleep while maintaining a constant pressure with my preferred thumb on a 'deadman switch'. Each nap will end after I have been asleep for 15 minutes, or after I have laid in bed for 20 minutes without falling asleep.

(d) I will be asked to sit quietly with my eyes alternately open and closed for a total of 8 minutes at 9 a.m., 11 a.m., 1 p.m., 3 p.m., and 5 p.m.

(e) Approximately every 30 minutes, I will be asked to rate my level of sleepiness on two short scales.
(7) I have been informed that an experimenter will be present in the Sleep Research Unit at all times if anything is needed throughout the study.

(8) I have been informed that I will a $50 honorarium for participation in this study.

(9) I understand that I may withdraw from this study at any time without prejudice. In addition, I may withdraw any information that will be collected about me during this study.

(10) I understand that all data collected from me during this study will be seen only by the investigators concerned, and will be regarded as confidential. I understand that the results reported from my data will in no way identify me personally as a participant.

I have read and understood the above statement, and I freely consent to participate in this research.

Signature: ________________________________
Name (printed): __________________________
Date: ________________
Address: ________________________________
Telephone: ______________________________

I have explained the nature of this study to the participant and believe that s/he understands it.

Signature of Investigator: ____________________

Investigators:
Christi Alloway, B.Sc.
Robert Ogilvie, Ph.D.
Colin Shapiro, Ph.D.
Appendix B-1

Brock University Sleep Laboratory
Screening Questionnaire
Multiple Daytime Nap Study 1994

Name: ____________________________________________________________

Address: __________________________________________________________

______________________________________________________________

Home Phone: _________  Business Phone: _________

Age: _____  Sex: ___  Date: ________________

1. How would you rate your daytime level of alertness?

___ Mainly alert.  ___ Continual sleepiness.
___ Alertness mixed with some periods of sleepiness.
___ Sleepiness mixed with some periods of alertness.

At what age did your daytime sleepiness become excessive? ________.

2. Please place a slash mark (i.e., /) on the line below, to indicate how alert you typically feel throughout the day.

Wide awake. ________________________________ Exhausted. Too sleepy to function.
Refeshed, ________________________________ energetic.

3. Do you take voluntary naps throughout the day?

___ I have no need to nap.
___ I want to take a nap, but cannot sleep.
___ I take naps twice a week or less.
___ I take naps on 3-5 days per week.
___ I take naps daily, or almost daily.
___ I take several naps per day.

How long do your naps typically last? ________.
Do you wake feeling relatively alert? ________.
4. How often do you stop an activity because of an irresistible need to sleep?
   ___ Never. ___ Sometimes. ___ Often.

5. Do you fall asleep *unintentionally* during the day while engaged in any of the following activities?

<table>
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<th>Activity</th>
<th>Never</th>
<th>Monthly or less</th>
<th>Weekly</th>
<th>Daily</th>
<th>Several times daily</th>
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<td>reading</td>
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<td>eating</td>
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<td>watching t.v.</td>
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<td>social activity</td>
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<td>during talks or lectures</td>
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<td>in movie theater</td>
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<tr>
<td>travelling</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>in a car</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>standing</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>other activities</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Do you wake feeling relatively alert? ____.
6. When laughing, becoming glad or angry, or in an exciting situation have you experienced the following symptoms?

<table>
<thead>
<tr>
<th>Manifestation</th>
<th>Never</th>
<th>1-5 times during lifetime</th>
<th>Monthly</th>
<th>Weekly</th>
<th>Daily/ almost daily</th>
</tr>
</thead>
<tbody>
<tr>
<td>Falling down</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knees sagging</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mouth opening</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Head nodding</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

How long do these symptoms last? ___________.
At what age did these symptoms first appear? _____.

7. When falling asleep, or waking up have you ever felt unable to move or speak?

___ Never.
___ 1-5 times during lifetime.
___ Monthly.
___ Weekly.
___ Daily or almost daily.

At what age did these experiences first begin? _____.

8. Have you ever had unusual experiences when falling asleep or waking up; that is, have you seen, heard or felt something that did not really exist?

___ Never.
___ 1-5 times during lifetime.
___ Monthly.
___ Weekly.
___ Daily or almost daily.

At what age did these experiences first begin? _____.

9. Each day I usually drink ____ cups of caffeinated coffee.
Each day I usually drink ____ cups of tea.
Each week I usually drink ____ glasses of cola.
Each week I usually drink ____ glasses of wine.
Each week I usually drink ____ bottles of beer.
Each week I usually drink ____ ounces of liquor.

