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**Electroencephalographic Correlates of Sleep Onset in
Chronic Psychophysiological Insomniacs and Normal Sleepers**

Richard A. Bonato, M.A., R.Psg.T.

A thesis submitted to the Faculty of Graduate
Studies and Research of Carleton University
in partial fulfillment of the requirements
for the degree of Doctor of Philosophy

Department of Psychology

Carleton University

Ottawa, Ontario

Canada

May 1997

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Electroencephalographic Correlates of Sleep Onset in
Chronic Psychophysiological Insomniacs and Normal Sleepers

submitted by

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in partial fulfillment of the requirements
for the degree of Doctor of Philosophy

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Carleton University
May 1997

Abstract

This study compared the sleep onset periods of chronic psychophysiological insomniacs with those of sex- and age-matched good sleepers. The insomniac sample included 15 medically healthy chronic psychophysiological insomniacs recruited from the population of patients regularly referred to the Royal Ottawa Hospital Sleep Disorders Laboratory. The control group of 15 normal individuals was also free from any neurological or physical illness. Each carefully screened subject slept for three consecutive nights in the sleep laboratory while both electroencephalographic and a behavioural response (auditory stimuli) measurement of sleep/wakefulness were recorded.

Subjective sleep onset measures were significantly longer in both groups than the traditional stage 1 definition of sleep onset, and within the control group, a behavioural response definition of sleep onset latency was significantly longer than the 90 second definition of sleep onset. This brings the usefulness and accuracy of the 90 second definition of sleep onset into question. The behavioural definition of sleep onset was aligned between stages 1 and 2 sleep, which was interpreted within the concept of a sleep onset period. As predicted, insomniacs showed significantly more stage reversals prior to the first 15 minutes of continuous sleep (excluding stage 1 sleep). This finding suggested that the tendency for insomniacs to overestimate sleep onset latency may, in part, be accounted for by a combination of a lighter sleep and a greater frequency of stage reversals than occurs in good sleepers during the sleep onset period.

Sleep/wakefulness was accurately assessed in both groups by an economical behavioural response system which showed overall concordance with traditional polysomnograms of greater than 93%. The high degree of multivariate overlap between polysomnographic and behavioural sleep/wake measurements, as shown by the significant canonical correlations, was compelling evidence indicating that the set of behavioural sleep/wake measures were strongly associated with the set of polysomnographic sleep/wake indices.

Period-amplitude analysis (PAA) of sleep stages during the transition from wake to sleep revealed a decrease in beta, sigma, and alpha values, and an increase in theta and delta values. More interestingly, beta EEG values during early stage 2 sleep were greater

in the insomniac group than in the control group, and alpha measures during the sleep onset period were greater within the insomniac group than within the control group, suggesting the presence of alpha intrusion as an indicator of an impaired sleep onset process within the insomniac group. This suggests that insomniacs have a higher level of cortical arousal which impairs the ability to initiate sleep. The finding that control subjects showed more delta activity during sleep onset than the insomniac group confirms the utility of digital analytical techniques because it revealed that controls proceed more deeply down the arousal continuum than insomniacs.

Concerning the reaction time (RT) measures, 10-second samples of pretone EEG were sampled and subjected to a PAA algorithm. The analyses revealed a novel dissociation between EEG amplitude and incidence during the sleep onset period. Although there were no changes in EEG amplitude as responses slowed (RT Bins 1 through 4) and finally ceased (RT Bin 5), differences in EEG incidence were observed as sleep was entered. These PAA findings extend and clarify the traditional belief, based on fast Fourier transform (FFT) analysis, that increased neuronal synchronization is consistently associated with increased amplitude as sleep begins. The dissociation of amplitude and incidence observed herein indicates that the neuronal activity in the brain begins to slow down before any significant increase in EEG amplitude is observed, suggesting that incidence change is an excellent indicator of sleepiness.

In conclusion, this study provides new and important information about alterations in EEG which occur very early in the sleep onset process: brain frequencies begin to slow down before there is an increase in amplitude and this change in EEG frequency, rather than amplitude, is an excellent index of sleepiness and sleep onset. In addition, this study reveals that the sleep onset process is fundamentally different between insomniacs and good sleepers: insomniacs show more alpha EEG activity, more beta EEG activity during stage 2 sleep, lower amounts of delta EEG, and more stage reversals than good sleepers during the sleep onset process. Thus, sleep onset in insomniacs is of a lighter nature and vastly different from sleep onset in controls. Finally, the results of the present study replicate and extend past results which have shown the validity and accuracy of the behavioural response system for the discrimination of sleep and wakefulness.

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Richard A. Bonato, Ph.D., M.A., R.Psg.T.

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Introduction

Insomnia may be broadly defined as the inability to initiate and/or maintain sufficient sleep followed by a decrease in daytime functioning. The International Classification of Sleep Disorders: Diagnostic and Coding Manual (ICSD) (1990) describes three primary insomnia subtypes in greater detail: (1) psychophysiological insomnia; (2) sleep state misperception; and (3) idiopathic insomnia. Psychophysiological insomnia, the focus of the present paper, can easily be differentiated from both sleep state misperception, and from idiopathic insomnia. The latter is a chronic condition of poor quality sleep which usually begins during childhood, whereas psychophysiological insomnia typically begins during the second or third decade of life and gradually exacerbates due to learned sleep-preventing associations. Psychophysiological insomnia can be readily distinguished from sleep state misperception using polysomnographic measures. The polysomnogram recorded from psychophysiological insomniacs will confirm the subjective complaint; however, polysomnographic measures recorded from individuals with sleep state misperception will yield results virtually identical to those of good sleepers although the subjective complaint of poor quality sleep will persist.

Psychophysiological insomnia has also been referred to as learned or conditioned insomnia (ICSD, 1990). For individuals who suffer from psychophysiological insomnia, learned sleep-preventing associations - either learned associations to internal cognitions or learned associations to external stimuli - directly interfere with sleep. Learned internal associations largely consist of an overconcern with sleep initiation and/or maintenance. Such individuals become preoccupied with their sleep performance per se, and will attempt to rectify the situation by trying harder to sleep. Unfortunately, a vicious cycle then develops and increased sleep effort leads to increased agitation and frustration, and in turn, increased sleeping difficulty (ICSD, 1990). It is often the case that falling asleep is easily achieved when no effort is made, as when reading or watching television.

Learned external factors causing psychophysiological insomnia develop from prolonged association of sleep-related behaviours and situations with sleepless nights (ICSD, 1990). Psychophysiological insomniacs with conditioned arousal will often sleep better when away from home, and may show a reverse first-night effect when in the sleep laboratory.

According to Spielman and Glovinsky (1991), predisposing, precipitating, and perpetuating factors contribute to the development of insomnia. A weak sleep-generating system is an example of a predisposing factor, and Hauri (1982) has speculated that this might be another factor necessary for the development of psychophysiological insomnia. Many psychophysiological insomniacs were only fair sleepers before their insomnia began, and consequently, it has been suggested that the occasional sleepless night has reinforced the learned sleep-preventing associations to the extent that elimination is difficult (ICSD, 1990). A precipitating factor is one which initiates or triggers the insomnia disorder; anxiety over a new job is often cited as an example of a precipitating factor. "Both internal and external associations are frequently learned during a period of poor sleep caused by other precipitating factors, such as depression, pain, poor sleep environment, or shiftwork" (ICSD, 1990, p. 29). Perpetuating factors - both mental states and behavioural practices which are not present during inception but appear as a consequence of coping - then follow and maintain the poor sleep performance (Spielman & Glovinsky, 1991). Excessive time in bed, irregular sleep and wake times, unpredictability of sleep, anxiety over daytime deficits, expectation of a bad night, sleep fragmentation, maladaptive conditioning, caffeine consumption, and substance dependence, are all examples of perpetuating factors. In summary, psychophysiological insomnia is characterized by the individual's preoccupation and overconcern about her/his sleep problem to such an extent that the heavy focus on the insomnia (analogous to a specific performance anxiety) continuously interferes with good sleep.

Psychophysiological insomnia is a disorder in which the insomnia is *the* primary feature

and must be distinguished from psychiatric disorders in which the insomnia is a symptom secondary to the psychopathology.

Morin (1993) offers an integrative model of insomnia which uses a multidimensional approach to the understanding of insomnia. In his model, chronic insomnia is a problem which is almost always preceded by situational insomnia - insomnia brought on by a stressful life event, such as the death of a loved one, which fails to improve after the stressor is removed. Morin considers hyperarousal (i.e., emotional, cognitive, physiological) to be a central mediating feature of insomnia and a common pathway for the development of insomnia. Thus, hyperarousal predisposes an individual to insomnia. Negative daytime events, cognitive ruminations, and unrealistic expectations then fuel bedtime arousal which increases sleeplessness. Concern about sleep itself then ensues (sleep performance anxiety) followed by attempts to try harder to fall asleep. As sleeplessness worsens, daytime functioning deteriorates, performance suffers, and falling asleep becomes associated with worry about daytime functioning. Eventually, a sense of learned helplessness develops and the cycle of emotional distress, cognitive arousal, and additional sleeplessness advances. In the attempt to cope with this dilemma, insomniacs develop maladaptive sleep habits such as spending more time in bed, daytime napping, and consuming alcohol to aid sleep onset. The scenario worsens over the long-term as insomnia becomes entrained, and emotional distress exacerbates the sleep disorder.

Insomnia has historically been considered to be an indicator of anxiety or depression (Hauri & Fisher, 1986; Haynes, Follingstad, & McGowan, 1974; Wexberg, 1949), but in 1979 the Association of Sleep Disorders Centers (ASDC, 1979) published a nosology of sleep and arousal disorders which described in detail a sleep disorder known as Persistent Psychophysiological Insomnia. This description suggested that insomnia may be the exclusive result of learned sleep-preventing associations rather than a symptom of some psychiatric or medical disorder. It is essential, therefore, to

differentiate psychophysiological insomnia from insomnia secondary to anxiety syndromes, phobias, obsessive-compulsive neurosis, major depression, or other DSM-III-R diagnosable psychopathologies (Hauri, 1986; ICSD, 1990). Research has failed to establish any clear association between psychophysiological insomnia and psychopathology, but patients with this kind of insomnia tend to be guarded, with denial and repression often being their main defences (ICSD, 1990).

The most common symptoms of insomnia include an increased latency to sleep onset, increased time awake after sleep onset, premature early morning awakening, decreased sleep efficiency (a ratio of time asleep to time in bed), and an associated complaint of decreased daytime functioning (ICSD, 1990). Decreased attention, concentration, energy, and vigilance, and a deterioration in mood and motivation are often associated with the increased fatigue and malaise resulting from the poor quality sleep (ICSD, 1990). Nationwide health surveys in the United States have shown a prevalence of insomnia ranging from 21 percent (Hammond, 1964) to 35 percent (Karacan et al., 1976); the difference in prevalence most likely due to the question subjects were asked. Specifically, the former asked about insomnia, whereas the latter questioned subjects concerning difficulty sleeping. Such surveys are not able to distinguish between the insomnia subtypes, so the prevalence of psychophysiological insomnia in the population is unknown (ICSD, 1990). According to the ICSD, approximately 15% of all people presenting to the sleep disorders centre with a complaint of insomnia will be diagnosed with psychophysiological insomnia.

Historically, there has been a tendency to use the electroencephalogram (EEG) as *the* criterion of sleep for insomniacs rather than investigating more precisely the dimensions of behaviour and experience that lead us to recognize sleep. In the literature, researchers frequently refer to the EEG as the “objective” or “true” measure of sleep. However, this reference ignores the fact that the validity of the EEG as an index of sleep

is based entirely on its covariation with subjective and behavioural indicators of sleep (Davis, Davis, Loomis, Harvey, & Hobart, 1938; Loomis, Harvey, & Hobart, 1936, 1937; Kamiya, 1961; Rechtschaffen, 1994). The use of the EEG as the criterion of sleep may lead to confusion when found to be at variance with behavioural and subjective indices. For example, parasomnias (sleepwalking, sleepwalking, etc.) involve a paradox between electrophysiology and observed behaviour. In the study of chronic insomnia, such instances of disagreement between polysomnographic measures and subjective impressions regarding sleep states are well documented (Carskadon, Dement, Mitler, Guilleminault, Zarcone, & Spiegel, 1976; Frankel, Coursey, Buchbinder, & Snyder, 1976; Hauri & Olmstead, 1983; Knab & Engel, 1988), and in its most severe form, the discordance between polysomnographic and subjective sleep results in a diagnosis of sleep state misperception.

Measuring Sleep Onset

The traditional approach to establishing sleep onset involves scanning each epoch (typically a page of 30 seconds duration recorded at a paper speed of 10 mm/sec) and assigning the epoch a categorical variable: (1) wakefulness; (2) stage 1 sleep; (3) stage 2 sleep; and more rarely (4) stage REM sleep (Rechtschaffen & Kales, 1968). An epoch is assigned a sleep stage score when 50 percent or more of the epoch is consistent with the criteria for that particular sleep stage (see below). Typical sleep onset for a healthy adult is from wakefulness to stage 1 sleep, and then stage 2 sleep.

Wakefulness, as defined by the EEG, consists of either high-frequency, low voltage desynchronous activity (beta waves, 16 Hz to 30 Hz) or 8 Hz to 12 Hz sine wave activity known as alpha waves. Alpha waves and beta waves were first identified and named by Berger in 1929. The alpha rhythm is most prominent over the occipital cortex (Loomis, Harvey, & Hobart, 1937) when the individual is relaxed with eyes closed.

