

**A Comparison of Electrophysiological Changes During the
Sleep Onset Period of Psychophysiological Insomniacs,
Psychiatric Insomniacs and Normal Sleepers**

by

Carole H. Lamarche

A thesis
submitted in partial fulfilment
of the requirements for the degree
Master of Arts

Department of Psychology
Brock University
St. Catharines, Ontario

July 11, 1995

© Carole H. Lamarche, 1995

Abstract

The EEG of the sleep onset period of psychophysiological insomniacs, psychiatric insomniacs and controls was compared using power spectral analysis (FFT). Eighteen drug-free subjects were equally divided into three groups according to their responses in the Brock Sleep and Insomnia Questionnaire, the Minnesota Multiphasic Personality Inventory and the Sleep Disorders Questionnaire. Group 1 consisted of psychophysiological insomniacs, group 2 included insomniacs with an indication of psychiatric disturbances, and group 3 was a control group. EEG, EOG and EMG were recorded for two consecutive nights. Power spectral analysis (FFT) of EEG at C4 from the sleep onset period (defined as lights out to the first five minutes of stage 2) was performed on all standard frequency bands, delta: .5-4 Hz; theta: 4-8 Hz; alpha: 8-12 Hz; sigma: 12-15 Hz beta: 15-25 Hz. Psychophysiological insomniacs had less alpha during wakefulness than the other two groups and did not show the dramatic drop in alpha across the sleep onset period, which characterizes normal sleep. They also had less delta, especially during stage 2 on night 2. They also showed less delta in the last quartile of the chronological analysis of the sleep onset period. Psychiatric insomniacs showed lower relative beta power values overall while psychophysiological insomniacs showed higher relative beta power values during wakefulness. This microanalysis

confirms that the sleep onset period is generally similar for psychiatric insomniacs and normal sleepers. This may be due to the sample of psychiatric insomniacs being heterogeneous or may reflect a sleep onset system that is essentially intact. Psychophysiological insomniacs have higher cortical arousal during the sleep onset period than do the psychiatric insomniacs and the controls. Clear differences in the sleep onset period of psychophysiological insomniacs exist. The dramatic changes in power values in these two groups are not seen in the psychophysiological insomniacs, which may make the discrimination between wakefulness and sleep more difficult.

Acknowledgements

First and foremost, I would like to thank my supervisor, Dr. Robert Ogilvie for his guidance, enthusiasm and support throughout the design, data collection, analysis and writing of this thesis. Through his encouraging words and gentle nudging, he inspired me to develop my own ideas and to think independently while still being part of his "sleep gang". I am grateful for the lessons learned.

I would also like to acknowledge my other thesis committee members, Nancy De Courville and Kathy Belicki, for statistical advice and for advice on clinical matters pertaining to my thesis, respectively. I would like to thank Tim Murphy and Danny Bernstein for their help with electrode hook-up and Iain Simons for his very helpful MQE lessons. And last but not least, I would like to thank my family for their continuing support.

To my Mom, the most wonderful person I know

Table of Contents

Introduction.....	1
Wakefulness and Sleep Stages.....	2
Sleep Onset.....	4
Behavioral and physiological indices.....	4
Event-related potential changes.....	8
Epidemiology of Insomnia.....	10
Aetiology of Insomnia.....	11
Primary Characteristics of Insomnia.....	14
Insomnia and Psychopathology.....	18
American Sleep Disorders Association Definitions.....	21
Insomnia Subtypes.....	23
Psychophysiological insomnia.....	23
Sleep state misperception.....	24
Psychiatric disorders and insomnia.....	26
Clinical Evaluation of Insomnia.....	28
Computer-Based EEG Analyses.....	33
Hypotheses.....	37
Method.....	39
Participants.....	39
Apparatus.....	41
Data acquisition and analysis of EEG.....	41
Slope changes.....	41
Screening questionnaires.....	42
Procedure.....	43
Sleep Stages.....	45

Table of Contents (continued)

FFT Analyses from the Sleep Onset Period.....	45
Sleep stage analysis.....	45
Consecutive epoch analysis.....	45
Statistical Analyses.....	46
Standard sleep parameters.....	46
Subjective/objective estimates of sleep onset latency.....	46
Sleep onset period "stage" analyses.....	46
Sleep onset period "consecutive epoch" analyses.....	47
Results.....	47
Sample characteristics.....	47
Standard Sleep Parameters.....	53
Subjective and Objective Estimates of Sleep Onset Latency.....	56
Sleep Onset Period Analyses.....	58
The sleep onset period in normal sleepers.....	58
Absolute power across stages.....	60
Relative power across stages.....	70
Absolute power across quartiles of the sleep onset period.....	79
Slope changes in power bands during the sleep onset period.....	85
Discussion.....	95
Visually-Scored Standard Sleep Parameters.....	96

Table of Contents (continued)

The Sleep Onset Period in Normal Sleepers.....98

The Sleep Onset Period in Psychophysiological
Insomniacs, Psychiatric Insomniacs and
Normal Sleepers.....101

 Delta activity.....102

 Alpha activity.....105

 Beta activity.....107

Future directions.....111

Summary.....112

References.....114

Appendix 1: Questionnaires.....128

List of Tables

Tables	page
1. Mean T-scores (standardized scores) on the MMPI-2...	49
2a. Mean scores on the Sleep Disorders Questionnaire and the Brock Sleep and Insomnia Questionnaire.....	51
2b. Characteristics of psychophysiological insomniacs and psychiatric insomniacs.....	52
3. Sleep variables by group.....	54
4. Summary hierarchical multiple regression table using EEG estimates and group membership as predictors of subjective sleep onset latency.....	57
5. Mean absolute power during wakefulness, stage 1 and stage 2 on night 2.....	61
6. Mean absolute power during wakefulness, stage 1 and stage 2 on night 2.....	62
7. Mean relative power during wakefulness, stage 1 and stage 2 on night 1.....	73
8. Mean relative power during wakefulness, stage 1 and stage 2 on night 2.....	74
9. Mean absolute power across quartiles of the sleep onset period on night 1.....	80
10. Mean absolute power across quartiles of the sleep onset period on night 2.....	82
11. Mean slope changes across quartiles of the sleep onset period on night 1.....	89

List of Tables (continued)

Tables	page
12. Mean slope changes across quartiles of the sleep onset period on night 2.....	91

List of Figures

Figures	page
1. The sleep onset period in normal sleepers.....	59
2a. Absolute delta power for groups on night 1.....	63
2b. Absolute delta power for groups on night 2.....	64
3. Absolute theta as a function of stage.....	65
4. Group by stage interaction for absolute alpha power.....	67
5. Absolute sigma power as a function of stage....	68
6. Absolute beta power as a function of stage....	69
7a. Total power on night 1.....	71
7b. Total power on night 2.....	72
8. Group by stage interaction for relative alpha power.....	76
9a. Relative beta power on night 1.....	77
9b. Relative beta power on night 2.....	78
10. Group by quartile interaction for absolute delta power.....	84
11. Group by quartile interaction for absolute alpha power.....	88
12. Group by night interaction for delta slope changes.....	93
13. Alpha slope changes as a function of group....	94

Introduction

Sleep has fascinated many throughout history. In ancient times, sleep was viewed as a mysterious time when one could communicate with the gods. Aristotle wrote several essays on sleep and wakefulness. Hippocrates even had his own theory of sleep. Today, many questions about sleep still remain. The functions of sleep and its role in psychological functioning are of primary importance not only because sleep occupies one-third of our lives but also because sleep disorders are becoming more prevalent in this fast-paced society (Bixler, Kales, Soldatos, Kales & Healy, 1979) as we continue to violate the circadian and psychological requirements for normal sleep.

The following review will address the most common sleep disorder, insomnia. It will begin with a brief history of modern sleep research and contemporary sleep stage scoring. Issues involved in defining sleep and sleep onset will be described and information on insomnia and its subtypes will be presented. It will conclude with statements about the goal of this investigation: to describe and contrast in great detail the sleep onset periods of normal and insomniac sleepers in the hope of learning more about the fundamental nature of the wake/sleep process.

Significant milestones in the history of modern sleep research might well begin with the publication in 1912 of Le problème physiologique du sommeil by Piéron. In the first

comprehensive, scientific work on sleep, he defined normal sleep as a suspension of sensory-motor activities characterized by an almost complete disappearance of spontaneous activity and reactions, and an increase in one's sensitivity threshold. Twenty-five years later, Loomis, Harvey and Hobart (1937) first used electroencephalography (EEG) to describe sleep in humans. In 1953, Aserinsky and Kleitman discovered rapid eye movement (REM) sleep. As knowledge increased, Rechtschaffen and Kales (1968) modified the Loomis et al. (1937) sleep/wake categories and the Dement and Kleitman (1957) scoring routines. Their scoring system of sleep stages for humans is the standard, visually-based system used today. In it, wakefulness was described and sleep was divided into five stages: 1, 2, 3, 4 and REM sleep, according to differences in frequency and amplitude of brain waves (EEG), eye movements (EOG) and muscle tone (EMG). Stages 1, 2, 3, and 4 are also referred to as non-REM (NREM) sleep.

Wakefulness and Sleep Stages

Wakefulness is primarily characterized by activity in the beta range (15 to 30 Hz). This fast frequency activity is present when an individual is actively engaged in a cognitive task and is thought to reflect heightened or waking arousal. Relaxed wakefulness is characterized by slower, alpha activity (8 to 12 Hz). Stage 1 is identified by a record with less than 50% alpha activity, vertex sharp

waves, activity in the theta range (2 to 7 Hz) and slow rolling eye movements. Stage 1 "sleep" is considered by many researchers to be a transition stage between wakefulness and sleep (Ogilvie, 1985). Stage 2 is marked by the appearance of sleep spindles (sigma activity in 12 to 14 Hz range), K-complexes (positive sharp wave immediately followed by a negative wave) and the first visible signs of delta wave activity. Stages 3 and 4 are often grouped under the name slow wave sleep. Slow wave sleep is essentially characterized by delta activity, which consists of slow (2 cycles per second or slower), high amplitude waves. Delta waves make up 20-50% of the EEG in stage 3 and 50% or more in stage 4. Delta activity is considered to be the primary index of the development of slow wave or non-REM sleep.

REM sleep is characterized by EEG patterns generally similar to stage 1, but containing unique theta frequency "saw-toothed" waves, low muscle tone, and periodic, rapid eye movements. Typically, REM sleep first appears approximately 90 minutes after sleep onset and REM periods get longer as the night progresses. Periods of REM and non-REM sleep alternate every 90 minutes. This 90-minute cycle seems to reflect an ultradian rhythm that is also present during wakefulness. Kleitman (1963) identified this cycle as the Basic Rest Activity Cycle (BRAC). In the normal young adult, stage 2 comprises approximately 50% of total sleep time (TST), SWS about 20 to 25%, and REM sleep 25%.

REM sleep, SWS and TST tend to decrease with age. The reader is referred to Horne (1988) for a more comprehensive review of normal sleep in adults.

Sleep Onset

Behavioral and physiological indices. There has been growing interest in the transition from wakefulness to sleep. This period is marked by the physiological, behavioral and cognitive changes that will be outlined below. The sleep onset period is important, not only because, as Harsh and Ogilvie (1994) point out "it links our two most fundamental states, wakefulness and sleep" (p.xviii) but also because sleep disorders of all kinds are most often linked to either difficulties in falling asleep or difficulties in remaining awake.

Sleep onset has been studied in detail in the normal adult. Many researchers disagree on the exact moment of sleep onset. Some define sleep onset as the first epoch of stage 1 (Rechtschaffen & Kales, 1968), others as the first sleep spindle or K-complex (Johnson, 1973) or the beginning of stage 2 (Agnew & Webb, 1972). Ogilvie (1985) has argued that falling asleep does not take place at a precise point in time but is better conceived of as occurring during an identifiable time period. This transition between relaxed wakefulness and unresponsive sleep can be referred to as the sleep onset period (SOP).

Ogilvie and Wilkinson (1984) found that changes in EEG,

reaction time to an auditory stimulus and respiration were orderly and rapid during the transition from wakefulness to sleep (stage 1). These changes occurred in close temporal relation to one another. They concluded that adding reaction time and respiratory measures to EEG criteria could improve the detection and definition of sleep onset.

Ogilvie, Wilkinson and Allison (1989) added a subjective measure, the Stanford Sleepiness Scale (SSS), and a continuous behavioral measure of arousal, to a reaction time measure, a respiration measure and EEG measures in order to further identify changes occurring at sleep onset. Sleep was defined behaviorally as the failure to respond to an auditory stimulus. They found that significant EEG changes occurred in all indices during the transition from wakefulness to stage 1 but that reaction time was most sensitive to further decreases in arousal occurring between stages 1 and 2.

Changes in EEG power (Fast Fourier Transformation) during the sleep onset period have also been examined. This type of analysis involves more sophisticated, computer-based techniques to quantify the EEG in humans. Power spectral analysis is performed using the Fast Fourier Transformation (FFT) to transform the digitized signal into the frequency domain (Ktonas & Gosalia, 1981). Hori (1985) used a chronological approach to the study of sleep onset period EEG changes. He examined the waking-sleeping transition in

one minute segments from ten minutes before stage 1 onset to 30 minutes after stage 1 onset. He computed power spectral activity in the five standard bands (delta: 1-3 Hz, theta: 4-7 Hz, alpha: 8-12 Hz, sigma 13-15 Hz and beta 16-19 Hz) as well as a coefficient of variation each band, which he defined as the standard deviation/average of power for each frequency band. This coefficient was used to estimate the variability of spectral power in each minute. Main effects for time were found for the power and the coefficient of variation of the delta band. The coefficient of variation increased at two points during the sleep onset period, one immediately after stage 1 onset and another two to three minutes after the end of the initial increase. Theta power and the coefficient of variation showed a similar increase just before or immediately after stage 1 onset. Alpha power was significantly different before and after stage 1 onset. Alpha decreased rapidly in the 10 minute period following stage 1 onset. The coefficient of variation for alpha increased three minutes before stage 1 onset to eight minutes after stage 1 onset then showed a sharp drop. Alpha showed another increase about 20 minutes after stage 1 onset. Sigma decreased immediately after stage 1 onset. The sigma coefficient of variation tended to increase one to three minutes prior to stage 1 onset. Beta activity did not show any significant changes. Hori (1985) concluded that significant power changes in EEG bands often did not

coincide with visually scored stages. Some EEG changes occurred before stage 1 while others occurred soon after visually scored stage 2, suggesting that the waking-sleeping transition may occur from stage 1 onset to stage 2 onset.

Badia, Wright and Wauquier (1994) also used power spectral analysis but they focused on a shorter portion of the transition to sleep. Using 30-second and 5-second epochs, they examined broadband (3-7 Hz, 8-12 Hz and 13-25 Hz) and single-Hertz changes for three minutes of continuous EEG activity, 1.5 minutes of wakefulness and 1.5 minutes of stage 1 sleep. The 30-second analyses showed that activity in the 8-12 Hz range was generally higher during wakefulness and activity in the 3-7 Hz range was higher during sleep. There was little change in 13-25 Hz activity. Power changes in broadbands generally occurred before visually scored stage 1. Single-hertz analyses revealed that 3 and 4 Hz showed the greatest increases and 9 and 10 Hz showed the greatest decreases between wakefulness and sleep. Analysis of the EEG using 5-second epochs revealed many fluctuations between wakefulness and sleep that were not evident in the 30-second analysis, suggesting that the transition from wakefulness to sleep was not smooth. Frequent fluctuations between wakefulness and sleep EEG activity were found in all subjects. Again, 3 Hz and 10 Hz were good indicators of momentary changes in wakefulness or sleep. Although this study describes changes that occur before and during stage

1, analyses of changes occurring from stage 1 to stage 2 would also have been useful in furthering understanding of the sleep onset period as a whole.

Event-related potential changes. Noldy, McGarry and Campbell (1988) used the late components (P1, N1, P2, N2) of auditory event-related potentials to examine changes in arousal during the sleep onset period. These components are thought to be affected by the relevance of the stimulus or the psychological state of the individual and vary as a function of arousal level. Campbell, Bell and Bastien (1992) have discussed their structure and significance. As reaction times to the auditory stimuli slowed, N1 became progressively smaller and N2 became larger in amplitude. P2 increased in amplitude as subjects entered stage 2.

Harsh, Voss, Hull, Schrepfer and Badia (1994) have also looked at changes in event-related potentials during the transition from wakefulness to sleep. They found that the amplitude of the P300 decreased during stage 1 and was not evident in stage 2, suggesting that the P300 is dependent on the subjects maintaining wakefulness. However, other positive peaks emerged that were related to the onset of sleep as well as a negative peak (N350) occurring between 250 and 400 milliseconds. They suggested that the N350 reflects a low level of information processing.

Ogilvie, Simons, Kuderian, MacDonald and Rustenburg (1991) examined both EEG and event-related potential changes

during the sleep onset period but they divided EEG and event-related potentials into bins according to a behavioral measure: the slowing and disappearance of responses in a reaction time task to an auditory stimulus. They used a behavioral definition of sleep onset: five consecutive, failed responses in the reaction time task. They found that delta, theta and sigma power increased and alpha and beta decreased as responses to the reaction time task slowed, but all bands showed significant increases in power at sleep onset. They suggested that EEG synchronization at sleep onset typically associated with the slower frequencies was also seen in the faster frequencies when careful behavioral differentiations of arousal level are used, allowing for precise separation of EEG segments. These changes were similar to those of Hori (1985), except for the increase in beta power at sleep onset. This is interesting considering that Hori used a chronological approach and Ogilvie et al. (1991) used a behavioral task to bin their EEG samples. Confirming Noldy et al. (1988), they found that N1 decreased as responses to the reaction time task slowed and practically disappeared at SO. P1, N2, P3 and N3 all increased at sleep onset. They concluded that behavioral differentiations of arousal allow for a precise discrimination of wakefulness and sleep.

As the above demonstrates, a growing body of research has been completed on normal sleep and sleep onset in order

to better understand its basic mechanisms and functions. Research on normal sleep processes was, and continues to be, the necessary precedent to research into abnormal sleep processes. As knowledge of normal sleep increased over the last four decades, instances of abnormal sleep became more apparent. There are now over 80 types and categories of sleep disorders listed in the International Classification of Sleep Disorders (ICSD) (American Sleep Disorders Association, 1990) ranging from those which mildly inconvenience the sleeper, such as time zone change syndrome and sleep talking, to others which can be life-threatening, like sleep apnea, narcolepsy and sudden infant death syndrome. Surprisingly, as the study of sleep disorders evolves, it is apparent that insomnia is the most prevalent and among the least studied and understood.

Epidemiology of Insomnia

Many definitions of insomnia have been proposed. It has been defined as a series of disorders involving initiating and maintaining sleep (DIMS) (American Sleep Disorders Association, 1990); as a group of related symptoms, which includes difficulty in initiating sleep, difficulty in maintaining sleep or nonrestorative sleep (Hartmann, 1988), and as a relative lack of sleep, an inadequate quality of sleep, or both (A. Kales & J. Kales, 1984).

It is estimated that insomnia affects up to one-third

of the population. In a large survey of over one million Americans, Hammond (1964) found that 21% suffered from insomnia. Bixler, A. Kales, Soldatos, J. Kales and Healy (1979) reported that 32.2% of people in the Los Angeles Metropolitan area complained of insomnia. In a study of Alachua County, Florida, (Karacan et al., 1976) 35% of the sample were found to suffer from sleep difficulties sometimes, often or all the time. Cirignotta, Mondini, Zucconi, Lenzi and Lugaresi (1985) reported that 13.4% of the 5713 people from their San Marino, Italy study declared that they rarely or never slept well. The differences in prevalence are probably due to the way questions were asked, with references to insomnia yielding smaller percentages than more general questions about difficulties in sleeping. Nevertheless, many millions of people are not sleeping well on any given night. Insomnia occurs more frequently with increasing age (Hammond, 1964; Karacan et al., 1976) and more often in women (Hammond, 1964; Bixler et al., 1979; Cirignotta et al., 1985). It is often associated with lower socioeconomic status (Karacan et al., 1976).

Aetiology of Insomnia

Many hypotheses concerning the aetiology of insomnia have been proposed, most of which include some disruption in the arousal system brought about by physiological, psychological or environmental factors. Espie (1991) and Morin (1993) have summarized these various hypotheses which

will be described below:

Central nervous system dysfunction may be responsible for insomnia. Specifically, lesions or particular sensitivity in the thalamic regions that mediate sleep activation or to the Reticular Activating System that are responsible for alertness may be involved in insomnia. Hauri (1975, as cited in Espie, 1991) has suggested that insomnia may be the result of a deficient serotonergic sleep system.

Autonomic arousal may also be implicated in insomnia. For example, Monroe (1967) found that compared to good sleepers, poor sleepers had higher rectal temperature, higher perspiration rate, more vasoconstrictions per minute, higher skin conductance and more body movements per hour prior and during sleep. Lack, Balfour and Kallucy (1985) reported higher mean rectal temperatures in poor sleepers than in good sleepers, from two hours before to four hours after sleep onset. Freedman and Sattler (1982) found that insomniacs had higher heart rates and frontalis and chin EMG levels prior to sleep onset than controls.

Psychological arousal involving cognitions (thinking, planning, rehearsing, etc.) and emotions (worry, anxiety, ruminations, etc.) may also play a role in causing and perpetuating insomnia. Lichstein and Rosenthal (1980) found that 55% of their sample of 300 chronic insomniacs attributed their poor sleep to cognitive arousal. It is not

so much the number of cognitions but their content and affect that is important in causing and perpetuating insomnia. Kuisk, Bertelson and Walsh (1989) found that subjective and psychophysiological insomniacs reported more negative thoughts during the sleep onset period than controls. Borkovec, Lane and Von Oot (1981) found similar results in their sample of insomniacs. Insomniacs often have dysfunctional beliefs, expectations and attributions about sleep, all of which lead to heightened emotional arousal and to the exacerbation of sleep difficulties (Morin, 1993). Morin, Stone, Trinkle, Mercer and Remsberg (1993) found that older (mean age 68.2 years, range from 55-88 years) insomniacs had more dysfunctional beliefs about sleep, expressed more hopelessness about the fear of losing control over their sleep and more helplessness about its unpredictability than the age-matched control group.

Others suggest that environmental factors are of primary importance in insomnia. Bootzin (1972) postulated that falling asleep is an instrumental act emitted to produce reinforcement; sleep being the reinforcing factor. Difficulties in falling asleep may be caused by an absence of discriminative stimuli for sleep (getting into bed, turning off the light, etc.) or the presence of discriminative stimuli that are not compatible with sleep (reading, television, etc.). Temporal factors for sleep (i.e., lack of a regular sleep-wake schedule, napping,

having a bedtime that is unrelated to level of tiredness) may also contribute to insomnia. Therapies (i.e., stimulus control therapy) (Bootzin, 1972; Lacks, Bertelson, Gans & Kunkel, 1983) aimed at strengthening the relationship between the bedroom and sleep have produced reductions in sleep onset latency.

There appears to be evidence to support each arousal model of insomnia. This suggests that certain factors might be more important in the cause of insomnia while others might play a more important role in its maintenance, which makes the study of different models of insomnia a complex issue.

Primary Characteristics of Insomnia

The sleep of insomniacs has several defining features. The two primary characteristics of insomnia are extended sleep onset latency and disrupted sleep period. Insomniacs have a significantly longer sleep latency than good sleepers (A. Kales et al., 1984; Monroe, 1967). Carskadon, Mitler, Billiard, Phillips and Dement (1975) compared total sleep time and sleep latency in insomniacs and normals. All-night sleep recordings were obtained from 109 individuals (55 females and 54 males) between the ages of 30 and 68. Male insomniacs slept longer than female insomniacs (385 minutes vs. 369 minutes) while male normal sleepers slept less than female normals (402 minutes vs. 422 minutes). Sleep latency decreased monotonically over age groups in the insomniac

group while sleep latency increased monotonically over these age groups in normals. The mean sleep latency for insomniacs from 30 to 39 years of age was 50.8 minutes while the latency for insomniacs from 60 to 69 years of age was 18.3 minutes. Mean sleep latency for normals aged 30 to 39 was 7.8 minutes whereas the mean latency for normals aged 60 to 69 was 12.4 minutes. Some researchers (Monroe, 1967; Gaillard, 1978) have found longer wakefulness after sleep onset (WASO) in insomniacs when compared to good sleepers while others have not (e.g., Coates et al., 1982).

Total wake time is higher in insomniacs (Kales et al., 1984; Coates et al., 1982). Kales and colleagues (1984) found that total wake time in insomniacs differed from age-matched controls in three age groups. For individuals under 30 years of age, mean wake time was 63 minutes for insomniacs and 40 minutes for controls; for individuals between 30 and 49 years of age, mean wake time was 84 and 48 minutes for insomniacs and controls respectively; and for individuals over 50 years of age, the amount of total wake time was 106 for insomniacs and 72 minutes for controls.

Insomnia is often characterized by night to night variability on most sleep parameters when compared with the sleep of normals (Coates, Strossen, Rosekind, & Thoresen, 1978). For example, Roth, Kramer and Lutz (1976) found within-subject variability in sleep latency for some patients to be as great as two hours. Kales et al. (1984)

found greater night to night variability in wake time after sleep onset (WASO). Several nights of poor sleep may be followed by one or more nights of sleep recovery. This can make the study of insomnia in the laboratory problematic since it may increase within-subject variability tremendously. Some insomniacs do not show what has been termed the "first night effect" (Agnew, Webb & Williams, 1966), that is, poorer sleep on the first night in the laboratory. Some actually sleep better in the laboratory, away from the "insomnia" environment (Hauri & Olmstead, 1989).

Other studies comparing sleep stages of insomniacs and good sleepers have also shown inconsistent results. Monroe (1967) found lower percentages of REM sleep in insomniacs while Kales et al. (1984) found no significant differences in REM sleep percentage. Some studies (e.g., Gaillard, 1978) have found lower percentages of stage 3 and 4 sleep while some have found no such differences (e.g., Stepanski, Zorick, Roehrs, Young & Roth, 1988). These discrepancies may be partly explained by differing methods or different definitions of insomnia. For example, Gaillard (1978) used an automatic sleep staging program to score his data and included only sleep maintenance insomniacs. This lack of consistent definitions and research protocols has seriously compromised the value of much of this earlier research.

Subjective reports of impaired daytime functioning are

common even though consistent differences have not been found between insomniacs and good sleepers on objective tests like the Multiple Sleep Latency Test (MSLT) (Seidel et al., 1984). Use of the MSLT has shown normal daytime wakefulness in young as well as old insomniacs (Dement, Seidel & Carskadon, 1982, 1984). Stepanski et al. (1988) also used overnight EEG measures and the MSLT. They found that insomniacs were significantly more alert on the MSLT than control subjects even though the insomniacs had experienced less nighttime sleep. This has been interpreted in different ways. Insomniacs are either not sleep deprived or sleep deprivation affects insomniacs in a way that is different from control subjects. Higher or normal alertness levels in insomniacs during the day is consistent with the hypothesis of a general hyperarousal mechanism that is not only active at night, when the individual is trying to sleep, but also during the day (Morin, 1993).

Insomniacs' subjective estimates of total sleep time and sleep latency tend to be inaccurate. Rechtschaffen (1968) found that when insomniacs were awakened after the first sleep spindle, they often reported that they had been awake while normal controls usually said that they had been asleep. Moore, Bonnet, Warm and Kramer (1980) compared estimates of sleep onset in insomniacs and controls. They found that, although objective sleep latencies did not differ, subjective estimates of controls were significantly

shorter than estimates of insomniacs. Borkovec (1982) suggests that excessive cognitive activity during the early stages of sleep and the lighter stages throughout the night may be the cause of insomniacs' perception of having been awake during those periods. The perception of time seems longer in an aversive situation, such as lying in bed awake, than in a relaxing situation. Hauri and Olmstead (1983) confirmed that, for most insomniacs, the subjective estimate of being asleep occurs later in the EEG-defined transition from waking to sleeping than it does for good sleepers. They compared subjective sleep onset latency estimates with three different criteria for defining EEG sleep onset. The first criterion was the first epoch to be scored as stage 2, the second was the beginning of the first 15 minutes of uninterrupted stage 2, and the third was the beginning of the first 30 minutes of uninterrupted stage 2. They found, in their sample of 56 insomniacs and 10 good sleepers, that insomniacs' estimates fit well with the second criterion used; that of 15 minutes of uninterrupted stage 2, while good sleepers' estimates fit with the first, earlier occurring and more traditional criterion.

Insomnia and Psychopathology

Relationships between insomnia and psychopathology have also been explored. The Minnesota Multiphasic Personality Inventory (MMPI) (Hathaway & Meehl, 1951) has been used extensively to assess psychopathology in various

populations. It consists of ten clinical scales and three validity scales. The severity of sleep difficulty and the degree of psychopathology as assessed by the MMPI were found to be positively related in insomniacs (A. Kales et al., 1984). Monroe (1967) reported significantly higher scores in poor sleepers on the Depression, Psychasthenia, Schizophrenia, Paranoia, Hypochondriasis, and two of the validity scales, the F scale and the K scale. Kales, Caldwell, Soldatos, Bixler and Kales, (1983) found that all eight clinical scales (Hypochondriasis, Depression, Hysteria, Psychopathic Deviate, Paranoia, Psychasthenia, Schizophrenia and Hypomania) and the F scale of the MMPI were significantly higher in a sample of 528 insomniacs when compared to controls. They also noted that insomniacs were unable to discharge anger outwardly and internalized their emotions. Unlike Monroe, the Kales group found lower scores on the K scale. Schneider-Helmert (1987) found higher scores on eight of the thirteen scales of the MMPI in a group of chronic insomniacs when compared to controls. In contrast, Mendelson, Garnett, Gillin and Weingartner (1984) found higher scores for insomniacs only on Depression, F and Social Introversion scales of the MMPI and lower scores on the K scale when compared to controls.

When individual questions from the MMPI were examined (J. Kales et al., 1984), a higher number of psychosocial difficulties, more physical problems commonly linked to

emotional factors and more tension, anxiety and worry at bedtime were found in insomniacs when compared to controls.

Tan, Kales, Kales, Soldatos and Bixler (1984) gave DSM-III diagnoses to their sample of 100 insomniacs. Diagnoses were made on axis I (psychiatric disorders) in 69 patients, on axis II (personality disorders) in 26 patients and on axis III (physical disorders) in 5 patients. The most common disorders were dysthymia (axis I), anxiety (axis I), somatoform (axis I), substance use (axis I) and compulsiveness (axis II).