Each day I usually smoke ____ cigarettes.
Each day I usually smoke ____ pipes.
Each day I usually smoke ____ cigars.

10. How often do you use the following drugs:

<table>
<thead>
<tr>
<th>Drug Description</th>
<th>Never</th>
<th>Sometimes</th>
<th>Often</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marijuana</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cocaine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LSD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stimulants (Upplers, speed)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcotics (morphine, heroin, opium)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

11. How fast do you usually fall asleep at night?

____ Longer than 40 minutes.
____ 31-40 minutes.
____ 21-30 minutes.
____ 10-20 minutes.
____ 5-10 minutes.
____ Less than 5 minutes.

12. How many times do you usually wake up during the night?

____ I usually do not wake up during the night.
____ I usually wake up 1-3 times during the night.
____ I usually wake up 4-6 times during the night.
____ I usually wake up more than 6 times during the night.

If you awaken at night, how often do you stay awake more than 30 minutes before you go to sleep again?

____ Never. ____ Sometimes. ____ Often. ____ Always.
13. How often would you describe your sleep at night as being:

<table>
<thead>
<tr>
<th>Sleep Quality</th>
<th>Never</th>
<th>Sometimes</th>
<th>Often</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>Light</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deep</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

14. How often do you work the following schedules?

<table>
<thead>
<tr>
<th></th>
<th>Never</th>
<th>Sometimes</th>
<th>Often</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day shift</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Evening shift</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Night shift</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

15. Have you been diagnosed with a sleep disorder?
   ____ No.    ____ Yes.

   Please specify:
   ____________________________________________________________
   ____________________________________________________________
   ____________________________________________________________
   ____________________________________________________________

16. Are you HLA-DR2 positive?
   ____ No.    ____ Yes.    ____ Don't know.

17. Do you have relatives with sleep disorders?
   ____ No.    ____ Yes.

   Please specify relation and sleep disorder:
   ____________________________________________________________
   ____________________________________________________________
   ____________________________________________________________
18. Please indicate the items in the following list that apply to you.

<table>
<thead>
<tr>
<th></th>
<th>During the past year</th>
<th>More than a year ago</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thyroid disorders</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epilepsy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychiatric illness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychiatric treatment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurologic disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kidney disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peptic ulcer, gastritis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intestinal disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Liver disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High blood pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Headache</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arthritis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Back pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asthma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pneumonia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enlarged tonsils/adenoids</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Repeated throat infection</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic sinusitus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other health problems</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(specify)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Hospitalization:
- 1 or 2 times
- 3 or 4 times
- More than 4 times

Surgery on mouth and/or nose (specify)

FOR WOMEN ONLY:
- Irregular menstrual periods
- Use of birth control pills
- Problems associated with the menopause
19. Are you currently taking any medications?
   ____ No.  ____ Yes.

For symptoms related to sleep/sleepiness/sleep disorder:

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
<th>Duration of treatment</th>
<th>Is the drug effective?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Medications for other symptoms (NOT related) to sleep:

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
<th>Duration of treatment</th>
<th>Is the drug effective?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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</tr>
</tbody>
</table>
20. We hope you would be willing to help us with our research on normal and narcoleptic sleep onset processes. If so, when might you be available for one night/day to come into the Brock Sleep Lab?

<table>
<thead>
<tr>
<th>Date</th>
<th>Available weekdays</th>
<th>Available weekends</th>
</tr>
</thead>
<tbody>
<tr>
<td>June 13-19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>June 20-26</td>
<td></td>
<td></td>
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<tr>
<td>June 27-30</td>
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<tr>
<td>July 4-10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>July 11-17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>August 1-7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>August 8-16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>August 15-21</td>
<td></td>
<td></td>
</tr>
<tr>
<td>August 22-28</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

21. Questions or comments?

References

Questions were adopted from the following sources:


INSTRUCTIONS:

Each morning for one week prior to your scheduled stay in the Sleep Lab please do the following:

1. Write in the date.
2. With an arrow pointing down, mark each time you got into bed yesterday.
3. With an arrow pointing up, mark each time you got out of bed.
4. With a plain vertical line, mark the time at which you began to sleep, and the time at which you woke up; then join the lines together to indicate periods of sleep.
5. Describe (on page 3) any events that influenced your sleep.