The disappearance of the alpha rhythm heralds the onset of stage 1 sleep: the 8 Hz to 12 Hz activity gives way to low-voltage, mixed frequency brain waves of 4 Hz to 8 Hz called theta waves. At one time, the theta frequency band was subsumed within the delta range, but early researchers considered an intermediate frequency band to be necessary (Niedermeyer, 1993). As a result, the term *theta* was chosen to describe EEG within the 4 Hz to 8 Hz range because of the presumed thalamic origin of this brain wave frequency. Vertex sharp waves may be seen in addition to the low-voltage, mixed frequency activity. Stage 1 sleep usually occupies 5-10 percent of the entire night.

After a few minutes of stage 1 sleep, a transient phenomenon such as a sleep spindle or a K-complex will occur thus defining stage 2 sleep. A sleep spindle consists of 12 to 14 Hz EEG activity with a minimum duration of .5 seconds. A well-delineated negative component (tradition has dictated that negative is up) followed immediately by a positive deflection describes the morphology of a K-complex. According to Niedermeyer (1993), sleep spindles are also known as sigma waves or sigma activity, and are typically characterized by a group of rhythmic waves which gradually increase and decrease in amplitude (similar in appearance to a football). Unfortunately, the waxing and waning 12 Hz to < 16 Hz waves are often poorly formed. Sleep spindles are only observed in the EEG of healthy adults during stage 2 sleep which comprises about 40-50 percent of the night. Functionally, sleep spindles and K-complexes remain a mystery with some suggesting an endogenous arousal significance (Niedermeyer, 1993), and others suggesting evidence for bona fide sleep and loss of consciousness (Steriade & McCarley, 1990).

The reader should note that electro-oculographic (EOG) and electromyographic (EMG) measurements are made in conjunction with EEG recordings, and that these additional parameters are used to assist in the identification of certain sleep stages such as REM sleep (Rechtschaffen & Kales, 1968). With regard to the EOG and EMG

measurements, the EOG is characterized by slow movements during stage 1 sleep (i.e., SEMs); rapid movements during stage REM sleep; and relative inactivity for the remainder of the sleep stages. The EMG is at its highest amplitude during wakefulness and progressively decreases from stage 1 through to stage 4 sleep. The EMG is at its lowest during REM sleep, which is characterized by atonia.

The Perception of Sleep

It is frequently reported that insomniacs tend to overestimate sleep onset latency and time awake, and underestimate the number of awakenings (i.e., reporting they were awake the entire duration), thereby underestimating the amount of sleep (Carskadon, Dement, Mitler, Guilleminault, Zarcone, & Spiegel, 1976; Frankel, Coursey, Buchbinder, & Snyder, 1976; Hauri & Olmstead, 1983; Knab & Engel, 1988). Carskadon et al. compared polysomnographic measures to the self-reports of 122 drug-free chronic insomniacs, and found that while the polysomnogram generally confirms the poor sleep reported by patients, insomniacs overestimated sleep onset latency and the durations of waking periods after sleep onset, and underestimated the number of awakenings. These findings were substantiated by Frankel et al. who examined the polysomnographic and self-report data of 18 chronic insomniacs. Knab and Engel (1988) hypothesized that good sleepers “smooth” their perceptions of sleep and wakefulness in the direction of sleep, whereas the reverse is true for insomniacs. While these findings suggest that some discrepancy exists between EEG sleep and the self-reports, it is also possible that the definition of polysomnographic sleep onset used was too liberal. For example, Carskadon et al. defined “objective” sleep onset as the time from lights out to the first three consecutive epochs of stage 1 sleep or the first epoch of stage 2 sleep. However, research into “normative” sleep has established that sleep onset is not a straightforward and unitary phenomenon (Bonnet & Moore 1982; Kamiya, 1961; Ogilvie & Wilkinson, 1984,

1988).

Kamiya (1961) reported a study in which 20 “sleepers” were awakened at the first sign of a sleep spindle - the EEG criterion of stage 2 sleep in humans and the point usually taken as definite indication of sleep onset (Johnson, 1973) - and asked whether they had been asleep. On 19 of 47 occasions subjects reported that they had definitely been awake prior to being questioned. Only on 12 occasions did subjects respond in the affirmative. Reports of imagery occurred on four occasions, and uncertainty of the subjective state was reported upon 12 “awakenings.” This study clearly indicates the imperfect relationship between electroencephalographic and subjective indices of sleep/wakefulness.

Bonnet and Moore (1982) examined the perception of sleep and wakefulness in normal sleepers (six women and six men) during EEG-defined sleep onset. Immediately following the first sleep spindle, a tone was produced and subjects were asked whether they had been asleep or awake when the tone was presented. The results are particularly interesting. Approximately 35 percent of subjects reported being asleep following the first sleep spindle; sleep was reported 50 percent of the time within 2 to 4 minutes of the initial sleep spindle (i.e., 2-4 minutes after the beginning of stage 2 sleep). The authors termed this period the sleep onset threshold - the time when sleep is reported by 50 percent of the subjects. This is clear evidence that EEG-associated changes are not necessarily coincident with subjective alterations in state. The authors concluded that subjective sleep onset in “normal” sleepers is a time during which state perception is blurred and uncertain. In light of such research results, it would be unreasonable to expect that insomniacs’ perception of sleep onset would be any better than that of normals. Consequently, one might wonder whether the finding that insomniacs overestimate sleep onset latency is robust given the instability of the phenomenon in good sleepers; that is, that 50 percent of good sleepers will report sleep onset to occur 2-4 minutes after the

initial sleep spindle. The issue of sleep onset measurement for insomniacs was considered by Hauri and Olmstead (1983).

Hauri and Olmstead (1983) examined sleep onset in the following insomniac groups: psychophysiological insomniacs (n=20); subjective insomniacs (n=18); medical insomniacs (n=8); insomniacs with depression (n=10); and controls (n=10).

Psychophysiological insomniacs were considered to be those who reported insomnia persisting for at least two years in addition to a polysomnographic sleep onset latency of 30 minutes or more, and a polysomnographic sleep efficiency of less than 86%.

Subjective insomniacs were defined as those who complained about serious insomnia for two years, but had both a polysomnographic sleep onset latency of less than 30 minutes, and a polysomnographic sleep efficiency greater than 90% on at least two of the three laboratory nights.

The results of Hauri and Olmstead's (1983) study indicated that most controls identified their sleep onset latency as being within the first five minutes of stage 2 sleep whereas the insomniac groups identified sleep onset as occurring approximately 15 minutes into stage 2 sleep. Unfortunately, no tests of statistical significance were reported, but rather, the groups were matched with time of sleep onset based on group averages. Nevertheless, the results of this study suggest that the identification of sleep onset, particularly in insomniacs, is not straightforward and requires further investigation.

Behavioural Measures of Sleep/Wakefulness

Given that research has identified the limitations associated with EEG-based and subjective definitions of sleep/wakefulness, what alternatives are available which would contribute additional information regarding sleep and wakefulness in insomnia?

Researchers have successfully applied various behavioural measures of sleep/wakefulness to overcome the limitations of EEG-based definitions. Obtaining data

from behavioural tasks is typically accomplished within one of three paradigms: (1) a passive system (as in a “dead” switch) in which loss of tonus releases a thumb-switch; (2) an active, uncued signal system in which the participant actively indicates perceived wakefulness by the depression of a thumb-switch; and (3) an active, cued response system whereby the subject responds to a faint tone (e.g., a 1000 Hz tone 5 dB above background noise) with the depression of a thumb-switch. Such behavioural measures of sleep/wakefulness evaluate the sleeper's responsiveness to external stimuli (predominantly active, cued responding) and subjective judgement regarding the sleep state (primarily active, uncued signals). These paradigms have most frequently been used to evaluate sleep-associated alterations in EEG coincident with behavioural phenomena.

The first documented study which simultaneously measured behavioural and electroencephalographic indications of sleep was conducted by Davis, Davis, Loomis, Harvey, and Hobart (1938). These pioneers recorded 20 nights of sleep from 10 different subjects during a study which specifically examined behavioural, electroencephalographic ($C_z-A_1+A_2$, $O_z-A_1+A_2$), and subjective measures of sleep onset. Subjects were required to squeeze a “bulb” (i.e., an active, uncued behavioural signal) whenever they felt they had momentarily “floated” or “drifted” off. In general, the results indicated agreement between the measures; however, episodes of disagreement were observed. These investigators concluded that the process of falling asleep is not a unitary function, and that the moment of falling asleep can only be precisely defined if a particular operational definition is used. “Otherwise the problem [of determining sleep onset] is just as vague and difficult as that of determining the exact moment of death.” (p. 35)

Decades later, Campbell and Webb (1981) used a similar active, uncued signalling system (single button press), to study the perception of sleep in 40 healthy males (mean age was 55.3 years, and range was 50-60 years). Subjects were instructed to

press a button whenever they perceived themselves to be awake anytime during the night. The authors reported that 10 percent of the total number of behavioural responses recorded occurred during EEG-defined sleep. Moreover, subjects failed to respond from 27 percent of EEG-defined wake periods which were one minute in duration or longer. Thus, EEG-defined wakefulness (alpha EEG) was neither required for behavioural wakefulness, nor did it ensure that behavioural signalling would occur. According to Campbell and Webb, active, uncued signals contributed important information concerning arousals during sleep, and these researchers suggested that such a technique may be useful in clinical settings where sleep maintenance is a component of the sleep disturbance.

Anch, Salamy, McCoy, and Somerset (1982) used an active, uncued behavioural response system and compared it with EEG-defined wakefulness (alpha EEG). This study attempted to overcome the possibility of false positives by modifying the single button press technique used by Campbell and Webb (1981). Anch et al. instructed subjects to signal wakefulness by pressing a palm-mounted button three times; less than three consecutive button presses were not considered to be intentional attempts to indicate wakefulness and were excluded from any analyses. Seven healthy men ($\bar{x}$ age = 24.0 years) slept for three consecutive nights in the sleep lab while EEG (C_3-A_2 & O_3-P_2) and behavioural responses were recorded. A total of 111 triple button presses were obtained during the 21 recording nights, and the average per night response rate across subjects was 5.29. Using this triple button press measure of sleep/wakefulness, Anch et al. found that behavioural responses either coincided almost exactly with the appearance of alpha activity, occurred shortly after alpha activity, or occurred following intermittent bursts of alpha and non-alpha EEG. In either case, behavioural responses did not occur greater than 20 seconds following the presence of alpha activity. Unfortunately, the investigators did not report whether any responses failed to occur during EEG-defined wakefulness

(alpha activity) so it is not possible to determine the likelihood of false negatives.

Birrell (1983) used an active, cued behavioural response measure in addition to subjective and sleep EEG measures. Nine women and eight men slept in the sleep laboratory for three nights, and were required to respond to auditory tones at two minutes, five minutes, or random intervals. Average response rates to the tone presentations were 100 percent during wakefulness, 81 percent during stage 1 sleep, and eight percent during stage 2 sleep. Interestingly, the behavioural and stage 2 sleep index of sleep onset latency and sleep duration were virtually identical. Most of the stage 2 sleep responding was during the first five minutes of stage 2 sleep, and this is consistent with Bonnet and Moore's observation that the threshold for sleep onset - when 50 percent of subjects will report sleep - occurs within a four-minute period after the initial sleep spindle. The subjective estimates provided the longest measure of sleep onset, whereas a stage 1 sleep onset latency definition provided a time estimate significantly shorter than all other measures of sleep onset latency (i.e., behavioural, stage 2 sleep, subjective). From a behavioural perspective, these data strongly suggest that it is stage 2 sleep which should be considered genuine sleep; stage 1 sleep should be considered an intermediate stage between wakefulness and sleep.

In another application of an active, cued behavioural response measure, Ogilvie and Wilkinson (1984) reported physiological and behavioural convergence. Following a night of total sleep deprivation, five men and seven women slept in the sleep laboratory for two 45-minute test sessions spaced one hour apart. During the testing sessions, subjects were presented with faint (5 dB above background noise) 1000 Hz tones randomly produced from one to ten seconds apart and lasting a maximum of eight seconds. Subjects were instructed to press a button located in their palm if they heard the tone, and to continue to press the button as long as they were awake and heard the tone; however, participants were told that they should not be concerned about trying to stay

awake. The results of this investigation revealed a general agreement between behavioural responsiveness and the EEG (C_z-A_1 , P_z-A_1). Subjects responded to more than 99 percent of tones during EEG-defined stage wake, and failed to respond to 28 percent and 76 percent of tones presented during EEG-defined stages 1 and 2 sleep, respectively. Behavioural responses occurred during approximately five percent of tones presented during stage 3 sleep. Although this does suggest a gradual and progressive “shift” towards convergence, the divergence is remarkable. The authors interpreted the results of this study to suggest convergence, but the generalizability of the findings is limited because this study was conducted within a sleep deprivation paradigm.

Perry and Goldwater (1987) used a passive behavioural device to examine sleep onset. These investigators had 13 men and seven women use a behavioural device in which loss of extensor tension in the finger depressed a key and closed a circuit. A strong association was found between a loss of alpha EEG and key closure events (behavioural sleep). Overall percent concordance between EEG-defined wakefulness and the behavioural measure was 96 percent, with a range from 90 percent to 100 percent agreement. Overall percent concordance between the passive behavioural measure and stage 1 sleep was 63, with individuals ranging from 22 percent to 95 percent agreement. Mean latencies between alpha loss and key closure ranged from -1.8 to 18.4 seconds, with a median of 1.0 second. While Perry and Goldwater concluded that a very clear association exists between the EEG (stage 1 sleep) and a behavioural index of sleep onset, other researchers have found that behavioural sleep best agrees with stage 2 sleep (Ogilvie and Wilkinson, 1984, 1988; Viens, DeKoninck, Van den Bergen, Audet, and Christ, 1988).