Inconsistent results in several sleep studies on insomnia may have been the result of differing methods and definitions but may also have been due to the fact that great variability exists within and between subjects. While most of the earlier studies on insomnia have considered insomniacs as a homogeneous group, some authors have recently been making distinctions among insomniacs in order to better define and treat them. For example, distinctions have been made between sleep-onset (initiation) and sleep maintenance insomniacs, between objective and subjective insomniacs (confirmed by polysomnography or showing normal polysomnographic records), and between primary and secondary insomniacs (associated with a medical or psychiatric disorder). In sum, comparisons across types of insomniacs are becoming meaningless because of confusion caused by these various distinctions.

American Sleep Disorders Association Definitions

In an attempt to clarify these distinctions, the American Sleep Disorders Association (ASDA) has differentiated among different types of insomnia and other sleep disorders along broad pathophysiological lines. The ASDA has produced the International Classification of Sleep Disorders (1990), a diagnostic and coding manual. The manual has been used in clinical settings since its introduction, but little evaluation of its usefulness and of the validity of its classifications has been carried out. The only study to date (Buysse et al., 1994) compared clinicians' diagnoses of 216 insomniacs using the International Classification of Sleep Disorders (ICSD) (1990), the Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV) and the tenth edition of the International Classification of Diseases (ICD-10). The ICSD contains over 50 specific definitions of insomnia while the DSM-IV and the ICD-10 have two broad definitions of insomnia. There was correspondence on the diagnosis of the two most frequent disorders across all classification systems. Sleep disorders associated with mood disorder and psychophysiological insomnia were the most common ICSD diagnoses; insomnia related to another mental disorder and primary insomnia were the most common disorders using the DSM-IV; and insomnia due to emotional causes and insomnia of organic origin were the most common ICD-10 diagnoses.

Psychophysiological insomnia, primary insomnia and insomnia of organic origin corresponded roughly to the same disorder across classification systems. The diagnosis of sleep disorder associated with mood disorder, insomnia related to another mental disorder and insomnia due to emotional causes also corresponded to the same disorder across systems. The more narrow ICSD diagnoses (described below) nested logically within the broader DSM-IV and ICD-10 categories.

The ICSD manual divides sleep disorders into three broad categories: the dyssomnias, the parasomnias and sleep disorders associated with medical and psychiatric disorders. The dyssomnias category is further divided into three sections: intrinsic sleep disorders, extrinsic sleep disorders and circadian rhythm sleep disorders. Intrinsic sleep disorders are thought to have causes from within the body. Extrinsic sleep disorders have environmental causes and circadian rhythm sleep disorders are caused by temporal and biological rhythm factors. There are five disorders in the intrinsic sleep disorders section that list insomnia (DIMS) as an essential feature. They include psychophysiological insomnia, sleep state misperception, idiopathic insomnia, periodic leg movement disorder and restless legs syndrome. Those of particular interest to this study are psychophysiological insomnia and insomnia associated with psychiatric disturbances, identified earlier as the two most frequently encountered subtypes. These will

now be described in more detail. Since the diagnosis of sleep state misperception is usually made by polysomnographic assessment, and cannot be ruled out until polysomnographic assessment is made, it will also be further discussed.

Insomnia Subtypes

Psychophysiological insomnia. In the ICSD (American Sleep Disorders Association, 1990), psychophysiological insomnia is defined as "a disorder of somatized tension and learned sleep-preventing associations that results in a complaint of insomnia and associated decreased function during wakefulness". Hauri and Fisher (1986) have described psychophysiological insomnia in detail. Psychophysiological insomniacs experience chronic somatized tension, which not only manifests itself in insomnia but in other symptoms such as tension headaches, palpitations and low back pain. The more they try to sleep, the more aroused they get. When not trying to fall asleep (e.g. when watching television), they fall asleep easily. The insomnia often begins during a period of stress and remains after the stressor is gone. Diagnosis is usually achieved if the individual presents with the above symptoms and other disorders such as sleep state misperception (described below), insomnia related to psychiatric conditions, and medical conditions or circadian rhythms are ruled out.

Hauri and Fisher (1986) compared psychophysiological

insomniacs with normal sleepers and individuals who had insomnia associated with dysthymic disorders. Normal sleepers showed greater sleep efficiency than the psychophysiological insomniacs and the dysthymic insomniacs. The two insomniac groups showed similar sleep and sleep disturbances. The only differences were longer sleep latency to the first sleep spindle and less REM sleep in the psychophysiological insomniac group. Twelve of the 22 psychophysiological insomniacs and 8 of the 22 normal sleepers had excessive alpha activity during sleep. Psychophysiological insomniacs showed higher scores than normal sleepers on the Hysteria and Lie scales of the MMPI. The individuals with dysthymic disorders showed the highest MMPI scores on all scales. Lichstein, Wilson, Noe, Aguillard and Bellur (1994) compared three different measures of daytime sleepiness: the MSLT, pupillometry and the Stanford Sleepiness Scale (SSS) in psychophysiological insomniacs and controls. They found that the three measures could not distinguish insomniacs from controls. They concluded that insomniacs do not exhibit more daytime sleepiness than controls, either because they are satisfying their biological sleep need despite poor sleep, or chronic hyperarousal is preventing them from both sleeping and experiencing sleepiness.

Sleep state misperception. The ICSD (American Sleep Disorders Association, 1990) defines sleep state

misperception as a "disorder in which a complaint of insomnia or excessive sleepiness occurs without objective evidence of sleep disturbance" . There has been some criticism of this diagnostic category, especially of its prevalence. Trinder (1988) has criticized the procedures used to define the disorder. He believes that it is a pseudo diagnostic classification because there were methodological problems with the studies used to help define the disorder. Many did not determine whether patients recognized whether they had slept well in the laboratory, did not consider the relation between number of nights studied and the likelihood of a patient's sleeping adequately in the laboratory, did not identify sleep disturbance not reflected in standard sleep measurements and did not adequately screen for psychopathology. McCall and Edinger (1992) suggest that sleep state misperception represents a distinctive but rare sleep disorder but that the diagnostic criteria may need refinements to improve the meaningfulness of the diagnosis. There is evidence from research and clinical literature that sleep state misperception represents a distinct sleep disorder.

McCall and Edinger (1992) describe two cases of sleep state misperception in detail. For the 28 year old male, 14 days of sleep diaries revealed no sleep for 12 nights and 1 hour of sleep on the other two nights. Polysomnographic data revealed a sleep latency of 27 minutes and 443 minutes

of sleep with 17 minutes of WASO. However, the individual reported no sleep on the night of the recording. He showed no signs of major psychopathology. For the 39 year old woman, there were reports of no sleep in the last 13 years. She denied sleepiness or daytime naps during this period. Her psychiatric history and MMPI scores were well within normal limits. Her polysomnographic recording showed a sleep latency of 26 minutes, 411 minutes of sleep, 82 minutes of WASO and otherwise normal sleep. She reported no sleep on the night of the recording and did not believe the polysomnographic results. The authors suggest that the diagnosis needs to be made not from polysomnographic findings but from other criteria, as is the case with other sleep disorders where clinical impression is then followed by polysomnographic recording. Such criteria could be the presentation of a complaint that is physiologically improbable, such as unrelenting total insomnia which would suggest an extreme misperception of the sleep state. Sugeran, Stern and Walsh (1985) have shown that these individuals have much greater impairment of daytime vigilance than other insomnia subgroups or normal controls. They also show more restless sleep as measured by wrist actigraphy than do other insomniacs (Hauri & Wisbey 1989).

Psychiatric disorders and insomnia. Insomnia is typically a feature of psychiatric disorders such as depression and anxiety. Depressed individuals show specific

abnormalities in sleep continuity and architecture, especially REM sleep. Gillin, Duncan, Pettigrew, Frankel, and Snyder (1979) compared all-night EEG data of 41 normal sleepers, 56 depressed individuals and 18 insomniacs. An insomnia diagnosis was made if the individual had insomnia on at least four nights a week for at least two years, were not medically or mentally ill, and had dysphoric feelings related to their sleeping difficulties. Group comparisons showed that normals had greater total sleep, shorter sleep latency, more delta sleep and greater sleep efficiency when compared to insomniacs. Comparisons between normals and depressed subjects showed these same differences as well as less early morning awake time, less awake time, and longer REM latency for normals. Depressed patients showed greater early morning awake time, shorter REM latency, greater REM index and greater REM density than insomniac subjects. They concluded that EEG sleep data could be a useful biological measure that can help discriminate between depressed, insomniac and normal individuals.

Individuals with anxiety disorders show sleep onset or maintenance insomnia due to excessive anxiety. Individuals with anxiety disorders usually show longer sleep latencies, more stage 1 and 2, less SWS, and a reduced sleep efficiency (Reynolds, Shaw, Newton, Coble & Kupfer, 1983). Reynolds, Taska, Sewitch, Restifo, Coble and Kupfer (1984) compared 10 psychophysiological insomniacs with 10 individuals suffering

from generalized anxiety disorder and 20 individuals with primary major depression. All showed similar difficulties in initiating and maintaining sleep. Comparisons between groups showed that depressed individuals had lower percentage of stage 2, higher REM sleep percentage, smaller REM latencies, more REM time, more REM activity and higher REM density than insomniac or anxious individuals. Anxious and insomniac individuals differed only on REM density, with anxious individuals having higher REM density.

It is interesting to note that throughout this analysis of insomnia and its subtypes, virtually every set of comparisons to date has been restricted to basic human scored sleep/wake parameters such as described in the paragraph above. It is the major prediction of the present study that the in-depth analyses of the EEG, particularly during the sleep onset period, will unmask fundamental differences in the sleep onset mechanisms of normal sleepers, psychophysiological insomniacs and psychiatric insomniacs.

Clinical Evaluation of Insomnia

There are several methods for identifying sleep disorders such as insomnia. More traditional methods include sleep history, polysomnographic assessment in the sleep laboratory, assessment of daytime functioning, sleep diaries and psychological testing. Interestingly, there is disagreement in the sleep literature on the importance of

the role of polysomnographic assessment in insomnia. Kales, Bixler, Soldatos, Vela-Bueno, Caldwell and Cadieux (1981) found that medically-based sleep problems like apnea and periodic leg movements are so infrequent in insomnia patients that polysomnographic assessment is rarely needed. In 200 consecutive insomniac patients, they found no cases of apnea and only 5% with periodic leg movements. However, Coleman, Roffwarg and Kennedy (1982) found that 6.2% of their national sample of 1214 patients had sleep apnea and 12.2% had periodic leg movement (PMS) or restless leg syndrome. Edinger, Hoelscher, Webb, Marsh, Radtke and Erwin (1989) evaluated the importance of polysomnographic assessment of DIMS patients. Their sample consisted of 100 (54 females and 46 males) patients presenting with a complaint of insomnia at a sleep disorders centre. The age ranged from 21 to 85 (mean 46.1). Diagnostic impression of the sleep problem was given by a psychiatrist and a psychologist. Six DIMS subtypes were identified in their sample: psychiatric, periodic leg movement/restless leg syndrome, psychophysiological, drug/alcohol dependency, subjective and sleep apnea. Twenty-five percent had a periodic leg movement/restless leg syndrome diagnosis, 3% had sleep apnea and 6% had subjective insomnia. Polysomnographic assessment yielded important information in 65% of the sample. Only 14 of the 25 cases of PMS were predicted from clinical evaluation. One in three cases of

sleep apnea was predicted and 1 in 6 cases of subjective insomnia was predicted from the clinical evaluation. They suggest that the age of sleep disordered individuals is important in identifying their sleep problems. Persons over 40 have a relatively high incidence of disorders that need polysomnography for definite diagnosis and routine use of polysomnography with this age group may be justified.

Jacobs, Reynolds, Kupfer, Lovin, and Ehrenpreis (1988) reviewed charts of 123 patients evaluated at a sleep disorders centre over a 5-year period. Seventy-eight (63%) of the 123 had present or past mental illness (40 affective disorders, 27 with anxiety disorders). Clinical impression of the problem was given. Sixty of the 123 (49%) clinical diagnoses required substantial modification based on sleep laboratory findings. Lab findings either refuted, added new and unsuspected information, or ruled out a possibility in these cases. They suggest that polysomnographic assessment be used in cases of chronic insomnia where the individual has been treatment resistant.

The Standards of Practice Committee of the American Sleep Disorders Association commissioned a review to assess the importance of polysomnography in the evaluation of insomnia. The authors of this review, Reite, Buysse, Reynolds and Mendelson (1995), concluded that polysomnography has a limited role in the evaluation of patients with insomnia complaints, unless there is strong

clinical evidence of a sleep-related breathing disorder or periodic limb movements, or if the patient has failed a comprehensive behavioral or pharmacologic treatment program for insomnia. Since insomnia is such a prevalent problem in the general population, routine polysomnography for the evaluation of insomnia is impractical in most cases.

Recently, behavioral methods have been developed to allow easier assessment of certain sleep parameters. Wrist actigraphy (Kripke, Mullaney, Messin and Wyborney, 1978; Hauri & Wisbey, 1989), the Sleepscope (Bonato & Ogilvie, 1989; Kuderian, Ogilvie, McDonnell & Simons, 1991), and the Nightcap (Mamelak & Hobson, 1989; Pace-Schott, Kaji, Stickgold & Hobson, 1994; Stickgold & Hobson, 1994) have been compared to standard measures in the laboratory as well as in the home.

Several studies have compared EEG measures with wrist actigraph data in normal sleepers. Kripke and colleagues (1978) found that the two estimates of total sleep time were correlated (+0.98). Mullaney, Kripke and Messin (1980) reported a +0.89 correlation between EEG and actigraph measures. Hauri and Wisbey (1992) used the two measures with insomniacs of different subtypes. They found that, compared to EEG measures, the actigraph disagreed by an average of 49 minutes. The actigraph overestimated sleep in the psychophysiological and psychiatric groups and either underestimated or was accurate in estimating sleep in the

sleep-state misperception group.

Ogilvie and Wilkinson (1984) developed a behavioral response (BR) system designed to assess sleep onset. Subjects responded to a faint tone with a hand-held switch while falling asleep and throughout the night. The BR system was in 93% agreement with EEG measures of sleep and wakefulness. Bonato and Ogilvie (1989) and Kuderian et al. (1991) used a portable variation of the BR system, the Sleepscope, to assess different sleep parameters (sleep onset latency, total sleep time, number of microarousals and macroarousals) in good and fair or poor sleepers. Bonato and Ogilvie (1989) found that fair sleepers obtained less sleep, had more microarousals and more macroarousals than good sleepers. Kuderian et al. (1991) also found these differences between good and poor sleepers as well as an increased sleep onset latency.

Mamelak and Hobson (1989) developed a home-based sleep monitoring system, the Nightcap, to predict NREM and REM sleep using eye movements and body movements. Eye movements were measured using a transducer attached to the eyelid. There was 85.57% agreement between sleep records and nightcap data. The Nightcap has also been used by self-described good and poor sleepers in the home. Significant differences have been found in sleep latency and sleep efficiency as measured by the Nightcap in these two groups (Pace-Schott et al., 1994).

There has also been increased effort to develop questionnaires that will allow for adequate discrimination between good sleepers and insomniacs, to rule out other sleep disorders and to successfully classify insomniacs into ICSD DIMS subtypes that would eventually allow for accurate diagnosis without having to bring subjects or patients into the sleep laboratory for polysomnographic recording. The Sleep Disorders Questionnaire (Douglass, Bornstein, Nino-Murcia, 1986; Douglass et al., 1994) has been under development for several years. Acceptable reliability has been established for the SDQ (Douglass et al., 1990; Douglass et al., 1994). Douglass et al. (1992) have found a significant correlation ($r=.70$) between the PSYCH scale (items relating to psychiatric sleep disorder) of the SDQ and another measure of depression the Carroll Depression Scale, suggesting that the PSYCH scale of the SDQ is valid.

Recently, Côté and Ogilvie (1993) have devised the Brock Sleep and Insomnia Questionnaire (BSIQ). The BSIQ assesses sleep in good sleepers and insomniacs and attempts to discriminate among ICSD DIMS subtypes. Small samples have precluded any attempts to subtype insomniacs, however the questionnaire does adequately discriminate between insomniacs and controls (Cote & Ogilvie, 1993). Further validation of the BSIQ is currently under way.

Computer-based EEG Analyses

Sophisticated computer-based EEG analyses routines such

as power spectral analysis and period-amplitude analysis, have been used to study normals during sleep onset and during sleep. However, these techniques are just beginning to be used to study clinical populations (Ogilvie & Harsh, 1994).

Period amplitude analysis has been used to study sleep changes in depressed individuals during sleep onset (Armitage, Hudson, Fitch & Pechacek, 1994). Analysis of half-wave zero-cross, first derivative and full-wave zero-cross revealed higher beta activity in the depressed group when compared to controls. Slower frequencies were higher in normals.

Only four studies have reported using power spectral analysis with the EEG of insomniacs. Freedman (1986) compared the EEG from the first unambiguous minute of wakefulness and each sleep stage from the first sleep cycle in sleep-onset insomniacs and age-matched normal sleepers. The criteria for insomnia was a sleep latency of at least 1 hour 4 times a week and a sleep latency of at least 30 minutes in the laboratory. He found that, during wakefulness, insomniacs had significantly more beta activity, more 1 Hz activity and less 9Hz (alpha) than normals. During stage 1 and REM sleep, insomniacs had significantly more beta activity than normals. There were no differences in stages 2, 3 and 4. Freedman suggests that physiological (cortical) arousal, as indicated by beta

activity, may be higher in insomniacs than in normals. There are problems with this study, however. Freedman only used one minute of EEG from each stage; he did not screen for psychopathology nor did he discriminate among insomnia subtypes.

Mérica and Gaillard (1992) have looked closely at changes in the EEG during the sleep onset period (SOP) in 12 psychophysiological insomniacs with sleep maintenance insomnia, and 23 normal controls. They looked at beta, delta and the beta/delta ratio (activity index) measured at temporal lobe sites. The SOP was defined as the beginning of the stage 1 episode which immediately preceded stage 2 sleep. It was of a duration of approximately 3 minutes. However, 20 minutes from each night were included in the analysis to ensure that the SOP was preceded by wakefulness and followed by stage 2 or 3. Samples were taken at seven different times, one before the SOP, 2 during the SOP and 4 after the SOP. The SOP was marked by a period of rapid change. Beta activity decreased and delta activity increased in both groups. The rates of change in delta and the activity index were significantly different in insomniacs and controls. Changes subsided when stage 2 was reached. Insomniacs had more EEG activity during the SOP, activity which persisted in the first few minutes of stage 2. This higher activity in the insomniacs resulted from higher beta activity and lower delta activity. Discriminant

analysis showed that it was possible to discriminate between insomniacs and normals using the activity index, beta, delta and interhemispheric differences in activity as variables. Although Mérica and Gaillard (1992) described changes that occurred during twenty minutes of the sleep onset period, they only used three minutes of data in the analysis.

Jacobs, Benson and Friedman (1993) used power spectral analysis of the EEG along with other measures to assess the efficacy of a behavioral intervention for chronic sleep-onset insomnia. Insomniacs had higher relative beta power than normal sleepers before treatment. Insomniacs had reduced beta power after treatment, although not to the level of controls. There were still differences between the groups at this time.

Björnsson and Hetta (1994) have also looked at the SOP in chronic primary insomniacs. EEG variability and an activity index (beta power/delta power) were calculated for the SOP. They found that increased EEG spectral irregularity and activity during the SOP lead to increased variability and increased alpha and beta throughout the night's sleep, even though normal cyclicity and sleep architecture were maintained. This increased variability coincided with subjective reports of disturbed sleep. They did not describe how they defined this variability.

Though sleep onset has been studied in some detail in normals (Badia et al., 1994; Ogilvie & Wilkinson 1984;

Ogilvie et al., 1989, 1991), the studies by Merica and Gaillard (1992) and Björnsson and Hetta (1994) are the only ones to have looked at it in insomniacs. This is ironic, considering that the sleep onset period is the most problematic period for many insomniacs, and that sleep onset latency is a commonly used clinical measure, not only for insomniacs but for disorders of excessive sleepiness (Richardson et al., 1978).

The present study's goal was to describe the sleep onset characteristics of the two largest insomnia subtypes of the International Classification of Sleep Disorders (ICSD) (American Sleep Disorders Association, 1990) Manual using electrophysiological measures. This was achieved by first using the ICSD's definitions to identify subtypes and then predicting fundamental electrophysiological differences among individuals with psychophysiological insomnia, insomnia associated with psychiatric disturbances and normal controls in accordance with the evidence summarized above. Specifically, it was hypothesized that:

- 1) Psychophysiological insomniacs would show lower delta activity than controls and psychiatric insomniacs during wakefulness (eyes closed) prior to sleep onset.
- 2) Psychophysiological insomniacs would show lower delta activity than controls and psychiatric insomniacs during stage 1 and that these differences would persist into stage 2.

3) Psychophysiological insomniacs would show lower alpha than controls and psychiatric insomniacs during wakefulness (eyes closed) prior to sleep onset.

4) Psychophysiological insomniacs would show higher beta than controls and psychiatric insomniacs during wakefulness (eyes closed) prior to sleep onset.

5) Psychophysiological insomniacs would show higher beta activity than controls and psychiatric insomniacs during stage 1 and that these differences would persist into the beginning of stage 2.

(Hypotheses 3, 4 and 5 would reflect higher arousal levels during the sleep onset period for the psychophysiological insomniacs)

6) The sleep onset periods of psychophysiological insomniacs and psychiatric insomniacs would have more variability (as measured by slope changes) in power measures than controls. These power changes would occur in both absolute and relative power spectral analyses.

METHOD

Participants

Twenty-four participants were recruited through newspaper advertisements, posters in health centres and in the university. Participants were divided into three groups of eight, consisting of psychophysiological insomniacs, psychiatric insomniacs and a control group, and were age-matched (to two years) across groups. Group membership was determined according to scores on a health and history questionnaire, on the Sleep Disorders Questionnaire (Douglass et al., 1994), which screened for general sleep pathologies, on the Brock Sleep and Insomnia Questionnaire (Côté & Ogilvie, 1993), which helped identify insomnia subtypes, and on the Minnesota Multiphasic Personality Inventory-2 (MMPI-2), which screened for psychiatric stability. Screening criteria for the two insomniac groups were either sleep onset (latency of 45 minutes), maintenance (multiple awakenings), or early morning awakening insomnia on 3 or more nights per week with a duration of at least 6 months as reported on the Brock Sleep and Insomnia Questionnaire. Additional screening criteria for the psychophysiological insomniacs included a normal MMPI-2 profile, and scales within normal limits on the psychiatric scales of the Sleep Disorders Questionnaire and the Brock Sleep and Insomnia Questionnaire. Additional screening criteria for the psychiatric insomniacs were

elevations (above one standard deviation) on one or more clinical scales of the MMPI-2, and on the psychiatric scales of the Sleep Disorders Questionnaire and the Brock Sleep and Insomnia Questionnaire.

Screening criteria for the control group included a sleep onset latency of 20 minutes or less and no reported sleep difficulties. The control group also had a normal MMPI-2 profile, and scores within normal limits on the Sleep Disorders Questionnaire and Brock Sleep and Insomnia Questionnaire psychiatric scales. None of the participants was taking sleep medication or other drugs that interfered with sleep and none had any serious medical conditions that interfered with sleep. All participants had scores within normal limits on the Sleep Disorders Questionnaire Narcolepsy and Sleep Apnea scales and on the Brock Sleep and Insomnia Questionnaire Apnea scale. All participants received a \$30 honorarium for participation. Six participants (two from each group) were excluded because of excessive movement artifact on one or both nights of polysomnographic recording. The first group consisted of 3 men and 3 women who had psychophysiological insomnia (mean age 27.8, $sd = 10.28$), the second of 3 men and 3 women who had insomnia associated with psychiatric disorders (mean age 31.5, $sd = 10.21$) and the control group of 2 men and 4 women (mean age 27.8, $sd = 9.60$).

Apparatus

Data acquisition and analysis of EEG. All electrophysiological parameters were amplified on two 14-channel Nihon-Kohden (model 4314B and model A) electroencephalographs (EEG) and displayed and recorded using ACT Premium 386/25 and 486/33 computers, both with a 12-bit National Instruments A/D board (model APX-5200) and a 486/33 computer Microcomputer Quantitative Electrophysiology (Imaging Research Inc.). MQE is a computer program that allows data acquisition, scoring and Fast Fourier Transform (FFT) analysis of EEG activity. Data were backed up using a Panasonic optical disk drive.

Slope changes. AROUSE (Ogilvie, 1994), a custom designed DOS-based program was used to calculate slope changes (points at which continually increasing or decreasing changes in power were reversed) in power separately for each frequency band. FFT power spectrum output from the MQE program was the input for the AROUSE program. The sleep onset period record was divided into quartiles and the number of slope changes per quartile was calculated. The number of slope changes was taken to indicate the number of reversals of movement toward wakefulness or sleep. In order to reduce noise in the slope change data, only changes greater than 30% of one standard deviation of the power band being analyzed were considered in the analysis.

The participant's sleep room was illuminated by a red filtered 40W light source. The room was a three meter by three meter electrically shielded and sound attenuated bedroom equipped with a single bed, a dresser, a mirror, a closet, and a night table. Communication between the participant and the experimenter was possible through an intercom system in the participant's room. A Sony Trinitron monitor with split-screen capabilities and two RCA CCTV low illumination video cameras allowed simultaneous and continuous monitoring of both the participant and physiological measures during the sleep session.

Screening questionnaires. The MMPI-2 (Hathaway & Meehl, 1951) is a 567-item questionnaire designed to assess psychiatric problems. The clinical scales are: Hypochondriasis (Hs), Depression (D), Conversion hysteria (Hy), Psychopathic deviate (Pd), Masculinity-Femininity (Mf), Paranoia (Pa), Psychasthenia (Pt), Schizophrenia (Sc), Hypomania (Ma) and Social Introversion (Si). The validity scales include the Lie (L) scale which was designed to detect individuals who are trying to present themselves in a favourable light; the Infrequency (F) scale which was designed to detect individuals who are approaching test-taking in an atypical or deviant manner; and the Correction (K) scale which is designed to identify clinical defensiveness (Graham, 1987).

The Sleep Disorders Questionnaire (Douglass et al.,

1994) is a 175-item questionnaire is designed to predict the presence of narcolepsy, sleep apnea, periodic leg movement disorder and psychiatric DIMS, especially depression.

The Brock Sleep and Insomnia Questionnaire (Côté & Ogilvie, 1993) contains 175 items divided into 11 scales designed to assess sleep quality, sleep history, drug intake, and eight of the International Classification of Sleep Disorders of Initiating and Maintaining Sleep (DIMS) subtypes: DIMS associated with psychiatric disorders, psychophysiological insomnia, breathing disorders (e.g., apnea), movement disorders (e.g., restless leg syndrome, periodic limb movement disorder), hypnotic-dependent sleep disorder, idiopathic insomnia, delayed sleep phase syndrome and medically related DIMS.

The pre-sleep questionnaire screened for any unusual events, drug, alcohol and caffeine intake, daytime functioning and mood.

The post-sleep questionnaire assessed participants' sleep quality on the laboratory nights as well as an estimate of their sleep onset latency.

Procedure

Participants were given a complete tour of the Sleep Laboratory and a full orientation with respect to the procedure and apparatus involved in the study prior to signing consent. They were informed that the study was for research purposes only and not for treatment. In the first

phase of the study, each participant filled out a health and history questionnaire, the Brock Sleep and Insomnia Questionnaire, the MMPI-2 and the Sleep Disorders Questionnaire. Only participants who fell into the DIMS subtypes of interest, or qualified as a normal sleepers, were accepted for the second phase of the study, which consisted of two consecutive nights at the Sleep Lab. Participants kept a sleep diary for seven days prior to their nights at the Sleep Lab. They were instructed to refrain from drinking alcohol and taking naps, and to limit caffeine intake the day before and the days of scheduled nights in the Sleep Lab. On the Sleep Lab nights, participants came in two hours before their regular bed time. They were required to fill out a pre-sleep questionnaire.

Two central EEG (C3-A2 and C4-A2) and O2-A2 channels, two EOG channels (referenced to A2) and one bipolar submental EMG were used. Silver disk electrodes were filled with electrode cream and secured using either Micropore surgical tape or collodion-soaked gauze (for the scalp electrodes). Regular bedtimes and wake times were maintained. Each participant had his/her own room and comfort (e.g., pillow, room temperature) was respected as much as possible. Upon awakening in the morning, participants completed a post-sleep questionnaire.

Following the two nights of sleep assessment,

participants were given an information booklet that contained information on how to improve sleep hygiene and sleep quality. Participants also received information on psychological services available in the region.

Sleep Stages

Sleep stages were scored in 14-second epochs according to Rechtschaffen and Kales (1968) criteria.

FFT analyses from the sleep onset period

The sleep onset period was defined as lights-out to the first five minutes of stage 2 sleep.

Sleep stage analysis. The sleep onset period was analyzed in 14-second artifact-free epochs and sorted according to sleep/wake stage (wakefulness, stage 1, stage 2). Epochs within each stage bin were subjected to an FFT analysis. Root mean square power (absolute) for each of the five standard EEG bands (delta: 5.-4 Hz, theta: 4-8 Hz, alpha: 8-12 Hz, sigma: 12-15 Hz, beta: 15-25 Hz) was averaged within each bin. Relative power was also calculated as a ratio of each particular power band to the total power.

Consecutive epoch analysis. Each consecutive artifact-free epoch was FFT analyzed individually. Root mean square power (mV^2) for each of the five standard EEG bands was calculated for each epoch. These files were then read into the AROUSE program. The record was divided into quartiles and the jitter factor was set at 30% of each participant's

standard deviation for the particular power band being analyzed. The 30% value was selected empirically after preliminary analyses using higher and lower values. Average power and number of slope changes for each power band were calculated for each quartile.

Statistical Analyses

Standard sleep parameters. A one-way ANOVA with group as the independent variable was performed for all the standard sleep variables: sleep latency, percentage of each sleep stage, sleep efficiency, WASO, movement time and total sleep time.

Subjective/objective estimates of sleep onset latency. It was expected that the two insomniac groups would overestimate their sleep latency compared to EEG measures while the normal sleepers would either underestimate or correctly estimate their sleep latency. The relationship among the groups between subjective estimates of sleep onset latency from the post-sleep questionnaire and objective measures of sleep latency to stage 2 on night 2 (according to EEG criteria) was examined using hierarchical multiple regression.