Example:

<table>
<thead>
<tr>
<th>DATE</th>
<th>6</th>
<th>8</th>
<th>10</th>
<th>12</th>
<th>2</th>
<th>4</th>
<th>6</th>
<th>8</th>
<th>10</th>
<th>12</th>
<th>2</th>
<th>4</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>June 14</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

Lights out 11:30 am/pm  Total sleep time 9 hrs
Circadian Rhythm Questionnaire
J.A. Horne & O. Ostberg

Instructions

1. Please read each question very carefully before answering.

2. Answer ALL questions.

3. Answer questions in numerical order.

4. Each question should be answered independently of the others. Do NOT go back and check your answers.

5. All questions have a selection of answers. For each question place a cross alongside ONE answer only. Some questions have a scale instead of a selection of answers. Place a cross at the appropriate point along the scale.

6. Please answer each question as honestly as possible. Both your answers and the results will be kept in strict confidence.

7. Please feel free to make any comments in the section provided below each question.

Please supply the information requested below:
(1) Name: __________________________
(2) Age: ________
(3) Sex: ________
1. Considering only your own "feeling best" rhythm, at what time would you get up if you were entirely free to plan your day?

<p>| | | | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>11</td>
<td>12</td>
<td></td>
</tr>
</tbody>
</table>

2. Considering only your own "feeling best" rhythm, at what time would you go to bed if you were entirely free to plan your evening?

<p>| | | | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>9</td>
<td>10</td>
<td>11</td>
<td>12</td>
<td>a.m.</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

3. If there is a specific time at which you have to get up in the morning, to what extent are you dependent on being woken up by an alarm clock?

- Not at all dependent
- Slightly dependent
- Fairly dependent
- Very dependent

4. Assuming adequate environmental conditions, how easy do you find getting up in the morning?

- Not at all easy
- Not very easy
- Fairly easy
- Very easy

5. How alert do you feel during the first half hour after having woken in the morning?

- Not at all alert
- Slightly alert
- Fairly alert
- Very alert

6. How is your appetite during the first half-hour after having woken in the morning?

- Very poor
- Fairly poor
- Fairly good
- Very good

Please turn to next page...
7. During the first half-hour after having woken in the morning, how tired do you feel?

- Very tired
- Fairly tired
- Fairly refreshed
- Very refreshed

8. When you have no commitments the next day, at what time do you go to bed compared to your usual bedtime?

- Seldom or never later
- Less than one hour later
- 1-2 hours later
- More than two hours later

9. You have decided to engage in some physical exercise. A friend suggests that you do this one hour twice a week and the best time for him is between 7.00 - 8.00 a.m. Bearing in mind nothing else but your own "feeling best" rhythm, how do you think you would perform?

- Very tired
- Fairly tired
- Fairly refreshed
- Very refreshed

- Would be on good form
- Would be on reasonable form
- Would find it difficult
- Would find it very difficult

10. At what time in the evening do you feel tired and as a result in need of sleep?

<table>
<thead>
<tr>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12 a.m.</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

11. You wish to be at your peak performance for a test which you know is going to be mentally exhausting and lasting for two hours. You are entirely free to plan your day and considering only your own "feeling best" rhythm which ONE of the four testing times would you choose?

- 8.00 - 10.00 a.m.
- 11.00 a.m. - 1.00 p.m.
- 3.00 - 5.00 p.m.
- 7.00 - 9.00 p.m.

Please turn to next page ...
12. If you went to bed at 11.00 p.m. at what level of tiredness would you be?

- Not at all tired
- A little tired
- Fairly tired
- Very tired

13. For some reason you have gone to bed several hours later than usual, but there is no need to get up at any particular time the next morning. Which ONE of the following events are you most likely to experience?

- Will wake up at usual time and will NOT fall asleep
- Will wake up at usual time and will doze thereafter
- Will wake up at usual time but will fall asleep again
- Will NOT wake up until later than usual

14. One night you have to remain awake between 4.00 - 6.00 a.m. in order to carry out a night watch. You have no commitments the next day. Which ONE of the following alternatives will suit you best?

- Would NOT go to bed until watch was over
- Would take a nap before and sleep after
- Would take a good sleep before and nap after
- Would take ALL sleep before watch

15. You have to do two hours of hard physical work. You are entirely free to plan your day and considering only your own "feeling best" rhythm which ONE of the following times would you choose.

- 8.00 - 10.00 a.m.
- 11.00 a.m. - 1.00 p.m.
- 3.00 - 5.00 p.m.
- 7.00 - 9.00 p.m.