Viens et al. (1988) compared a sleep onset latency clock (a passive behavioural measure) to EEG criteria. This sleep onset latency clock device consisted of a time base counter and a coded display module. Initial circuit closure (depression of thumb-switch in

preferred hand) caused the screen to go blank thus preventing subjects from viewing real time. When the circuit was opened (tonus was lost), a coded sleep onset latency value was displayed, and was recorded by the subject on the post-sleep questionnaire the following morning. Viens et al. had three healthy women and one female insomniac sleep in the laboratory for three series of four consecutive nights and two series of three consecutive nights, respectively. The passive behavioural measure was compared to two EEG definitions of sleep onset: stage 1 sleep latency (Rechtschaffen & Kales, 1968); and stage 2 sleep latency (Johnson, 1973). Significant differences were found between the behavioural measure and latency to stage 1 sleep for the insomniac subject, and for two of the three healthy sleepers. No significant differences were found between the clock monitor and latency to stage 2 sleep. This finding is further evidence indicating that behavioural sleep more closely aligns itself to stage 2 sleep, than to the initial alterations in EEG found during stage 1 sleep.

Knab and Engel (1988) used a behavioural task (active, uncued double button press) in addition to the EEG and self-reports, and compared the sleep of insomniacs to that of good sleepers. The sleep logs of insomniacs indicated that they slept poorly, with a longer sleep onset latency, more wakefulness after sleep onset, and less total sleep time. These subjective reports were confirmed by polysomnographic data which also revealed that insomniacs had more wakefulness after sleep onset, less total sleep time, and a lower sleep efficiency. They found that most behavioural signals occurred during EEG-defined wakefulness (alpha waves) and concluded that alpha activity is necessary, but not sufficient, for the perception of awakenings at night. Although the investigators suggest that alpha activity is not sufficient for the perception of wakefulness since many awakenings were not signalled (71% and 67% for insomniacs and controls, respectively), the measure may have been too conservative. It is precisely for this reason that other investigators prefer an active, cued behavioural device rather than an uncued system as

used by Knab and Engel.

Ogilvie and Wilkinson (1988) conducted a follow-up study to their 1984 paper already described. Therein they reported “behavioural wakefulness” to occur within EEG-defined stages 1 and 2 “sleep”; responsiveness to the external stimulus was much less likely to occur during stages 3, 4, and REM sleep. This latter finding is contrary to Campbell and Webb's (1981) observation for stage REM sleep signalling, and suggests the existence of false positives in Campbell and Webb's study. It is unlikely that the tones used by Ogilvie and Wilkinson were excessively intrusive and caused subjects to “awaken” and respond because the tones measured only 5 dB above a background noise of 27 dB. Previous studies have found that auditory awakening thresholds rise sharply at sleep onset and remain high, ranging from 15-100 dB above waking levels throughout sleep (Rechtschaffen, Hauri, & Zeitlin, 1966). As a result of these extensive investigations, Ogilvie and colleagues (Ogilvie & Wilkinson, 1984, 1988; Ogilvie et al., 1989, 1991) have recommended adoption of the term “sleep onset period” to describe the transition from wakefulness to sleep. These investigators concluded that data from electroencephalographic, behavioural, and subjective measures supported the notion of a “sleep onset period” and refuted the common view that stage 1 represents sleep. According to these investigators, initial stage 1 sleep occurs during the “sleep onset period.” Incidentally, this recommendation was first made in the 1930s by the pioneers Davis and Loomis.

A subsequent study by Ogilvie and colleagues (Ogilvie, Simons, Kuderian, MacDonald, & Rustenburg, 1991) used the fast Fourier transform method and found that an increase in EEG power (C_4-A_2) across all EEG bandwidth frequencies (alpha, beta, delta, theta, and sigma) was associated with progressive lengthening of response time. The increase in power across all bandwidth frequencies was interpreted by Ogilvie et al. to suggest that a behavioural definition of sleep/wakefulness was the most powerful

sleep/wake discriminator yet identified. This strongly suggests that the combination of behavioural and electroencephalographic sleep measures can provide additional information not obtained by traditional polysomnographic indices.

Variations on the auditory stimulus-response paradigm have included vibratory stimulation (Kemp & MacLean, 1994; MacLean, Arnedt, Biedermann, & Knowles, 1992). Kemp and MacLean reported a relationship between the least and most stringent behavioural definitions and the least and most stringent polysomnographic definitions. They also observed behavioural responses to occur during polysomnographically defined stage 1 sleep, and concluded that a behavioural measure of sleep contributes important information not obtained with polysomnographic methods.

Most researchers who have investigated behavioural and electroencephalographic indices of sleep have arrived at the same conclusion: it would be fruitless to reconcile all differences in these measures by distinguishing between “physiological sleep” and “behavioural sleep.” These findings merely point out the imperfect validity of the physiological indicator, and the imperfect relationship between the various sleep indices. With regard to insomnia, the literature suggests that the combination of the behavioural signalling/response paradigm with traditional polysomnographic measures may contribute additional information to more accurately reflect the nature of psychophysiological insomnia (i.e., cases in which dysfunction is primarily due to insomnia, and is not secondary as in depression). The application of a signal transformation technique, such as Fast Fourier Transform (FFT) or digital Period-Amplitude Analysis (PAA), will provide additional precision which may reveal a physiological abnormality not observed through the application of the traditional Rechtschaffen and Kales (1968) sleep stage schema.

The application of behavioural measures to assess insomnia has primarily been used in home settings. Bonato and Ogilvie (1989), and Kuderian, Ogilvie, McDonnell,

and Simons (1991) used an active, cued behavioural measure of sleep/wakefulness to differentiate good from poor sleepers in the home: however, neither of these studies used either a clinical sample or electroencephalographic measures. A literature search revealed only two studies, Cote and Ogilvie (1994) and Knab and Engel (1988), which used both behavioural and electroencephalographic measures to define sleep in poor sleepers.

Concerning the Cote and Ogilvie (1994) study, the insomniac subjects were not obtained from a clinical sleep disorders center, but rather were obtained from a university student population, and thus may not have been representative of chronic psychophysiological insomniacs. In that study, subjects were required to press a thumb-switch whenever they heard a low-level tone; that is, a button press would indicate wakefulness whereas response failure would indicate sleep. The authors observed that concordance between behavioural and polysomnographic indices of sleep/wakefulness was lowest during stage 1 sleep, and increased from stage wake, through stages 2 and REM sleep to reach the highest level of concordance (approximately 100%) during slow wave sleep. In addition, Cote and Ogilvie found that arousals were just as likely to occur *before* the auditory stimulus as they were to occur *after* the auditory stimulus. This allowed them to argue that low level auditory stimuli presented during sleep do not cause subjects to awaken and respond to stimuli.

Knab and Engel (1988) used both behavioural and electroencephalographic measures to define sleep in a clinical sample of insomniac subjects. Recall that Knab and Engel used an active, uncued double-button press to indicate perceived wakefulness; however, in the only study comparing passive and active behavioural measures of sleep/wakefulness, Ogilvie (1985) found that an active, cued system was best. A passive measure is the least intrusive behavioural test used to evaluate sleep/wakefulness, but a shortcoming of this technique is that subjects infrequently resume the task once initial sleep onset has occurred. Consequently, a passive behavioural measure of sleep

preferentially detects sleep onset (Perry & Goldwater, 1987; Viens et al., 1988) and is only suited for the detection of the loss of tonus which coincides with EEG-defined stage 1 sleep according to Perry and Goldwater (1987) and Blake, Gerard and Kleitman (1939). An active, uncued behavioural test of sleep/wakefulness also does not intrude upon sleep, and offers the ability to detect perceived wakefulness after initial sleep onset.

Unfortunately, subjects frequently fail to indicate wakefulness during the night, most likely due to the absence of any cue (Anch et al., 1982; Campbell & Webb, 1981; Knab & Engel, 1988). An active, cued behavioural measure of sleep/wakefulness possesses the most desirable characteristics of any behavioural device. It is precisely because cued responding relies on periodic probes (low intensity sound) that this method provides a more accurate and continuous description of sleep/wakefulness than active, uncued systems.

It is unlikely that such tones are excessively intrusive and cause subjects to “awaken” and respond because, as already indicated, auditory awakening thresholds rise sharply at sleep onset and remain high, ranging from 15-100 dB above waking levels throughout sleep (Rechtschaffen, Hauri, & Zeitlin, 1966). In a study of auditory threshold at sleep onset, Bonnet and Moore (1982) found that the threshold rapidly increased during sleep onset and had reached 64% of the maximum amount within one minute of stage 2 sleep entry. Additional work investigating auditory awakening thresholds in psychophysiological insomniacs, subjective insomniacs, and controls (Bonnet & Johnson, 1978; Johnson, Church, Seales, & Rossiter, 1979; Haynes, Fitzgerald, Shute, & O'Meara, 1985) has consistently found no difference in the auditory awakening thresholds of good and poor sleepers. Haynes et al. (1985) presented an ascending series of 1000 Hz tones to psychophysiological insomniacs ($n = 10$), subjective insomniacs ($n = 9$), and noninsomniac controls ($n = 22$), and found no statistically significant between-group differences in electrophysiologically determined auditory awakening thresholds. Using

polysomnography to define an awakening, they reported that the mean awakening threshold for the psychophysiological, subjective, and noninsomniacs was 65.2 ± 11.8 , 61.8 ± 12.2 , and 60.5 ± 12.0 decibels, respectively. Using subjective experience to define an awakening, they found that the mean awakening threshold for the psychophysiological, subjective, and noninsomniacs was 76.8 ± 9.2 , 73.6 ± 18.9 , and 75.5 ± 23.8 decibels, respectively. This is strong evidence which further negates the notion that an active, cued behavioural response device - producing a tone in the neighbourhood of 30 decibels - is excessively intrusive and causes subjects to “awaken” and respond to the tone. Such research also refutes the suggestion that insomniacs are more sensitive to auditory stimuli than controls, and thus more susceptible to nocturnal awakenings. Clearly, the literature reviewed thus far indicates that the use of an active, cued behavioural response device in combination with electrophysiological measures would be the most productive approach towards an understanding of the sleep onset process in insomnia. Although an active, cued behavioural response device will not cause subjects to “awaken” and respond to the tone, some habituation both to the device and to the sleep environment is typically required (Bonato & Ogilvie, 1989).

First Night Effect

The quality of sleep is often worse during the first night in a sleep laboratory than that experienced at home. This transient alteration in sleep is said to be the result of habituation to an unfamiliar environment, and has been termed the first-night effect. Consequently, many researchers frequently omit first-night data from statistical analyses (Agnew & Webb, 1966). The first-night effect is characterized by decreased sleep efficiency and increased stage 1 sleep, increased latency to sleep onset, stage 4, and REM sleep, and less REM sleep during the first night in the sleep laboratory than on subsequent nights (Agnew & Webb, 1966; Bonato et al., 1992; Webb & Campbell,

1979). A decrease in the number of sleep stage changes on subsequent nights may also be observed. However, some researchers have demonstrated that the first-night effect is not ubiquitous (Coble, McPartland, Silva, & Kupfer, 1974; Browman & Cartwright, 1980). With regard to the first-night effect and the behavioural response device, research has found that the behavioural response device will produce (and can detect) a first-night effect (Bonato & Ogilvie, 1989; Kuderian et al., 1991). For example, both Bonato and Ogilvie (1989) and Kuderian et al. (1991) found statistically significant increases in behaviourally-defined sleep efficiency from nights one to two. Sleep efficiency was found to stabilize thereafter. It is general practice, then, to use night one as an adaptation night to both the sleep laboratory and to the behavioural response device (e.g., Ogilvie & Wilkinson, 1988).

Digital Analytical Techniques

The use of digital computers in the analysis of sleep is an increasingly popular method with which to overcome the insufficient representation of sleep by a series of stages. The efficiency of computers has continued to increase and cost has continued to decrease so much so that virtually any sleep laboratory can now obtain a data acquisition program. Computers are excellent devices for accurately collecting, manipulating, storing and retrieving large amounts of data at high speed. Importantly, analog-to-digital (A/D) conversion yields data suitable for multivariate parametric analysis. Typical computer quantification paradigms for EEG involve: (1) A/D conversion of EEG at sufficiently fast sampling rates for good band-width resolution; and (2) submitting this digitized data to an algorithm devised to produce one or more measures per epoch. Some of these algorithms include: fast Fourier transforms (FFTs), period-amplitude analysis (PAA), auto-regression, coherence analysis, and phase analysis. Of these, the two most commonly used are FFTs (i.e., spectral analysis), and PAA. The former is a method of

EEG analysis which is mainly in the frequency domain, and the latter is a method predominantly in the time domain. Vociferous arguments have occurred (and frequently persist) between those using the frequency domain and those preferring the time domain concerning which measure was the better. Most research has found that these measures are essentially describing the same creature through the use of a different language. For example, research has revealed canonical correlations of 0.96 and 0.97 between FFTs and PAA performed on the EEG of awake and sleeping subjects, respectively (Pigeau et al., 1981). A recent study has found significant multivariate overlap between the FFT method and PAA and has concluded that the FFT power measure and the PAA amplitude measure essentially report the same results (Armitage, Hoffmann, Fitch, Morel, & Bonato, 1995). However, this study also found that PAA provides additional time-in-frequency data not obtained through the application of the FFT method. A more detailed discussion of the FFT and PAA is therefore appropriate.

In 1965, Cooley and Tukey described an algorithm that substantially reduced the computational effort involved in Fourier transforms. It became known as the FFT, and along with the digital computer it has made the Fourier analysis of large sets of data relatively routine. Fast Fourier Transform techniques are used to compute power spectra. The FFT analysis separates the EEG waveform into sine and cosine components producing a power measure (area under the rectified curve) for each frequency of each epoch, or mean power values for bandwidth frequencies. The time window of the EEG data, and the sampling rate determine the spectrum frequency range and the resolution. The dependent variable, power, is given in either absolute units or relative units.