Sleep onset period "stage" analyses. The sleep onset period analyses were designed to test the study's main hypotheses relating to beta, alpha and delta power. Power values for each frequency band from the stage analysis of the sleep onset period were analyzed separately. The

analyses consisted of 3(group) by 2(night) by 3(stage) ANOVAs (with night and stage as repeated measures factors) for each band both for absolute and relative power.

Sleep onset period "consecutive epoch" analyses. Power values and slope changes were again analyzed using mixed ANOVAs (night and quartiles were the repeated measures factors). A 3x2x4 (group by night by quartile) ANOVA was performed for each frequency band. Since the study's hypotheses related to the delta, alpha and beta power bands only, and no specific group differences were expected, significance levels for theta and sigma analyses were set at .01.

Results

Sample characteristics

Individuals were grouped according to scores on the screening questionnaires. One-way ANOVAs were performed on all questionnaire data (MMPI-2, Sleep Disorders Questionnaire and the Brock Sleep and Insomnia Questionnaire) in order to verify the validity of the groups. ANOVAs were followed by Scheffé tests set at $p = .05$. The mean ages of the three groups did not differ significantly. Table 1 lists T-scores (standardized scores: scale mean=50, sd=10) for the three groups on the MMPI-2. Overall, the psychiatric group showed higher scores on one of the validity scales and five of the ten clinical scales. They differed significantly from the other two groups on the

infrequency scale $F(2, 15) = 11.32, p=.001$ the hypochondriasis scale $F(2, 15) = 10.58, p=.001$, the depression scale $F(2, 15) = 34.49, p=.00001$, the schizophrenia scale $F(2, 15) = 10.86, p=.001$ and the social introversion scale $F(2, 15) = 4.23, p=.035$. They differed from the control group on the psychasthenia scale $F(2, 15) = 8.37, p=.004$

Table 1
 Mean T-Scores (standardized scores) of Psychophysiological Insomniacs, Psychiatric Insomniacs and Controls on the MMPI-2.

	Group		
	Psychophysiological Insomniacs	Psychiatric Insomniacs	Controls
n	6	6	6
Scale	Mean (SD)	Mean (SD)	Mean (SD)
Lie	54.0 (11.8)	51.0 (14.7)	45.3 (11.0)
Infrequency	46.8 (2.9) _b	60.5 (6.3) _a	48.0 (1.9) _b
Correction	52.0 (8.5)	47.3 (7.2)	54.5 (6.7)
1-Hypochondriasis	49.3 (8.8) _b	64.2 (5.7) _a	46.5 (6.5) _b
2-Depression	49.7 (7.6) _b	69.0 (4.9) _a	42.2 (4.2) _b
3-Conversion Hysteria	53.3 (10.2)	57.3 (13.5)	47.7 (8.8)
4-Psychopathic Deviate	45.8 (3.3)	62.0 (22.4)	48.3 (7.0)
5-Masculinity- femininity	51.6 (8.1)	52.3 (17.9)	54.2 (9.9)
6-Paranoia	53.0 (8.5)	58.3 (9.9)	48.7 (6.9)
7-Psychasthenia	57.5 (7.3)	69.2 (12.0) _a	47.8 (6.9) _b
8-Schizophrenia	49.3 (5.5) _b	67.7 (11.8) _a	48.3 (5.2) _b
9-Hypomania	56.0 (5.9)	54.5 (10.2)	55.5 (9.6)
10-Social Introversion	43.8 (8.7) _b	58.8 (16.6) _a	40.2 (8.2) _b

Note. Group means with different subscripts are significantly different at the .05 level.

Table 2a shows scores from the SDQ and the BSIQ scales. The scores of the three groups did not differ on the Sleep Apnea scale or the Narcolepsy scale of the SDQ. Both insomniac groups, however, showed significantly higher scores on the periodic limb movement $F(2, 15) = 11.17$, $p=.001$ scale than the control group. The psychiatric group showed higher scores on the psychiatric $F(2, 15) = 6.23$, $p=.011$ scale of the SDQ than the control group. The three groups did not differ on the BSIQ sleep apnea scale. The psychiatric group scored significantly higher on the BSIQ psychiatric scale $F(2, 15) = 24.65$, $p=.0001$ than the other two groups. The psychiatric group showed higher scores on the psychophysiological scale $F(2, 15) = 5.608$, $p=.015$ and on the periodic limb movement scale $F(2, 15) = 3.86$, $p=.044$ of the BSIQ than the control group. The psychophysiological insomniacs did not differ from the other two groups on these scales. The psychophysiological insomniac group reported a higher sleep onset latency $F(2, 14) = 19.374$, $p=.0001$ and a trend for more nightly awakenings $F(2, 15) = 2.747$, $p=.096$ on the BSIQ than the other two groups. Both insomniac groups reported greater difficulty in getting to sleep $F(2, 15) = 17.13$, $p=.0002$ as rated on a 5-point BSIQ scale than the control group.

Individual insomnia and psychological characteristics are listed in Table 2b for psychophysiological insomniac and psychiatric insomniacs.

Table 2a
 Mean Scores of Psychophysiological Insomniacs, Psychiatric Insomniacs and Controls on the Sleep Disorders Questionnaire (SDQ) and the Brock Sleep and Insomnia Questionnaire (BSIQ).

	Group		
	Psychophysiological Insomniacs	Psychiatric Insomniacs	Controls
n	6	6	6
Scales	Mean (SD)	Mean (SD)	Mean (SD)
SDQ Sleep Apnea	18.8 (1.9)	22.3 (6.1)	18.8 (4.5)
SDQ Narcolepsy	21.3 (3.7)	24.8 (4.9)	19.8 (3.1)
SDQ Periodic Limb Movement	22.7 (2.4) _b	22.2 (6.0) _b	12.3 (3.6) _a
SDQ Psychiatric	19.5 (5.9)	21.8 (1.5) _b	14.2 (2.9) _a
BSIQ Sleep Apnea	23.8 (12.4)	25.7 (9.0)	16.3 (6.7)
BSIQ Periodic Limb Movement	8.4 (1.9)	9.5 (2.1) _b	6.8 (1.0) _a
BSIQ Psychiatric	32.8 (7.0) _a	48.7 (5.0) _b	29.0 (3.7) _a
BSIQ Psycho-physiological	22.6 (3.0)	23.3 (4.3) _b	17.5 (2.3) _a
BSIQ Sleep Latency	90.0 (0) _a	38.8 (33.5) _b	13.8 (7.0) _b
BSIQ Nightly Awakenings	2.17 (2.1)	2.67 (1.88)	0.5 (0.6)
BSIQ Difficulty in Getting to Sleep	4.8 (0.4) _b	3.7 (1.2) _b	1.7 (0.8) _a

Note. Group means with different subscripts are significantly different at the .05 level.

Table 2b. Characteristics of psychophysiological insomniac and psychiatric insomniacs.

Group	Sleep Onset Insomnia	Sleep Maintenance Insomnia	Duration of Insomnia	SDQ Score PSYCH Scale	MMPI-2 Clinical Elevations (1 sd or more)	
1	PPI	yes	yes	1 year	12	-
2	PPI	yes	yes	10 years	14	-
3	PPI	yes	no	3 years	19	-
4	PPI	yes	no	2 years	22	-
5	PPI	yes	yes	15 years	28	-
6	PPI	yes	yes	12 years	22	-
7	PSY	yes	yes	2 years	22	Hs, D, Hy
8	PSY	no	yes	12 years	20	D
9	PSY	yes	yes	8 years	23	D, Sc, Si
10	PSY	no	yes	20+ years	20	D, Pd, Mf, Pa, Pt, Sc, Ma, Si
11	PSY	yes	no	6 years	23	Mf, Pt, Sc
12	PSY	yes	yes	20 years	23	Hs, D, Hy, Pt, Sc, Si

Note. PPI = psychophysiological insomniacs; PSY = psychiatric insomniacs; Hs= Hypochondriasis; D = Depression; Hy = Conversion Hysteria; Pd = Psychopathic Deviate; Mf = Masculinity-Femininity; Pa = Paranoia; Pt = Psychasthenia; Sc = Scizophrenia; Ma = Hypomania; Si = Social Introversion.

Standard sleep parameters

A 3(group) by 2(night) ANOVA of sleep onset latency revealed no significant differences in sleep onset latency to stage 1 among the groups as measured in the sleep laboratory. A 3x2 (group by night) ANOVA of latency to stage 2 revealed no significant group effect. However, there was a night effect for latency to stage 2, $F(1, 15) = 4.75$, $p=.046$, with mean latencies being higher on the first night. The means and standard deviations are presented in Table 3.

A series of one-way ANOVAs with group as the factor was conducted on a set of standard, visually-scored sleep/wake parameters. There were no significant differences in sleep stage (stages 1, 2, 3, 4 and REM) percentages, wakefulness after sleep onset, total sleep time, time in bed, sleep efficiency or movement time among the three groups. The means and standard deviations for these variables are presented in Table 3.

Table 3. Sleep variables by group.

	Group		
	Psychophysiological Insomniacs	Psychiatric Insomniacs	Controls
n	6	6	6
	Mean (SD)	Mean (SD)	Mean (SD)
Stage 1 Latency on Night 1	26.97 (19.30)	12.21 (11.76)	18.46 (13.95)
Stage 2 Latency on Night 1	53.36 (27.43)	25.57 (15.78)	28.80 (17.05)
Stage 1 Latency on Night 2	19.31 (13.33)	8.21 (3.37)	13.34 (9.88)
Stage 2 Latency on Night 2	27.30 (15.70)	24.06 (18.11)	24.96 (13.43)
Latency Estimate on Night 1	58.33 (39.77)	51.67 (26.20)	21.67 (6.83)
Latency Estimate on Night 2	43.33 (27.69)	40.83 (22.89)	16.67 (7.53)
Stage 1 %	16.25 (12.57)	10.83 (6.24)	10.33 (2.80)
Stage 2 %	57.41 (4.13)	57.83 (10.36)	57.33 (6.15)
Stage 3 %	9.34 (4.00)	8.17 (4.31)	9.0 (2.45)
Stage 4 %	1.62 (0.87)	5.33 (4.13)	3.50 (3.14)
REM %	21.50 (3.73)	18.00 (3.57)	19.67 (3.33)
Wake After Sleep Onset (minutes)	31.74 (24.12)	19.93 (26.08)	9.92 (5.96)
Total Sleep Time (minutes)	394.80 (28.20)	392.19 (65.18)	390.41 (52.04)
Time In Bed (minutes)	461.64 (24.79)	436.65 (50.47)	428.59 (50.61)
Sleep Efficiency	85.83 (6.11)	89.50 (7.56)	91.17 (3.54)

Movement Time	67.00	67.83	40.67
(# of epochs)	(36.37)	(51.48)	(8.76)

Subjective and objective estimates of sleep onset latency.

Sources of variation among the groups in the subjective estimate of sleep onset latency were investigated using hierarchical multiple regression. The criterion variable was the subjective estimate. Predictor variables were EEG sleep latencies (objective) and two dummy coded variables representing group differences. The first group variable contrasted insomniacs and controls while the second group variable contrasted the two insomniac groups. In order to assess the importance of group membership in determining subjective estimates, EEG estimates of sleep latency were partialled out in the first step of the analysis. The dummy coded group variables were then entered in step two. It was expected that group differences would account for variance in the subjective estimate over and above the variance accounted for by the EEG estimate. As can be seen in Table 4, this was the case. The first group variable (comparing insomniacs to normal sleepers) accounted for a significant proportion of variance in the subjective estimate (Step 2), over and above that accounted for by the EEG estimate (Step 1).

Table 4. Summary of Hierarchical Multiple Regression Analysis using EEG Estimates and Group Membership as Predictors of Subjective Sleep Onset Latency Estimate.

--Variable	B	SE B	β	t	p

--Step 1					
EEG latency	.76	.34	.48	2.49	.03
Step 2					
Group 1 (Both types of insomniacs vs. controls)	8.29	3.04	.51	2.72	.02
Group 2 (Psychophysiological vs. Psychiatric)	.05	5.29	.002	.01	.99

Note. $R^2 = .24$ for step 1, $p = .04$; $\Delta R^2 = .26$ for step 2, $p = .051$.

Sleep Onset Period Analyses

The sleep onset period in normal sleepers. Absolute EEG power changes in normal sleepers were first analyzed in order to characterize the normal sleep onset period. For the stage analysis, 2 (night) by 3 (stage) ANOVAS with repeated-measures on both factors were calculated separately for delta, theta, alpha, sigma and beta power. These changes are illustrated in Figure 1.

Analysis of the delta band revealed a significant stage effect, $F(2, 10) = 62.43$, $p=.0001$. Delta power increased from wakefulness to stage 1 and dramatically from stage 1 to stage 2. There were no night effects or interactions. Theta power showed a similar progression to delta power. Theta increased significantly across stages, $F(2, 10) = 20.23$, $p=.0001$, especially from stage 1 to stage 2.

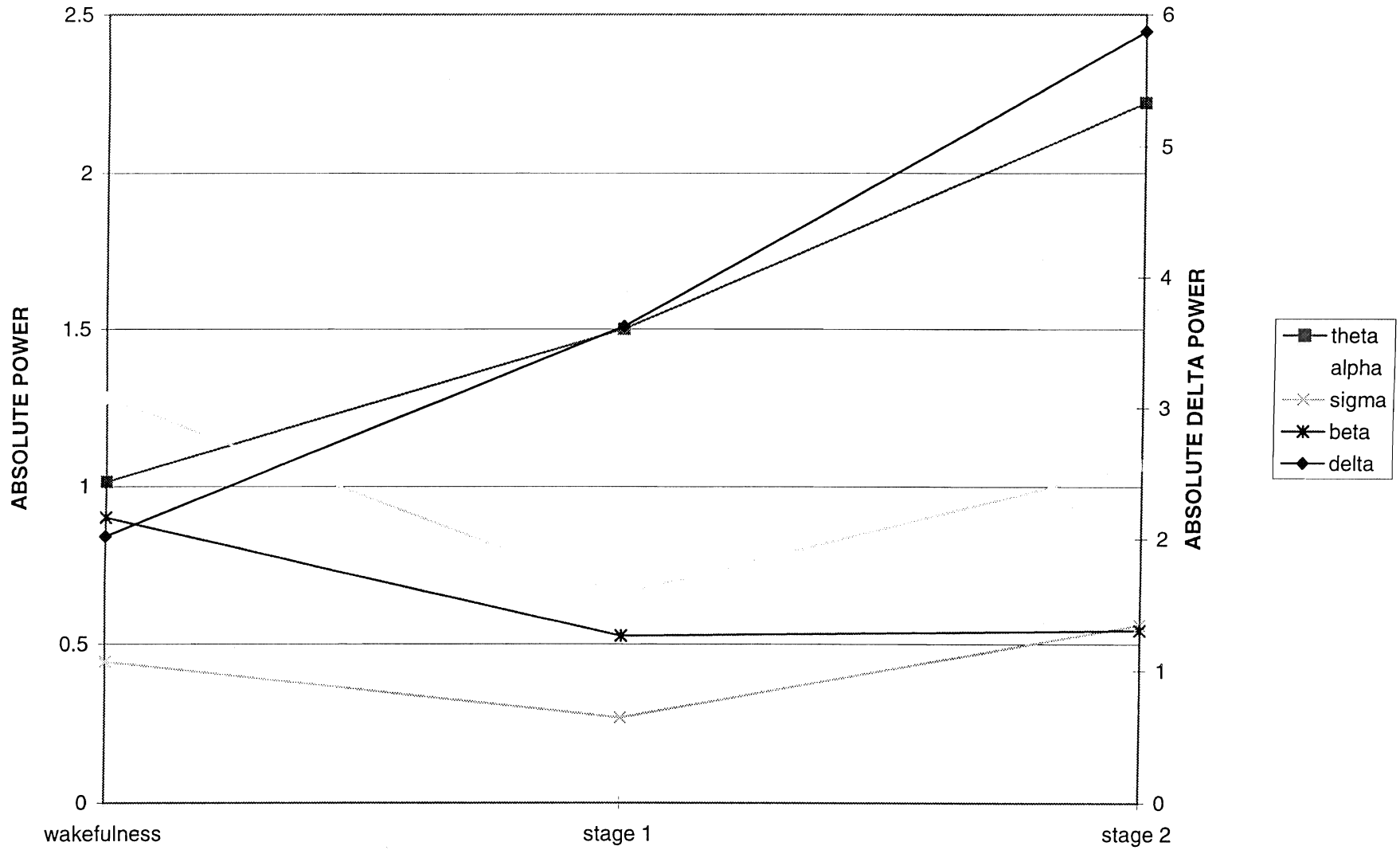
There were no significant effects for absolute alpha. Alpha power appeared to decrease from wakefulness to stage 1 then showed a slight increase from stage 1 to stage 2, though these changes did not reach statistical significance.

Analysis of sigma power revealed a significant main effect for stage, $F(2,10) = 4.22$, $p=.047$. Sigma decreased from wakefulness to stage 1 then increased from stage 1 to stage 2.

Beta power showed a significant main effect for stage, $F(2,10) = 6.64$, $p=.015$. Beta decreased from wakefulness to stage 1 and did not change from stage 1 to stage 2.

Figure 1

The sleep onset period in normal sleepers



Absolute power across stages. Power spectral analyses are presented separately for delta, theta, alpha, sigma and beta. Three (group) by 2 (night) by 3 (stage) ANOVAs with night and stage as repeated measures were performed for each band as well as for total power. Group main effects or group interactions were followed up by the Newman-Keuls test with alpha set at $p=.05$. The means and standard deviations are presented in Table 5 (night 1) and Table 6 (night 2).

There was a significant group effect for delta $F(2, 15) = 3.70, p=.049$, and a main effect for stage $F(2, 30) = 110.08, p=.0001$, as one would expect, with power values for delta increasing from wakefulness to stage 1 to stage 2. There was also a trend for a group by stage interaction $F(4, 30) = 2.43, p=.070$ and the group by night by stage interaction was significant, $F(4, 30) = 4.59, p=.005$. As can be seen in Figures 2a and 2b, delta power increased more in the psychiatric and control groups than it did in the psychophysiological insomniac group, and significantly so during stage 2 on night 2. Although reduced delta power in the psychophysiological insomniacs was expected for all stages (hypotheses 1 and 2), the delta power differences during stage 2 on night two were in the predicted direction.

There was a significant main effect for stage in the theta power band $F(2, 30) = 28.45, p=.0001$, with theta increasing from wakefulness to stage 1 to stage 2, as expected (see Figure 3).

Table 5. Mean absolute power in delta, theta, alpha, sigma and beta bands during wakefulness, stage 1 and stage 2 on night 1.

	Group		
	Psychophysiological Insomniacs	Psychiatric Insomniacs	Controls
	n=6 mean (sd)	n=6 mean (sd)	n=6 mean (sd)
Delta			
Wakefulness	2.36 (.74)	2.68 (.71)	2.29 (.51)
Stage 1	4.00 (1.54)	5.83 (2.52)	4.76 (1.65)
Stage 2	7.61 (2.32)	10.81 (3.82)	8.05 (2.32)
Theta			
Wakefulness	2.00 (.47)	2.09 (1.55)	1.69 (.80)
Stage 1	1.52 (.51)	3.16 (2.80)	2.06 (.62)
Stage 2	2.52 (.78)	3.49 (2.37)	2.56 (.78)
Alpha			
Wakefulness	1.45 (1.07)	4.59 (2.63)	4.92 (2.10)
Stage 1	.63 (.28)	1.23 (.70)	1.12 (.49)
Stage 2	.89 (.42)	1.23 (.52)	1.36 (.46)
Sigma			
Wakefulness	.51 (.26)	.41 (.16)	.57 (.24)
Stage 1	.31 (.17)	.29 (.12)	.49 (.25)
Stage 2	.68 (.36)	.81 (.60)	.87 (.38)
Beta			
Wakefulness	1.08 (.40)	.63 (.22)	1.08 (.70)
Stage 1	.64 (.45)	.38 (.12)	.72 (.58)
Stage 2	.56 (.62)	.25 (.04)	.93 (.73)

Table 6. Mean absolute power in delta, theta, alpha, sigma and beta bands during wakefulness, stage 1 and stage 2 on night 2.

	Group		
	Psychophysiological Insomniacs	Psychiatric Insomniacs	Controls
	n=6 mean (sd)	n=6 mean (sd)	n=6 mean (sd)
Delta			
Wakefulness	2.02 (.61)	2.59 (.68)	2.20 (.56)
Stage 1	3.62 (1.48)	5.90 (1.67)	4.54 (1.49)
Stage 2	5.87 (1.50)	9.95 (3.18)	9.99 (2.25)
Theta			
Wakefulness	1.02 (.51)	1.99 (1.34)	1.60 (.69)
Stage 1	1.50 (.46)	2.96 (2.55)	1.94 (.62)
Stage 2	2.22 (.62)	2.97 (1.82)	2.91 (1.18)
Alpha			
Wakefulness	1.28 (.97)	3.98 (2.2)	4.74 (2.89)
Stage 1	.66 (.34)	1.05 (.67)	.99 (.57)
Stage 2	1.07 (.67)	1.07 (.52)	1.47 (1.07)
Sigma			
Wakefulness	.45 (.30)	.38 (.17)	.52 (.17)
Stage 1	.27 (.16)	.29 (.11)	.44 (.24)
Stage 2	.56 (.28)	.61 (.31)	.98 (.53)
Beta			
Wakefulness	.91 (.52)	.60 (.20)	1.19 (.61)
Stage 1	.53 (.40)	.30 (.07)	.81 (.63)
Stage 2	.54 (.61)	.21 (.04)	.85 (.91)

Figure 2a

63

DELTA POWER ON NIGHT 1 AS A FUNCTION OF GROUP AND STAGE

—◆— PSYCHOPHYSIOLOGICAL INSOMNIACS —■— PSYCHIATRIC INSOMNIACS —▲— CONTROLS

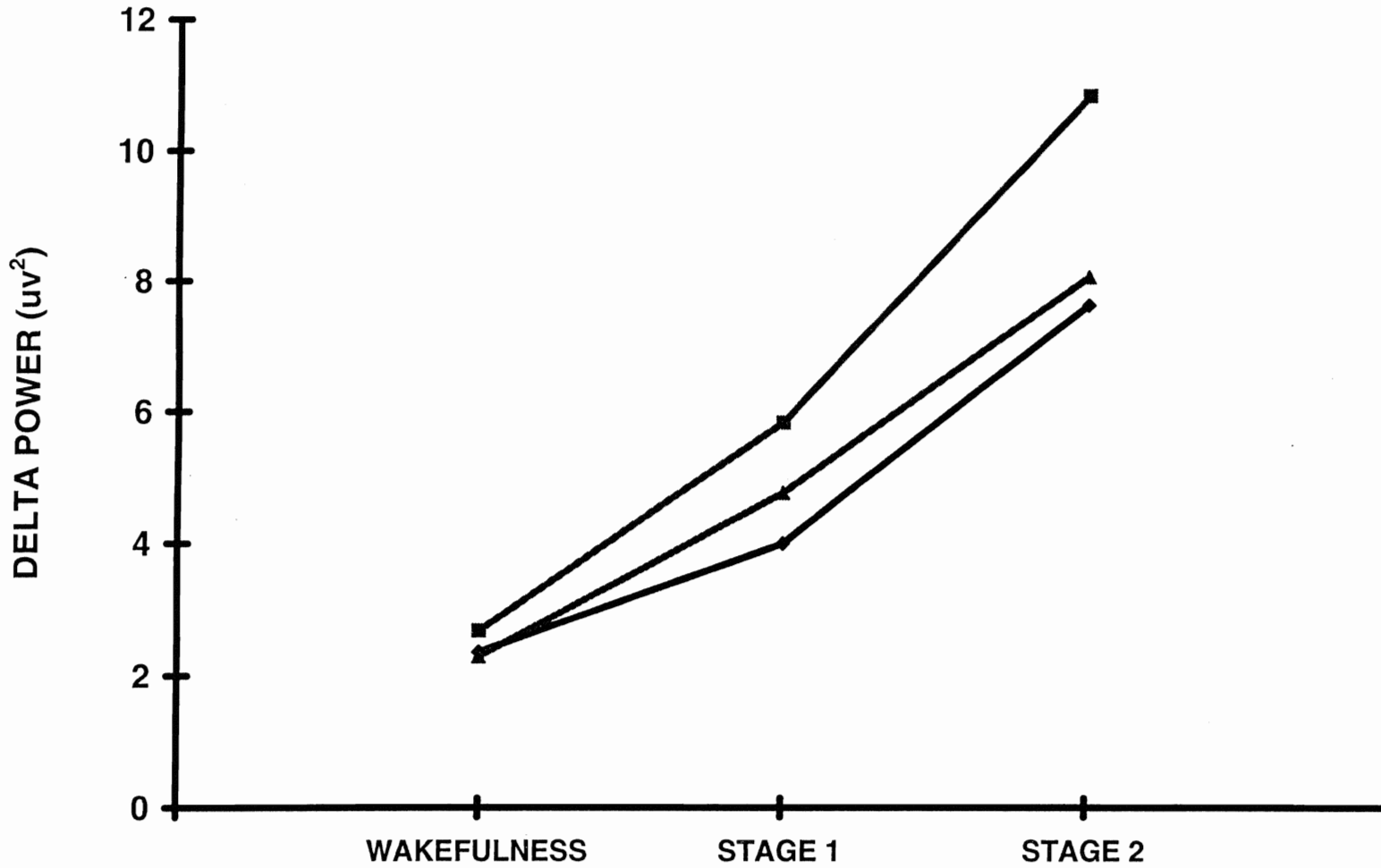


Figure 2b

64

DELTA POWER ON NIGHT 2 AS A FUNCTION OF GROUP AND STAGE

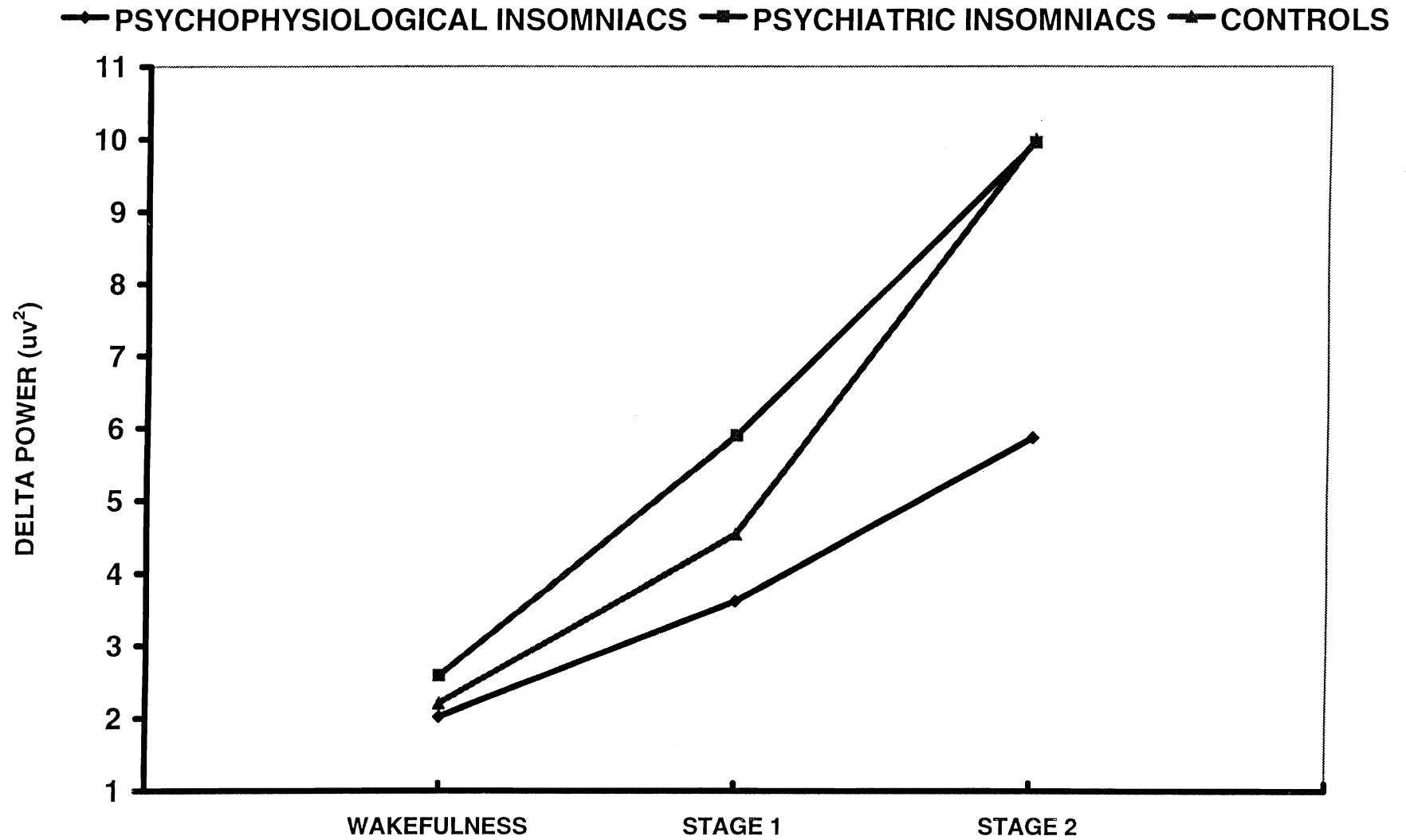
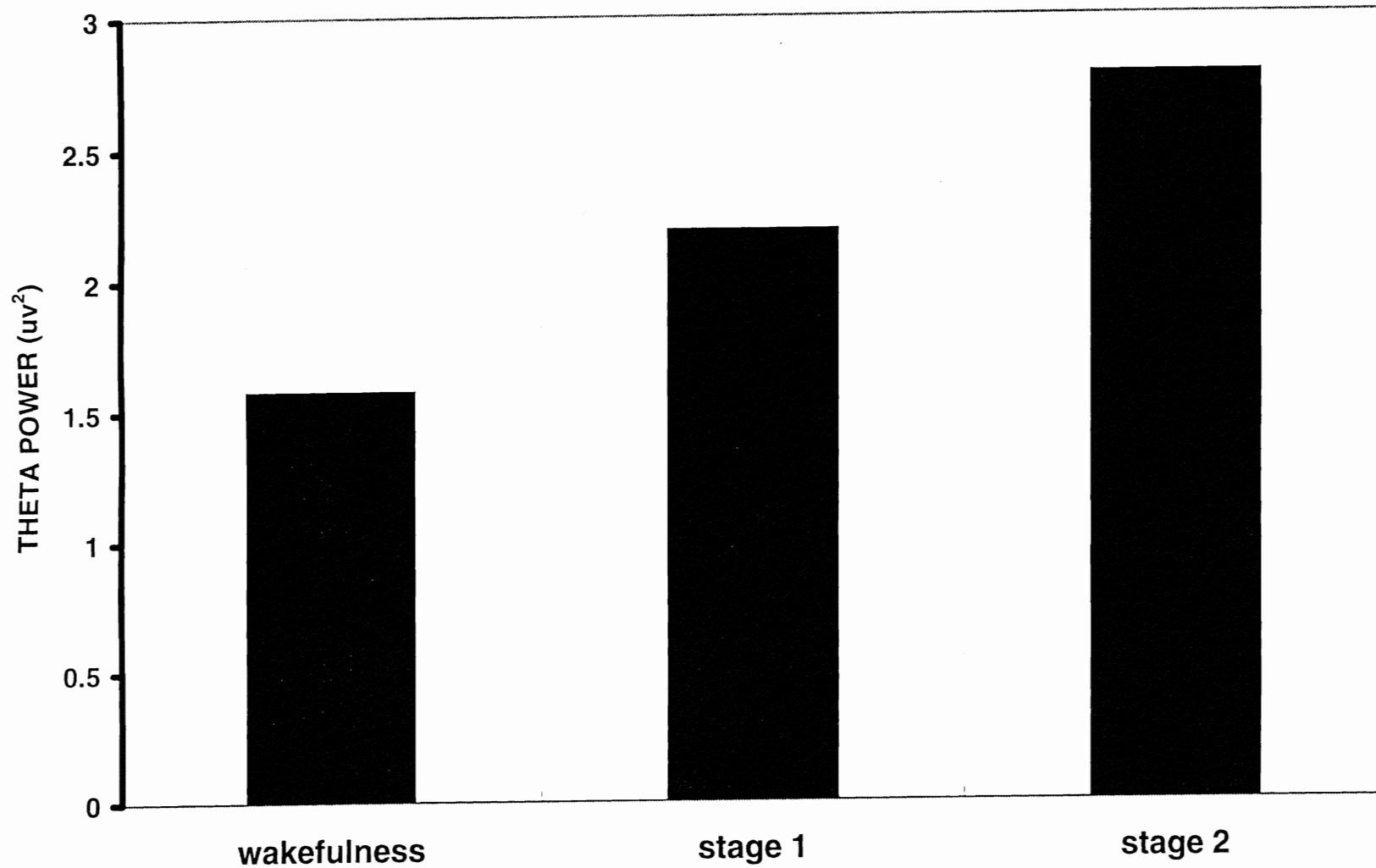


Figure 3

65

THETA POWER AS A FUNCTION OF STAGE



Analysis of the alpha band revealed significant main effects for group $F(2, 15) = 5.50$, $p=.016$ and stage $F(2, 30) = 25.48$, $p=.0001$. Figure 4 illustrates the predicted, significant group by stage interaction $F(4, 30) = 3.89$, $p=.012$. Psychophysiological insomniacs tended to have lower overall alpha power than the other two groups, but this difference was only significant between the controls and the psychophysiological insomniacs. This reduced alpha was only significant during wakefulness. Alpha levels were similar in all groups during stage 1 and stage 2.