16. You have decided to engage in hard physical exercise. A friend suggests that you do this for one hour twice a week and the best time for him is between 10.00 - 11.00 p.m. Bearing in mind nothing else but your own "feeling best" rhythm how well do you think you would perform?

- Would be on good form
- Would be on reasonable form
- Would find it difficult
- Would find it very difficult

Please turn to next page...
17. Suppose that you can choose your own work hours. Assume that you worked a FIVE hour day (including breaks) and that your job was interesting and paid by results. Which FIVE consecutive hours would you select?

```
12 1 2 3 4 5 6 7 8 9 10 11 12
Midnight Noon Midnight
```

18. At what time of the day do you think that you reach your "feeling best" peak?

```
12 1 2 3 4 5 6 7 8 9 10 11 12
Midnight Noon Midnight
```

19. One hears about "morning" and "evening" types of people. Which ONE of these types do you consider yourself to be?

```

Definitely a "morning" type [ ]
Rather more a "morning" type [ ]
than an "evening" type [ ]
Rather more an "evening" [ ]
than a "morning" type [ ]
Definitely an "evening" type [ ]
```
### BECK DEPRESSION INVENTORY

Appendix B-4

<table>
<thead>
<tr>
<th>Number</th>
<th>Name</th>
<th>Date</th>
</tr>
</thead>
</table>

1. □ 0 I do not feel sad.  
1 I feel sad.  
2 I am sad all the time and I can't snap out of it.  
3 I am so sad or unhappy that I can't stand it.

2. □ 0 I am not particularly discouraged about the future.  
1 I feel discouraged about the future.  
2 I feel I have nothing to look forward to.  
3 I feel that the future is hopeless and that things cannot improve.

3. □ 0 I do not feel like a failure.  
1 I feel I have failed more than the average person.  
2 As I look back on my life, all I can see is a lot of failures.  
3 I feel I am a complete failure as a person.

4. □ 0 I get as much satisfaction out of things as I used to.  
1 I don't enjoy things the way I used to.  
2 I don't get real satisfaction out of anything anymore.  
3 I am dissatisfied or bored with everything.

5. □ 0 I don't feel particularly guilty.  
1 I feel guilty a good part of the time.  
2 I feel quite guilty most of the time.  
3 I feel guilty all of the time.

6. □ 0 I don't feel I am being punished.  
1 I feel I may be punished.  
2 I expect to be punished.  
3 I feel I am being punished.

7. □ 0 I don't feel disappointed in myself.  
1 I am disappointed in myself.  
2 I am disgusted with myself.  
3 I hate myself.

8. □ 0 I don't feel I am any worse than anybody else.  
1 I am critical of myself for my weakness or mistakes.  
2 I blame myself all the time for my faults.  
3 I blame myself for everything bad that happens.

9. □ 0 I don't have any thoughts of killing myself.  
1 I have thoughts of killing myself, but I would not carry them out.  
2 I would like to kill myself.  
3 I would kill myself if I had the chance.

10. □ 0 I don't cry anymore than usual.  
1 I cry more now than I used to.  
2 I cry all the time now.  
3 I used to be able to cry, but now I can't even though I want to.

11. □ 0 I am no more irritated now than I ever am.  
1 I get annoyed or irritated more easily than I used to.  
2 I feel irritated all the time now.  
3 I don't get irritated at all by the things that used to bother me.