There has been some criticism concerning spectral analysis. Ktonas & Gosalia (1981) reported that the FFT method confounds wave amplitude and incidence which will produce results indicating that certain EEG epochs have similar power spectra although very different electrographic morphology. Theoretically, an infinity of EEG records can

be associated with a given power spectrum. This criticism is related to the fact that spectral analysis is very sensitive to amplitude (PAA is not) and consequently the FFT technique is biased towards over-representing slow frequencies. Another criticism regarding spectral analysis concerns its assumption that brain waves are sine waves and some have questioned the appropriateness of applying the technique to the EEG. A fundamental assumption in the FFT method is the assumption of stationarity which, essentially, assumes that the waveform under analysis is unchanging and will look the same at whatever point in time observation begins. However, to circumvent the assumption of stationarity in the EEG signal (which, incidentally, is usually violated since the EEG is a very changeable waveform) and to produce a quasi-stationary signal from the EEG, it is common practice when using FFT analysis to break a 30-second epoch down into four second mini-epochs, perform an FFT on each two second mini-epoch, and then average the 15 mini-epochs to obtain FFT power values for the 30-second epoch. Additional work also raises some issues concerning the FFT method. A recent study (Armitage et al., 1995) reported that PAA measures could account for more variance in the FFT measures than vice-versa, and that while both measures generally agreed when examining normals, the amount of agreement dramatically decreased in a sample of depressed unipolar outpatients. This led the authors to question the appropriateness of the FFT method for the study of clinical subjects.

Period-amplitude analysis involves the detection of zero voltage crossing events (i.e., baseline crossings in full-waves or half-waves) and zero slope events (i.e., first-derivative events) in the EEG. The time between each occurrence of these events is measured and recorded as a time-in-frequency (percentage) measure. Because the computer is sampling at a known rate, conversion of the data from number of samples to the corresponding broadband frequency is easily accomplished. The addition of the successive squared unit voltages between half-wave zero crossings can be used to obtain

information about amplitude. Thus, for the one power density measure obtained from the FFT method, four measures are obtained with PAA which are expressed in either percentage activity (alpha, delta, etc.) or in microvolts squared (amplitude).

Criticisms concerning PAA have also been raised. Some (Berger, 1990) have erroneously suggested that the utility of PAA is limited because it is incapable of detecting fast-frequency, low-amplitude components of the EEG which do not produce zero-crossings yet occur simultaneously with low-frequency, high-amplitude activity (e.g., alpha-delta sleep). Contrary to what has been reported, PAA is very capable of detecting alpha-delta activity. To accomplish this, the first-derivative analysis must be used because this measure selectively detects the highest frequency in the EEG signal. Unlike the first-derivative measure, the full-wave measure preferentially detects mid-range components in the EEG; the half-wave measure preferentially detects the slowest waveform in the ongoing EEG. One criticism of PAA is valid: PAA is much more sensitive to noise, such as shifts in the D.C. level, but generally this does not pose a problem because of high-pass filters inherent in modern amplifier design.

Studies Using Digital Analytical Techniques

From a much broader perspective, it is important to closely scrutinize electrophysiological variables as the discriminators of sleep and wakefulness. Since the days of Loomis, Harvey and Hobart (1937) alterations in EEG patterns have been associated with concomitant subjective and behavioural phenomenology. On the other hand, it has been observed by various researchers that the relationship is not absolute. It is essential, therefore, to enhance the resolution of the recording technique through the application of digital polysomnography, and thereby more precisely investigate any association between behavioural, subjective, and electroencephalographic sleep measures. Application of such an approach to the study of insomnia should provide very

useful information regarding the sleep onset process in this sleep disorder. Interestingly, only a limited number of studies can be located which specifically examine quantitative EEG measures during sleep onset. Most of these studies used the FFT method; however, one study was located (Armitage et al., 1994) which used period-amplitude analysis (PAA) to examine sleep onset in depressed outpatients.

In a study of power spectral analysis of sleep onset in 12 chronic insomniacs and 12 control subjects, Freedman (1986) examined the first unambiguous, artifact-free minute of C_3-A_2 and O_1-A_2 EEG from each sleep/wake stage obtained from lights-out (wakefulness) to the end of the first NREM-REM sleep cycle. He found significantly more beta EEG activity in insomniacs than in controls during wakefulness, stage 1 sleep, and REM sleep, but not during stages 2, 3, and 4 NREM sleep. He also reported less alpha EEG activity during wakefulness in insomniacs than in control subjects. The data were interpreted to suggest heightened presleep physiological arousal in chronic insomniacs.

In another examination of sleep onset in chronic insomniacs, Merica and Gaillard (1992) used a beta/delta ratio obtained from the temporal sites of 12 psychophysiological insomniacs and 23 normal control subjects. The sleep onset period lasted for an average duration of three minutes beginning with stage 1 sleep immediately preceding stage 2 sleep. As expected, beta activity decreased and delta EEG activity (beta/delta ratio) increased in both groups, but the rate of change in EEG activity differed: insomniacs showed a higher EEG activity index (beta/delta ratio) during the sleep onset period than the controls, and this activity persisted into the first few minutes of stage 2 sleep. The authors concluded that it is possible to distinguish between the two groups based on beta and delta spectral values obtained in the EEG during the sleep onset period.

Jacobs, Benson, and Friedman (1993) conducted a study to examine the sleep of chronic insomniacs pre- and post-treatment. The treatment consisted of a combination of

behavioural techniques including sleep restriction, sleep hygiene, stimulus control, and relaxation. The pre-sleep EEG activity of the 12 insomniacs and 14 controls were compared both pre- and post- treatment using power spectral analysis. Greater pre-sleep CNS arousal was observed pre-treatment in the form of significantly greater beta activity in the insomniac group. Post-treatment, the authors reported a 75% reduction in sleep-onset latency in the insomniac group which was not significantly different from the age- and sex-matched control group. Furthermore, the level of beta activity in the insomniac group was reduced post-treatment, but remained statistically significantly greater than that of the control group.

Hori, Hayashi, and Morikawa (1994) used both nine EEG stages (equivalent to stages wake, 1 and 2 sleep in the Rechtschaffen & Kales criteria), and the FFT method to examine the EEG associated with hypnagogic experiences. A 50 dB 1000 Hz tone was presented five times during each of the nine EEG stages¹ associated with sleep onset. Subjects were instructed to respond to the tone by pressing a button within five seconds of tone onset. Subjects were awakened if tone failure was observed, and were asked to indicate whether they had been asleep or awake prior to the tone. The authors concluded that reaction time, subjective assessment, and spectral power change as a function of the nine EEG sleep onset stages. Reaction time significantly increased from EEG stage 1 (stage wake according to Rechtschaffen & Kales) to EEG stage 9 (stage 2 according to Rechtschaffen & Kales). By EEG stage 9, 26% of subjects still reported being awake, and 44% thought they were asleep. The authors reported that EEG power decreased between EEG stage 3 (alpha intermittent) and EEG stage 4 (EEG flattening). While this research produced some important findings regarding phenomenology and reaction time during

¹Stage 1: trains of alpha waves (traditional stage wake sleep). Stage 2: Intermittent alpha waves > 50% of epoch (stage wake). Stage 3: Intermittent alpha waves < 50% of epoch (traditional stage one sleep). Stage 4: EEG flattening (stage one). Stage 5 (stage one): ripples in the EEG. Stage 6 (stage one): Solitary hump in the EEG. Stage 7 (stage one): Trains of humps in the EEG. Stage 8 (stage 1): Humps in the EEG with incomplete spindles. Stage 9 (traditional stage two sleep): Fully formed sleep spindles.

the sleep onset period, the authors both defined sleep onset according to changes in the ongoing EEG waveform (the nine EEG stages), and then subsequently measured EEG alterations using Fourier transforms. This is analogous to performing a visual spectral analysis to assign stage scores, then using the Fourier method to analyze the EEG, and concluding - not surprisingly - that there are changes in EEG spectral power associated with visually determined changes in the EEG pattern. Unfortunately, additional researchers have also defined sleep onset as changes in EEG, and measured sleep onset by examining alterations in the ongoing EEG waveform.

Badia, Wright, and Wauquier (1994) examined fluctuations in broadband and single-hertz EEG activity during the sleep onset period in a study involving 14 university undergraduate students. Sleep onset was defined as three consecutive 30-second epochs of any sleep stage according to Rechtschaffen and Kales (1968). Fast Fourier Transforms were computed on the EEG for the last three 30-second epochs of wakefulness and for the first three 30-second epochs of stage 1 sleep. The authors reported a significant increase in theta EEG (3 - 7 Hz) beginning prior to the first 30-second epoch of sleep. Furthermore, they observed a significant decrease in alpha EEG (8 - 12 Hz), but no significant changes in beta EEG (13 - 25 Hz) associated with sleep onset. Single-hertz analyses revealed large variation with the greatest increase in relative power occurring at 3 Hz, and the largest decrease in relative power occurring at 10 Hz. The conclusion was virtually the same as that reported by Davis, Davis, Loomis, Harvey, and Hobart (1938): EEG changes at sleep onset in normal sleepers primarily involve an increase in theta EEG, a decrease in alpha EEG, and involves a transitory process in which some cortical sites may show EEG-defined sleep while others may show EEG-defined wakefulness.

More recently, Lamarche (1995) used the FFT technique to analyze C₄ EEG from the sleep onset period (defined as the time from lights-out to the first five minutes of stage 2 sleep) in three groups of subjects: (1) six psychophysiological insomniacs; (2) six

psychiatric subjects; and (3) six controls. She observed that psychophysiological insomniacs had significantly less alpha EEG during wakefulness than the other two groups, and the psychophysiological insomniacs did not show the typical drop in alpha EEG usually observed during sleep onset. Delta EEG power was also significantly lower in the psychophysiological group during stage 2 sleep on night two. Confirming Jacobs et al. (1993), Lamarche observed significantly more beta power during wakefulness in psychophysiological insomniacs than in the other two groups. The results of this study were interpreted to suggest that psychophysiological insomniacs have a higher level of cortical arousal during the sleep onset period than both psychiatric insomniacs and normal controls. Furthermore, changes in EEG power values observed during the sleep onset period in the psychiatric insomniacs and controls, but not in the psychophysiological insomniacs, were interpreted to suggest that the discrimination of sleep and wakefulness is more difficult for psychophysiological insomniacs. Although Lamarche's study involved a detailed examination of sleep and quantitative EEG measures in chronic insomniacs and healthy controls, the lack of statistical significance in a number of parameters (e.g., alpha EEG change at sleep onset) may have been a function of low statistical power due to the small sample sizes ($n = 6$).

In a study of period-amplitude analysis (PAA) of sleep onset, Armitage, Hudson, Fitch, and Pechacek (1994) examined the sleep-wake transition in eight healthy, normal volunteers, and 28 depressed (unipolar) outpatients. Armitage et al. found that delta half-wave zero-cross was higher in controls than depressed patients, and that beta half-wave zero-cross was greater in the depressed subjects. Beta first derivative analysis and beta full-wave zero-cross were both statistically significantly greater in the depressed individuals than in the control subjects. With regard to amplitude measures, both delta amplitude and theta amplitude were higher in the controls than in the depressed subjects.

Hypotheses

The primary purpose of the study proposed herein is to more precisely identify sleep onset in both insomniacs and normals and to investigate, as suggested by Hauri and Olmstead (1983), whether the perception of sleep onset by insomniacs lags behind the perception of sleep onset by good sleepers. Researchers have used various definitions of sleep onset. Recall that Carskadon et al. (1976) defined sleep onset as the time from lights out to the first three consecutive epochs of stage 1 sleep or the time until the first epoch of stage 2 sleep. Johnson (1973) defined sleep onset as the beginning of stage 2 sleep because the spindles that occur therein are unique to sleep. Hauri and Olmstead (1983) investigated sleep onset measures and considered sleep onset as the time from lights out to the first five minutes of continuous stage 2 sleep and also as the time from lights out to the first 15 minutes of continuous stage 2 sleep. Consequently, several definitions of electrographic sleep onset will be investigated: (1) the time from lights out to the first three consecutive epochs of stage 1 sleep; (2) the time from lights out to the first five minutes of continuous sleep including stage 1 sleep; (3) the time from lights out to the first epoch of stage 2 sleep; (4) the time from lights out to the first five minutes of continuous sleep (stage 2 sleep, but excluding stage 1); and (5) the time from lights out to the first 15 minutes of continuous sleep (stage 2 sleep, but excluding stage 1). These electrographic definitions of sleep onset will be compared to both subjective reports, and to a behavioural definition of sleep onset defined as five consecutive response failures. As mentioned earlier, a behavioural measure of sleep onset can be obtained through the use of an active, cued response system whereby the subject responds to a faint tone (e.g., a 1000 Hz tone 5 dB above background noise) with the depression of a thumb-switch. Five consecutive response failures is an operational definition for behavioural sleep onset which has been used within this paradigm for over a decade (Bonato & Ogilvie, 1989; Kuderian et al., 1991; Cote & Ogilvie, 1994; Ogilvie & Wilkinson, 1988).

These various measures of sleep onset will allow for a more precise discrimination of the sleep onset period, and permit an examination of the various sleep onset indices in both normal and insomniac sleepers. This is a theoretically important issue because it is commonly believed that insomniacs' perception of sleep onset is excessively delayed; that is, that they overestimate sleep onset latency. One possible explanation for this common belief may involve the use of sleep stage scoring to evaluate the sleep onset period in insomnia. Electroencephalographic definitions of sleep using 30-second epochs are gross measures of tonic brain activity which may ignore any phasic or tonic events indicative of wakefulness (Borkovec, Lane, & VanOot, 1981). Consequently, the use of a behavioural measure, in addition to traditional electrophysiological variables, is advantageous because it enables the researcher or clinician to identify possible arousals otherwise ignored due to the limitations of the traditional sleep stage scoring schema.