Psychophysiological insomniacs did not show the dramatic drop in alpha power from wakefulness to stage 1 that the other two groups had.

There was a significant stage effect for the sigma power band $F(2, 30) = 15.94$, $p=.0001$. As can be seen in Figure 5, sigma power decreased from wakefulness to stage 1 then increased from stage 1 to stage 2 to higher levels than seen in wakefulness.

Contrary, to expectation, hypotheses 4 and 5 were not supported. Absolute beta power did not show any group differences. There was a significant stage effect for beta power $F(2, 30) = 17.59$, $p=.0001$. Beta power decreased from wakefulness to stage 1 then increased slightly from stage 1 to stage 2 (See Figure 6).

Figure 4

67

GROUP BY STAGE INTERACTION FOR ALPHA POWER

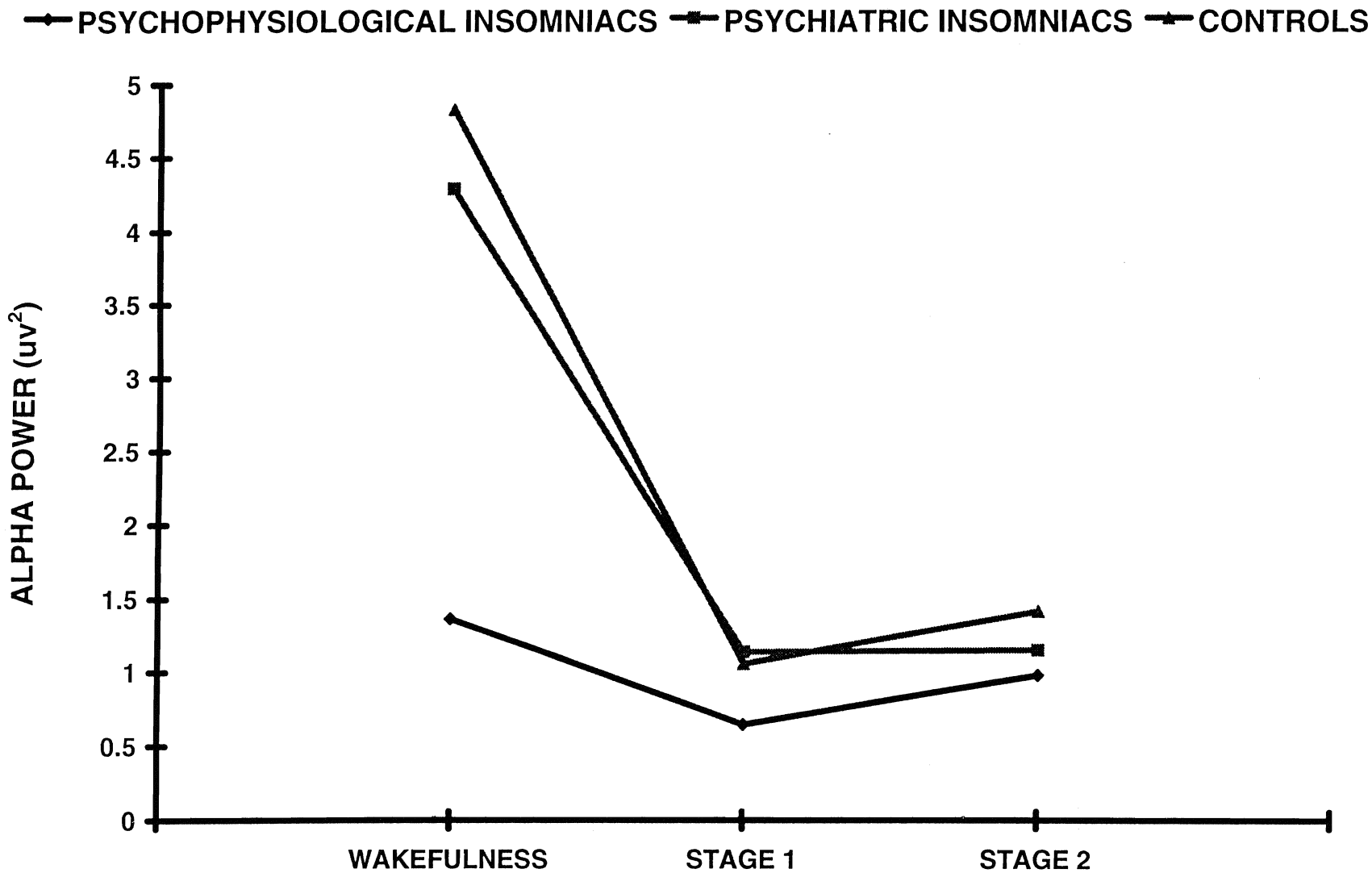


Figure 5

SIGMA POWER AS A FUNCTION OF STAGE

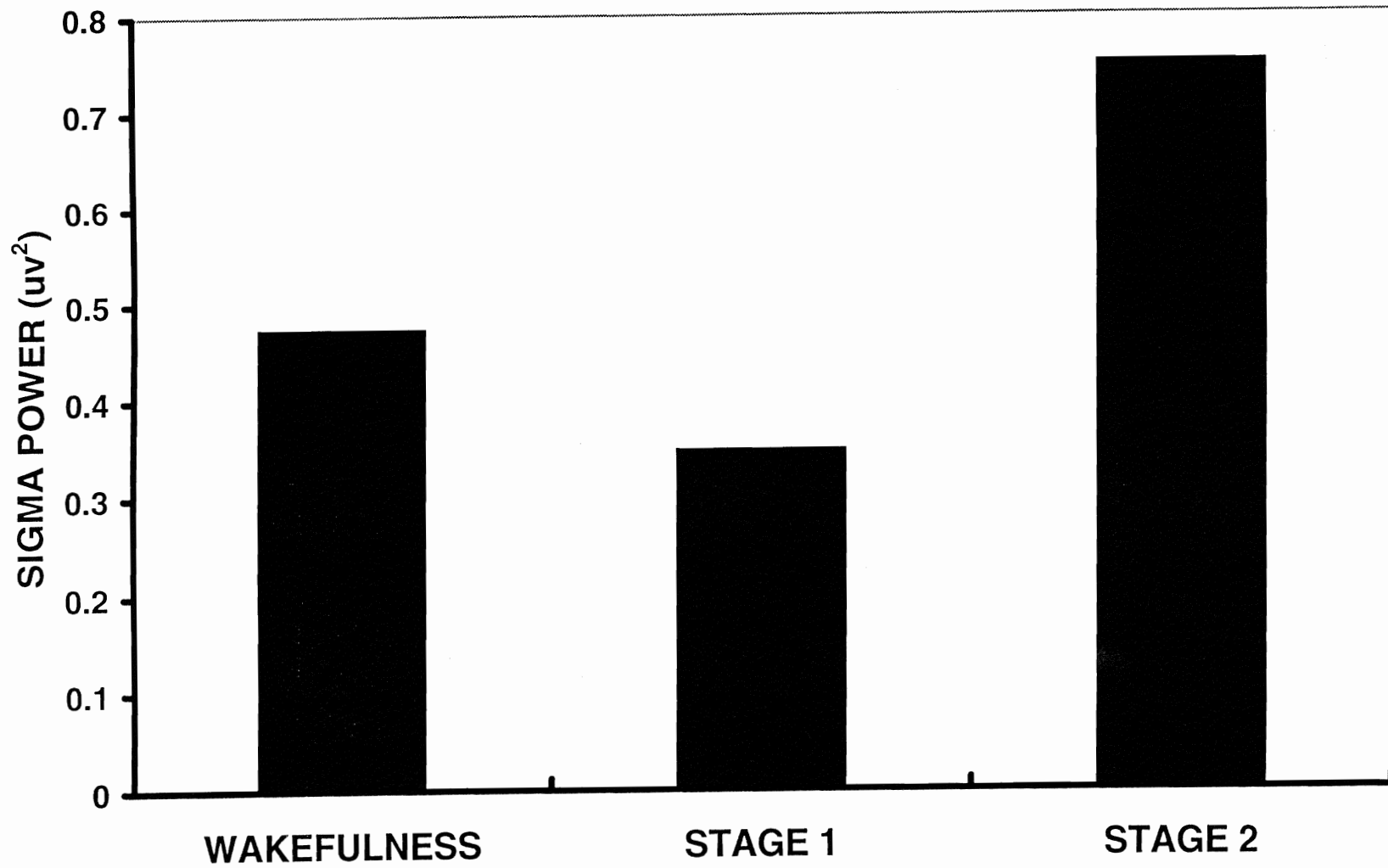
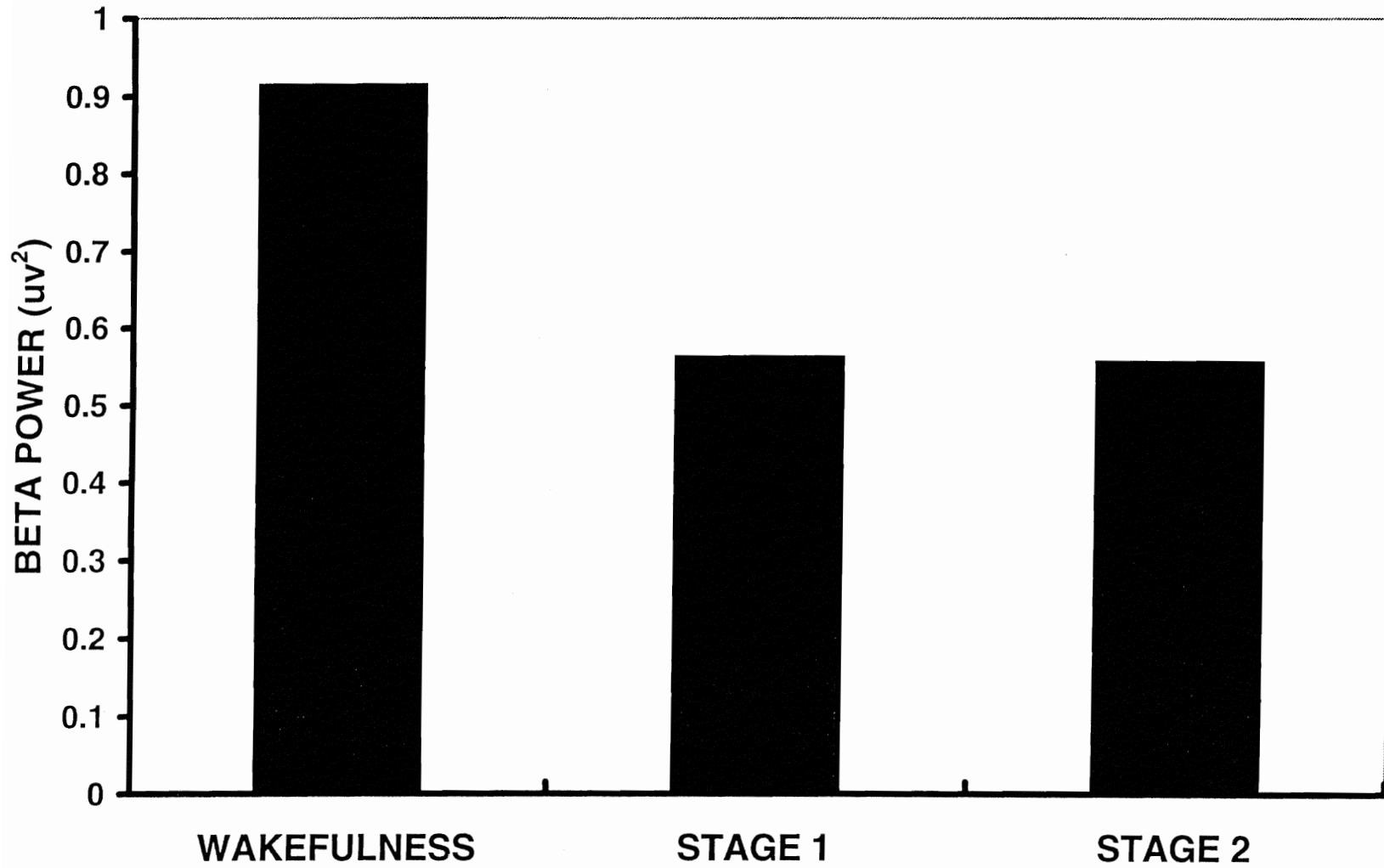


Figure 6

69

BETA POWER AS A FUNCTION OF STAGE



There was a significant group effect $F(2, 15) = 3.78$, $p=.047$ for total power. Psychophysiological insomniacs had reduced total power when compared to the other two groups. There was a significant stage effect $F(2, 30) = 49.87$, $p=.0001$, as well as a group by night by stage interaction effect for total power $F(4, 30) = 2.97$, $p=.035$ (see Figures 7a and 7b). Psychophysiological insomniacs had less total power during wakefulness on both nights, did not differ from the other groups during stage 1 and had less total power during stage 2 on night 2. All groups showed a dramatic increase in total power from stage 1 to stage 2.

Relative power across stages. Similar types of analyses were conducted to examine the bands as a percentage of total power. Group main effects or group interactions were followed up by Newman-Keuls post-hoc comparisons with alpha set at $p=.05$. Means and standard deviations are presented in Table 7 (night 1) and Table 8 (night 2).

A group by night by stage ANOVA for delta revealed a significant effect for stage $F(2, 30) = 94.89$, $p=.0001$ and a trend for an interaction between group and stage $F(4, 30) = 2.37$, $p=.075$. Psychophysiological insomniacs had significantly higher relative delta power during wakefulness, the three groups were comparable during stage 1 and the psychiatric insomniacs had higher relative delta power during stage 2.

Figure 7a

71

TOTAL POWER ON NIGHT 1 AS A FUNCTION OF GROUP AND STAGE

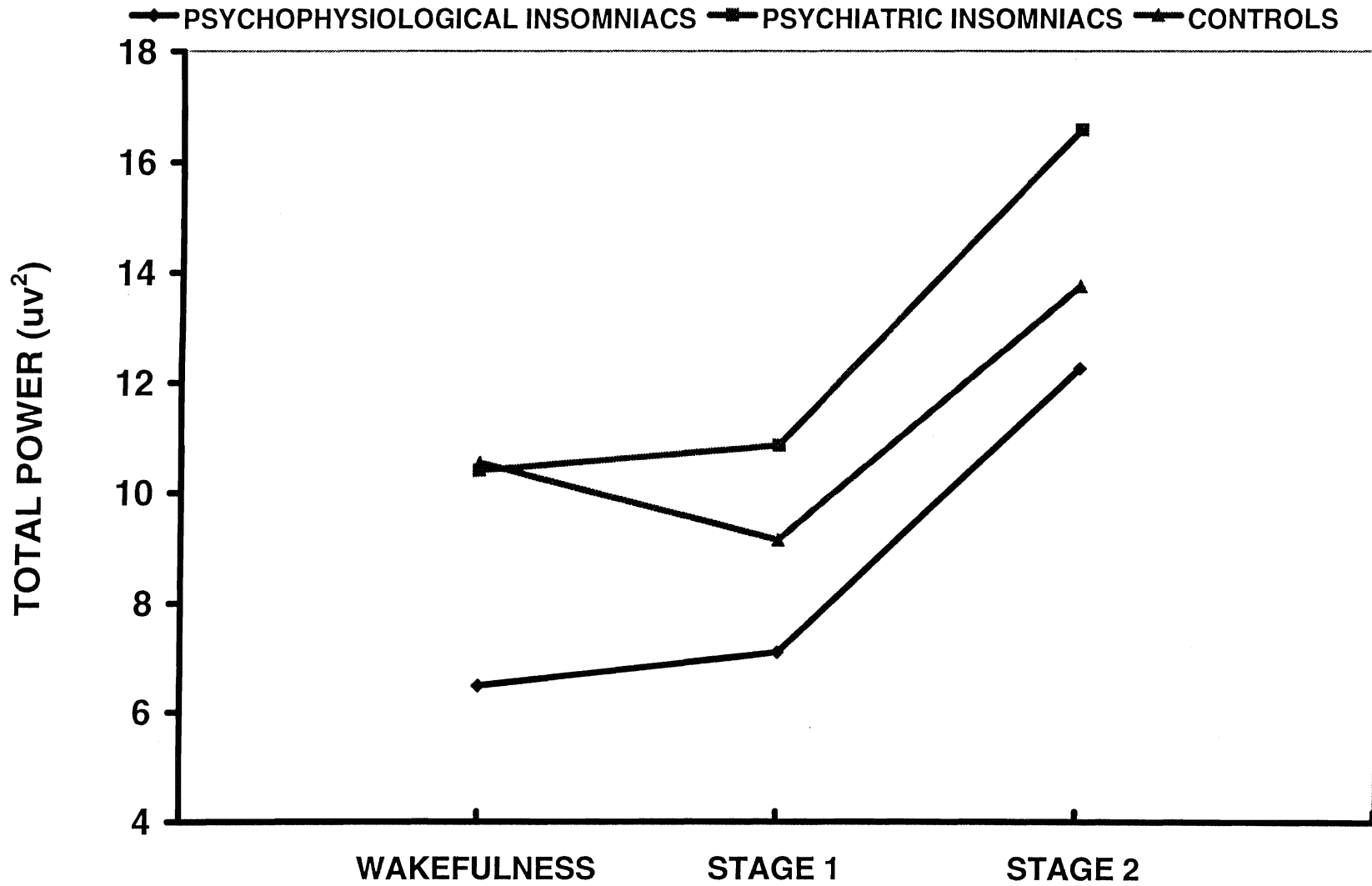


Figure 7b

72

TOTAL POWER ON NIGHT 2 AS A FUNCTION OF GROUP AND STAGE

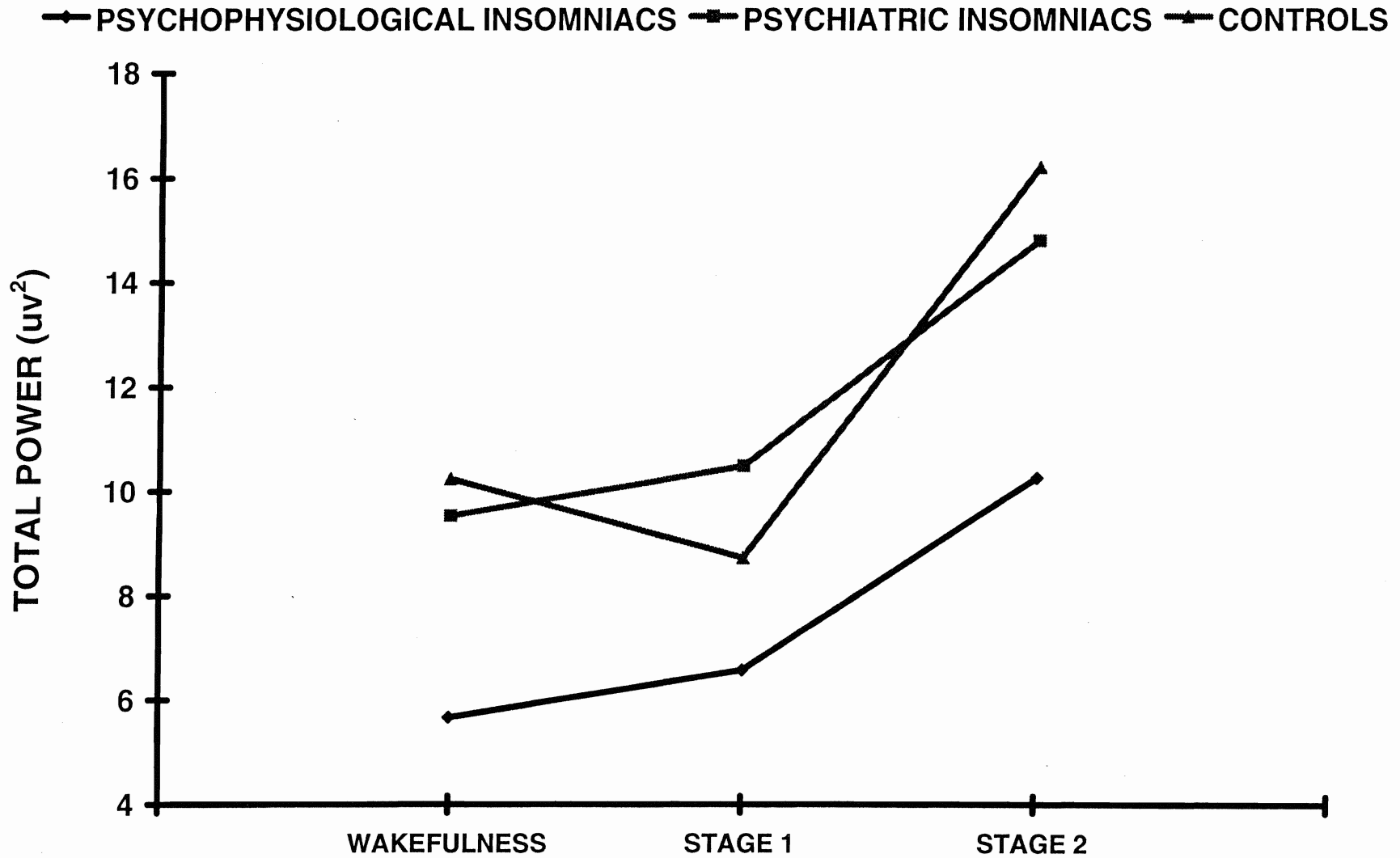


Table 7. Mean relative power in delta, theta, alpha, sigma and beta bands during wakefulness, stage 1 and stage 2 on night 1.

	Group		
	Psychophysiological Insomniacs	Psychiatric Insomniacs	Controls
	n=6 mean (sd)	n=6 mean (sd)	n=6 mean (sd)
Delta			
Wakefulness	.37 (.11)	.28 (.12)	.22 (.05)
Stage 1	.56 (.13)	.55 (.12)	.52 (.12)
Stage 2	.62 (.14)	.66 (.06)	.58 (.14)
Theta			
Wakefulness	.17 (.04)	.20 (.12)	.16 (.04)
Stage 1	.21 (.03)	.26 (.10)	.22 (.22)
Stage 2	.20 (.05)	.20 (.05)	.19 (.05)
Alpha			
Wakefulness	.21 (.12)	.42 (.22)	.46 (.10)
Stage 1	.09 (.05)	.12 (.06)	.12 (.04)
Stage 2	.07 (.04)	.08 (.03)	.10 (.04)
Sigma			
Wakefulness	.08 (.04)	.04 (.02)	.06 (.03)
Stage 1	.05 (.03)	.03 (.01)	.05 (.02)
Stage 2	.06 (.03)	.05 (.04)	.06 (.03)
Beta			
Wakefulness	.17 (.08)	.06 (.03)	.10 (.05)
Stage 1	.09 (.07)	.04 (.03)	.08 (.06)
Stage 2	.05 (.05)	.02 (.01)	.07 (.06)

Table 8. Mean relative power in delta, theta, alpha, sigma and beta bands during wakefulness, stage 1 and stage 2 on night 2.

	Group		
	Psychophysiological Insomniacs	Psychiatric Insomniacs	Controls
	n=6 mean (sd)	n=6 mean (sd)	n=6 mean (sd)
Delta			
Wakefulness	.38 (.11)	.29 (.13)	.23 (.06)
Stage 1	.55 (.12)	.59 (.10)	.53 (.12)
Stage 2	.58 (.11)	.68 (.05)	.63 (.12)
Theta			
Wakefulness	.18 (.04)	.20 (.10)	.16 (.02)
Stage 1	.23 (.03)	.26 (.09)	.22 (.03)
Stage 2	.22 (.04)	.19 (.04)	.18 (.04)
Alpha			
Wakefulness	.21 (.11)	.40 (.20)	.43 (.12)
Stage 1	.10 (.04)	.10 (.04)	.11 (.04)
Stage 2	.10 (.06)	.07 (.02)	.09 (.04)
Sigma			
Wakefulness	.08 (.04)	.04 (.02)	.06 (.03)
Stage 1	.04 (.02)	.03 (.01)	.05 (.03)
Stage 2	.05 (.02)	.04 (.03)	.06 (.03)
Beta			
Wakefulness	.16 (.09)	.06 (.02)	.13 (.07)
Stage 1	.08 (.06)	.03 (.01)	.09 (.06)
Stage 2	.05 (.05)	.01 (.01)	.05 (.04)

There was a significant stage effect for theta $F(2, 30) = 17.59$, $p=.0001$. Theta increased across stages for all three groups.

Analysis of the alpha relative power revealed a trend for a group effect $F(2, 15) = 3.5$, $p=.057$, a significant stage effect $F(2, 30) = 62.81$, $p=.0001$ and the predicted, significant group by stage interaction $F(4, 30) = 4.95$, $p=.003$. As can be seen in Figure 8, alpha relative power showed the same pattern as seen for alpha absolute power, that is, psychophysiological insomniacs had reduced alpha power during wakefulness, but the three groups were not significantly different during stages 1 and 2.

There were no significant effects for sigma.

The beta 3 (group) by 2(night) by 3(stage) ANOVA revealed the predicted trend for the group effect $F(2, 15) = 2.97$, $p=.082$, a significant stage effect $F(2, 30) = 35.54$, $p=.0001$, a significant group by stage interaction $F(4, 30) = 3.48$, $p=.019$, and a significant group by night by stage interaction $F(4, 30) = 4.63$, $p=.005$. During wakefulness, psychophysiological insomniacs had higher relative beta power followed by controls and psychiatric insomniacs, supporting hypothesis 4. The latter had lower relative beta power but only the insomniac groups differed significantly from each other during wakefulness on night 1 (see Figure 9a) and showed a similar trend during wakefulness on night 2 (see Figure 9b).