Continued on reverse...
<table>
<thead>
<tr>
<th>Question</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>12.</td>
<td>I have not lost interest in other people.</td>
<td>I am less interested in other people than I used to.</td>
<td>I have lost most of my interest in other people.</td>
<td>I have lost all of my interest in other people.</td>
</tr>
<tr>
<td>13.</td>
<td>I make decisions about as well as I ever could.</td>
<td>I put off making decisions more than I used to.</td>
<td>I have greater difficulty in making decisions than before.</td>
<td>I can't make decisions at all anymore.</td>
</tr>
<tr>
<td>14.</td>
<td>I don't feel I look any worse than I used to.</td>
<td>I am worried than I am looking old or unattractive.</td>
<td>I feel that there are permanent changes in my appearance that make me look unattractive.</td>
<td>I believe that I look ugly.</td>
</tr>
<tr>
<td>15.</td>
<td>I can work about as well as before.</td>
<td>It takes an extra effort to get started at doing something.</td>
<td>I have to push myself very hard to do anything.</td>
<td>I can't do any work at all.</td>
</tr>
<tr>
<td>16.</td>
<td>I can sleep as well as usual.</td>
<td>I don't sleep as well as I used to.</td>
<td>I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.</td>
<td>I wake up several hours earlier than I used to and cannot get back to sleep.</td>
</tr>
<tr>
<td>17.</td>
<td>I don't get more tired than usual.</td>
<td>I get tired more easily than I used to.</td>
<td>I get tired from doing almost anything.</td>
<td>I am too tired to do anything.</td>
</tr>
<tr>
<td>18.</td>
<td>My appetite is no worse than usual.</td>
<td>My appetite is not as good as it used to be.</td>
<td>My appetite is much worse now.</td>
<td>I have no appetite at all anymore.</td>
</tr>
<tr>
<td>19.</td>
<td>I haven't lost much weight, if any, lately.</td>
<td>A. I am purposely trying to lose weight by eating less</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>20.</td>
<td>I am no more worried about my health than usual.</td>
<td>I am worried about physical problems such as aches and pains; or upset stomach; or constipation.</td>
<td>I am very worried about physical problems and it's hard to think of much else.</td>
<td>I am so worried about my physical problems, that I cannot think about anything else.</td>
</tr>
<tr>
<td>21.</td>
<td>I have not noticed any recent change in my interest in sex.</td>
<td>I am less interested in sex than I used to be.</td>
<td>I am much less interested in sex now.</td>
<td>I have lost interest in sex completely.</td>
</tr>
</tbody>
</table>
Centre for Sleep and Chronobiology
The Toronto Hospital
Appendix C-1

Name: ________________________________ Time: __________

Date: ______________ Health Card No.: __________________

PRE-SLEEP QUESTIONNAIRE

At what time did you awaken today? __________ a.m.  p.m.

Has today been an unusual day in any way? No ___  Yes ___. If yes, explain:

Did you fall asleep or take a nap today? No ___ Yes ___. If yes, when and for how long:

Did you drink any alcohol today? No ___  Yes ___. If yes, when ________, how much _______ ______?

Did you, or will you, use any medications (prescription or non-prescription) today? No ___ Yes ___. If yes, specify type and amount:

Have you used any prescription medication in the last 2 weeks? No ___ Yes ___. If yes, specify type and amount:

Please indicate how many cups or glasses of the following that you have consumed today:

____ coffee, ____ decaffeinated coffee, ____ tea, ____ cola, ____ chocolate drinks

At what time did you drink your last caffeinated beverage? __________ a.m.  p.m.
Put a check mark (✓) in the appropriate column to indicate if you are experiencing any of the following, **right now**.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Not at All</th>
<th>Slightly</th>
<th>Moderately</th>
<th>Intensely</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
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<tr>
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Use the chart below to indicate the severity of any pains, aches, or stiffness that you may be experiencing right now.

On the chart a check (✓) in the row labelled '0' indicates no discomfort.

a check (✓) in the row labelled '6' indicates the worst possible discomfort.

<table>
<thead>
<tr>
<th>HEAD</th>
<th>NECK</th>
<th>SHOULDERS</th>
<th>UPPER LIMBS</th>
<th>CHEST</th>
<th>UPPER BACK</th>
<th>LOWER BACK</th>
<th>ABDOMEN</th>
<th>HIPS</th>
<th>LOWER LIMBS</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
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</tr>
</tbody>
</table>

**FATIGUE SCALE**

Please check (✓) the statement which best describes your present state of physical energy or fatigue.

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Full of energy: enough to tackle my usual physical activities.</td>
</tr>
<tr>
<td>2</td>
<td>Energy level is quite high but not at its peak: most physical activities would pose no problem.</td>
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<tr>
<td>3</td>
<td>Energy level is such that one would prefer to be doing very light or sedentary tasks at this point.</td>
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<td>4</td>
<td>Energy level is adequate for only routine activities at a leisurely pace.</td>
</tr>
<tr>
<td>5</td>
<td>Energy level is such that it would be preferable to rest before doing any routine activity.</td>
</tr>
<tr>
<td>6</td>
<td>Energy level is quite low: would strongly prefer to rest rather than do anything else.</td>
</tr>
<tr>
<td>7</td>
<td>Totally physically exhausted: unable to undertake the least activity.</td>
</tr>
</tbody>
</table>
### STANFORD SLEEPINESS SCALE

Please check (☑) the statement which best describes your state of sleepiness. (Choose only one statement)