The aforementioned sleep onset measures are also useful because they will permit an examination of the frequency of stage reversals. In an examination of the phenomenology of sleep in both normals and insomniacs, Borkovec et al. (1981) reported that the only variable found to predict the occurrence of a wakefulness report from the sleep onset period was the frequency of stage reversals prior to the first consecutive five minutes of stage 2 sleep. If the frequency and duration of arousals dictate the perception of sleep, then a closer examination of stage reversals in combination with a behavioural measure should provide very important information leading to a better understanding of the sleep onset period in insomnia. Borkovec et al. suggest that a smooth progression through the early sleep stages may be necessary to experience and report sleep, and that frequent arousals or stage reversals may eliminate the experience leaving the individual with the experience of wakefulness. Such an interpretation of the sleep onset process is consonant with empirical evidence provided by Bonnet and Moore (1982) who observed

that about 50% of normal sleepers reported experiencing sleep within five minutes of the first sleep spindle.

The various electrographic definitions of sleep onset (see above) will be compared to both subjective reports, and to a behavioural definition of sleep onset defined as five consecutive response failures. It is anticipated that a stage 1 sleep definition of sleep onset will underestimate and precede sleep onset according to subjective and behavioural measures (*hypothesis #1*). The behavioural definition of sleep onset (Ogilvie & Wilkinson, 1988) for both groups should align itself between stage 1 sleep and stage 2 sleep definitions of sleep onset (*hypothesis #2*). Insomniacs should show longer sleep onset latencies given the subject selection criteria; however, it is expected (Hauri & Olmstead, 1983) that insomniacs will also have a longer sleep onset period in which they will have more intervening periods of stage wake or stage 1 sleep from the first epoch of stage 2 sleep to the first 15 minutes of continuous sleep than good sleepers (*hypothesis #3*). In other words, it is hypothesized that insomniacs will show more stage reversals (i.e., from stage 2 sleep back to stage wakefulness or stage 1 sleep) prior to the first fifteen minutes of continuous sleep than good sleepers.

Another purpose of this study is to compare the simultaneous use of the two sleep/wake measurement techniques (i.e., polysomnography vs. behavioural response), and to assess the agreement between these measurements in both chronic psychophysiological insomniacs and good sleepers. As reported by Cote and Ogilvie (1994), it is anticipated that the overall percent concordance between the polysomnographic and behavioural data will exceed 90% with percent agreement between behavioural sleep/wakefulness and sleep stages increasing from stage 1 sleep, wakefulness, stage 2 sleep, REM sleep, stage 3 sleep, to stage 4 sleep (*hypothesis #4*). The polysomnographic set will include total recording time (TRT), total sleep time (TST), total time awake (TTA), sleep onset latency defined as time from lights out to the

first epoch of five consecutive minutes of any sleep stage (SOL), and number of awakenings following sleep onset (NA). The behavioural sleep/wake equivalents will be used and include total number of trials (BR-TRT), total number of trials without response (BR-TST), behavioural sleep onset latency defined as the time from lights out to the first of five consecutive response failures (BR-SOL), number of microarousals defined as incidences of less than five consecutive responses following initial sleep onset (BRMICRO), and number of macroarousals defined as incidences of more than five consecutive responses following initial sleep onset (BRMACRO). It is also hypothesized that there will be a high degree of multivariate overlap between polysomnographic and behavioural sleep/wake measurements (*hypothesis #5*). In addition, it is anticipated that insomniacs will show shorter response latencies than controls during the sleep onset period perhaps due to increased cognitive arousal (*hypothesis #6*).

To test the hypothesis that the EEG may reveal a physiological abnormality in the insomniacs not observed through the application of traditional sleep stage criteria, and to concomitantly evaluate current polysomnographic assessment techniques, the sleep EEG of both groups during the sleep onset period will be period-amplitude analyzed using 30-second epochs regardless of behavioural response. Consistent with findings of Lamarche (1995), it is anticipated that insomniacs will show less delta, theta and alpha EEG activity, and more beta EEG than controls during the sleep onset period (*hypothesis #7*). Hypothesis eight is based on the findings of Freedman (1986) and Lamarche (1995): the period-amplitude analyzed EEG of insomniacs is hypothesized to show more beta EEG activity and less alpha EEG activity during wakefulness than that of controls.

While it is accepted practice within the sleep research community to both define sleep as changes in EEG, and to measure sleep by examining EEG alterations (see Hori et al., p. 25, and Badia et al., p. 26), substantial information is obtained by examining brain electrical activity associated with changes in arousal as defined by behavioural response

latency. Using this approach, behavioural response latency can be used to measure arousal, and associated changes in brain electrical activity during the sleep onset period are thereby examined. As in Ogilvie et al. (1991), it is anticipated that neuronal synchronization at sleep onset will be very rapid given the separation of EEG by behavioural response latencies; however, the use of period-amplitude analytical techniques will enable this researcher to separate amplitude and incidence measures confounded by the Fourier analysis method. Pretone electroencephalographic data will be used to evaluate changes in EEG associated with behavioural responding. The EEG will be sampled 10 seconds prior to stimulus onset to evaluate the previous finding (Ogilvie et al., 1991) that increased EEG synchrony occurs in all bandwidth frequencies as behavioural sleep onset approaches (i.e., longer response latencies will be associated with increased pretone EEG synchrony). It is hypothesized that chronic psychophysiological insomniacs will show less EEG synchronization than controls - specifically less delta amplitude and incidence, less theta amplitude and incidence, less alpha amplitude and incidence, and more beta EEG amplitude and incidence - as a result of heightened arousal during the sleep onset period (*hypothesis #9*). Moreover, it is hypothesized (*hypothesis #10*) that there will be significant increases in PAA amplitude across all EEG frequencies (Ogilvie et al., 1991) during the transition from slow responding (i.e., slow reaction time or RT) to response cessation (i.e., behavioural sleep).

Method

Subjects

Two groups of subjects were used in this study: (1) 15 chronic psychophysiological insomniacs and (2) 15 approximate age-matched ($\pm$ two years) normal or good sleepers. The insomniac sample included 15 medically healthy (i.e., free from any neurological or physical illness), right-handed, chronic psychophysiological

insomniacs (nine women and six men). The insomniac sample was recruited from the population of patients regularly referred to the Royal Ottawa Hospital Sleep Disorders Laboratory, and recommended for study based on the assessment performed by the Clinical Director of the Sleep Laboratory (Dr. D. Radonjic). Subjects ranged in age from 22 to 50 years (mean age = 34.0 years, ± 8.4), and were free from psychopathology. The mean insomnia duration was 4.55 years. A control group of 15 normal ($\bar{x}$ age = 34.1 years ± 8.2) age- and sex-matched, right-handed individuals free from any neurological or physical illness was used for statistical comparison. This group of good sleepers was obtained both from the local university student population, and from the Royal Ottawa Hospital staff. Informed, written consent (see appendix C) was obtained from all subjects prior to entry into the study. Refusal to participate in no way affected the regular clinical treatment received by referred patients. Four insomniacs did not continue to participate in the study: one did not continue because of significant periodic limb movements during sleep (PLMs); one could not continue due to technical problems; and two would not continue because they felt the behavioural response apparatus interfered with their sleep. All control subjects successfully completed their participation in the study.

Calculation of a suitable sample size (i.e., statistical power analysis) was according to information provided by Marascuilo and Serlin (1988). The power analysis for a one-way ANOVA was based on a beta value of .30; an alpha value of .05; number of groups (K) being 5 (behavioural response bins); and an eta-squared value of .15. The appropriate sample size based on the formula outlined by Marascuilo and Serlin was 13 subjects per group. When using a beta value of .20 (all else identical), the appropriate sample size was 15 subjects per group. As a result of these power calculations, this study used a sample size of 15 individuals per group.

Inclusion criteria for insomniacs were based on the International Classification of Sleep Disorders criteria (ICSD, 1990). Insomniac participants met all of the following conditions:

- A) A complaint of insomnia combined with a complaint of decreased functioning during wakefulness for a duration of 6 months or longer (i.e., chronic).
- B) Indications of learned sleep-preventing associations:
 - 1. Trying too hard to sleep, suggested by an inability to fall asleep when desired, but ease of falling asleep during other activities, such as watching television or reading.
 - 2. Conditioned arousal to bedroom or sleep-related activities, indicated by sleeping poorly at home, but sleeping better away from the home or when not engaging in bedtime routines.
- C) A self-reported sleep-onset latency of 45 minutes or longer.
- D) An average polysomnographic sleep onset latency of 30 minutes or longer during two of the three nights of polysomnography.
- E) An average polysomnographic sleep efficiency of less than or equal to 85% during two of the three nights of polysomnography.
- F) Completion of two-week sleep/wake log.
- G) No evidence of other medical or psychiatric disorders that would account for the sleep disturbance. Assessment of potential for any psychiatric disorder was based upon an interview conducted by an experienced psychiatrist (Dr. D. Radonjic), and by paper and pencil tests (see Materials subsection and Appendices A through M).

Exclusion criteria for insomniacs:

- A) Life-long or early childhood insomnia (Idiopathic Insomnia).
- B) Current use of medications.
- C) Habitual napping.
- D) Shiftwork.
- E) Sleep apnea or periodic limb movements during sleep (assessed by routine polysomnography on night 1).
- F) Evidence of alpha-delta sleep.
- G) History of alcohol and/or drug abuse.

Inclusion criteria for good sleepers:

- A) A self-reported good sleeper with no sleep disturbance.
- B) A self-reported sleep-onset latency of 20 minutes or less.
- C) An average polysomnographic sleep onset latency of less than 20 minutes on two of the three nights of polysomnography.
- D) An average polysomnographic sleep efficiency of greater than 90% during two of the three nights of polysomnography.
- E) A self-reported mean sleep duration of 7-9 hours nightly.
- F) Completion of Two-week Sleep/Wake Log.

Exclusion criteria for good sleepers:

- A) Current use of medications.
- B) Habitual napping.
- C) Shiftwork.
- D) Sleep apnea or periodic limb movements during sleep (assessed by polysomnography on night 1).
- E) History of alcohol and/or drug abuse.
- F) Evidence of any medical or psychiatric disorder.

Materials

Assessment Instruments:

- A) Initial Subject Assessment Questionnaire
- B) Beck Depression Inventory
- C) Beck Anxiety Inventory
- D) MMPI - critical items only
- E) General Sleep History Questionnaire
- F) Two-week Sleep Log
- F) Stanford Sleepiness Scale
- G) ROH Post-sleep Questionnaire
- H) Behavioural Response Post-sleep Questionnaire
- I) An interview conducted by an experienced psychiatrist (Dr. D. Radonjic) prior to the first recording night to assess the potential for any psychiatric disorder.

The Initial Subject Assessment Questionnaire (see Appendix B) was a brief checklist given to study candidates to quickly evaluate their suitability for participation. For example, one of the inquiries concerns hearing difficulties, others ask about shiftwork, left handedness, and sleep apnea. Individuals were asked to read this during their orientation to the sleep laboratory immediately following their interview with Dr. D. Radonjic.

The Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) was completed by subjects and consists of 21 groups of statements in which individuals select one or more statements from each group that best describes the way they had been feeling in the past week (see Appendix D). The statements in each group are scored from 0 to 3 and overall Beck Depression Inventory scores may range from 0 to 63. A score between 1 and 10 indicates an individual is free from depression; 11 to 16 indicates mild mood disturbance; 17 to 20 indicates borderline clinical depression; moderate depression is indicated by a score between 21 to 30 whereas 31 to 40 indicates severe depression; an individual who scores over 41 is extremely depressed. Any individual who scored higher than 16 on this scale was excluded from the study. The BDI was completed following the initial clinical interview; that is, prior to the first recording night.

The Beck Anxiety Inventory (BAI) (Beck, Epstein, Brown, & Steer, 1988) was completed by subjects and consists of 21 items, each describing a common symptom of anxiety. Individuals are asked to rate, on a scale from 0 (not at all) to 3 (severely), how much they have been bothered by the particular item during the past week (see Appendix E). The items are summed to obtain a total anxiety score that can range from 0 to 63. Any individual who scored higher than 16 on this scale was excluded from the study. The BAI was completed following the initial clinical interview and prior to initial polysomnographic assessment.

The Minnesota Multiphasic Personality Inventory (MMPI) critical item set is an abridged version of the MMPI which is considered adequate to screen for any major psychopathology (Lachar & Wrobel, 1979), and was intended as an adjunct to the routine psychiatric assessment normally performed by the laboratory director (see Appendix F). The scale contains: 11 items concerning anxiety and tension; 11 items dealing with deviant thinking and experience; 15 items about deviant beliefs (i.e., reality distortion); 16 items dealing with depression and worry; and 23 items on somatic symptoms. Subjects who scored higher than the following were excluded from the study: 6 on anxiety and tension; 2 on deviant thinking and experience; 0 on deviant beliefs (reality distortion); 6 on depression and worry; and 6 on somatic symptoms. The MMPI critical items questionnaire was completed following the initial clinical interview.

The General Sleep History Questionnaire was routinely given to patients referred to the sleep disorders laboratory to obtain additional information regarding health and sleep history. Individuals were requested to consult their sleep partners when completing this questionnaire as bed partners are frequently a valuable source of information. This questionnaire, which is presented in Appendix G, was completed by subjects at home prior to the first recording night.

All individuals recorded in the sleep disorders laboratory were also asked to maintain a Two-week Sleep Log (see Appendix H). The sleep log detailed subjects' daily activities, including the amount and time of any coffee/tea consumption, and the time and duration of sleep periods. The Two-week Sleep Log was completed prior to the first recording night.