Figure 8

76

RELATIVE ALPHA POWER AS A FUNCTION OF GROUP AND STAGE

—◆— PSYCHOPHYSIOLOGICAL INSOMNIACS —■— PSYCHIATRIC INSOMNIACS —▲— CONTROLS

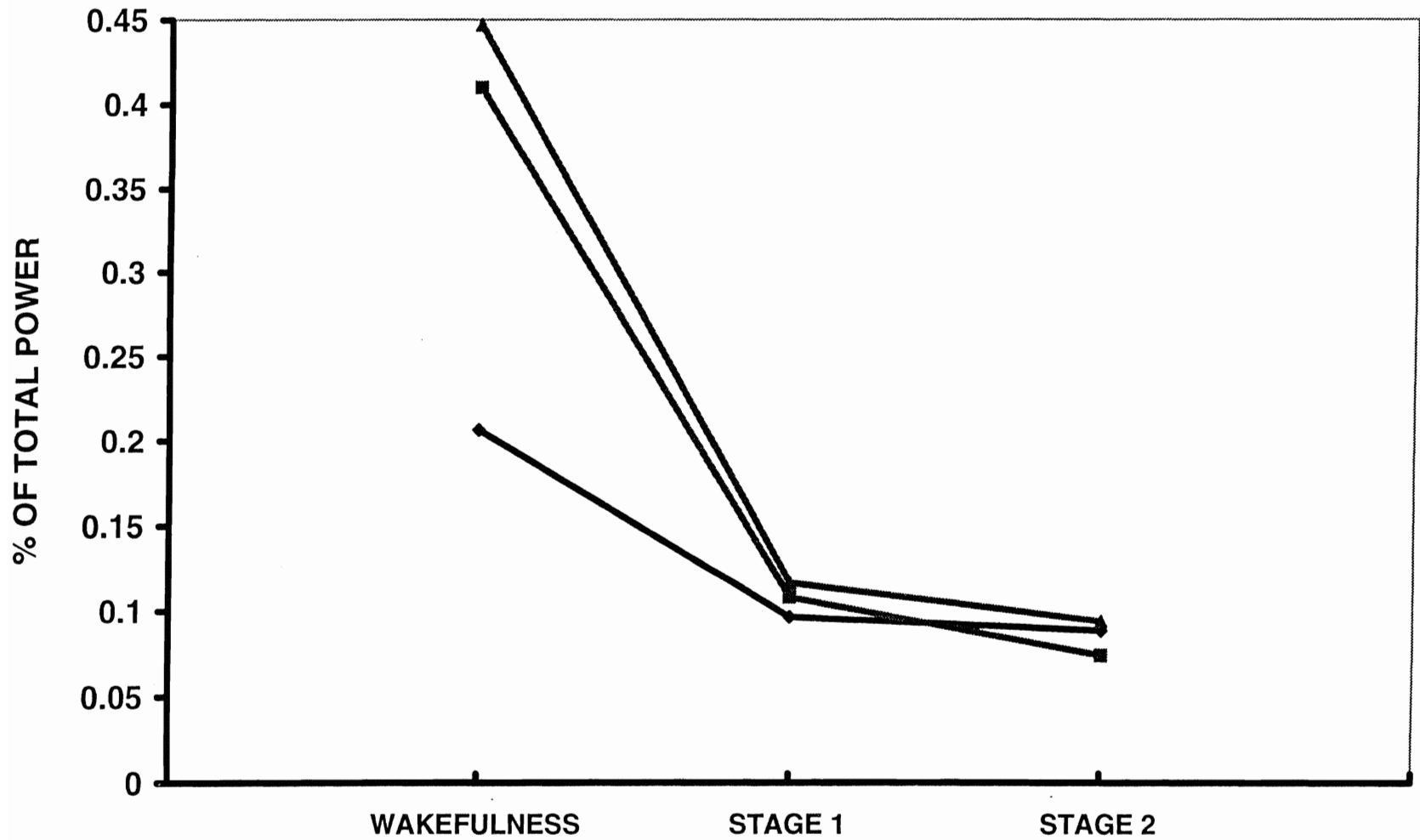


Figure 9a

77

RELATIVE BETA POWER ON NIGHT 1 AS A FUNCTION OF GROUP AND STAGE

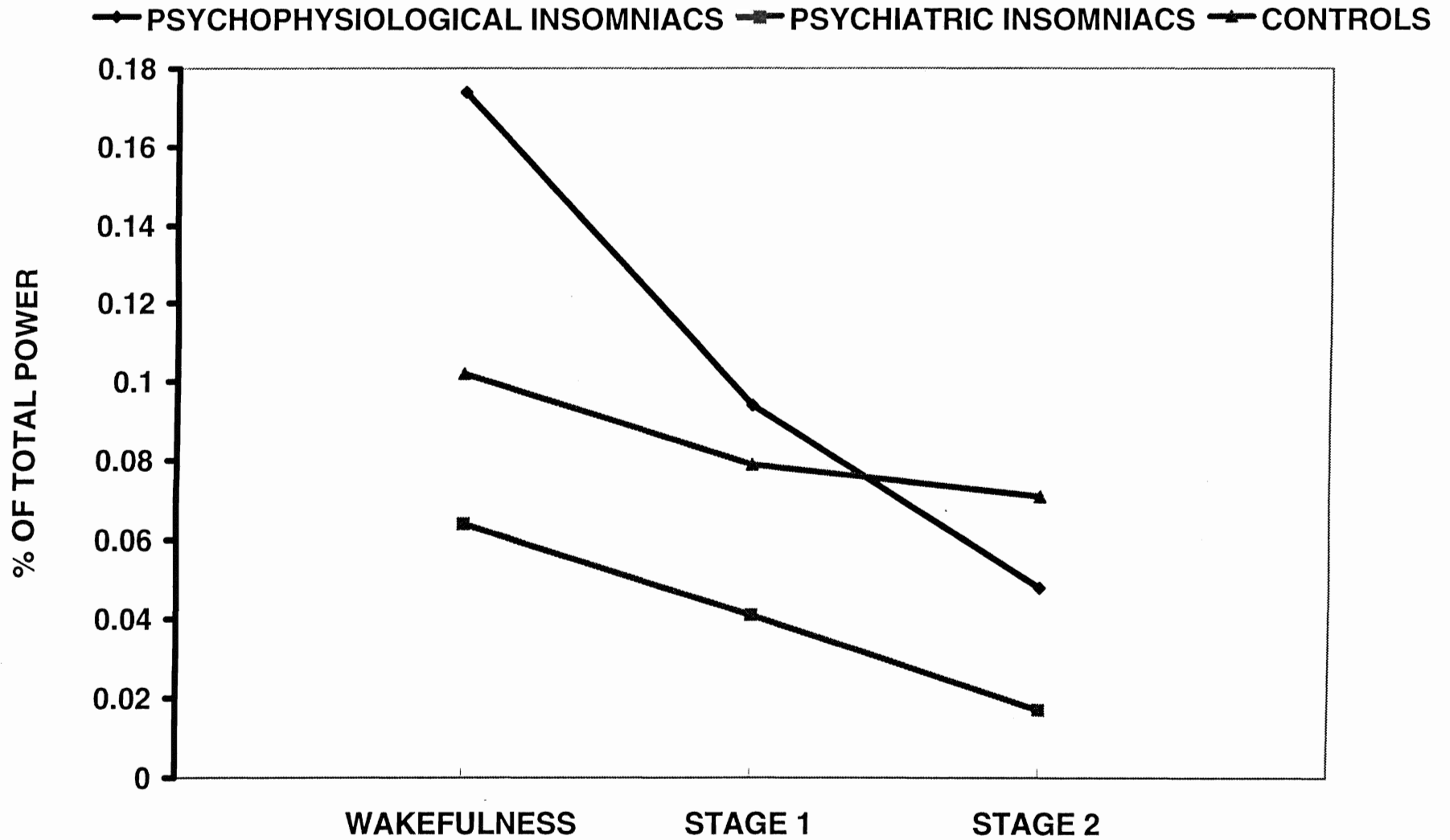
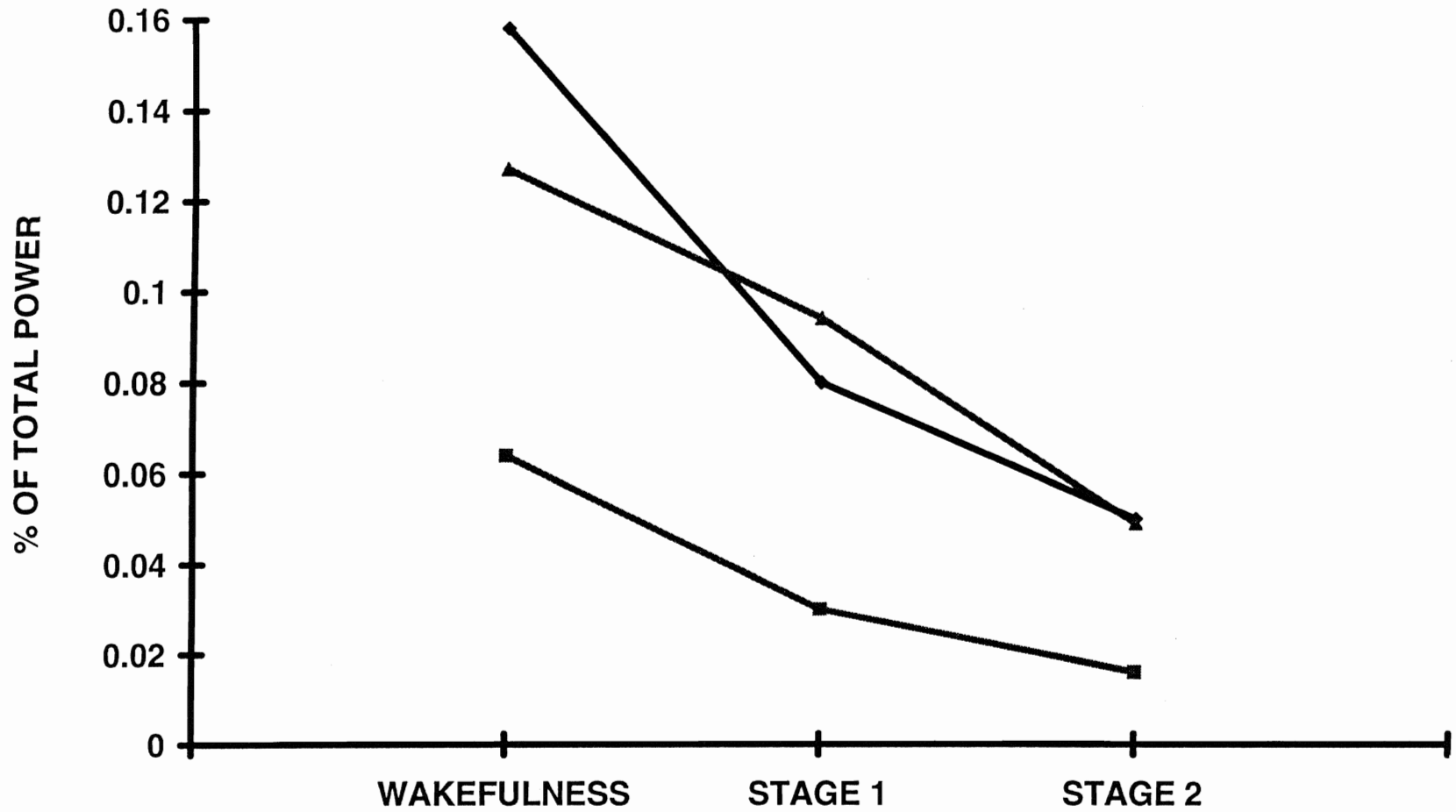


Figure 9b

78

RELATIVE BETA POWER ON NIGHT 2 AS A FUNCTION OF GROUP AND STAGE

◆ PSYCHOPHYSIOLOGICAL INSOMNIACS ■ PSYCHIATRIC INSOMNIACS ▲ CONTROLS



Absolute power across quartiles of the sleep onset period. For the following set of analyses, the sleep onset period was divided into quartiles in order to study temporal changes in absolute power across the sleep onset period. The quartiles represent four successive temporal divisions which were used to preserve order effects while also allowing for differences in the lengths of the sleep onset periods. A series of 3(group) by 2(night) by 4(quartile) ANOVAs with night and division as repeated measures factors were calculated separately for each power band. Group main effects or interactions were followed by Newman-Keuls post-hoc analyses with a probability level set at .05. Group means and standard deviations are presented in Table 9 (night 1) and Table 10 (night 2).

There was a significant quartile effect $F(3, 45) = 54.83$, $p=.0001$ and a significant group by quartile interaction for mean delta power $F(6, 45) = 2.37$, $p=.045$, summarized in Figure 10. Psychophysiological insomniacs did not show as consistent increases in delta power across the first three quartiles as did the other two groups. Although all three groups showed an increase in delta power from the third to the last quartile of the sleep onset period, the psychophysiological insomniacs had lower delta power values than did the other two groups.

Table 9. Mean absolute power in delta, theta, alpha, sigma and beta bands across quartiles of the sleep onset period on night 1.

	Group		
	Psychophysiological Insomniacs	Psychiatric Insomniacs	Controls
	n=6 mean (sd)	n=6 mean (sd)	n=6 mean (sd)
Delta			
1st Quartile	3.51 (1.44)	3.09 (.10)	2.64 (.57)
2nd Quartile	4.22 (2.02)	6.87 (6.08)	4.03 (1.39)
3rd Quartile	4.40 (1.15)	6.99 (3.24)	6.59 (2.44)
4th Quartile	7.52 (3.16)	9.89 (3.72)	9.33 (3.28)
Theta			
1st Quartile	1.12 (.52)	2.00 (1.66)	1.50 (.59)
2nd Quartile	1.36 (.74)	2.60 (1.92)	1.69 (.76)
3rd Quartile	1.36 (.56)	2.92 (2.0)	2.09 (.94)
4th Quartile	1.96 (.61)	3.12 (2.30)	2.22 (.99)
Alpha			
1st Quartile	1.07 (.69)	4.01 (2.38)	4.65 (3.01)
2nd Quartile	.86 (.45)	2.92 (2.21)	2.21 (1.07)
3rd Quartile	.92 (.49)	2.11 (1.33)	1.33 (.89)
4th Quartile	.74 (.27)	1.07 (.43)	1.19 (.77)
Sigma			
1st Quartile	.39 (.20)	.34 (.14)	.41 (.13)
2nd Quartile	.43 (.24)	.30 (.13)	.51 (.16)
3rd Quartile	.44 (.20)	.55 (.54)	.43 (.19)
4th Quartile	.45 (.14)	.60 (.34)	.54 (.23)

Beta

1st Quartile	.86 (.32)	.58 (.22)	.76 (.33)
2nd Quartile	.69 (.43)	.46 (.18)	.60 (.29)
3rd Quartile	.63 (.22)	.37 (.15)	.55 (.28)
4th Quartile	.46 (.25)	.24 (.08)	.39 (.21)

Table 10. Mean absolute power in delta, theta, alpha, sigma and beta bands across quartiles of the sleep onset period on night 2.

	Group		
	Psychophysiological Insomniacs	Psychiatric Insomniacs	Controls
	n=6 mean (sd)	n=6 mean (sd)	n=6 mean (sd)
Delta			
1st Quartile	3.06 (.58)	3.06 (1.14)	2.36 (.65)
2nd Quartile	2.75 (.59)	4.44 (3.05)	2.82 (1.19)
3rd Quartile	3.85 (1.71)	6.82 (1.97)	5.03 (3.34)
4th Quartile	6.26 (1.78)	9.32 (2.28)	8.43 (2.06)
Theta			
1st Quartile	.93 (.38)	1.93 (1.51)	1.43 (.62)
2nd Quartile	1.08 (.61)	2.50 (2.21)	1.41 (.68)
3rd Quartile	1.42 (.64)	2.62 (2.29)	1.62 (.85)
4th Quartile	1.83 (.49)	2.71 (2.02)	2.17 (1.31)
Alpha			
1st Quartile	1.04 (.71)	4.27 (2.81)	5.89 (3.90)
2nd Quartile	1.04 (.73)	3.00 (2.06)	3.77 (2.27)
3rd Quartile	.86 (.41)	1.15 (.72)	1.23 (.57)
4th Quartile	.79 (.34)	1.12 (.51)	.91 (.25)
Sigma			
1st Quartile	.33 (.21)	.33 (.16)	.36 (.13)
2nd Quartile	.37 (.27)	.52 (.47)	.41 (.13)
3rd Quartile	.33 (.14)	.39 (.17)	.32 (.14)
4th Quartile	.41 (.11)	.57 (.31)	.52 (.23)

Beta

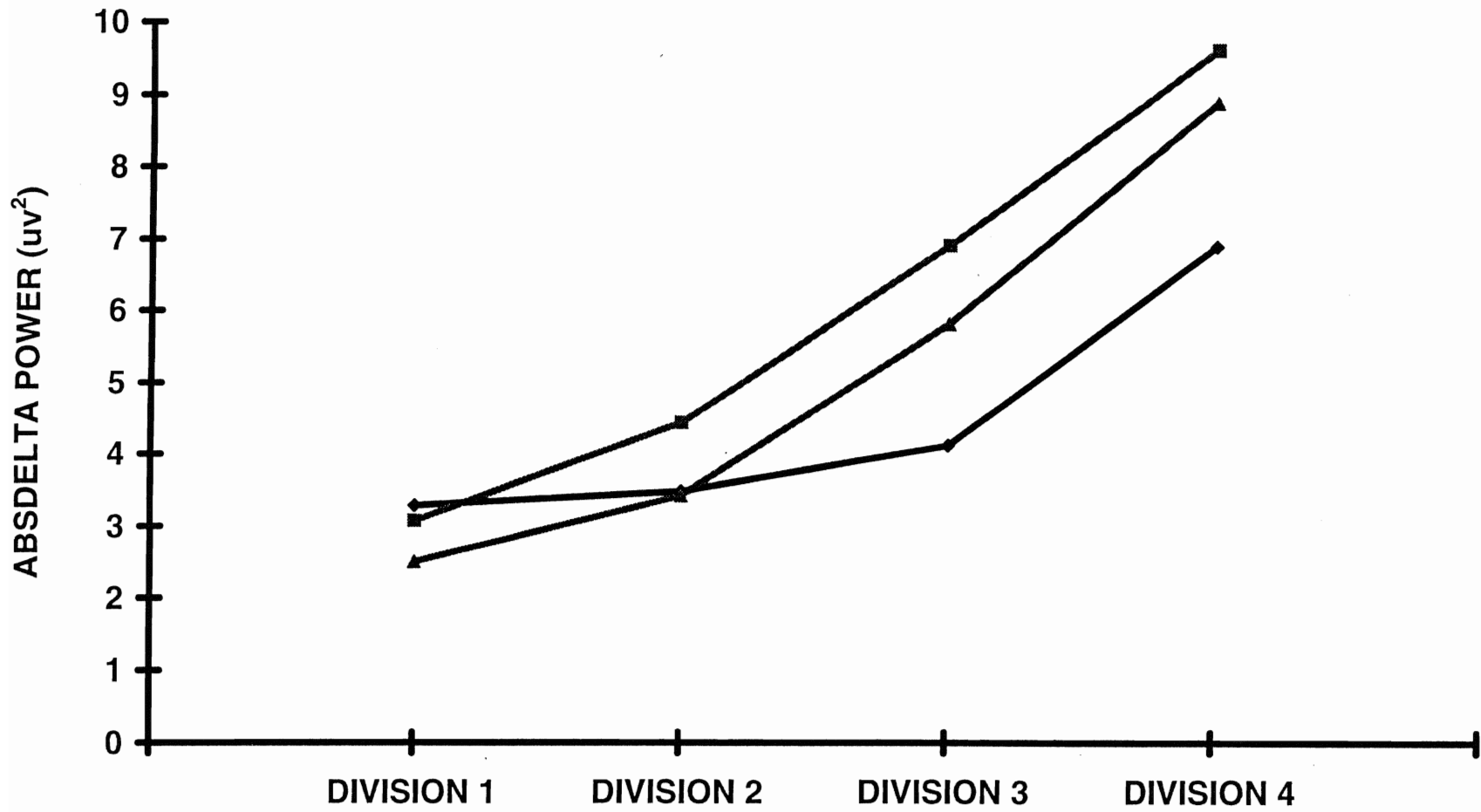
1st Quartile	.76 (.45)	.55 (.25)	.76 (.29)
2nd Quartile	.72 (.45)	.41 (.19)	.69 (.38)
3rd Quartile	.57 (.25)	.25 (.07)	.51 (.31)
4th Quartile	.27 (.14)	.19 (.09)	.36 (.19)

Figure 10

84

ABSOLUTE DELTA POWER ACROSS THE SLEEP ONSET PERIOD AS A FUNCTION OF GROUP

◆ PSYCHOPHYSIOLOGICAL INSOMNIACS ■ PSYCHIATRIC INSOMNIACS ▲ CONTROLS



There was a significant quartile effect for theta power $F(3, 45) = 20.01$, $p=.0001$. Power increased across the sleep onset period in all groups, marking theta as a key index to sleep onset period changes.

There was a significant group effect for alpha $F(2, 15) = 5.41$, $p=.017$, a significant quartile effect $F(3, 45) = 16.34$, $p=.0001$ and as predicted, a significant group by quartile interaction $F(6, 45) = 3.75$, $p=.004$. As seen in Figure 11, alpha power in the psychophysiological insomniacs did not show any changes across quartiles while psychiatric insomniacs and controls showed a sharp decrease, especially across the first three quartiles.

There were no significant effects for sigma power.

There was a significant quartile effect for beta power $F(3, 45) = 24.31$, $p=.0001$. Beta power values decreased across the sleep onset period for all groups, but did not vary significantly among groups.

Slope changes in power bands during the sleep onset period. Slope changes were calculated for delta, theta, alpha, sigma and beta. The AROUSE program allowed the sleep onset period record to be divided into quartiles as well as permitting the selection of a jitter or noise factor, that is, which EEG changes would be regarded as actual slope changes or simply as "noise". Since slope changes would necessarily be affected by the length of the sleep onset period, and the sleep onset periods were not of equal length

in all participants, slope changes were analyzed as a proportion of the number of epochs in each quartile (each sleep onset period was separated into successive time periods). A series of 3x2x4 (group by night by quartile) ANOVAs with night and division as repeated measures factors were calculated separately for each power band. Group main effects or interactions were followed by Newman-Keuls post-hoc analyses with a probability level set at .05. Means and standard deviations for slope changes are presented in Table 11 (night 1) and Table 12 (night 2).

There was a significant night effect for delta slope changes $F(1, 15) = 6.21, p=.025$ and a significant group by night interaction for delta slope changes $F(2, 15) = 4.37, p=.032$. As can be seen in Figure 12, both insomniac groups showed a smaller proportion of slope changes on night 1 than on night 2, while the controls showed a higher proportion of slope changes on night 1 than on night 2. There was also a significant quartile effect for delta slope changes $F(3, 45) = 57.86, p=.0001$, with delta slope changes increasing consistently across the quartile of the sleep onset period.

There was significant quartile effect for theta $F(3, 45) = 20.09, p=.0001$. Theta slope changes increased across quartiles of the sleep onset period for all groups.

There was a significant group effect for alpha slope changes $F(2, 15) = 3.98, p=.041$. As can be seen in Figure 13, psychophysiological insomniacs had a higher proportion

of alpha slope changes than the other two groups. There was also significant quartile effect $F(3, 45) = 2.85, p=.048$, with alpha slope changes decreasing across the sleep onset period, most dramatically from the second to the third quartile.

There was also a quartile effect for sigma $F(3, 45) = 14.36, p=.0001$. Sigma slope changes increased dramatically from the first to the second quartile of the sleep onset period and again from the third to the fourth quartile.

There were no significant effects for beta slope measures.

Figure 11

88

ALPHA POWER ACROSS THE SLEEP ONSET PERIOD AS A FUNCTION OF GROUP

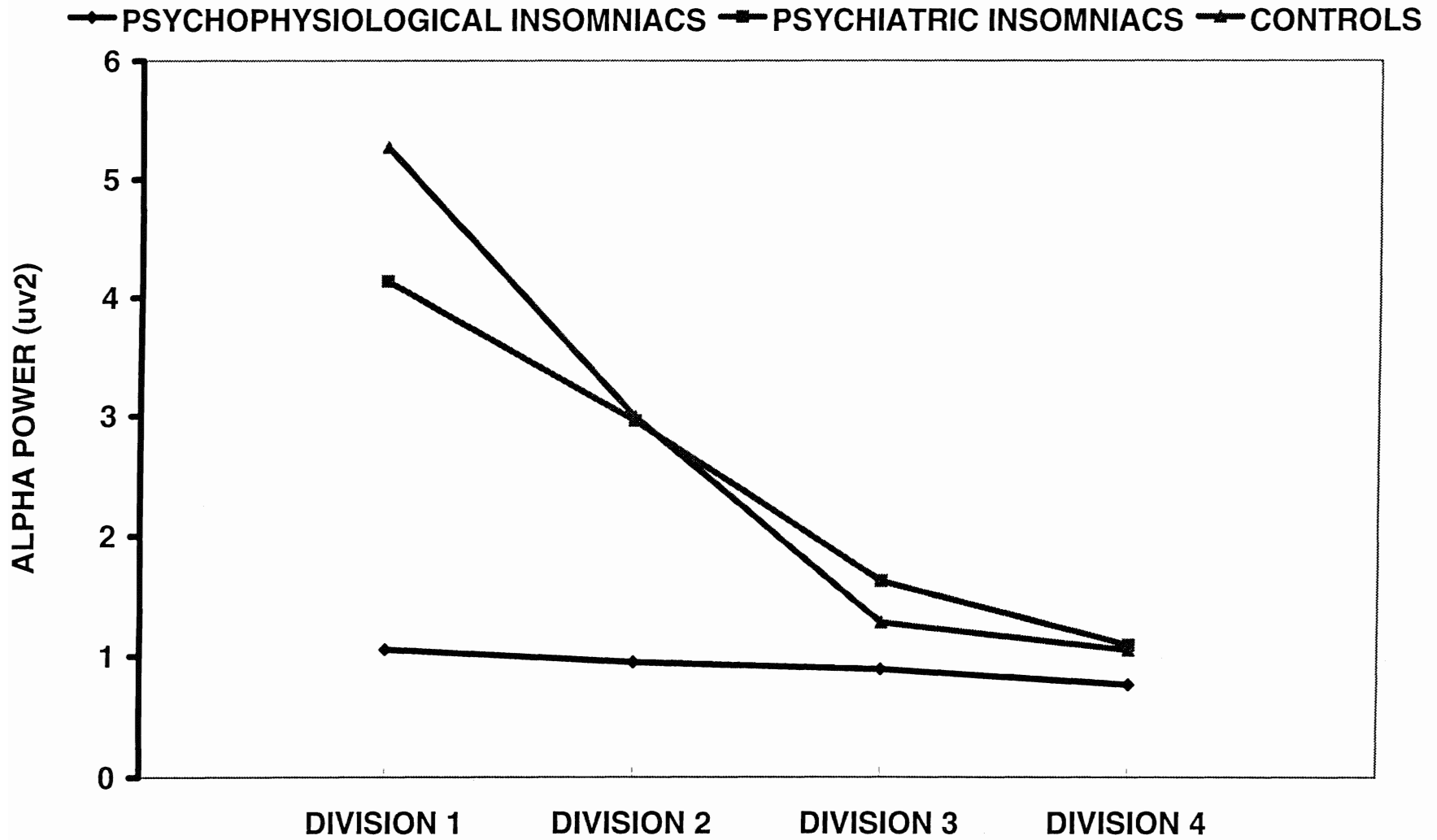


Table 11. Mean slope changes in delta, theta, alpha, sigma and beta bands across quartiles of the sleep onset period on night 1.

	Group		
	Psychophysiological Insomniacs	Psychiatric Insomniacs	Controls
	n=6 mean (sd)	n=6 mean (sd)	n=6 mean (sd)
Delta			
1st Quartile	.19 (.08)	.20 (.12)	.12 (.17)
2nd Quartile	.32 (.21)	.24 (.06)	.23 (.15)
3rd Quartile	.34 (.18)	.38 (.11)	.42 (.12)
4th Quartile	.49 (.17)	.49 (.18)	.64 (.16)
Theta			
1st Quartile	.31 (.20)	.37 (.16)	.48 (.07)
2nd Quartile	.36 (.14)	.49 (.18)	.51 (.13)
3rd Quartile	.35 (.12)	.49 (.19)	.53 (.10)
4th Quartile	.50 (.16)	.50 (.10)	.63 (.10)
Alpha			
1st Quartile	.50 (.11)	.45 (.11)	.38 (.13)
2nd Quartile	.55 (.10)	.38 (.25)	.39 (.17)
3rd Quartile	.54 (.11)	.47 (.16)	.28 (.19)
4th Quartile	.54 (.10)	.25 (.30)	.28 (.12)
Sigma			
1st Quartile	.41 (.17)	.31 (.18)	.35 (.15)
2nd Quartile	.50 (.07)	.38 (.26)	.53 (.11)
3rd Quartile	.49 (.06)	.48 (.16)	.48 (.18)
4th Quartile	.55 (.14)	.56 (.20)	.52 (.13)

Beta

1st Quartile	.47 (.12)	.48 (.14)	.41 (.21)
2nd Quartile	.41 (.22)	.44 (.22)	.42 (.23)
3rd Quartile	.59 (.09)	.44 (.11)	.47 (.21)
4th Quartile	.41 (.12)	.42 (.15)	.49 (.18)

Table 12. Mean slope changes in delta, theta, alpha, sigma and beta bands across quartiles of the sleep onset period on night 2.