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Feel active and vital; alert; wide awake</td>
</tr>
<tr>
<td>2</td>
<td>Functioning at a high level, but not at peak; able to concentrate</td>
</tr>
<tr>
<td>3</td>
<td>Relaxed; awake; not at full alertness; responsive</td>
</tr>
<tr>
<td>4</td>
<td>A little foggy; not at peak; let down</td>
</tr>
<tr>
<td>5</td>
<td>Fogginess; beginning to lose interest in remaining awake; slowed down</td>
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<td>Almost in reverie; sleep onset soon; lost struggle to remain awake</td>
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</table>
POST-SLEEP QUESTIONNAIRE

Name: ________________________________________________________________

Date: ___________________________                Time: ___________________________

Please complete immediately upon the final awakening

How long did it take you to fall asleep last night: ________ minutes

How much sleep do you think you got last night: ________ hours

Please indicate with an X on the line:

[ ] Best Possible Sleep                          [ ] Worst Possible Sleep

How many times do you think you woke up last night: ________ times.

How did last night differ from your usual night's sleep, taking into account that you slept in a different bed, with electrodes, etc.

Any comments or suggestions:
Please mark each line with an 'X'.

**Going to bed**

- Asleep quickly
- Felt very physically tense
- No worries on my mind
- Many thoughts
- Felt very sleepy
- Felt very exhausted
- Had many physical ailments
- Went to bed in a very bad mood

- Long time awake
- Felt very physically relaxed
- Many worries on my mind
- No thoughts
- Felt very wide awake
- Not exhausted at all
- Had no physical ailments
- Went to bed in a very good mood

**During the night**

- Frequently awakened
- No noises
- Very comfortable room temp.
- Very uncomfortable bed
- Little or no body movement
- Awakened and took an extremely long time to go back to sleep
- Lightest sleep possible

- Uninterrupted sleep
- Very noisy
- Extremely hot or cold
- Very comfortable bed
- Tossed and turned all night
- Awakened but immediately went back to sleep
- Deepest sleep possible
## During the night (Continued)

<table>
<thead>
<tr>
<th>Adequate amount of sleep</th>
<th>Not enough sleep at all</th>
</tr>
</thead>
<tbody>
<tr>
<td>Many thoughts</td>
<td>No thoughts</td>
</tr>
<tr>
<td>Felt very physically relaxed</td>
<td>Felt very physically tense</td>
</tr>
<tr>
<td>Had many physical ailments</td>
<td>Had no physical ailments</td>
</tr>
<tr>
<td>Extremely pleasant dreams</td>
<td>Extremely unpleasant dreams</td>
</tr>
<tr>
<td>Many dreams</td>
<td>No dreams</td>
</tr>
</tbody>
</table>

## Upon awakening

<table>
<thead>
<tr>
<th>Woke up long before or after I expected</th>
<th>Woke up exactly when I expected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Woke up extremely tired</td>
<td>Woke up as rested as possible</td>
</tr>
<tr>
<td>Had a very hard time awakening</td>
<td>Woke up as easily as possible</td>
</tr>
<tr>
<td>Woke up in a very good mood</td>
<td>Woke up in a very bad mood</td>
</tr>
<tr>
<td>Remembered extremely unpleasant dreams</td>
<td>Remembered very pleasant dreams</td>
</tr>
<tr>
<td>Woke up feeling as physically poor as possible</td>
<td>Woke up feeling as physically good as possible</td>
</tr>
<tr>
<td>Woke up with no worries on my mind</td>
<td>Woke up with many worries</td>
</tr>
<tr>
<td>Woke up with no thoughts on my mind</td>
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Put a check mark (✓) in the appropriate column to indicate if you are experiencing any of the following, right now.

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FATIGUE SCALE

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</table>
HOW DO YOU FEEL?

example: CALM | IRRITABLE

CALM

HAPPY

ENERGETIC

RELAXED

IRRITABLE

SAD

SLUGGISH

TENSE

STANFORD SLEEPINESS SCALE

Please check (✓) the statement which best describes your state of sleepiness. (Choose only one statement)

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Appendix D-1

STANFORD SLEEPINESS SCALE (SSS)
Hoddes, Dement & Zarcone (1972)

Please indicate the number of the statement below which best describes your state of sleepiness at this time.

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

VISUAL ANALOGUE SCALE (VAS)
Folstein and Luria (1973)

Please place a slash mark (i.e., /) on the line at the point corresponding to your state of sleepiness at this time.

Example:

\[\begin{array}{c}
\text{VERY ALERT}\\
\rule{4cm}{0.1pt}\\
\text{VERY SLEEPY}
\end{array}\]