The Stanford Sleepiness Scale (Hoddes, Zarcone, & Smythe, 1973) is a Likert-type scale which was used to quantify subjective sleepiness. Subjects were asked to rate their sleepiness within 5 minutes of lights out and within 5 minutes of morning wake-up during each of the three recording nights. The Stanford Sleepiness Scale is included in

Appendix I.

Two post-sleep questionnaires were used in this study to obtain subjective information regarding subjects' sleep. The ROH Post-sleep Questionnaire (see Appendix J) was completed within 15 minutes of morning wake-up immediately after subjects returned from the bathroom and used to obtain subjective information concerning subjects' sleep. The Behavioural Response Post-Sleep Questionnaire was used to obtain information regarding subjects' experiences with the behavioural response system (see Appendix K). The Behavioural Response Post-Sleep Questionnaire was completed following the ROH Post-sleep Questionnaire.

Data Collection & Analysis

Grass gold plated electrodes were used to record the electrophysiological data, and were affixed using either collodion impregnated gauze or Transpore™ surgical tape depending upon the site of placement. A thermocouple was used to measure oral/nasal airflow (ONA), and piezo-electric crystals were used to monitor respiratory effort. SANDMAN™ (a commercial paperless polysomnographic system) was used for both data acquisition and analysis using an 80486 DX2 66 MHZ ALR microcomputer equipped with LabMaster 12 bit A/D converters for digitization, and an optical disk drive for permanent storage. Grass amplifiers (model 7P511J) were used to filter and amplify the bioelectrical signals: C_3-A_2 EEG, O_1-A_2 EEG, EOG, submental EMG, bilateral anterior tibialis EMG, lead II EKG, oral/nasal airflow, thoracic and abdominal respiratory effort. Electrical contact was provided by 10-20 electrode paste applied after abrasion of the skin, and electrode impedance was initially below 2 kohms for scalp electrodes and reference sites, and below 5 kohms for all other electrode sites. All amplifiers were calibrated prior to each recording session. An Ohmeda pulse oximeter was used to simultaneously collect pulse rate and SaO_2 from the finger, and was fed directly into the

multiplexer. The base sampling rate was 320 Hz with specific channel sampling rates ranging from 10 Hz (SaO₂ and respiration) to 320 Hz for EEG (see Table 1).

Data analysis was performed on a 20" monitor using a screen resolution of 1280 x 1024 pixels, and was done according to traditional criteria (Rechtschaffen & Kales, 1968) by a registered polysomnographic technologist blind to behavioural response/nonresponse. Blind scoring was achieved by removing the behavioural response/nonresponse channel from the display screen. Our previous work has indicated satisfactory agreement between paper recordings and our current paperless recording system (Jeakins, Loewy, & Bonato, 1993). Period-amplitude analytical techniques were used to analyze the EEG (Hoffmann et al., 1979). Period-amplitude analysis involves the detection of zero voltage crossing events (i.e., full-wave and half-wave baseline crossing) and zero slope events (i.e., first derivative). The time between each occurrence of these events is measured and recorded as a time-in-frequency (percentage) measure. Conversion of the data from the number of samples to the corresponding bandwidth frequency is easily accomplished because the computer is sampling at a known rate. The addition of the squared successive unit voltages between half-wave zero crossings can be used to obtain information about amplitude. Thus, this analytical technique yields four measures for each bandwidth frequency (alpha, beta, delta, theta, and sigma). A recent study has reported significant multivariate overlap in shared variance between amplitude analysis and FFT power measures, and has indicated that the two signal transformation techniques are generally comparable (Armitage, Hoffmann, Fitch, Morel, & Bonato, 1995).

In addition to electroencephalographic and traditional polysomnographic measures, subjects used a behavioural response system to monitor sleep/wakefulness patterns. This system is a modified version of that developed by Ogilvie and Wilkinson (1984). Previous research (Cote & Ogilvie, 1994; Ogilvie & Wilkinson, 1988) has found

that this system reliably discriminates between sleep and wakefulness throughout the night. In the present version, 1000 Hz tones are produced at random intervals ranging from 10 to 40 seconds throughout the night. These tones were delivered to the subject via an earphone and were sent to the 80486 acquisition computer through a Grass DC Driver Amplifier (Model 7DAF). The tones were five seconds in duration unless terminated by the subject through the depression of a palm-mounted thumb-switch. Any response within the five second period was used to define wakefulness, whereas failure to respond

Table 1. Sampling rates and amplifier settings for electrophysiological data^a

Bioelectrical Signal	Sampling Rate (Hz)	LFF (Hz)	HFF (Hz)	S ($\mu\text{V/mm}$)
EEG	320	.3	100	7.5
EOG	60	.1	100	15
EMG	125	10	100	1
EKG	50	.3	100	20
Oral/Nasal Airflow	10	.1	100	5 ^b
Behavioural Response	10	---DC Amplifier---		15

Note. ^aBand-stop filter was used; roll-off = 6 dB/octave. ^bmV/cm

defined sleep. The intensity of the tone was adjusted to be no greater than 35 dB(A). A Beltone Audiometer (Model 119) was used to assess subjects' hearing, and a Brüel & Kjær Sound Level Meter (Type 2209) was used to calibrate the intensity of the tones. Calibrations indicated that the tone ranged from 26 - 35 dB(A) depending upon the auditory acuity of the subject. Because the auditory threshold increases rapidly at sleep onset (Bonnet & Moore, 1982; Rechtschaffen, Hauri, & Zeitlin, 1966), the repetitive tones do not excessively intrude upon sleeping behaviour; however, a first night effect is often experienced by subjects (Bonato & Ogilvie, 1989). Concerning auditory thresholds, Rechtschaffen et al. (1966) reported that the auditory awakening threshold ranges from 15-100 dB above waking levels throughout sleep. In addition, Bonnet and Moore (1982)

reported that the auditory awakening threshold had reached 64 percent of maximum within one minute of stage 2 sleep entry.

Procedure

The insomniac group consisted of patients referred to the sleep laboratory by area physicians; sex-matched and approximate age-matched ($\pm$ two years) control subjects were obtained from the local university student population, and from the Royal Ottawa Hospital staff. All insomniacs referred to the sleep disorders laboratory underwent an initial presleep interview with the sleep laboratory director as part of the regular laboratory protocol. An introduction to the sleep laboratory and to the behavioural response system was given to study candidates following the interview. Individuals who agreed to participate were then asked to complete the informed consent form, the BDI, BAI, MMPI critical items, the General Sleep History Questionnaire, and Two-week Sleep Log prior to their return (for their scheduled recordings) to the sleep laboratory. Hearing adequacy was briefly tested following questionnaire presentation. As part of the routine clinical procedure, all individuals referred to the sleep laboratory were also asked to eliminate medication intake for two weeks prior to the first recording night, and to keep a constant 23:00-07:00 sleep-wake schedule (as much as possible) for one week prior to the first scheduled sleep recording. Avoidance of daytime naps was stressed to all subjects. Subjects were required to refrain from alcohol and caffeine ingestion during the three days of the study.

Subjects slept in the sleep laboratory for three consecutive recording nights during which time electrophysiological and behavioural response data were recorded. Electrode placements remained identical during the three nights; however, the finger probe used to record arterial oxygen saturation, the oral/nasal thermocouple, and the thoracic respiratory belt were worn by subjects only during the first night (the abdominal belt was

still used). The finger probe, oral/nasal thermocouple and respiratory effort channels were used to screen for sleep apnea. Electrodes placed over the anterior tibialis muscles (leg electrodes) were used to screen for periodic limb movements during sleep. Participants arrived at the sleep laboratory around 21:00 so that they could become familiar with their sleep environment, and to provide sufficient time for electrode placement. Subjects were again asked to read some introductory Information on the Behavioural Response System (see Appendix A), and the Initial Subject Assessment Questionnaire (see Appendix B).

During the first night of recording, all subjects used the behavioural response device, and the tone intensity was adjusted once immediately prior to lights-out (around 23:00). Subjects were permitted to practice with the device for five minutes and were then given the option to decrease/increase the volume so as to make it clearly audible, but as unintrusive as possible. The tone intensity was not adjusted on subsequent nights. The thumb-switch was both velcro-strapped and taped to the palm, and participants wore the earphone in the preferred ear. In addition, all participants were screened during night one for both sleep apnea syndrome and for periodic leg movements. Subjects who showed either significant sleep apnea or periodic leg movements (i.e., ≥ 5 per hour in accordance with ICSD criteria) were excluded from the remainder of the study (one subject had a PLM-Arousal index of 16.76 and had to be excluded). Subject exclusion in no way affected the regular clinical treatment received by patients. In addition to being a screening night, night one also served to reduce the first night effect commonly associated with both polysomnographic and behavioural sleep/wake recordings (Agnew & Webb, 1966; Bonato et al., 1992; Kuderian et al., 1991; Webb & Campbell, 1979).

Electroencephalographic electrodes were secured to the subjects' scalps with collodion at the following sites: C_3 and O_1 each referred to the A_2 contralateral mastoid. Transpore tape was used to affix all remaining electrodes. Two electrodes recorded EOG activity (a bipolar derivation): both outer canthus, one left infraorbital and one right

supraorbital site. A pair of electrodes were used to measure EMG from the submental location, and a pair of electrodes recorded the EKG using a lead II placement (right trapezius-right anterior tibialis). Bilateral anterior tibialis activity was recorded using one electrode on each leg. An oral/nasal cannula thermocouple was used to record oral/nasal airflow, and piezo-electric respiratory belts were placed around the thorax and abdomen to record respiratory effort. Electrical contact was provided by 10-20 electrode paste applied after abrasion of the skin, and electrode impedance was initially below 2 kohms for scalp electrodes and below 5 kohms for all other electrode sites. All amplifiers were calibrated prior to each recording session. Recording sessions were from 6.5 to 8 hours in duration.

For each recording session, the Stanford Sleepiness Scale was completed within five minutes of lights-out (approximately 23:00), and within five minutes of morning wake-up (around 07:00). Subjects were also given the regular ROH Post-sleep Questionnaire, and the Behavioural Response Post-sleep Questionnaire within 15 minutes following morning wake-up. Subject debriefing took place following completion of the study.

Results

There are at least two philosophies, and therefore statistical approaches, with which to consider overall analyses. The traditional approach is to use an omnibus, or overall single (M)ANOVAs analysis. In this case, a 2 (groups) x 3 (nights) x 2 (electrode site) x 1 (PAA method)² x 5 (EEG frequency) design would be required. However, this approach would result in a large number of unpredicted and uninteresting comparisons (e.g., site by night; site by PAA method), would produce unnecessary complexity involving third and higher order interactions, and would exhaust the degrees of freedom. Furthermore, many of the comparisons from such an approach would be meaningless with poor external validity. For these reasons, this approach will not be used. The second, alternative approach used herein involves the examination of specific hypotheses only. These tests, which are reported on the following pages, are of importance because they allow for the testing/expansion of specific hypotheses based entirely on the results of previous studies. Thus, the complexity, external validity and statistical issues associated with approach number one are averted with the use of the second approach.

To address the issue of increased Type I errors associated with the number of planned analyses, the alpha critical level was set at .05 for all hypothesized tests and at the more conservative .01 level for all serendipitous findings and unpredicted post-hoc analyses. Unless indicated otherwise, all analyses involving multivariate main effects or interactions, and all univariate breakdowns involving beta, alpha, theta, and delta EEG measures following a MANOVA will be evaluated using a critical alpha level of .05. Analyses dealing with sigma EEG measures will be evaluated using a critical alpha level of .01. Any additional ANOVAs will be evaluated using a critical alpha level of .01. This method of setting the alpha critical level according to hypothesized tests was preferred

²Due to multicollinearity/singularity, it is typical practice to analyze the period-amplitude indices (i.e., half-wave, full-wave, etc) separately.

over a Bonferroni correction because a balance was desired between Type I and Type II errors. According to Stevens (1992), more attention should be paid to Type II errors and he suggests that for studies where n is low (i.e., less than 20 subjects per group), the researcher should seriously consider adopting “a more lenient α level, perhaps $\alpha = .10$ or $\alpha = .15$ ” (p.175). While Stevens’ position was not entirely adopted within this study, his suggestion for striking a balance between Type I and Type II errors was implemented.

Before proceeding with the sleep data analyses, a brief comment on the Beck Depression, Beck Anxiety, and MMPI scales would be appropriate. Recall that the purpose of these tools was to serve as an adjunct to the psychiatric interview and to further screen primary insomniacs from those suffering from secondary insomnia (e.g., insomnia secondary to depression). To ensure that the insomniac and control groups were homogeneous with respect to depression and anxiety, one-way between-groups ANOVAs were performed on the results of the Beck Depression and Anxiety Scales. One-way ANOVAs did not show any statistically significant differences in the Beck Depression Scale, $F(1, 28)=1.07$, $p = .31$, or in the Beck Anxiety Scale, $F(1, 28)=3.33$, $p = .08$. For the insomniac group, depression and anxiety values were $6.53 (\pm 3.23)$ and $7.73 (\pm 3.47)$, respectively. For the control group, depression and anxiety values were $5.27 (\pm 3.74)$ and $5.4 (\pm 3.52)$, respectively. Concerning the MMPI critical items, only items concerned with deviant thinking (11 items) and deviant beliefs (15 items) were examined because anxiety and depression levels were already assessed using the Beck Scales. All of the subjects (i.e., both controls and insomniacs) scored zero on deviant beliefs (reality distortion). One male control subject responded to two items on the deviant thinking items, and two female insomniacs each responded to one item within the set of deviant items. The paper and pencil questionnaires confirmed the results of the psychiatric interview; that is, all subjects were essentially free from any psychiatric diagnosis. Thus, the main analyses proceeded with the reassurance that any group differences could not be

attributable to any affective disorder, to a general disturbance of thought, or to any specific psychiatric disorder.