	Group		
	Psychophysiological Insomniacs	Psychiatric Insomniacs	Controls
	n=6 mean (sd)	n=6 mean (sd)	n=6 mean (sd)
Delta			
1st Quartile	.30 (.12)	.24 (.05)	.11 (.10)
2nd Quartile	.43 (.08)	.30 (.16)	.22 (.07)
3rd Quartile	.42 (.16)	.44 (.13)	.38 (.22)
4th Quartile	.65 (.08)	.68 (.08)	.56 (.05)
Theta			
1st Quartile	.33 (.24)	.43 (.12)	.42 (.13)
2nd Quartile	.37 (.23)	.54 (.11)	.48 (.12)
3rd Quartile	.43 (.24)	.58 (.15)	.53 (.08)
4th Quartile	.58 (.06)	.66 (.21)	.64 (.09)
Alpha			
1st Quartile	.44 (.20)	.43 (.09)	.43 (.11)
2nd Quartile	.49 (.22)	.37 (.15)	.41 (.11)
3rd Quartile	.51 (.19)	.29 (.23)	.27 (.22)
4th Quartile	.50 (.26)	.30 (.32)	.22 (.20)
Sigma			
1st Quartile	.46 (.16)	.28 (.22)	.42 (.18)
2nd Quartile	.48 (.22)	.51 (.07)	.58 (.11)
3rd Quartile	.41 (.20)	.52 (.16)	.41 (.09)
4th Quartile	.59 (.16)	.58 (.20)	.67 (.19)

Beta

1st Quartile	.40 (.22)	.50 (.10)	.52 (.06)
2nd Quartile	.46 (.23)	.55 (.16)	.54 (.10)
3rd Quartile	.42 (.21)	.42 (.14)	.54 (.15)
4th Quartile	.33 (.22)	.40 (.22)	.50 (.15)

Figure 12

93

DELTA SLOPE CHANGES AS A FUNCTION OF GROUP AND NIGHT

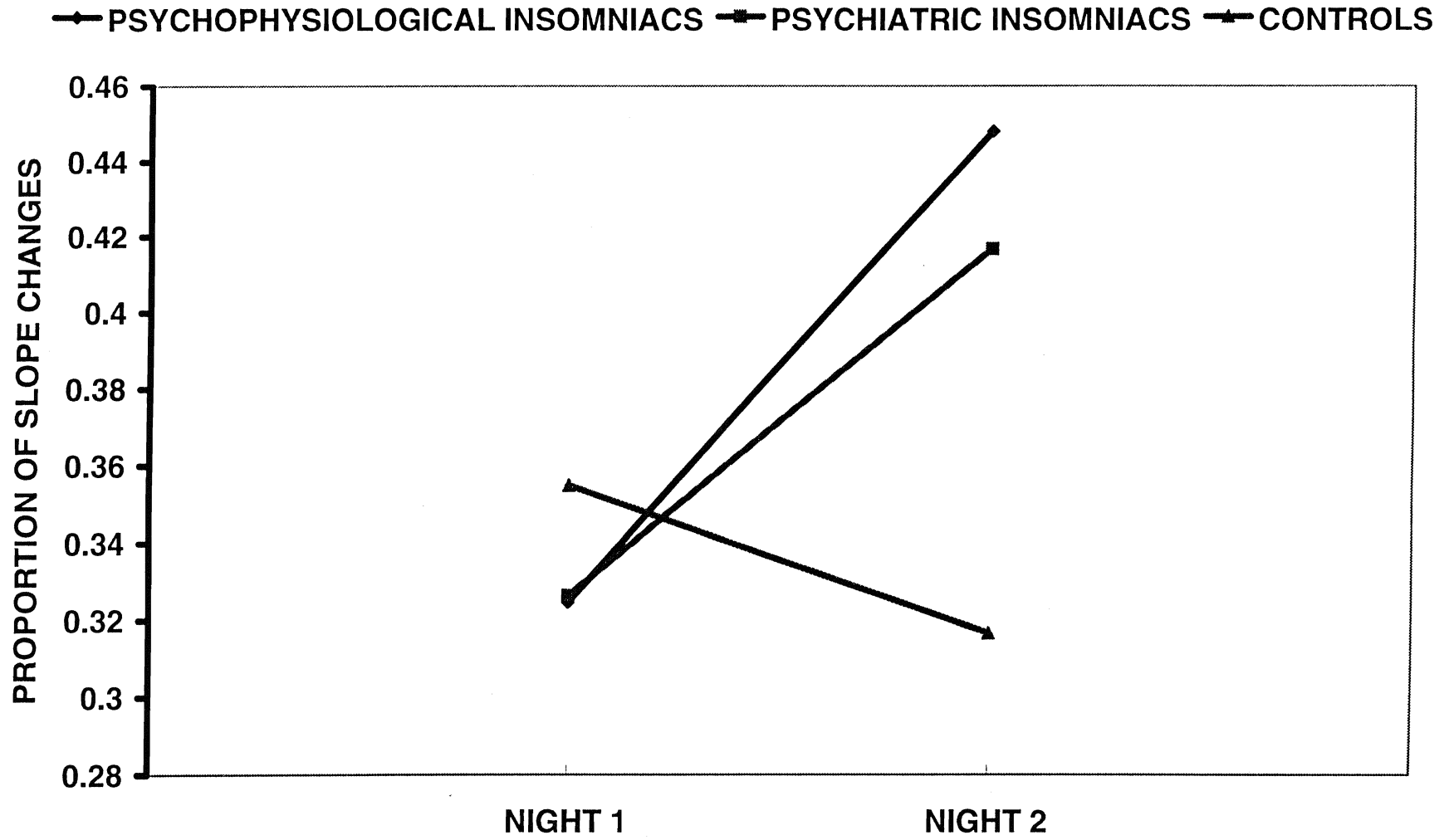
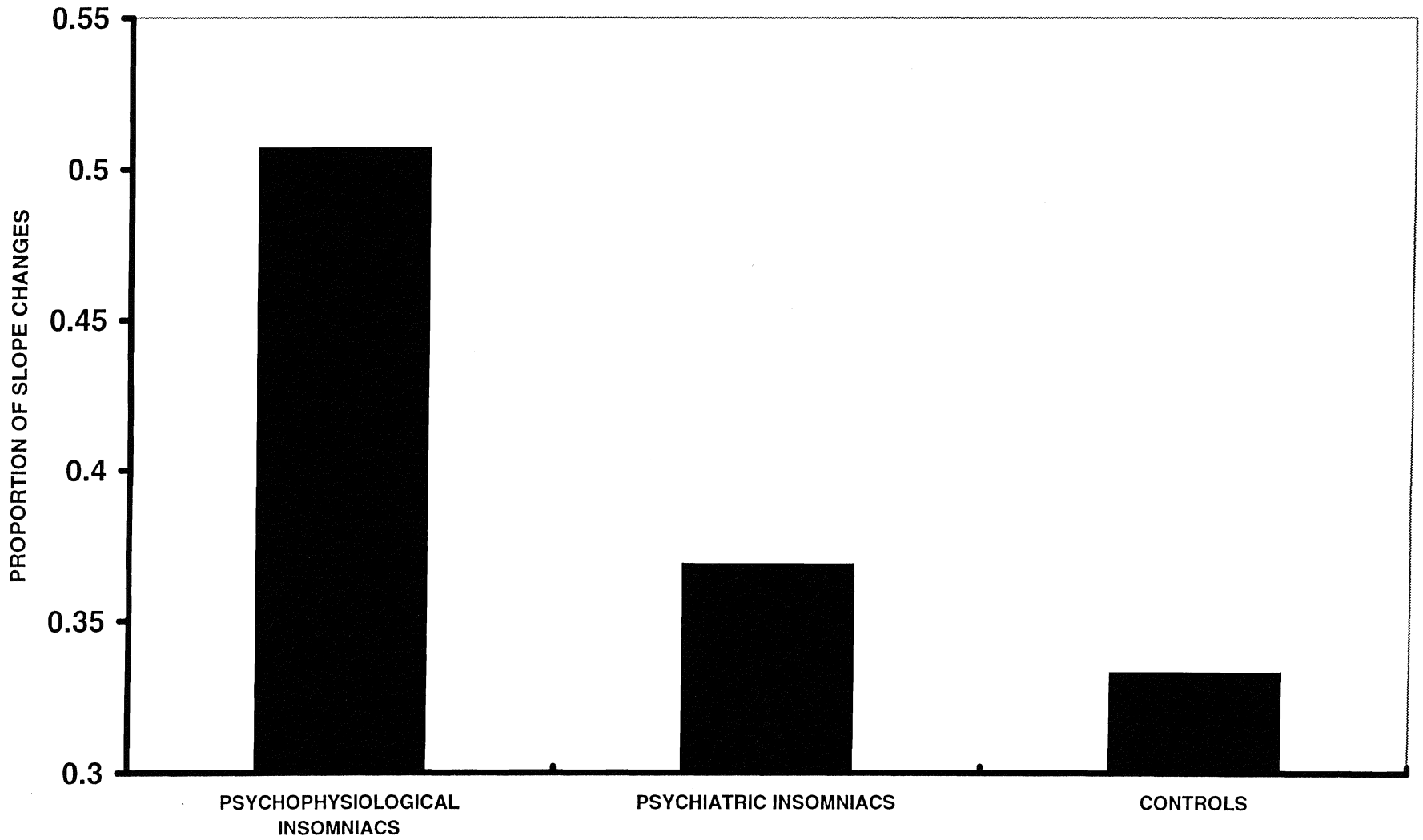


Figure 13

94

ALPHA SLOPE CHANGES AS A FUNCTION OF GROUP



Discussion

The present study represents the most detailed analysis yet attempted of the sleep onset period in insomniac and normal sleep. Using an FFT-based computer program, analyses of the five standard EEG bands in terms of absolute and relative power are described for the entry into sleep for epochs identified visually as representative of wakefulness and stages 1 and 2 sleep. These are supported by a chronological analysis of consecutive 14-second epochs of the sleep onset period, organized into four time periods or quartiles. This same chronological approach was also used to study fluctuations in slope changes in the arousal continuum as sleep was entered. Thus, we have employed four complimentary measures of changing EEG activity per frequency band in an effort to accomplish two tasks: first, to further characterize the process of entering sleep and second, in so doing, to provide the framework for distinguishing among the sleep onset patterns of normal sleepers and two subtypes of insomniacs. This last task was undertaken in an effort to identify and further understand the electrophysiology of insomnia. The results of the traditionally scored sleep variables will be discussed, followed by a brief description of the sleep onset period in normal sleepers and a finally by a comparison of the sleep onset periods of insomniacs and normal sleepers.

Visually-scored standard sleep parameters

There was a significant first night effect on sleep latency to stage 2, with latencies being shorter on the second night than on the first. This first night effect was stronger for the psychophysiological insomniacs than for the other two groups. Although self-reported differences existed between the two insomniac groups and the controls on the BSIQ and in the sleep diaries on such measures as difficulty in getting to sleep, sleep onset latency and total sleep time, these differences were not present in the traditional sleep variables measured by polysomnography. As reported by Reite et al. (1994), the magnitude of insomniac-control differences is not always striking and subjective reports do not always correspond to objective polysomnography.

There were no differences in other objective indices like sleep onset latency, sleep stages or other sleep variables such as total sleep time and sleep efficiency. The absence of differences in all of these sleep parameters speaks to the issue of their usefulness in the study of insomnia and the need for more in-depth and sensitive analyses of the EEG and perhaps to a focusing of attention on sleep onset EEG differences. The lack of differences in sleep latencies between the two insomniac groups and the control group is likely due to the high variability in the psychophysiological insomniacs' sleep latencies, to the

inclusion of insomniacs with both sleep onset and maintenance difficulties, and to the control group sleeping more poorly in the laboratory than what would have been expected from sleep diary and questionnaire estimates. Some psychophysiological insomniacs sleep better away from the home or in the laboratory (Hauri & Olmstead, 1989) (a phenomenon known as a reversed first night effect) and it was certainly the case for at least one of the participants in the present study who was prepared to move into the laboratory to obtain better sleep! Many past studies (e.g. Freedman, 1986; Hauri & Fisher, 1986; Jacobs et al., 1993) have established "laboratory criteria" for insomnia, that is, they have only included insomniacs who had a sleep latency of at least 30 minutes or good sleepers with a sleep latency of 15 minutes or less, and therefore, have probably exaggerated the sleep latency differences actually existing between insomniacs and good sleepers.

The multiple regression analysis of subjective and EEG estimates of sleep onset latency shows that both insomniac groups indeed displayed the characteristic subjective overestimation when compared to EEG sleep onset latency (Hauri & Olmstead, 1983; Moore et al., 1980). Group membership (insomniac versus normal sleepers) accounted for a significant amount of variance in subjective sleep latency estimate above that accounted for by the EEG.

The absence of differences in sleep stages is also

consistent with previous studies (Freedman, 1986). For example, Lichstein et al. (1994) did not find any differences in stage 1 latency, in sleep stage percentages or in sleep efficiency on the sleep laboratory night before daytime sleepiness assessment in psychophysiological insomniacs and controls. The only difference between the two groups was latency to stage 2. They noted that this lack of difference in sleep variables (total sleep time, sleep stage percentages, and sleep efficiency) between the groups is common when sampling from the community. Stage percentages for slow wave sleep are lower in all groups than established norms (Feinberg, 1974). This may be due to the fact that smaller epochs were scored and a more precise picture of actual sleep stage percentages was drawn.

The overall absence of striking differences in all-night visually-scored polysomnographic sleep parameters among insomnia subtypes and controls makes the present discovery of consistent computer-based EEG differentiation during the sleep onset period very exciting. Perhaps some of the most outstanding features of insomnia do revolve around the entry into sleep.

The sleep onset period in normal sleepers

The sleep onset period will first be described in the normal sleepers in order to provide a reference against which to contrast the sleep onset periods of the two insomniac groups.

Absolute delta power increased across the sleep onset period in the normal sleepers. These increases were more dramatic from stage 1 to stage 2 in the stage analysis and from the second through to the last quartile in the quartiles analysis of the sleep onset period. Relative delta power showed similar increases in the stage analysis. These increases in delta activity are consistent with Ogilvie et al. (1991) and Hori (1985).

Delta activity, which is not apparent in the visual analysis of the sleep onset period, can be seen by FFT analysis of the EEG. Delta activity is a major marker of synchronized or slow wave sleep. FFT analyses are able to trace this synchronization, not from stage 2 sleep, as in visually-scored analyses, but earlier in its development, throughout the sleep onset period. This may be particularly important because it may provide a synchronization index which is continuous from wakefulness through deep sleep. Perhaps delta measures sleepiness during waking and the sleep onset period.

Absolute theta power increases across the sleep onset period paralleled those of delta power. Theta power increased consistently from wakefulness through to stage 2 in the stage analysis of the sleep onset period and steadily across each quartile in the chronological analysis. Again, these changes during the sleep onset period are consistent with previous work (Ogilvie et al., 1991), but extend the

work because the earlier work from this lab was based on behavioral reference points while the present study traces continuous EEG changes. Relative theta power increased from wakefulness to stage 1, then decreased slightly from stage 1 to stage 2. The present study clearly maps theta and confirms it to be an important frequency band, one subject to dramatic changes, as sleep is entered.

Relative and absolute alpha power were high in the normal sleepers during wakefulness and dropped dramatically from wakefulness to stage 1, although these changes were only significant for relative power. Absolute alpha power increased slightly from stage 1 to stage 2 while relative alpha power decreased slightly. In the chronological analysis, alpha power decreased dramatically from the first to the third quartiles, then decreased only slightly from the third to the last quartile. These findings are also consistent with Ogilvie et al. (1991) and Hori (1985). High alpha is associated with relaxed wakefulness when the eyes are closed. There is a drop in alpha and a slowing of the EEG as sleep onset is approached with lower frequencies such as theta and delta become more prominent .

There was a drop in both absolute and relative sigma power from wakefulness to stage 1, then a dramatic increase from stage 1 to stage 2. Sigma power increased from the first to the second quartile, decreased from the second to the third quartile and increased from the third to the last

quartile in the chronological analysis. This complex pattern is perhaps suggestive of the involvement of two EEG processes. Decreases in sigma across the first three quartiles may mark the decline of processes showing this frequency during wakefulness, while the reversal of this decrease in the fourth quartile is likely due to the sensitivity of the FFT analysis to the early signs of sleep spindle activity.

Beta decreased from wakefulness to stage 1 and remained essentially the same from stage 1 to stage 2. These changes are also consistent with Ogilvie et al. (1991) who found decreases in beta as subjects became sleepier (as defined by a slowing of responses to a reaction time task), and an increase in beta at sleep onset. Relative beta decreased from wakefulness through to stage 2. A chronological analysis of beta power revealed consistent decreases in beta power across all quartiles of the sleep onset period. Decreases in beta are thought to represent decreases in arousal level.

Sleep Onset Period in Psychophysiological Insomniacs, Psychiatric Insomniacs and Normal Sleepers

EEG power spectral analyses of the sleep onset period were much more sensitive to differences among the groups than visual scoring of the EEG across the night. Virtually none of the standard, visually-scored sleep/wake parameters varied significantly across the night (see Table 3). On the

other hand, most of the spectral parameters distinguished among the sleep onset processes of psychophysiological insomniacs and the other two groups.

Delta activity. Delta power was lower in the psychophysiological insomniacs in both the stage analysis and the temporal analysis of the sleep onset period, but these differences were more obvious in the later part of the sleep onset period. Although lower delta power in the psychophysiological insomniacs had been predicted throughout the entire sleep onset period, this group only differed significantly from the other two groups during stage 2 on night 2 and during the last half of the sleep onset period in the chronological analysis. The lowered delta in the psychophysiological insomniacs is consistent with Mérica and Gaillard's (1992) findings of reduced delta in this group. This reduced delta activity appears to reflect an abnormality in the sleep onset process and may indicate problems within sleep. If these delta differences persist into slow wave sleep, then psychophysiological insomniacs may have a lesser quality sleep, which would not necessarily be reflected in lowered percentages of slow wave sleep but simply in the amount of delta power. Such delta differences have been found in depressives (Borbély et al. (1984). Borbély had earlier proposed a model of sleep regulation in normals (1982). This model specifies that sleep propensity is determined by two factors, process S, which is sleep-wake

dependent and increases exponentially during wakefulness; and process C which is a circadian process that is independent of wakefulness. Borbély and his colleagues proposed that the build-up of process S during waking is deficient in depressives and they do not show the increases in delta power that normals do. A similarly deficient process might be at work in the psychophysiological insomniacs. They had little stage 4 sleep, although this amount was not significantly lower than in the other two groups. Delta power did not show the dramatic increases that it should at sleep onset which may, in turn, account for their perception of being awake. The synchronization of delta activity which increases throughout the sleep onset period in the normal sleepers may be impaired in the psychophysiological insomniacs.

As we will see, the absence of the normally dramatic EEG changes seen in the sleep onset period is a feature of other frequencies as well, all of which may reduce discriminability of sleep and wakefulness in psychophysiological insomniacs. The reduced delta power may help to explain why they overestimated their sleep onset latency. It is interesting to note, however, that both insomniac groups overestimated their sleep onset latency. In the initial screening, all of the psychophysiological insomniacs reported sleep onset difficulties and four also had sleep maintenance difficulties. In the psychiatric

group, three of the participants reported sleep onset and maintenance difficulties, one reported sleep onset difficulties exclusively and two participants had maintenance insomnia exclusively. These differences are reflected in the sleep onset latencies of the two groups and may also be reflected in delta power. The sleep onset difficulties of the psychophysiological insomniacs may be related to a delayed increase in delta power, while in the psychiatric insomniacs, delta changes are essentially intact and no different from the normal sleepers. However, the psychiatric insomniacs appeared to have higher overall delta power than both groups on the first night, though this was not significant. The curves of psychiatric insomniacs and normal sleepers for delta power throughout the sleep onset period were more similar to each other than to the psychophysiological insomniacs. This may be due to the fact that the psychiatric group was heterogeneous, presenting with a wide array of clinical problems. It is possible that hospitalized psychiatric individuals might have specific sleep onset EEG differences. Higher delta power during wakefulness has been associated with psychiatric disturbances such as schizophrenia (Sponheim, Clementz, Iacono & Beiser, 1994) and bipolar disorder (Clementz, Sponheim & Beiser, 1994). Participants in the present study were classified according to the MMPI-2 but numbers precluded the use of particular psychiatric subtypes.

Despite scoring on the pathological end of the MMPI-2, none of the 6 participants in this group reported ever having been diagnosed with a psychiatric disorder.

The slope change measure was devised as an index of a fundamental sleep onset characteristic, variability in oscillatory activity toward and away from sleep. This is an idea first expressed by Kleitman (1963). He noted that humans did not enter smoothly into sleep but that sleep onset was better described as an oscillatory descent down the arousal continuum. Hori (1985) found that the coefficient of variation, that is, the standard deviation/average of a power band captured some of this phenomenon. The present slope change index quantifies the number of movements toward and away from sleep, studying them separately for each quartile of the sleep onset period.

Both insomniac groups had more delta slope changes on night 2 than on night 1 while the controls showed the opposite effect. This perhaps reflects a reversed first night effect. There were fewer oscillations in delta on the first night, suggesting a smoother descent into sleep, while the reverse was true for good sleepers.

Alpha activity. It was predicted that there would be differential changes in alpha activity during the sleep onset period depending on the insomnia subtype. The alpha band showed the predicted interaction between group and stage. Psychophysiological insomniacs had reduced absolute

and relative alpha power when compared to the psychiatric insomniacs and controls during wakefulness and showed similar power to the two groups during stage 1 and stage 2. The chronological analysis of the alpha band revealed the same pattern. Alpha levels remained rather constant in the psychophysiological insomniacs during the sleep onset period. Again, alpha power measures during the sleep onset period in the psychiatric insomniacs were more similar to the normal sleepers than to the psychophysiological insomniacs. This may suggest that their sleep problems are secondary to their psychiatric problems.

Decreased alpha power during wakefulness when eyes are closed may have two alternative explanations. It may mean that the psychophysiological insomniacs were sleepier than the other two groups or it may mean the opposite. Unfortunately, as one descends the arousal continuum from active wakefulness to sleep, alpha first rises during initial drowsiness, then lowers as sleepiness continues. Torsvall, Akerstedt and Gilberg (1987) examined EEG power changes during sleep deprivation. They found that, during a drowsiness test, periods of increasing sleepiness as selected by subjective sleepiness ratings were marked by an increase in alpha, theta and delta power density during an eyes open condition and a rapid decrease of alpha and a doubling of theta power during an eyes closed condition.

Freedman (1986) found decreased relative 9Hz (alpha)

activity during wakefulness in insomniacs when compared to good sleepers during both eyes open and eyes closed conditions. He interpreted this finding, coupled with the increased relative beta power, as meaning that insomniacs were more cortically aroused during this period than normal sleepers. In the present study, psychophysiological insomniacs showed differences in power measures that are consistent with the hypothesis of heightened arousal. They had reduced relative alpha as well as increased relative beta activity during wakefulness.

Alpha slope changes were the only ones that were sensitive to group differences. A greater proportion of slope changes were thought to reflect a less smooth transition from wakefulness to sleep and therefore a more problematic sleep onset period. Alpha changes were higher in the psychophysiological insomniacs but contrary to initial expectation, the psychiatric insomniacs did not show these same elevations. As mentioned above, changes in the psychiatric insomniacs were similar to changes in the normal sleepers. Since participants were included in the psychiatric insomniac group if they demonstrated an elevation on any of the ten clinical scales of the MMPI-2, this group was comprised of individuals showing a number of psychiatric problems, which increased variability on most measures.

Beta activity. Relative beta power during wakefulness

was higher in psychophysiological insomniacs than in the other two groups, especially on night 1. Again, cortical arousal, as measured by beta activity was higher in the psychophysiological insomniacs and may reflect their reported difficulties with sleep onset latency and their delayed perception of sleep. These differences are consistent with Freedman (1986), Jacobs et al. (1993), and Mérica and Gaillard (1992). Relative beta activity appeared lower in the psychiatric insomniacs, though not significantly. This may be due to a lower arousal level at sleep onset than the other two groups and is consistent with their tendency toward shorter sleep latencies, although latencies did not differ between the groups. It is puzzling that absolute power measures did not show this same pattern. In light of the consistently useful changes in other frequency bands (delta and alpha), the present findings suggest that the lower bands provide the most important and consistent evidence of group differences during the sleep onset period.

Psychophysiological insomniacs have characteristics that are indicative of heightened arousal such as tension headaches, faster heart rates and higher EMG levels (Freedman & Sattler, 1982), and higher body temperature (Lack et al., 1985; Monroe, 1967). This hyperarousal is experienced not only at night before sleep onset but also during the day. In studies assessing daytime sleepiness,

psychophysiological insomniacs are no more sleepy than controls (Lichstein et al., 1994) and, often, are more alert during the day than controls (Stepanski et al., 1988). The elevated beta and lowered alpha power in the psychophysiological insomniacs in this study lend further support to the hypothesis of heightened arousal, be it peripheral, physiological, or cortical. These differences in power bands may be unrelated to their perception and reflect more fundamental and physiological differences or as suggested by Méric and Gaillard (1992), they may help to explain why psychophysiological insomniacs tend to overestimate their sleep onset latencies. Recent findings have shown that psychophysiological insomniacs differ from controls on other physiological dimensions like immune system function and metabolic rate. For example, insomniacs have reduced killer cell activity when compared to controls and depressed individuals (Irwin et al., 1995) as well as increased metabolic rate (Bonnet & Arand, 1995).

As expected, all bands showed significant stage effects and most of them showed quartile effects. As anticipated, theta and sigma bands showed changes that are fundamental to normal sleep onset and did not differ among groups. The power spectral analyses were sensitive when used traditionally, that is, to distinguish among stages and consecutively to preserve the chronology of EEG changes during the sleep onset period.

The present findings also suggest that it may be fruitful to examine other subtypes of insomnia, such as sleep state misperception and idiopathic insomnia. Three nights of assessment could be used in order to counter first night effects of sleeping in the laboratory. It would also be interesting to analyze other sleep onset periods that occur after awakenings throughout the night. The present results however, show much promise with respect to computer-based EEG analytic techniques and their sensitivity to differences between insomniacs and good sleepers.

Power spectral analysis of the sleep onset period offers valuable information on the EEG of insomniacs and controls that is often not present in visual scoring of all night EEG recordings. A recent report from the American Sleep Disorders Association (1995) has called the usefulness of polysomnography into question. It noted that traditional polysomnography does not include sufficient or appropriate measures to adequately determine altered physiology in insomniacs. More detailed analyses of the EEG such as power spectral analysis yields this important information. There appears to be less differentiation in power measures during the sleep onset period in psychophysiological insomniacs than in the other two groups. This indicates that this group has a problem at the electrophysiological level. Psychophysiological insomniacs show power differences consistent with heightened cortical arousal. Also

interesting are studies suggesting that cognitive-behavioral methods can be effective in improving psychophysiological insomnia (Morin, Stone, McDonald & Jones, 1994). The disrupted sleep mechanism evident herein is amenable to psychological treatment, the efficacy of which might be measured in part employing the EEG analyses used in the present study.

In the future, EEG analysis of the sleep onset period could perhaps replace overnight polysomnography in the assessment and diagnosis of insomnia. Considering that there were no differences among groups in subjective, objective and physiological daytime sleepiness, power spectral analyses of the EEG of Multiple Sleep Latency Test naps might show similar differences to the ones found in this study, whereas simply looking at the latencies of these tests is not very informative (Lichstein et al., 1994), much like looking at the latencies of overnight polysomnography. Latency measures may be informative in disorders of excessive daytime sleepiness, however, they are not sensitive enough to differences between insomniacs and normal sleepers. For example, past studies (Freedman, 1986; Hauri and Fisher, 1986) that have established laboratory criteria for insomnia (i.e., have excluded insomniacs on the basis of short sleep latencies in the sleep laboratory). These studies may not give an accurate picture of the true insomniac, and differences, at least in

visually scored sleep parameters, may have been exaggerated. Sleep latency by itself may not be a sensitive parameter to differences between insomniacs and normal sleepers whereas power spectral analyses of the EEG during the sleep onset period have revealed fundamental differences that may also be present during the day, given that hyperarousal is thought to be present on a 24-hour basis in insomniacs (Morin, 1993).

In summary, power spectral analysis (FFT) allows for a more detailed and thorough investigation of insomniac sleep onset patterns. This type of analysis yields important information on the EEG of the sleep onset period, not only for normal sleepers but for insomnia and its subtypes. The sleep onset period in normal sleepers is characterized by dramatic changes; increases in delta and theta power and decreases in beta and alpha. This analysis also shows that the EEG during the sleep onset period is generally similar for normal sleepers and psychiatric insomniacs. A number of explanations for these similarities must be considered. It may be that the psychiatric group does not differ from normal sleepers during sleep entry, or it is possible that more stringent inclusion criteria for the psychiatric group (i.e., current hospitalization) may have shown differences from normal sleepers. Only further research can answer these important questions. The EEG of both groups changes substantially as sleep is entered,

whereas a different and less dramatic picture of EEG activity is shown by the psychophysiological insomniacs. Electrophysiological changes in insomniacs with psychiatric disorders needs further consideration. The differentiation primarily of psychophysiological insomniacs from the other two groups shows that independent electrophysiological processes of sleep entry exist, as predicted. In-depth analyses of the EEG such as the ones described have contributed to our understanding of the pathophysiology of the insomnia complaint.

References

Agnew, H., & Webb, W. (1972). Measurement of sleep onset by EEG criteria. American Journal of EEG Technology, 12, 127-134.

Agnew, H., Webb, W., & Williams, R. (1966). The first night effect: an EEG study of sleep. Psychophysiology, 2(3), 263-266.

American Sleep Disorders Association. (1990). International classification of sleep disorders: diagnostic and coding manual. Rochester, Minnesota: American Sleep Disorders Association.

American Sleep Disorders Association (1995). Practice parameters for the use of polysomnography in the evaluation of insomnia. Sleep, 18(1), 55-57.

Armitage, R., Hudson, A., Fitch, T., Pechacek, P. (1994). Period analysis of sleep onset in depressed outpatients and normal control subjects. In R. Ogilvie and J. Harsh (Eds.), Sleep onset: Normal and abnormal processes (pp. 185-200). Washington: American Psychological Association.

Aserinsky, E., & Kleitman, N. (1953). Regularly occurring periods of eye motility and concomitant phenomena during sleep. Science, 118, 273-274.

Badia, P., Wright, K.P. Jr., & Wauquier, A. (1994). Fluctuations in single-hertz EEG activity during the

transition to sleep. In R. Ogilvie and J. Harsh (Eds.), Sleep onset: Normal and abnormal processes (pp. 201-218). Washington: American Psychological Association.

Bixler, E.O., Kales, A., Soldatos, C.R., Kales, J.D., & Healy, S. (1979). Prevalence of sleep disorders in the Los Angeles Metropolitan area. American Journal of Psychiatry, 136, 1257-1262.

Björnsson, J., & Hetta, J. (1994). The role of sleep-onset EEG parameters in chronic insomnia. Journal of Sleep Research, 3, Supplement 1, 26.

Bonato, R.A., & Ogilvie, R.D. (1989). A home evaluation of a behavioral response measure of sleep/wakefulness. Perceptual and Motor Skills, 68, 87-96.

Bonnet, M., & Arand, D. (1995). Sleep state misperception: misperception by clinicians? Sleep Research, 24, 200.

Bootzin, R. (1972). Stimulus control treatment for insomnia. Proceedings of the American Psychological Association, 7, 395-396.

Borbély, A. (1982). A two-process model of sleep regulation. Human Neurobiology, 1, 195.

Borbély, A., Tobler, I., Loepfe, M., Kupfer, D., Ulrich, R., Grochocinski, V., Doman, J., & Matthews, G. (1984). All-night spectral analysis of the sleep EEG in untreated depressives and normal controls. Psychiatry Research, 12, 27-33.

Borkovec, T. (1982). Insomnia. Journal of Consulting and Clinical Psychology, 50, 880-895.

Borkovec, T., Lane, T., & Von Oot,, P. (1981). Phenomenology of sleep among insomniacs and good sleepers: wakefulness experience when cortically asleep. Journal of Abnormal Psychology, 90, 607-609.

Buysse, D., Reynolds, C., Kupfer, D., Thorpy, M., Bixler, E., Manfredi, R., Kales, A., Vgontzas, A., Stepanski, E., Roth, T., Hauri, P., & Mesiano, D. (1994). Clinical diagnoses in 216 insomnia patients using the International Classification of Sleep Disorders (ICSD), DSM-IV and ICD-10 categories: a report from the APA/NIMH DSM-IV field trial, Sleep, 17(7), 630-637.

Carskadon, M.A., Mitler, M.M., Billiard, M., Phillips, R., & Dement, W.C. (1975). A comparison of insomniacs and normals: total sleep time and sleep latency. Sleep Research, 4 , 212.

Cirignotta, F., Mondini, S., Zucconi, M., Lenzi, P.L., & Lugaresi, E. (1985). Insomnia: an epidemiological survey. Clinical Neuropharmacology, 8, suppl I, S49-S54.

Clementz, B., Sponheim, S., & Beiser, M. (1994). Resting EEG in first-episode schizophrenia patients, bipolar psychosis, and their first-degree relatives. Psychophysiology, 32,

Coates, T., Killen, J., Marchini, E., Silverman, S., Hamilton, S., & Thoresen, C. (1982). Discriminating good

sleepers from insomniacs using all-night polysomnograms conducted at home. Journal of Nervous and Mental Disease, 170, 224-230.

Coates, T.J., Strossen, R.J., Rosekind, M.R., & Thoresen, C.E. (1978). Obtaining all-night sleep recording data: How many nights are needed? Sleep Research, 7, 285.

Coleman, R., Roffwarg, H., & Kennedy, S. (1982). Sleep-wake disorders based on a polysomnographic diagnosis: a national cooperative study. Journal of the American Medical Association, 247, 997-1003.

Côté, K., & Ogilvie, R. (1993). The Brock Sleep and Insomnia Questionnaire: phase 1. Sleep Research, 22, 356.

Dement, W.C., & Kleitman, N. (1957). The relation of eye movements during sleep to dream activity: an objective method for the study of dreaming. Journal of Experimental Psychology, 53, 339-346.

Dement, W.C., Seidel, W., & Carskadon, M.A. (1982). Daytime alertness, insomnia and benzodiazepines. Sleep, 5 (Suppl.1), 28-45.

Dement, W.C., Seidel, W., & Carskadon, M.A. (1984). Issues in the diagnosis and treatment of insomnia. Psychopharmacology Supplement, 1, 12-43.

Douglass, A., Bornstein, R., Nino-Murcia, G., Keenan, S. (1986). Creation of the ASDC Sleep Disorders Questionnaire. Sleep Research, 15, 117.

Douglass, A., Bornstein, R., Nino-Murcia, G., Keenan,

S., Miles, L., Zarccone, V., Guilleminault, C., Dement, W., & Abelseth, D. (1990). Item test-retest reliability of the Sleep Disorders Questionnaire (SDQ), Sleep Research, 19, 215.

Douglass, A., Bornstein, R., Nino-Murcia, G., Keenan, S., Miles, L., Zarccone, V., Guilleminault, C., Dement, W. (1994). The Sleep Disorders Questionnaire I: creation and multivariate structure of SDQ. Sleep, 17(2), 160-167.

Douglass, A.B., Shipley, J.E., Nino-Murcia, G., Keenan, S., Miles, L., Guillemineault, C., Zarccone, V.P., & Dement, W.C. (1992). Validation of the "Psych" scale of the Sleep Disorders Questionnaire (SDQ). Sleep Research, 21, 150.

Edinger, J.D., Hoelscher, T.J., Webb, M.D., Marsh, G.R., Radtke, R.A., & Erwin, C.W. (1989). Polysomnographic assessment of DIMS: Empirical evaluation of its diagnostic value. Sleep, 12(4), 315-322.

Espie, C. (1991). The psychological treatment of insomnia. Chichester, England: Wiley and Sons.

Feinberg, I. (1974). Changes in sleep cycle patterns with age. Journal of Psychiatry Research, 10, 283-306.

Freedman, R.R. (1986). EEG power spectra in sleep-onset insomnia. Electroencephalography and Clinical Neurophysiology, 63, 408-413.

Freedman, R., & Sattler, H. (1982). Physiological and psychological factors in sleep onset insomnia. Journal of Abnormal Psychology, 91, 380-389.

Gaillard, J.M. (1978). Chronic primary insomnia: Possible physiopathological involvement of slow-wave sleep deficiency. Sleep, 1, 133-147.

Gillin, J.C., Duncan, W., Pettigrew, K.D., Frankel, B.L., & Snyder, F. (1979). Successful separation of depressed, normal and insomniac subjects by EEG sleep data. Archives of General Psychiatry, 36, 85-90.

Graham, J. (1987). The MMPI: a practical guide, 2nd edition, Oxford University Press.

Hammond, E.C. (1964). Some preliminary findings on physical complaints from a prospective study of 1,064,004 men and women. American Journal of Public Health, 54, 11-23.

Harsh, J., Voss, U., Hull, J., Schrepfer, S., & Badia, P. (1994). ERP and behavioral changes during the wake-sleep transition. Psychophysiology, 31, 244-252.

Hartmann, E. (1988). Insomnia: Diagnosis and treatment. In R.L. Williams, I. Karacan, & C.A. Moore (Eds.), Sleep Disorders Diagnosis and Treatment (2nd ed.) (pp.29-46). New York: Wiley and Sons.

Hathaway, S., & Meehl, P. (1951). An atlas for the clinical use of the MMPI. Minneapolis: University of Minnesota Press.

Hauri, P., & Fisher, J. (1986). Persistent psychophysiological (learned) insomnia. Sleep, 9(1), 38-53.

Hauri, P., & Olmstead, E. (1983). What is the moment

of sleep onset for insomniacs? Sleep, 6(1), 10-15.

Hauri, P., & Olmstead, E. (1989). Reverse first night effect in insomnia. Sleep, 12(2), 97-105.

Hauri, P., & Wisbey, J. (1992). Wrist actigraphy in insomnia. Sleep, 15(4), 293-301.

Hori, T. (1985). Spatiotemporal changes of EEG activity during waking-sleeping transition period. International Journal of Neuroscience, 27, 101-114.

Horne, J. (1988). Why we sleep. New York: Oxford University Press.

Irwin, M., Fortner, M., Clark, C., McClintick, J., Costlow, C., White, J., Ancoli-Israel, S., & Gillin, C. (1995). Reduction of natural killer cell activity in primary insomnia and in major depression. Sleep Research, 24, 256.

Jacobs, E.A., Reynolds, C.F. the III, Kupfer, D.J., Lovin, P.A., & Ehrenpreis, A.B. (1988). The role of polysomnography in the differential diagnosis of chronic insomnia. American Journal of Psychiatry, 145(3), 346-349.

Jacobs, G., Benson, H., Friedman, R. (1993). Home-based central nervous system assessment of a multifactor behavioral intervention for chronic sleep-onset insomnia. Behavior Therapy, 24, 159-174.

Johnson, L. (1973). Are stages of sleep related to waking behaviour? American Scientist, 61, 326-338.

Kales, A., Bixler, E.O., Vela-Bueno, A., Cadieux, R.J.,

Soldatos, C.R., & Kales, J.D. (1984). Biopsychobehavioral correlates of insomnia, III: Polygraphic findings of sleep difficulty and their relationship to psychopathology. International Journal of Neuroscience, 23, 43-56.

Kales, A., Bixler, E.O., Soldatos, C.R., Vela-Bueno, A., Caldwell, A.B., & Cadieux, R.J. (1981). Biopsychobehavioral correlates of insomnia, part I: Role of sleep apnea and nocturnal myoclonus. Psychosomatics, 23, 589-600.

Kales, A., Caldwell, A.B., Soldatos, C.R., Bixler, E.O., & Kales, J. (1983). Biopsychobehavioral correlates of insomnia. II. Pattern specificity and consistency with the Minnesota Multiphasic Personality Inventory. Psychosomatic Medicine, 45(4), 341-356.

Kales, J.D., Kales, A., Bixler, E.O., Soldatos, C.R., Cadieux, R.J., Kashurba, G.J., Vela-Bueno, A. (1984). Biopsychobehavioral correlates of insomnia, V: Clinical characteristics and behavioral correlates. American Journal of Psychiatry, 141(11), 1371-1376.

Karacan, I., Thornby, J.I., Anch, M., Holzer, C.E., Warheit, G.J., Schwab, J.J., & Williams, R.L. (1976). Prevalence of sleep disturbance in a primarily urban Florida county. Social Science and Medicine, 10, 239-244.

Kleitman, N. (1963). Sleep and wakefulness. Chicago: University of Chicago Press.

Kripke, D.F., Mullaney, D.J., Messin, S., & Wyborney,

V.G. (1978). Wrist actigraphy measures of sleep and rhythms. Electroencephalography and Clinical Neurophysiology, 44, 674-676.

Kuderian, R.H., Ogilvie, R.D., McDonnell, G., & Simons, I.A. (1991). Behavioural response home monitoring of good and insomniac sleepers. Canadian Journal of Psychology, 45(2), 169-178.

Ktonas, P.Y., & Gosalia, A.P. (1981). Spectral analysis vs. period-amplitude analysis of narrowband EEG activity: A comparison based on the sleep delta frequency band. Sleep, 4, 193-206

Kuisk, L.A., Bertelson, A.D., & Walsh, J.K. (1989). Presleep cognitive hyperarousal and affect as factors in objective and subjective insomnia. Perceptual and Motor Skills, 69, 1219-1225.

Lack, L., Balfour, R., Kallucy, R. (1985). The circadian rhythm of body temperature in poor sleepers. Sleep Research, 14, 301.

Lacks, P., Bertelson, A., Gans, L., & Kunkel, J. (1983). The treatment of sleep-maintenance insomniac with stimulus control techniques. Behaviour Research and Therapy, 21, 291-295.

Lichstein, K., & Rosenthal, T. (1980). Insomniacs' perceptions of cognitive versus somatic determinants of sleep disturbance. Journal of Abnormal Psychology, 89, 105-107.

Lichstein, K., Wilson, N., Noe, S., Aguillard, R., & Bellur, S. (1994). Daytime sleepiness in insomnia: behavioral, biological and subjective indices. Sleep, 17(8), 693-702.

Loomis, A.L., Harvey, E.N., & Hobart, G.A. (1937). Cerebral states during sleep, as studied by human brain potentials. Journal of Experimental Psychology, 21, 127-144.

Mamelak, R. & Hobson, J.A. (1989). Nightcap: a home-based sleep monitoring system, Sleep, 12(2), 157-166.

McCall, W.V., & Edinger, J.D. (1992). Subjective total insomnia: an example of sleep state misperception. Sleep, 15(1), 71-73.

Mendelson, W., Garnett, D., Gillin, J. C., & Weingartner, H. (1984). The experience of insomnia and daytime and night time functioning. Psychiatry Research, 19, 1261-1264.

Merica, H., & Gaillard, J.-M. (1992). The EEG of the sleep onset period in insomnia: A discriminant analysis. Physiology and Behavior, 52, 199-204.

Monroe, L.J. (1967). Psychological and physiological differences between good and poor sleepers. Journal of Abnormal Psychology, 72(3), 255-264.

Moore, S.E., Bonnet, M.H., Warm, J.S., & Kramer, M. (1980). Perception of sleep latency in the morning and at sleep onset in insomniac and normal subjects. Sleep

Research, 10, 220.

Morin, C. (1993). Insomnia: psychological assessment and management. New York: Guilford Press.

Morin, C., Stone, J., McDonald, K., & Jones, S. (1994). Psychological management of insomnia: a clinical replication series with 100 patients. Behavior Therapy, 25, 291-309.

Morin, C.M., Stone, J., Trinkle, D., Mercer, J., & Remsberg, S. (1993). Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. Psychology and Aging, 8(3), 463-467.

Mullaney, D.J., Kripke, D.F., and Messin, S. (1980). Wrist-actigraphic estimation of sleep time. Sleep, 3, 83-92.

Noldy, N., McGarry, P., & Campbell, K. (1988). Late auditory evoked potentials as indicators of sleep onset. W. Koella, F. Obal, H. Schulz, P. Visser (Eds.). Sleep '86. Stuttgart, Germany: Gustav Fischer Verlag.

Ogilvie, R.D. (1985). Behavioral and physiological investigations of sleep onset. Unpublished doctoral dissertation. University of Cambridge, Cambridge, U.K.

Ogilvie, R., & Harsh, J. (1994). Sleep onset: Normal and abnormal processes. Washington: American Psychological Association.

Ogilvie, R.D., Simons, I.A., Kuderian, R.H., MacDonald,

T., & Rustenburg, J. (1991). Behavioral, event-related potential, and EEG/FFT changes at sleep onset. Psychophysiology, 28(1), 54-63.

Ogilvie, R.D., & Wilkinson, R.T. (1984). The detection of sleep onset: Behavioral and physiological convergence. Psychophysiology, 21(5), 510-509.

Ogilvie, R.D., Wilkinson, R.T., & Allison, S. (1989). The detection of sleep onset: Behavioral, physiological and subjective convergence. Sleep, 12(5), 458-474.

Pace-Schott, E., Kaji, J., Stickgold, R., & Hobson, J.A. (1994). Nightcap measurement of sleep quality in self-described good and poor sleepers. Sleep, 17, 214-219.

Piéron, H. (1913). Le problème physiologique du sommeil. Paris: Masson.

Rechtschaffen, A. (1968). Polygraphic aspects of insomnia. In H. Gastaut, (Ed.) The abnormalities of sleep in man. Bologna, Italy: Aul. Gaggi Editore.

Rechtschaffen, A., & Kales, A. (1968). A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects. Los Angeles: UCLA, Brain Information Service/Brain Research Institute.

Reite, M., Buysse, D., Reynolds, C., Mendelson, W. (1995). The use of polysomnography in the evaluation of insomnia: An American Sleep Disorders Association Review. Sleep, 18(1), 58-70.

Reynolds, C.F. the III, Shaw, D.M., Newton, T.F.,

Coble, P.A., & Kupfer, D.J. (1983). EEG sleep in outpatients with generalized anxiety: A preliminary comparison with depressed outpatients. Psychiatry Research, 8, 81-89.

Reynolds, C.F. the III, Taska, L.S., Sewitch, D.E., Restifo, K., Coble, P.A., & Kupfer, D.J. (1984). Persistent psychophysiological insomnia: Preliminary research diagnostic criteria and EEG sleep data. American Journal of Psychiatry, 141(6), 804-805.

Richardson, G., Carskadon, M., Flagg, W., Van Den Hoed, J., Dement, W., & Mitler, M. (1978). Excessive daytime sleepiness in man: multiple sleep latency measurement in narcoleptic and control subjects. Electroencephalography and Clinical Neurophysiology, 45, 621-627.

Roth, T., Kramer, M., & Lutz, T. (1976). The nature of insomnia: A descriptive summary of a sleep clinic population. Comprehensive Psychiatry, 17, 217-220.

Schneider-Helmert, D. (1987). Twenty-four hour sleep-wake function and personality patterns in chronic insomniacs and healthy controls. Sleep, 10(5), 452-462.

Seidel, W.F., Ball, S., Cohen, S., Patterson, N., Yost, D., & Dement, W. (1984). Daytime alertness in relation to mood, performance, and nocturnal sleep in chronic insomniacs and noncomplaining sleepers. Sleep, 7(3), 230-238.

Sponheim, S., Clementz, B., & Iacono, W., & Beiser, M. (1994). Resting EEG in first-episode and chronic

schizophrenia. Psychophysiology, 31, 37-43.

Stepanski, E., Zorick, F., Roehrs, T., Young, D., & Roth, T. (1988). Daytime alertness in patients with chronic insomnia compared with asymptomatic control subjects. Sleep, 11(1), 54-60.

Stickgold, R., & Hobson, J.A. (1994). Home monitoring of sleep onset and sleep-onset mentation using the nightcap. In R. Ogilvie and J. Harsh (Eds.) Sleep onset: normal and abnormal processes (pp.141-159). Washington: American Psychological Association.

Sugerman, J.L., Stern, J.A., & Walsh, J.K. (1985). Daytime alertness in subjective and objective insomnia: Some preliminary findings. Biological Psychiatry, 20, 741-750.

Tan, T.L., Kales, J.D., Kales, A., Soldatos, C.R., & Bixler, E.O. (1984). Biopsychobehavioral correlates of insomnia, IV: Diagnosis based on DSM-III. American Journal of Psychiatry, 141(3), 357-362.

Torsvall, L., Akerstedt, T., & Gilberg, M. (1987). EEG/EOG approaches to sleepiness in laboratory and field studies. In M. Carskadon (Chair), Behavioral Significance of Sleepiness. Symposium conducted at the 5th International Congress of Sleep Research, Copenhagen.

Trinder, J. (1988). Subjective insomnia without objective findings: A pseudo diagnostic classification? Psychological Bulletin, 103(1), 87-94.

Appendix

**BROCK UNIVERSITY
INSOMNIA STUDY 1994
CONSENT FORM**

I, _____, hereby agree to spend two consecutive nights in the Brock University Sleep Laboratory. I have been fully oriented to the procedure and understand the following points:

1) The study consists of two phases. After completing the first phase, I may be asked to return for the second phase. Both phases are for research purposes only and do not involve any treatment.

2) I understand that in the first phase of the study, I will be required to complete a health and history questionnaire, the Brock Sleep and Insomnia Questionnaire (BSIQ), the Sleep Disorders Questionnaire and the Minnesota Multiphasic Personality Inventory (MMPI). In the second phase of the study, I will spend two consecutive nights in the Brock Sleep Laboratory. I will be asked to complete a presleep questionnaire (to screen for any daytime experiences that might render sleep atypical) on the days scheduled for participation, and a postsleep questionnaire (a qualitative report of my night's sleep and an estimate of my sleep onset latency) each morning.

3) 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 and computer.

4) I will wear two respiration belts on my first night in the lab, one at navel level and one at chest level.

5) I will have my own private room and my comfort will be respected as much as possible (i.e., pillow, room temperature, etc.).

6) A person will be on call in the laboratory if anything is needed throughout the night. S/he may be contacted via a special intercom system installed in the subject's room.

7) I have also been informed that, based on prior research, there is no danger whatsoever to my health. However, I have been told that if I have a past health history which suggests that complications could occur because of participation in this study, my physician and the experimenter should be consulted prior to the beginning of the experiment.

8) I have been informed that I will receive a \$30 honorarium for participation in this study.

9) I understand that I may withdraw from this study at anytime (without prejudice) even after signing this form and may withdraw any information that will be collected about me during this study. I understand that any information collected about me during this study will be kept confidential.

Participant's Signature

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

Signature of Investigator

Investigators:

Carole Lamarche, B.A.--Principle Investigator 688-5550 X3795

Robert Ogilvie, Ph.D.--Principle Investigator 688-5550 X3573

HEALTH & HISTORY QUESTIONNAIRE

Subject's ID: _____

Date: _____

Name of interviewer: _____

131

First, I would like to get some general background information:

Age: Sex: D.O.B.: Handedness:

Current address: Permanent:
Home: Phone:

Marital Status:

Current living arrangements (with family? friends? alone?)

Currently Employed? _____ Describe type of work/hours/duties, etc.
Last employed?

Education to date? (Grade completed? Degrees? Special training?)

In general, how would you (have) describe(ed) yourself as a student? (A B C)

Best subjects?

Worst subjects?

Major Hobbies? Current/Past?

→

Now I would like to ask you some questions about your health. Have you had any...

132

- Serious childhood diseases?
- Injuries, falls, broken bones?
- Sports injuries?
- High fevers?
- Serious infections?
- Diabetes?
- Liver problems?
- Kidney problems?
- Problems with arteries?
- Stroke?
- Seizures?
- Hypertension?
- Heart Problems? Angina?
- Blood problems?
- Breathing problems?
- Asthma, Emphysema?
- Tuberculosis?
- Skin disorders?
- Serious Allergies?
- Sleep problems?
- Cancer? Treatment?
- Surgery?
- Psychiatric problems?
- Anxiety or depression?
- Problems with vision?
- Hearing Problems?
- Paralysis or numbness?
- Fainting or dizziness?
- Serious headaches?
- Blurred vision?
- Serious viral/immune disorders? Treatment?
- Stomach problems? Digestion? Ulcers?
- Bowel or bladder problems?
- Movement problems, arthritis, sore joints?

If yes to any of the above, please explain. When, how serious, long-term effects, nature of treatment (e.g., chemo-therapy)?

Are you taking any prescribed or "over-the-counter" medications? Which ones? Purpose?

How would you describe your use of caffeine _____, alcohol _____, other recreational drugs _____ (none, mild, moderate, heavy)?

Current/Past? Changes?

PRE-SLEEP QUESTIONNAIRE

133

This form must be completed each night before retiring for bed.
 ID: _____ Date: _____ Night#: 1 2 3

Today was:	1	2	3	4	5	
Calm	--	--	--	--	--	Busy
Pleasant	--	--	--	--	--	Unpleasant
I felt:						
Depressed	--	--	--	--	--	Happy
Anxious	--	--	--	--	--	Relaxed

<u>Intake:</u>	<u>#taken today</u>	<u>Time last taken</u>
Cups of coffee	_____	_____
Cups of tea	_____	_____
Beer or wine	_____	_____
Hard alcohol	_____	_____
Cigarettes	_____	_____

<u>Medication:</u>	<u># today</u>	<u># regularly</u>	<u>Specify type</u>
Vitamins	_____	_____	_____
Tranquilizers	_____	_____	_____
Sleeping pills	_____	_____	_____
Hormones	_____	_____	_____
Aspirin	_____	_____	_____
Antidepressants	_____	_____	_____
Other	_____	_____	_____

Daily Routine:

Did you take any NAPS today? Y or N, How many _____
 Time taken? From _____ to _____?

Did you exercise today? Y or N, What time? _____

Was your day typical? Y or N, Explain. _____

Was your day unusually stressful? Y or N, Why? _____

Rate the quality of your daytime functioning:
 poor__ satisfactory__ average__ good__ exceptional__

Rate your job performance (use above scale) _____

What proportion of the day did you feel tired?
 not at all__ very little__ half__ most__ all day__

Do you feel tired now? Y or N

Do you anticipate a good night's sleep? Y or N

Describe briefly any events, good or bad, that occurred today,
 which you feel may affect your mood or sleep patterns _____

At what time are you retiring to bed this evening? _____

SWEET DREAMS!

POST-SLEEP QUESTIONNAIRE

This is to be completed each morning upon awakening.

subject I.D. _____ Date: _____ Night 1 2

What time did you go to bed last night? _____ hrs. _____ mins.

How long did it take you to fall asleep? _____ hrs. _____ mins.

How many times did you wake up during the night? _____

How long did you sleep? _____ hrs. _____ mins.

Did you wake up spontaneously? yes or no

Did you feel confused or disoriented upon awakening? yes or no

Did you wake up with a headache? yes or no

Did you wake up with slight amnesia? yes or no

Was your sleep:

non-existent _____ light _____ satisfactory _____

average _____ good _____ deep _____

How well did you sleep:

very poor _____ poor _____ fair _____ well _____ very well _____

How did you feel this morning:

drowsy _____ slightly drowsy _____ clear-headed _____ alert _____

How satisfied were you with your sleep:

not at all _____ slightly _____ fairly _____ very _____ completely _____

How tired do you feel:

extremely _____ very _____ slightly _____ hardly _____ not at all _____

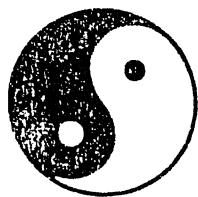
Please elaborate on any difficulty you may have had sleeping (e.g. possible reasons, temperature, noise, different bed, etc.)

BROCK SLEEP & INSOMNIA QUESTIONNAIRE (BSIQ)

By

Kimberly A. Cote, B.A.
&
Robert D. Ogilvie, Ph.D.

Brock University
St. Catharines, Ontario



Copyright © Cote/Ogilvie, 1993

BROCK SLEEP AND INSOMNIA QUESTIONNAIRE (BSIQ)

136

Subject ID: _____ Age: _____ Sex: _____ Date: _____

EEP QUALITY: Please answer the following questions with respect to a "typical" night's sleep.

How difficult is it for you to get to sleep at night?
easy 1 2 3 4 5 extremely difficult

How long do you lie awake before sleeping?(minutes) _____

Do you awake often throughout the night? Y or N
How many times do you wake up in the night? _____

What is the longest awakening? (minutes) _____
At what time does the longest awakening occur? _____

Do you wake up early in the morning and find you are unable to return to sleep? Y or N
How many nights per month do you awaken early and are unable to return to sleep? _____

What time do you usually retire for bed? _____
What time do you get up in the morning? _____
Does this time vary considerably? Y or N; By how much? (minutes) _____

How many hours on average do you think you sleep? _____
How many hours sleep do you feel you need each night? _____

Do you take naps? Y or N
How many times per week? 0 1 2 3 4 5 6 7 other _____
How many times per month? _____
How long do these naps usually last? (minutes) _____

How many days per week do you exercise? 0 1 2 3 4 5 6 7
Do you exercise before bed or in late evening? Y or N
What type of exercise? (ie:walking or aerobic) _____

When you go to bed do you usually have a "racing mind"? Y or N
Do you do most of your planning and thinking when you lie down to sleep? Y or N

Which of the following items describe your specific sleep difficulty: (You may check more than one)

- A. Initiating sleep or falling asleep
- B. Maintaining sleep
- C. Frequent awakenings throughout night
- D. Early morning awakenings without being able to return to sleep
- E. Excessive daytime sleepiness
- F. Non-restorative sleep or "unrefreshing" sleep
- G. Other: _____

INSTRUCTIONS

This questionnaire is intended to help you to describe your sleep-related experiences in detail. By answering all of the following questions, you will provide us with information which we will use to compare your particular pattern of answers to those of many other people experiencing either normal sleep or any of a number of sleep-related problems. Not all of the questions will seem relevant to your specific situation, but it is important to us that you try to use an answer provided which most closely describes your own sleep.

If you feel a YES or NO response does not accurately describe your answer to a specific question, please elaborate where necessary. If you feel that the space provided for an answer is not adequate, continue your answer on the reverse side of the page.

We realize that your sleep/wake schedules may vary considerably from time to time, so when answering questions such as, "What time do you retire for bed?", simply describe your typical night's sleep over the last month.

Please complete all sections of the questionnaire answering all questions as accurately as possible, so that we may make a proper assessment of your specific sleep problem.

Do not leave any questions blank. If the question does not apply to you, simply put "N/A" - for Not-applicable, so that we know you have not just missed the question.

All answers will be kept confidential.

If you have any questions, please feel free to ask at any time.

Please begin by printing your age, sex, and today's date at the top of the following page.

SLEEP HISTORY:

1. Do you ever suffer from insomnia? Y or N
Would you describe your as insomnia: transient___ intermittent___ persistent___ chronic___
2. How long have you had insomnia? years _____
months _____
3. At what age did the insomnia begin? _____
4. Do you recall a great deal of stress or any unusual events that occurred at the onset of the insomnia? (ie: job, relationships, finances) Y or N,
Explain: _____
5. Is there a history of insomnia in your family? Y or N; Relations? _____
6. How many nights per week on average do you have trouble sleeping? 0 1 2 3 4 5 6 7
7. Does your insomnia seem worse in times of high stress? Y or N
8. Have you ever seen a physician regarding your insomnia? Y or N
For how many months did you see your physician for the insomnia? _____
Did the doctor prescribe sleeping pills? Y or N
Did the doctor prescribe muscle relaxants? Y or N
Did your doctor ever specify what TYPE of insomnia you had or provide a reason for the sleep disturbance? Y or N. Explain: _____
9. Have you ever had a sleep lab evaluation? Y or N. If yes,
Where? _____
When? _____
Did they diagnose you with a particular sleep disorder? Y or N
-If so, what was the diagnosis? _____
-In your view, have subsequent events confirmed the diagnosis? Y or N
10. Do you feel your daytime functioning has declined since the insomnia began? Y or N
11. Has your job performance suffered? Y or N
Have you been unable to keep a job? Y or N
12. Have you ever sustained a head injury? Y or N (ie: car accident or fall).
Explain: _____

Have you ever been hospitalized for a head injury? Y or N
Have you ever been knocked unconscious? Y or N
How many minutes? _____ When? _____

RUG INVENTORY:

Are you currently taking any **PRESCRIPTION** sleeping aids? Y or N

What: _____

How many times per week: _____
per month: _____
per year: _____

Have you taken **PRESCRIPTION** sleeping aids in the past? Y or N

How long were you on prescription drugs? (days) _____ (weeks) _____ (months) _____

Has this use been intermittent__ or constant__?

Have you ever experienced difficulty going off a sleep drug? Y or N

Do you or did you find these prescription drugs helpful? Y or N

For how many days or weeks were they effective _____

Are you currently using any **OVER-THE-COUNTER** sleeping aids? Y or N

How many times per week: _____
per month: _____
per year: _____

Do you find these over the counter drugs helpful? Y or N

Have you used **OVER-THE-COUNTER** aids in the past? Y or N

For how long did you use them? _____

Do you ever take more than the prescribed amount of either prescription or over-the-counter sleeping pills? Y or N

How many times per month? _____
How much more? _____

Have you ever taken Aspirin as a sleeping aid? Y or N

Was it successful? Y or N
How many times per month? _____

Have you ever taken Antihistamines to help you sleep? Y or N

Was it successful? Y or N
How many times per month? _____

Are you currently taking any other type of drug or medication to help you sleep? If so, please specify what drug, duration taken, dosage, and effectiveness. _____

) Are you taking any other medication at all for any reason? Y or N

Specify: _____

11. Do you take any vitamins regularly? Y or N.

List _____

140

12. Are you currently taking hormones? Y or N.

List _____

13. Do you have any allergies?

Specify: _____

14. Do you use any illegal narcotics or street drugs?

Specify: _____

Have you used any of these substances in the past? Y or N.

Specify: _____

15. At the time the insomnia first appeared, were you doing any of the following:

taking any medication - Yes__ No__ Unsure__

using street drugs - Yes__ No__ Unsure__

drinking alcohol in excess - Yes__ No__ Unsure__

16. Do you smoke? Y__ or N__ or occasionally__

How many cigarettes do you smoke per day?_____

17. How many cups of coffee do you drink per day?_____

Do you regularly ingest any other substances with caffeine such as tea, cola, or chocolate? Y or N

How many times per day?_____

18. Do you take stimulants (ie/caffeine pills) to stay awake during the day? Y or N

19. How many beers or glasses of wine do you drink per day?_____

per week?_____

20. How many shots of hard alcohol do you drink per day?_____

per week?_____

SLEEP HYGIENE:

Do you have a "letdown" or relaxation period before retiring for bed? Y or N

141

Do you experience bodily tension (tension in muscles, anxiety feelings) before you fall asleep? Y or N

Do worrisome thoughts interfere with your ability to fall asleep? Y or N

Is your sleeping environment free of noise and other disturbances? Y or N, If no, explain.