To assess the presence or absence of a first-night effect, repeated measures ANOVAs were computed for sleep efficiency values (Agnew & Webb, 1966; Bonato et al., 1992; Webb & Campbell, 1979). Sleep onset latency values are not included at this time because they are tested in a separate analysis below (see test of hypotheses #1 and #2 below). Significance levels for repeated measures designs are based on Huynh-Feldt epsilon adjustments. A repeated measures ANOVA computed on sleep efficiency for the insomniacs (total sleep time in minutes $\div$ total recording time in minutes multiplied by 100) found a significant difference between nights, ($F(2, 28)=11.51, p = .0002$). Tukey's Honestly Significant Difference (HSD) test indicated that night one was significantly lower than both nights two and three, but no difference was detected in sleep efficiency between nights two and three. A repeated measures ANOVA computed on sleep efficiency for the controls revealed a significant difference between nights, ($F(2, 28)=4.00, p = .032$). Tukey's HSD test found that night one was significantly lower than night three, but no difference in sleep efficiency was observed between either nights one and two or between nights two and three.

A number of Pearson correlations and ANOVAs (see Tables 2, 3, 4, 5, 6) were computed on various sleep onset measures to determine whether defining sleep onset latency as 90 seconds of stage 1 sleep, as used by Carskadon et al. (1976), is an accurate estimate of sleep onset latency. Recall that it was hypothesized that a stage 1 sleep definition of sleep onset (i.e., three continuous epochs of stage 1 sleep as used by Carskadon et al. and described on page 28) would underestimate sleep onset length according to subjective and behavioural measures (*hypothesis #1*), and also that the behavioural definition of sleep onset for both groups would align itself between stage 1 sleep and stage 2 sleep definitions of sleep onset (*hypothesis #2*). The dependent

variables were as follows: (1) the time from lights out to the first three consecutive epochs³ of stage 1 sleep (90 SOL); (2) the time from lights out to the first five minutes of continuous sleep including stage 1 sleep (SOL); (3) the time from lights out to the first epoch of stage 2 sleep (S2 SOL); (4) the time from lights out to the first five minutes of continuous stage 2 sleep (excluding stage 1) (SOL 5); (5) the time from lights out to the first 15 minutes of continuous stage 2 sleep (excluding stage 1) (SOL 15); (6) a subjective estimate of sleep onset latency (Subj SOL); and (7) the behavioural sleep onset latency (BR SOL).

For the insomniac group on night one, neither the subjective (Subj SO) nor the behavioural measures (BR SOL N1) of sleep onset were significantly correlated with the three-epoch stage one sleep definition of sleep onset (see Table 2). Further, the subjective measure of sleep onset was only significantly correlated to the 15 minute (SOL 15) of continuous sleep index (excluding stage 1 sleep) of sleep onset. The behavioural measure was significantly correlated with four of the remaining six sleep onset indices, but tended to be higher with the first epoch of stage 2 sleep. With regard to the control group on night one (see Table 3), the behavioural measure was significantly correlated with five of the six sleep onset indices, and tended to be higher with the first epoch of stage 2 sleep. The subjective measure of sleep onset for the control group was significantly correlated to all sleep onset indices, but tended to be higher when sleep onset was defined as the latency to the first five minutes of sleep including stage 1 sleep.

On night two, the subjective measure of sleep onset for the insomniac group was only found to correlate with the behavioural index (see Table 2). The behavioural measure correlated with five of the six indices, the highest correlation was with latency to 90 seconds of stage 1 sleep. For the control group on night two (see Table 3), behavioural

³For each of the polysomnographic sleep onset latency (SOL) definitions described herein, SOL refers to the time from lights out to the first epoch of that particular sleep onset latency definition. For example, SOL refers to the time from lights out to the first 30-second epoch of five continuous minutes of any sleep stage.

sleep onset was only found to correlate with subjective sleep onset latency, while the subjective measure was significantly correlated to both the first epoch of stage 2 sleep, and to the first five minutes of continuous stage 2 sleep.

The correlations computed on the data from night three revealed that the subjective measure of sleep onset for the insomniac group was significantly correlated with all other indices; however, the correlations between the subjective measure and the additional measures are virtually identical with the exception of latency to the first 15 minutes of sleep (see Table 2). This pattern is also observed in the correlations between the behavioural measure and the remaining six indices, but in this instance the highest correlation was with latency to the first five minutes of stage 2 sleep. For the control group on night three (see Table 3), behavioural sleep onset was most strongly correlated with subjective sleep onset latency, although it was also correlated with latency to the first 90 seconds of stage 1 sleep, latency to the first five minutes of sleep including stage 1 sleep, and latency to the first epoch of stage 2 sleep. The subjective measure was significantly correlated to both the first 90 seconds of stage 1 sleep, and to the first epoch of stage 2 sleep.

To further examine whether a 90 second stage 1 sleep definition would underestimate both subjective and behavioural indices (*hypothesis #1*), and to determine whether the behavioural definition of sleep onset for both groups would align itself between stage 1 sleep and stage 2 sleep definitions of sleep onset (*hypothesis #2*), a groups (2) by sleep onset latency (SOL) type (7) by nights (3) ANOVA was computed. Groups was used as the between-subjects independent variable (IV), SOL type as a within-subjects IV, nights as a within-subjects IV, and the dependent variable (DV) was sleep onset latency (time in minutes). The mean latencies and standard deviations are

Table 2. Correlation matrix for sleep onset measures in insomniacs on nights 1, 2, 3

	3 epochs... SOLN1	S2 SOLN1	SOL 5 m...	SOL 15 ...	Subj SO...	BR SOL N1	
3 epochs ...	1						
S1SOLN1	.646**	1					
S2SOLN1	.639*	.993**	1				
SOL 5 mi...	.609*	.969**	.97**	1			
SOL 15 m...	.372	.599*	.663**	.671**	1		
Subj SOL ...	.17	.355	.385	.376	.642**	1	
BR SOL N1	.156	.67**	.687**	.617*	.511*	.282	1

	3 epochs...	SOL N2	S2 SOLN2	SOL 5 m...	SOL 15 ...	Subj SO...	BR SOL N2
3 epochs ...	1						
SOL N2	.955**	1					
S2 SOL N2	.952**	.986**	1				
SOL 5 mi...	.957**	.986**	.993**	1			
SOL 15 m...	.523*	.49	.506	.543*	1		
Subj SOL ...	.278	.261	.258	.241	.176	1	
BR SOL N2	.886**	.868**	.844**	.837**	.453	.548*	1

	3 epochs... SOL N3	S2 SOLN3	SOL 5 m...	SOL 15 ...	Subj SO...	BR SOL N3
3 epochs ...	1					
SOL N3	.969**	1				
S2 SOL N3	.962**	.995**	1			
SOL 5 mi...	.938**	.97**	.985**	1		
SOL 15 m...	.628*	.665**	.672**	.741**	1	
Subj SOL ...	.746**	.727**	.721**	.738**	.632*	1
BR SOL N3	.923**	.945**	.95**	.952**	.59*	.723**

Note. Correlations above .51 are statistically significant. * $p < .05$; ** $p < .01$

Table 3. Correlation matrix for sleep onset measures in controls on nights 1, 2, 3

	3 epochs...	SOL N1	S2 SOLN1	SOL 5 m...	SOL 15 ...	Subj SO...	BR SOL N1
3 epochs ...	1						
S1SOLN1	.818**	1					
S2SOLN1	.889**	.954**	1				
SOL 5 mi...	.829**	.932**	.97**	1			
SOL 15 m...	.66**	.535*	.509	.474	1		
Subj SOL ...	.732**	.912**	.874**	.817**	.597*	1	
BR SOL N1	.661**	.767**	.852**	.822**	.313	.679**	1

	3 epochs...	SOL N2	S2 SOLN2	SOL 5 m...	SOL 15 ...	Subj SO...	BR SOL N2
3 epochs ...	1						
SOL N2	.857**	1					
S2 SOL N2	.806**	.824**	1				
SOL 5 mi...	.793**	.792**	.976**	1			
SOL 15 m...	.343	.427	.496	.623*	1		
Subj SOL ...	.277	.502	.586*	.531*	.421	1	
BR SOL N2	.056	.146	.467	.474	.377	.517*	1

	3 epochs...	SOL N3	S2 SOLN3	SOL 5 m...	SOL 15 ...	Subj SO...	BR SOL N3
3 epochs ...	1						
SOL N3	.85**	1					
S2 SOL N3	.883**	.905**	1				
SOL 5 mi...	.648**	.857**	.696**	1			
SOL 15 m...	.504	.633*	.437	.807**	1		
Subj SOL ...	.669**	.498	.636*	.303	.315	1	
BR SOL N3	.749**	.519*	.73**	.23	.067	.837**	1

Note. Correlations above .51 are statistically significant. *p <.05; **p <.01

reported in Table 4. The groups by SOL measure by nights interaction was not statistically significant, $F(12, 336)=0.81$, $p = .64$. Since the three-way interaction was not statistically significant, the two-way interactions were examined. The group by SOL type interaction was statistically significant, $F(6, 168)=3.34$, $p = .004$. The group by nights interaction was not statistically significant, $F(2, 56)=1.57$, $p = .22$. The SOL type by nights interaction was not statistically significant, $F(12, 336)=1.53$, $p = .11$. Because the nights IV was not involved in a significant interaction, the main effect for nights was examined. A statistically significant main effect for nights was revealed, $F(2, 56)=4.71$, $p = .01$. Post-hoc multiple comparisons using Tukey's HSD indicated an overall first-night effect (collapsed across both groups and SOL type) with the mean sleep onset latency for night one (37.05 minutes) significantly greater than night two (21.83 minutes), but neither night one nor night two differed from night three (28.03 minutes). It is clear from the analyses performed on both sleep efficiency (see above) and sleep onset latency, that a first-night effect is present within the data. Such a conclusion is consistent with previous research (Agnew & Webb, 1966; Bonato et al., 1992; Webb & Campbell, 1979). Due to the significant two-way interaction for group by SOL type, the simple effects were analyzed.

The simple effects analysis of SOL type within the insomniac group was statistically significant, $F(6, 84) = 7.87$, $p = .00006$ (see Figure 1).⁴ Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that 90 SOL (90 seconds of continuous stage 1 sleep) significantly differed from five minutes of continuous stage 2 sleep (SOL 5), from 15 minutes of continuous stage 2 sleep (SOL 15), and from subjective sleep onset latency (Subj SOL) (see Figure 1 and Table 4). This indicated that within the insomniac group, the 90 seconds of continuous stage 1 sleep definition (90 SOL) underestimates subjective sleep onset latency. In addition, five continuous minutes

⁴Significance levels for repeated measures analyses are based on Huynh-Feldt epsilon adjustments.

of sleep (SOL), latency to stage 2 sleep (S2 SOL), and behavioural sleep onset latency (BR SOL) differed from 15 minutes of continuous stage 2 sleep (SOL 15). Thus, the results of these statistical tests suggest that, within the insomniac group, a behavioural definition of sleep onset is most closely aligned with a (late) stage 1 and (early) stage 2 definition of sleep (see Figure 1). The simple effects analysis of SOL type within the control group was statistically significant, $F(6, 84) = 15.57, p = .00001$ (see Figure 1). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that 90 SOL (90 seconds of continuous stage 1 sleep) significantly differed from latency to stage 2 sleep (S2 SOL), five minutes of continuous stage 2 sleep (SOL 5), 15 minutes of continuous stage 2 sleep (SOL 15), subjective sleep onset latency (Subj SOL), and from behavioural sleep onset latency (BR SOL). This confirmed that within the control group, the 90 seconds of continuous stage 1 sleep definition (90 SOL) underestimates both subjective and behavioural sleep onset latency. In addition, SOL (5 continuous minutes of any sleep stage) significantly differed from five minutes of continuous stage 2 sleep, 15 minutes of continuous stage 2 sleep (SOL 15), and from subjective sleep onset latency (Subj SOL) (see Table 2). Both latency to stage 2 sleep (S2 SOL) and behavioural sleep onset latency (BR SOL) differed from 15 minutes of continuous stage 2 sleep (SOL 15). Again, these results suggest that, within the control group, a behavioural definition of sleep is most closely aligned with an early stage 2 sleep definition of sleep onset (see Figure 1). Latency to stage 2 sleep (S2 SOL) was found to significantly differ from 15 minutes of continuous stage 2 sleep (SOL 15). Not surprisingly, all simple effects analysis of group within sleep onset latency type are statistically significant with insomniacs showing consistently longer sleep onset latencies.

The simple effect analysis of group within 90 seconds of continuous stage 1 sleep (90 SOL) was statistically significant, $F(1, 88) = 24.00, p = .0001$, (see Figure 1). The simple effect analysis of group within five minutes of continuous sleep (SOL) was

statistically significant, $F(1, 88) = 31.82$, $p = .0001$, (see Figure 1). The simple effect analysis of group within latency to stage 2 sleep (S2 SOL) was statistically significant, $F(1, 88) = 25.82$, $p = .0001$, (see Figure 1). The simple effect analysis of group within latency to five minutes of continuous stage 2 sleep (S2 SOL) was statistically significant, $F(1, 88) = 29.28$, $p = .0001$, (see Figure 1). The simple effect analysis of group within latency to fifteen minutes of continuous stage 2 sleep (S2 SOL) was statistically significant, $F(1, 88) = 33.18$, $p = .0001$, (see Figure 1). The simple effect analysis of group within subjective sleep onset latency (Subj SOL) was statistically significant, $F(1, 88) = 26.61$, $p = .0001$, (see Figure 1). The simple effect analysis of group within behavioural sleep onset latency (BR SOL) was statistically significant, $F(1, 88) = 23.48$, $p = .0001$, (see Figure 1). Additional information is provided by the change in slope of the lines within Figure 1. Specifically, the slope changes within the insomniac group as it approaches latency to 15 continuous minutes of stage 2 sleep. Within the control group, however, there is no dramatic change in slope suggesting that the insomniacs take longer to reach this measure of sleep onset even after they reach S2 SOL or SOL 2 (Hauri & Olmstead, 1983). A planned analysis will specifically address this issue within the next two paragraphs.