Do you do strenuous exercise within four hours of going to bed? Y or N

Do you use any of the following substances the during the day or before going to sleep?

Caffeine (coffee, tea, pop, chocolate)

Nicotine

Alcohol

Recreational drugs

How long do you lie in bed awake if you cannot achieve sleep?(minutes) _____

How long do you lie in bed awake if you wake-up early in the morning and cannot return to sleep? (minutes) _____

Do you have a regular wake up time? Y or N, Time: _____

Does this wake up time apply across seven days a week? Y or N

If no, does the wake time apply to work days only? Y or N

Do you awaken to an alarm clock? Y or N

Do you have a regular bedtime? Y or N, Time: _____

. Do you sometimes retire for bed, NOT because you are tired, but only because it is a particular time at night and you feel that you should be sleeping? Y or N

. Do you eat heavy meals late in the evening? Y or N

. Do you watch TV in bed to help you get to sleep? Y or N

PART I:

1. Rate how you feel RIGHT NOW.(1=very little; 3=average; 5=alot)

142

Depressed 1 2 3 4 5
Anxious 1 2 3 4 5

2. How would you rate yourself IN GENERAL?

Depressed 1 2 3 4 5
Anxious 1 2 3 4 5

3. Have you ever seen a counsellor for any reason? Y or N

When? _____ For what purpose? _____

4. Have you ever been diagnosed with a psychiatric disorder? Y or N,

Specify: _____

5. Is there any history of psychiatric disorder in your family? Y or N.

Specify: _____

6. Do you sleepwalk? Y or N, How frequently? _____

Do you talk in your sleep? Y or N, How frequently? _____

Do you grind your teeth in your sleep? Y or N, How frequently? _____

7. Do you have nightmares? Y or N

How many nights per week? 0 1 2 3 4 5 6 7 OR per month? _____

8. How stressful is your life (1=low 5=average; 10=highly stressful):

1 2 3 4 5 6 7 8 9 10

9. Rate your ability to cope with stress.(1=poor; 10 good)

1 2 3 4 5 6 7 8 9 10

10. Are there any major life stresses in your life right now? Y or N. If yes, please explain:

11. Have you ever taken tricyclic antidepressants for your insomnia? Y or N.

Were they successful? Y or N

12. Answer the following questions using this scale: (1=never; 3=sometimes; 5=often)

___ Do you consider yourself more nervous than others?

___ Do you feel like "you are ready to go to pieces"?

___ Do you feel life is a strain?

___ Do you think you are less happy than others?

___ Are you lacking self-confidence?

___ Do you feel lonely most of the time?

___ Do you feel the future is hopeless?

ART II:

Do you ever have difficulty breathing during the night? Y or N

143

Do you snore? Y or N. If yes, how often?

1-2 times/month__ 1 night/week__ 2-3 nights/week__ nightly__

Does this snoring occur throughout the entire night? Y or N

Do sleepers in other rooms or neighbours ever complain of loud snoring? Y or N

How many years have you been snoring regularly?_____

Do you ever experience morning headaches? Y or N

How many times per week___ month___?

Do you ever feel confused or disoriented in the morning? Y or N

How many times per week___ month___?

Do you ever suffer from slight amnesia in the morning? Y or N

How many times per week___ month___?

Your current weight is _____ pounds or _____ kg

Your current height is ___ ft. ___ inches or ___ cm

Would you describe yourself as overweight? Y or N; By how much?_____

Is there a family history of ear, nose, and throat disease? Y or N

1. Do you experience frequent upper respiratory tract infections? Y or N.

How many times per year?_____

2. Do you experience recurrent middle ear disease? Y or N

Do you have a history of pneumonia? Y or N

3. Do you have a hearing problem? Y or N; If yes, Left ear__ right__ both__

4. Do you experience excessive daytime sleepiness? Y or N

How many days per week?_____

Does it interfere with your daytime performance? Y or N

5. What is your usual sleep position? stomach__ side__ back__

6. Do you currently have or have you ever been treated for the following:

asthma__ chronic bronchitis__ emphysema__

7. What is your current smoking status?

smoker__ occasional smoker__ ex-smoker__ never__

If you are a current or ex-smoker, please specify the length of time and the amount smoked:_____

PART III:

144

1. Do you frequently wake up during the night? Y or N
2. Do you often wake up with the blankets kicked off the bed or onto the floor? Y or N
3. Do you suffer pain or stiffness in the morning? Y or N
4. Do you get complaints from a partner of violent kicking or excessive movement in the night? Y or N
5. Do you suffer from excessive daytime sleepiness? Y or N
6. Does your sleep seem "unrefreshing" even when you get enough sleep? Y or N
7. Are you currently taking any of the following drugs:
Tricyclic antidepressants ___
Monoamine oxidase inhibitors___
8. Have you ever suffered withdrawal from any of the following:
Anticonvulsants ___
Benzodiazepines ___
Barbiturates ___
Hypnotics ___
9. Have you ever been diagnosed with epilepsy? Y or N
10. Do you ever feel a "creeping" sensation in your legs either at night or during the day?
Y or N
If yes, how many times per month? _____
11. Do you ever experience involuntary limb movement while awake? Y or N.
If yes, how many times per month? _____
12. Do you feel aches or discomfort in the legs (usually calves) that can only be alleviated by movement? Y or N
If yes, how many times per month? _____

ART IV:

Do you ever take sleeping aids during the day? Y or N
How many times per month? _____

145

Do you take any of the following during the day?

- ___ tranquilizers
- ___ anti-depressants
- ___ stimulants

Do you nap during the day? Y or N

If you are taking prescription or over the counter drugs, has the amount of medication needed to sleep increased over time? Y or N

If you remained at the same dosage level, was there a decrease in the amount of sleep?
Y or N

How long before you retire to bed do you normally take the hypnotics?
immediately ___ 1/2 hour ___ 1 hour ___ more(specify) _____

Do you ever use alcohol to get to sleep? Y or N
How many times per month? _____

Have you ever taken alcohol with sleeping pills?
How many times per month? _____

Do you repeat the dose if sleep is not achieved? Y or N
After how long? (minutes) _____

1. Have you experienced difficulty getting off drugs? Y or N

2. Do you experience nightmares? Y or N

3. Have you ever had any type of seizure or gone into convulsions? Y or N
How many times? _____ How often? _____

4. Do you have an alcohol dependence? Y or N

5. Do you experience NO problem falling asleep, but wake up in the night and cannot RETURN to sleep? Y or N

PART V:

146

1. How many years have you experienced insomnia? _____
2. Did the insomnia begin before puberty? Y or N
If so, was it serious enough to be a concern of your parents or your physician? Y or N
3. Have you ever been diagnosed with a neurological disorder? Y or N,
If yes, please describe. _____
4. As a child, did you experience any of the following:
sleep walking Y or N
sleep talking Y or N
nightmares Y or N
bed wetting Y or N
5. As a child were you hyperactive? Y or N
Did you take medication for it? Y or N
6. Were you ever told you had dyslexia? Y or N
7. Does the severity of the insomnia fluctuate over time? Y or N
8. Is the insomnia worse during times of high stress? Y or N
9. Do other family members have insomnia? Y or N
How many? _____
Relation? _____

ART VI:

Do you sleep an average number of hours, but cannot fall asleep at the desired clock time? (ie: sleep from 5am to 1pm) Y or N

Have you ever worked shift work?
For how many months? _____

Where there any circumstances in your life that required you to sleep irregular hours for a prolonged period of time? Y or N. _____

Which of the following do you experience problems with:

Falling asleep ___
Maintaining sleep ___
Waking up ___

Would you describe yourself as a night person? Y or N

When do you function your best or feel most alert?

__ morning
__ 10am
__ noon
__ 5pm
__ 8pm
__ after midnight

Do you experience 1 to 2 hour daily delays in sleep onset? (each night you fall asleep one to two hours later than the previous night - as if you were travelling across a different time zone each night) Y or N

Do you ever go WITHOUT sleep for 24 to 40 hours followed by prolonged sleep for 14 to 24 hours? Y or N

If yes, does this occur approximately once per month? Y or N

Do you find it difficult to work at a conventional nine to five job because you cannot always stay awake? Y or N

PART VII:

1. Was your childhood health poor? Y or N

148

2. Do you have poor health now? Y or N.

Specify: _____

3. Are you currently under your doctor's care for any reason? Y or N.

Specify: _____

4. Have you ever had an illness or physical symptoms which were attributed to emotional stress of psychological causes? (sometimes refers to as a psychosomatic illness) Y or N.

5. Is your work limited by illness? Y or N.

Specify: _____

6. Do you experience any chronic pain? Y or N; Where? _____

Does this pain keep you from sleeping? Y or N

PART VIII:

1. How tense do you feel in the muscles or body? (1=low 10=high)

1 2 3 4 5 6 7 8 9 10

2. Do you sleep better in a different environment? (ie: in a different bed, on vacation)? Y or N.

3. Do you try very hard to get to sleep at night? Y or N

4. At night, are your thoughts often about any of the following:

getting enough sleep _____

personal problems _____

work _____

health _____

death _____

relaxing _____

5. Do you get up at the same time every morning? Y or N

6. Do you ever watch T.V. while in bed? Y or N

7. Do you read in bed? Y or N

8. Do you lie in bed for hours hoping to fall asleep? Y or N

How many hours: _____

Thank you for taking the time to provide us with this information. It will be kept confidential.

Patient Name _____

Your weight _____

Date of Birth _____

Your height _____

Today's Date _____

Clinic Number _____

Doctor's name _____

SDQ

Sleep Disorders Questionnaire

(version 2.02)

Copyright © 1986-1989, 1991, 1992

All rights reserved worldwide, including rights of translation.

created by

Alan Douglass MD¹, Robert Bornstein PhD², German Nino-Murcia MD.³

Derived from a pool of questions (Sleep Questionnaire and Assessment of Wakefulness, "SQAW") created at Stanford University Sleep Disorders Clinic by Drs. Loughton Miles, Christian Guilleminault, Vincent Zarcone, and William Dement. The SQAW was copyrighted by Dr. Miles, 1979, and is used here by permission. The SDQ © is copyrighted by the seven above-named persons.

¹Dept. of Psychiatry, B2951 CFOB, University of Michigan, 1500 E. Medical Center Drive, Ann Arbor, MI, 48109-0704 (send correspondence to this address).

²Department of Psychiatry, Ohio State University, Columbus, OH

³Sleep Medicine and Neuroscience Institute, Palo Alto, CA

- | | | | | | |
|--|---|---|---|---|---|
| 1. I get too little sleep at night | 1 | 2 | 3 | 4 | 5 |
| 2. I often have a poor night's sleep | 1 | 2 | 3 | 4 | 5 |
| 3. I have trouble getting to sleep at night | 1 | 2 | 3 | 4 | 5 |
| 4. I wake up often during the night | 1 | 2 | 3 | 4 | 5 |
| 5. My bedtime varies a lot | 1 | 2 | 3 | 4 | 5 |
| 6. At bedtime, thoughts race through my mind | 1 | 2 | 3 | 4 | 5 |
| 7. At bedtime, I feel sad and depressed | 1 | 2 | 3 | 4 | 5 |
| 8. At bedtime, I worry about things | 1 | 2 | 3 | 4 | 5 |
| 9. At bedtime, I feel muscular tension | 1 | 2 | 3 | 4 | 5 |
| 10. At bedtime, I'm afraid of not being able to go to sleep | 1 | 2 | 3 | 4 | 5 |
| 11. When falling asleep, I feel paralyzed (unable to move) | 1 | 2 | 3 | 4 | 5 |
| 12. When falling asleep, I have "restless legs" (a feeling of crawling, aching, or inability to keep legs still) | 1 | 2 | 3 | 4 | 5 |
| 13. After waking at night, I fear I will not be able to get back to sleep | 1 | 2 | 3 | 4 | 5 |
| 14. My night sleep is restless and disturbed | 1 | 2 | 3 | 4 | 5 |
| 15. At night, my sleep disturbs my bed partner's sleep | 1 | 2 | 3 | 4 | 5 |
| 16. My night sleep is disturbed by light | 1 | 2 | 3 | 4 | 5 |
| 17. My night sleep is disturbed by noise | 1 | 2 | 3 | 4 | 5 |
| 18. My sleep is disturbed by severe heartburn and choking ("regurgitation", bringing up bitter stomach fluid) | 1 | 2 | 3 | 4 | 5 |
| 19. I often wake up because I am hungry | 1 | 2 | 3 | 4 | 5 |
| 20. I snore in my sleep | 1 | 2 | 3 | 4 | 5 |
| 21. I am told I snore loudly and bother others | 1 | 2 | 3 | 4 | 5 |
| 22. I am told I stop breathing ("hold my breath") in sleep | 1 | 2 | 3 | 4 | 5 |

***** Key for answers *****

1	2	3	4	5
NEVER	RARELY	SOMETIMES	USUALLY	ALWAYS
(strongly disagree)	(disagree)	(not sure)	(agree)	(agree strongly)

- 23. I awake suddenly gasping for breath, unable to breathe 1 2 3 4 5 2
- 24. At night my heart pounds, beats rapidly, or beats irregularly ("palpitations") 1 2 3 4 5
- 25. I sweat a great deal at night 1 2 3 4 5
- 26. I walk in my sleep 1 2 3 4 5
- 27. I grind my teeth while I sleep 1 2 3 4 5
- 28. I wake from sleep screaming, confused, and at times violent ("night terrors") 1 2 3 4 5
- 29. My sleep is disturbed because of pain in the neck, back, muscles, joints, legs or arms 1 2 3 4 5
- 30. My sleep is disturbed by chest pain (not angina) 1 2 3 4 5
- 31. My sleep is disturbed by "restless legs" (a feeling of crawling, aching, inability to keep legs still) 1 2 3 4 5
- 32. My sleep is disturbed by thoughts racing through my mind 1 2 3 4 5
- 33. My sleep is disturbed by sadness or depression 1 2 3 4 5
- 34. My sleep is disturbed by worrying about things 1 2 3 4 5
- 35. My sleep is disturbed by muscular tension 1 2 3 4 5
- 36. My sleep is disturbed by fears that I might not be able to get back to sleep if I should wake up 1 2 3 4 5
- 37. I often have a night full of intense vivid dreams 1 2 3 4 5
- 38. I have a lot of nightmares (frightening dreams) 1 2 3 4 5
- 39. I feel unable to move (paralyzed) after a nap 1 2 3 4 5
- 40. I have dream-like images (hallucinations) when I awaken in the morning even though I know I am not asleep 1 2 3 4 5
- 41. I am sometimes very sleepy in the daytime, and this seems to go in cycles at regular intervals 1 2 3 4 5
- 42. I have slept for several days at a time, or at least I have been overwhelmingly sleepy for that long 1 2 3 4 5
- 43. I have been unable to sleep at all for several days 1 2 3 4 5

***** Key for answers *****

1	2	3	4	5
NEVER	RARELY	SOMETIMES	USUALLY	ALWAYS
rongly disagree)	(disagree)	(not sure)	(agree)	(agree strongly)

- | | |
|--|-----------|
| 44. I feel that my sleep is abnormal | 1 2 3 4 5 |
| 45. I feel that I have insomnia | 1 2 3 4 5 |
| 46. As a child, I had difficulty waking up in the morning | 1 2 3 4 5 |
| 47. As a child, I had sleepiness during the day | 1 2 3 4 5 |
| 48. I have a problem because of headaches while sleeping | 1 2 3 4 5 |
| 49. As a child, I was fatigued during the day | 1 2 3 4 5 |
| 50. As a child, I rocked myself to get to sleep | 1 2 3 4 5 |
| 51. I used to bang my head as a child | 1 2 3 4 5 |
| 52. I used to sleepwalk in childhood | 1 2 3 4 5 |
| 53. As a child, I had convulsions (seizures) during sleep | 1 2 3 4 5 |
| 54. As a child, I would grind my teeth while asleep | 1 2 3 4 5 |
| 55. Now, I am very sleepy during the day and I struggle to stay awake | 1 2 3 4 5 |
| 56. In the past 6 months, I have fallen asleep accidentally in some of these situations: eating a meal, talking on the phone, talking to someone, riding in a bus or car, watching TV, at a theater, reading a book, at a lecture. | 1 2 3 4 5 |
| 57. I got bad grades in school because I was too sleepy | 1 2 3 4 5 |
| 58. I now have trouble doing my job because of sleepiness or fatigue | 1 2 3 4 5 |
| 59. I often have to let someone else drive the car because I am too sleepy to do it | 1 2 3 4 5 |
| 60. I see vivid dream-like images (hallucinations) either just before or just after a daytime nap, yet I am sure I am awake when they happen | 1 2 3 4 5 |
| 61. I have vivid dreams during my daytime naps | 1 2 3 4 5 |
| 62. I am often unable to move (paralyzed) when I am waking up in the morning | 1 2 3 4 5 |
| 63. Sometimes I realize I have driven my car to the wrong place, and I can't remember how I did it | 1 2 3 4 5 |
| 64. I find myself doing things which make no sense, such as writing nonsense instead of notes, or mixing together chocolate and gravy | 1 2 3 4 5 |

***** Key for answers *****

1	2	3	4	5
NEVER	RARELY	SOMETIMES	USUALLY	ALWAYS
gly disagree)	(disagree)	(not sure)	(agree)	(agree strongly)

- | | | | | | |
|---|---|---|---|---|---|
| 65. People tell me that I act strangely at times, and yet I was not aware of it when it happened | 1 | 2 | 3 | 4 | 5 |
| 66. I get "weak knees" when I laugh | 1 | 2 | 3 | 4 | 5 |
| 67. I get sudden muscular weakness (or even a brief period of paralysis, being unable to move) when laughing, angry, or in situations of strong emotion | 1 | 2 | 3 | 4 | 5 |
| 68. I am excessively sleepy during the daytime | 1 | 2 | 3 | 4 | 5 |
| 69. I have at some time had trouble with my bladder | 1 | 2 | 3 | 4 | 5 |
| 70. I have had problems with tonsils or adenoids | 1 | 2 | 3 | 4 | 5 |
| 71. I have high blood pressure (or once had it) | 1 | 2 | 3 | 4 | 5 |
| 72. My tonsils and/or adenoids have been removed | 1 | 2 | 3 | 4 | 5 |
| 73. I get pains in my abdomen (stomach) | 1 | 2 | 3 | 4 | 5 |
| 74. I have had a head injury | 1 | 2 | 3 | 4 | 5 |
| 75. I have been knocked unconscious (knocked out) | 1 | 2 | 3 | 4 | 5 |
| 76. I suffer from dizzy spells | 1 | 2 | 3 | 4 | 5 |
| 77. I have seizures ("fits", convulsions, epilepsy) | 1 | 2 | 3 | 4 | 5 |
| 78. I have problems with clumsiness, incoordination | 1 | 2 | 3 | 4 | 5 |
| 79. I feel that I have a sexual problem | 1 | 2 | 3 | 4 | 5 |
| 80. My desire or interest in sex is less than it used to be | 1 | 2 | 3 | 4 | 5 |
| 81. I have pain or discomfort during sexual intercourse | 1 | 2 | 3 | 4 | 5 |
| 82. I sleep better after having sex | 1 | 2 | 3 | 4 | 5 |
| 83. I am unhappy about my social life | 1 | 2 | 3 | 4 | 5 |
| 84. I am unhappy about loving relationships in my life | 1 | 2 | 3 | 4 | 5 |
| 85. I am unhappy about my sex life | 1 | 2 | 3 | 4 | 5 |
| 86. I am dissatisfied with my job | 1 | 2 | 3 | 4 | 5 |

***** Key for answers *****

1	2	3	4	5
NEVER	RARELY	SOMETIMES	USUALLY	ALWAYS
(strongly disagree)	(disagree)	(not sure)	(agree)	(agree strongly)

87. I have a problem with my sleep 1 2 3 4 5
88. I wake up in the morning with a headache 1 2 3 4 5
89. I have considered or attempted suicide 1 2 3 4 5
90. I feel I am useful and needed 1 2 3 4 5
91. I am sleeping more than I used to 1 2 3 4 5
92. Someone in my immediate family has trouble with insomnia (brother/sister, father/mother, son/daughter, grandparent) 1 2 3 4 5
93. Someone in my immediate family is very sleepy during the day 1 2 3 4 5
94. Someone in my immediate family has psychiatric or emotional illness (e.g.: depression, alcoholism) 1 2 3 4 5
95. Some of my other relatives have trouble with insomnia (uncles, aunts, cousins) 1 2 3 4 5
96. Some of my other relatives are very sleepy during the day 1 2 3 4 5
97. Some of my other relatives have psychiatric illness 1 2 3 4 5
98. Some family member has died suddenly in their sleep 1 2 3 4 5
99. Some family member has "restless legs" while sleeping (a feeling of crawling, aching, inability to keep the legs still) 1 2 3 4 5
100. A child in my family died from "crib death" (sudden infant death syndrome, SIDS) 1 2 3 4 5
101. Someone in my family has been hospitalized for a psychiatric illness or "nervous breakdown". 1 2 3 4 5
102. People in my family seem to be worriers 1 2 3 4 5
103. Someone in my family has diabetes 1 2 3 4 5
104. Someone in my family has had a stroke ("apoplexy") 1 2 3 4 5
105. I often use alcohol in order to get to sleep 1 2 3 4 5
106. I use alcohol to steady my nerves 1 2 3 4 5
107. While drinking alcohol, I have carried out actions without being aware of them, and not remembered them the next day 1 2 3 4 5

***** Key for answers *****

1	2	3	4	5
NEVER	RARELY	SOMETIMES	USUALLY	ALWAYS
(strongly disagree)	(disagree)	(not sure)	(agree)	(agree strongly)

108. I smoke tobacco within two hours of bedtime 1 2 3 4 5¹⁵⁶
109. I have used "street drugs" (marijuana, "uppers", "downers",
narcotics, hallucinogens, cocaine) 1 2 3 4 5
110. I have used tobacco to help me go to sleep 1 2 3 4 5
111. I have used marijuana to help me go to sleep 1 2 3 4 5
112. I currently take a non-prescription drug from the pharmacy in order to
help me sleep 1 2 3 4 5
113. I currently take a non-prescription drug to stop me being so sleepy
and fatigued in the daytime 1 2 3 4 5
114. I take a prescription drug which the doctor gave me mainly to help me
sleep (sleeping pills, anti-depressants, tranquilizers) 1 2 3 4 5
115. I take a prescription drug which the doctor gave me mainly to keep me
awake during the day (e.g.: ritalin) 1 2 3 4 5
116. I take some drugs at night for my other illnesses, not related to sleep,
yet I find they help me sleep 1 2 3 4 5
117. I have taken drugs for my heart 1 2 3 4 5
118. I use relaxation techniques or mental imagery (e.g.: counting sheep)
to help me sleep 1 2 3 4 5
119. I use non-drug therapies in order to get to sleep (e.g.: biofeedback,
acupuncture, electrosleep) 1 2 3 4 5
120. I exercise regularly 1 2 3 4 5
121. I was born as part of a multiple birth (twins, or triplets, etc. Includes
cases where the others died at birth or afterwards) 1 2 3 4 5
122. My family was emotionally close in my childhood 1 2 3 4 5
123. I got along well with my parents while growing up 1 2 3 4 5
124. I am currently unemployed 1 2 3 4 5
125. I am working at a job with rotating shifts 1 2 3 4 5
126. I have had a job where I worked at unusual times 1 2 3 4 5

***** Key for answers *****

1	2	3	4	5
NEVER	RARELY	SOMETIMES	USUALLY	ALWAYS
strongly disagree)	(disagree)	(not sure)	(agree)	(agree strongly)

127. I am presently living in a house 1 2 3 4 5¹⁵⁷
128. I get along well with my husband / wife / friend, who is currently living with me 1 2 3 4 5
129. Coffee, tea, or cola drinks seem to worsen my sleep 1 2 3 4 5
130. Mental stress, worry, or anxiety worsens my sleep 1 2 3 4 5
131. Physical exercise helps my sleep 1 2 3 4 5
132. A daytime nap worsens my nighttime sleep 1 2 3 4 5
133. Mental stress, worry, or anxiety makes me feel sleepy during the day 1 2 3 4 5
134. After a nap, I feel less sleepy in the daytime 1 2 3 4 5
135. Hot weather makes me sleepy during the day 1 2 3 4 5
136. When doing shift work, I am sleepy during the day 1 2 3 4 5
137. I have a small jaw, or other abnormality of the bones in my head or neck 1 2 3 4 5
138. I have a chronic chest disease (bronchitis, asthma, emphysema) 1 2 3 4 5
139. I have a problem with my nose blocking up when I am trying to sleep
(allergies, infections) 1 2 3 4 5
140. I wake up with "attacks" which are different from those described
anywhere else in this questionnaire 1 2 3 4 5
141. My snoring or my breathing problem is much worse if I sleep on my back 1 2 3 4 5
142. My snoring or my breathing problem is much worse if I fall asleep
right after drinking alcohol 1 2 3 4 5
143. My snoring or my breathing problem is much worse when I have an allergy
or infection in the nose, throat, or chest 1 2 3 4 5

***** Key for answers *****

- | | | | | |
|---------------------|------------|------------|---------|------------------|
| 1 | 2 | 3 | 4 | 5 |
| NEVER | RARELY | SOMETIMES | USUALLY | ALWAYS |
| (strongly disagree) | (disagree) | (not sure) | (agree) | (agree strongly) |

THE FOLLOWING QUESTIONS ARE FOR WOMEN ONLY:

144. I have gone through the menopause ("change of life") 1 2 3 4 5
145. My sleep at night is affected by my menstrual cycle 1 2 3 4 5
146. My daytime sleepiness worsens with pregnancy 1 2 3 4 5
147. My daytime sleepiness is worse since my menopause 1 2 3 4 5

THE FOLLOWING QUESTIONS ARE FOR MEN ONLY:

148. I often have problems getting an erection 1 2 3 4 5
149. I have trouble maintaining an erection 1 2 3 4 5
150. I have trouble with ejaculation (either I can't do it at all, or it happens too soon) 1 2 3 4 5
151. My erections are physically distorted 1 2 3 4 5
152. I often awaken with an erection during the night or in the morning 1 2 3 4 5

***** Key for answers *****

- | | | | | |
|---------------------|------------|------------|---------|------------------|
| 1 | 2 | 3 | 4 | 5 |
| NEVER | RARELY | SOMETIMES | USUALLY | ALWAYS |
| (strongly disagree) | (disagree) | (not sure) | (agree) | (agree strongly) |

IN THE NEXT SECTION, PLEASE CIRCLE THE ITEM (NUMBERED 1-5) WHICH BEST MATCHES YOUR ANSWER. 159

153. How many hours of sleep do you get at night, not including time spent awake in bed?

- ① Less than 4 hrs. ② Four to 5 hrs. ③ Six hrs.
④ Seven hrs. ⑤ Eight or more

154. How long is your longest wake period at night?

- ① Less than 5 min. ② Six to 19 min. ③ 20 to 59 min.
④ One to 2 hrs. ⑤ More than 2 hrs.

155. How many times in a night do you get up to urinate?

- ① None. ② One time ③ Two times
④ Three times ⑤ Four or more times

156. How many work accidents have you had as a result of sleepiness or fatigue?

- ① None ② One ③ Two
④ Three ⑤ Four or more

157. How many car accidents or "near misses" have you had because of excessive sleepiness?

- ① None ② One ③ Two
④ Three ⑤ Four or more

158. How many daytime naps (asleep for 5 minutes or more) do you take on an average working day?

- ① None ② One ③ Two
④ Three or four ⑤ Five or more

159. How many rest periods do you take on an average working day (but do not sleep during them)?

- ① None ② One ③ Two or three
④ Four or five ⑤ Six or more

160. How many times, in an average working day, do you try to nap but find that you can't fall asleep?

- ① None
- ② One
- ③ Two
- ④ Three
- ⑤ Four or more

161. How long do you remain restored (refreshed, alert) after a daytime nap?

- ① Less than 1 hr.
- ② One to 2 hours
- ③ Three hours
- ④ Four or 5 hours
- ⑤ Six hours or more

162. How long do you remain restored after a rest?

- ① Less than 30 min.
- ② 30-59 minutes
- ③ One to 2 hrs.
- ④ Three to 4 hrs.
- ⑤ Five hours or more

163. What is your current weight (in lb.)?

- ① 134 lb. or less
- ② 135-159 lb.
- ③ 160-183 lb.
- ④ 184-209 lb.
- ⑤ 210 lb. or more

164. What was your weight six months ago?

- ① 134 lb. or less
- ② 135-159 lb.
- ③ 160-183 lb.
- ④ 184-209 lb.
- ⑤ 210 lb. or more

165. What was your weight at age 20?

- ① 125 lb. or less
- ② 126-139 lb.
- ③ 140-155 lb.
- ④ 156-175 lb.
- ⑤ 176 lb. or more

166. How many cups of regular coffee do you have in a day?

- ① None
- ② One cup
- ③ Two cups
- ④ 3 to 5 cups
- ⑤ Six cups or more

167. How many of the coffees are within 2 hrs. of bedtime?

- ① None
- ② One cup
- ③ Two cups
- ④ 3 to 5 cups
- ⑤ Six cups or more

168. How many glasses/cans of cola drinks do you have in a day (do not include decaffeinated types)?

- ① None
- ② One can
- ③ Two cans
- ④ 3 to 5 cans
- ⑤ Six cans or more

169. How many of these colas are within 2 hrs. of bedtime?

- ① None
- ② One can
- ③ Two cans
- ④ 3 to 5 cans
- ⑤ Six cans or more

170. How many years were you a smoker?

- ① None
- ② One year
- ③ 2 to 12 years
- ④ 13 to 25 years
- ⑤ 26 years or more

171. How long does it take you to adjust after traveling across time zones (especially 4 or more zones)?

- ① No time at all
- ② One day
- ③ Two days
- ④ Three to 4 days
- ⑤ Five or more days

172. How tall are you?

- ① 63 in. or less
- ② 64 to 66.5 in.
- ③ 67 to 69.5 in.
- ④ 70 to 71 in.
- ⑤ 71.5 inches or taller

173. How old are you now?

- ① 25 or under
- ② 26-35 yr.
- ③ 36-44 yr.
- ④ 45-50 yr.
- ⑤ 51 yr. or older

174. How many years did you go to school? Include years of college and university too.

- ① 4 yr. or less
- ② 5-11 yr.
- ③ 12 yr.
- ④ 13-14 yr.
- ⑤ 15 yr. or more