Hypothesis three stated that insomniacs would have more intervening periods of stage wake or stage 1 sleep than good sleepers from the first epoch of stage 2 sleep to the first 15 minutes of continuous stage 2 sleep. In other words, it was anticipated that insomniacs would show more stage reversals (i.e., from stage 2 sleep back to wakefulness or stage 1 sleep) prior to the first fifteen minutes of continuous sleep (excluding stage 1 sleep) than good sleepers. To examine this hypothesis, a mixed ANOVA was computed using group (insomniac or control) as the between IV, night (night one, two, or three) as the repeated measure IV, and number of stage reversals prior to continuous 15 minutes of sleep as the DV.

Figure 1. Plot of group x SOL type interaction. Arrows indicate significant between group differences at $p < .05$. See also Table 4.

Table 4. Means and standard deviations for group by sleep onset latency types

GROUP	SOL-90	SOL	S2 SOL	SOL 5	SOL 15	Subj SOL	BR SOL
Insomniacs	28.15 (29.09)	39.12 (35.55)	43.57 (39.11)	47.44 (39.03)	64.34 (49.78)	54.96 (51.18)	37.33 (32.09)
Controls	6.41 (6.35)	8.41 (8.38)	13.17 (9.00)	15.05 (9.43)	19.83 (14.45)	14.84 (10.10)	12.99 (10.28)

Note. Standard deviation in parentheses.

Table 5. Mean sleep onset latency in minutes for 3 epochs S1, SOL, S2 SOL, 5 min S2 SOL, and BR SOL

SOL Measure	Night	$\bar{x}$ Insomniacs	sd	$\bar{x}$ Controls	sd
90 SOL	1	27.07	27.87	7.27	7.44
SOL	1	54.90	51.40	12.13	11.16
S2 SOL	1	57.17	51.45	15.53	12.02
SOL 5	1	63.03	49.48	17.07	11.47
SOL 15	1	84.50	63.67	23.73	19.18
Subj SOL	1	75.57	69.15	21.00	13.88
BR SOL	1	43.27	34.70	16.47	14.09
90 SOL	2	23.33	26.88	4.60	4.29
SOL	2	27.77	22.03	5.57	4.98
S2 SOL	2	32.47	25.15	9.93	6.45
SOL 5	2	34.10	24.77	10.67	7.08
SOL 15	2	50.23	29.55	16.17	12.76
Subj SOL	2	42.30	38.14	11.03	5.99
BR SOL	2	28.57	25.59	8.93	6.96
90 SOL	3	34.06	33.12	7.37	6.90
SOL	3	34.70	20.46	7.53	6.85
S2 SOL	3	41.07	34.51	14.03	7.10
SOL 5	3	45.20	35.63	17.43	8.17
SOL 15	3	58.30	46.61	19.60	9.72
Subj SOL	3	47.00	36.05	12.50	5.51
BR SOL	3	40.17	35.35	13.57	7.37

The results of the ANOVA are shown in Table 6. A statistically significant main effect for group was observed, $F(1, 28) = 4.27$, $p = .048$, but there was neither a significant main effect for night, $F(2, 56) = 0.92$, $p = .40$, nor a significant interaction, $F(2, 56) = 0.014$, $p = .99$. As expected, insomniacs showed significantly more stage reversals than controls (see Table 6).

Table 6. Mean number of reversals to stage wake/one during the sleep onset period

Repeated Mea...		Num S1 N1	Num S1 N2	Num S1 N3	Totals:
Group	Control	15 1.467	15 .6	15 .733	45 .933
	Insomniac	15 2.4	15 1.733	15 1.8	45 1.978
Totals:		30 1.933	30 1.167	30 1.267	90 1.456

To obtain information about the accuracy and validity of the behavioural response system when used by chronic insomniacs *vis à vis* conventional polysomnographic indices (Rechtschaffen & Kales, 1968), traditional sleep stage scoring using 30-second epochs was performed by a registered sleep technologist blind to behavioural response/non-response. Epochs contaminated with muscle and movement artifacts were omitted from statistical analyses. Following stage scoring, each epoch was then visually examined for behavioural response/nonresponse. If a trial was not presented during an epoch, the response/nonresponse from the immediately preceding trial was assigned to that epoch. In the event an epoch contained two trials, response/nonresponse was determined according to the trial preceding the midway point for that epoch.

Hypothesis four stated that the overall percent concordance (behavioural responding during stage wake; nonresponding during sleep stages) between the polysomnographic and behavioural data would exceed 90% with percent agreement

between behavioural sleep/wakefulness and sleep stages increasing from stage 1 sleep, wakefulness, stage 2 sleep, REM sleep, stage 3 sleep, to stage 4 sleep. In other words, concordance is expected to be lowest during stage 1 sleep and highest during slow wave sleep with no discernible difference between stages 3 and 4 sleep. For the insomniac group, a total of 35,889 trials were presented during 37,909 epochs of recording (45 nights of polysomnography), and both sleep/wakefulness indices simultaneously identified sleep/wake during 35,265 epochs. The percent concordance for stages wake, 1, 2, 3, 4, and REM sleep was 91.17, 61.84, 98.01, 99.76, 100, and 99.73, respectively (see Table 7). Collapsed across wakefulness and sleep stages, the percent concordance was 93.0. For the control group, a total of 35,695 trials were presented during 37,744 epochs of recording (45 nights of polysomnography), and both sleep/wake indices simultaneously identified sleep/wake during 35,662 epochs. The percent concordance for stages wake, 1, 2, 3, 4, and REM sleep was 91.91, 61.10, 98.33, 99.93, 100, and 99.82, respectively (see Table 7). Collapsed across all stages, the percent concordance was 94.5.

Descriptive statistics and Pearson correlations for the insomniac group are presented in Tables 8 and 9, respectively. The polysomnographic set included total recording time (TRT), total sleep time (TST), total time awake (TTA), sleep onset latency defined as time from lights out to the first epoch of five consecutive minutes of any sleep stage (SOL), and number of awakenings following sleep onset (NA). The behavioural sleep/wake equivalents were used and included total number of trials (BR-TRT), total number of trials without response (BR-TST), behavioural sleep onset latency defined as the time from lights out to the first of five consecutive response failures (BR-SOL), number of microarousals defined as incidences of less than five consecutive responses following initial sleep onset (BRMICRO), and number of macroarousals (BRMACRO) defined as more than five consecutive responses following initial sleep onset .

Table 7

Percent concordance^a between sleep stage scoring and the behavioral response system.

Stage	S1 ^b	S2 ^b	S3 ^b	S4 ^b	S5 ^b	S6 ^b	S7 ^b	S8 ^b	S9 ^b	S10 ^b	S11 ^b	S12 ^b	S13 ^b	S14 ^b	S15 ^b	χ^c	sd ^d
0	93.85	96.34	99.08	97.89	93.99	77.10	96.58	94.24	99.44	83.58	98.56	71.24	98.16	69.04	99.49	91.17	10.55
1	55.96	70.12	45.01	63.17	56.20	96.18	22.92	55.66	34.91	72.50	53.83	82.07	79.84	90.37	48.89	61.84	20.30
2	98.19	98.68	96.18	99.15	97.16	100	97.24	94.63	96.05	98.61	97.78	99.36	99.59	99.88	98.43	98.01	1.56
3	100	100	100	100	99.19	100	100	98.61	100	100	99.15	99.40	100	100	100	99.76	0.45
4	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	0.00
R	99.16	100	98.17	100	99.44	100	100	99.19	100	100	100	100	100	100	100	99.73	0.53

Note. ^aConcordance during wakefulness (stage 0) requires behavioral responding, whereas during sleep stages (1, 2, 3, 4, R) concordance requires the absence of behavioral responding. ^bAverage of subject's three nights. ^cAverage of 45 nights. ^dStandard deviation.

Percent concordance^a between sleep stage scoring and the behavioral response system.

Stage	S1 ^b	S2 ^b	S3 ^b	S4 ^b	S5 ^b	S6 ^b	S7 ^b	S8 ^b	S9 ^b	S10 ^b	S11 ^b	S12 ^b	S13 ^b	S14 ^b	S15 ^b	χ^c	sd ^d
0	90.30	96.59	93.31	88.89	99.69	94.29	99.67	89.77	86.01	88.19	67.31	96.11	96.11	93.97	98.37	91.91	8.02
1	66.96	77.40	72.14	54.10	57.38	45.06	49.27	64.03	35.84	65.49	83.80	60.06	60.06	72.94	48.41	61.10	12.99
2	99.92	99.61	98.82	98.83	99.20	96.31	99.19	97.88	90.05	98.67	99.69	98.92	98.92	99.43	99.17	98.33	2.44
3	100	100	100	100	100	98.91	100	98.61	100	100	99.15	99.40	100	100	100	99.93	0.28
4	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	0.00
R	99.63	100	100	99.72	100	99.62	100	99.65	99.03	100	100	100	100	100	100	99.82	0.27

Note. ^aConcordance during wakefulness (stage 0) requires behavioral responding, whereas during sleep stages (1, 2, 3, 4, R) concordance requires the absence of behavioral responding. ^bAverage of subject's three nights. ^cAverage of 45 nights. ^dStandard deviation.

Table 8. Descriptive statistics for polysomnographic and behavioural response data for insomniacs

Variable	Night	$\bar{x}$	sd	range
TRT	1	409.9 min	39.3	150
TST	1	283.1 min	68.1	229
Total Time Awake	1	118.4 min	61.1	216
SOL	1	54.9 min	51.4	204
Number of Awakenings	1	12.9	7.0	30
BR-TRT (Number Trials)	1	787.2 trials	88.6	357
BR-TST (No Responses)	1	502.7 trials	191.2	426
BR-SOL	1	43.3 min	34.7	131
BRMICRO	1	18.4	8.3	31
BRMACRO	1	3.7	2.2	7
TRT	2	423.3 min	42.3	155
TST	2	364.1 min	48.6	166
Total Time Awake	2	51.0 min	29.8	106
SOL	2	27.7 min	22.0	99
Number of Awakenings	2	9.1	4.7	16
BR-TRT (Number Trials)	2	794.0 trials	80.6	314
BR-TST (No Responses)	2	670.4 trials	95.5	326
BR-SOL	2	28.6 min	25.6	100
BRMICRO	2	16	6.7	22
BRMACRO	2	2.6	2.1	6
TRT	3	430.5 min	46.7	160
TST	3	346.1 min	89.7	354
Total Time Awake	3	74.1 min	61.8	238
SOL	3	34.7 min	20.4	111
Number of Awakenings	3	9.7	5.4	22
BR-TRT (Number Trials)	3	811.5 trials	91.8	272
BR-TST (No Responses)	3	653.3 trials	168.5	677
BR-SOL	3	40.1 min	35.4	111
BRMICRO	3	16	6.1	22
BRMACRO	3	2.5	1.6	6

Note. SOL = sleep onset latency defined as the time from lights out to the first epoch of five consecutive minutes of any sleep stage; BR-TST = number of failed responses following sleep onset; BR-SOL = time from lights out to first of 5 consecutive response failures; BRMICRO = number of microarousals defined as < 5 consecutive responses following initial sleep onset; BRMACRO = number of macroarousals defined as ≥ 5 consecutive responses following initial sleep onset.

Table 9. Pearson correlation matrices for insomniac subjects

Night one					
	TRT	TST	TTA	SOL	NA
BR-TRT	.92**	.45	.06	.39	-.39
BR-TST	-.08	.91**	-.79**	-.41	-.60*
BR-SOL	.22	-.32	.47	.67**	-.14
BRMICRO	.14	-.22	.32	.41	.35
BRMACRO	.12	-.24	.38	-.35	.76**
Night two					
	TRT	TST	TTA	SOL	NA
BR-TRT	.92**	.80*	.08	.42	-.13
BR-TST	.71**	.94**	-.58*	-.13	-.37
BR-SOL	.57*	.17	.49	.87**	-.23
BRMICRO	-.14	-.37	.36	.10	.47
BRMACRO	-.14	-.33	.36	-.14	.42
Night three					
	TRT	TST	TTA	SOL	NA
BR-TRT	.93**	.65**	-.28	.16	-.50
BR-TST	.72**	.95**	-.84**	-.44	-.38
BR-SOL	.24	-.35	.67**	.94**	-.08
BRMICRO	-.23	-.13	.02	-.22	.64**
BRMACRO	.03	-.18	.26	.10	.34

Note. TTA = total time awake; SOL = sleep onset latency defined as the time from lights out to the first epoch of five consecutive minutes of any sleep stage; NA = number of awakenings following initial sleep onset; BR-TST = number of failed responses following sleep onset; BR-SOL = time from lights out to first of 5 consecutive response failures; BRMICRO = number of microarousals defined as < 5 consecutive responses following initial sleep onset; BRMACRO = number of macroarousals defined as ≥ 5 consecutive responses following initial sleep onset. * $p < .05$, ** $p < .01$.

Descriptive statistics and Pearson correlations for the control group are shown in Tables 10 and 11, respectively. Correlations greater than .51 are statistically significant (with $n = 15$, $df = 13$). Note that strong positive correlations exist for each night across groups for the following variables : TRT-BRTTRT, TST-BRTST, and SOL-BRSOL.

Hypothesis five stated that there would be a high degree of multivariate overlap between polysomnographic and behavioural sleep/wake measurements. Canonical correlations were performed on data from each night's set of polysomnographic variables and each night's set of behavioural sleep/wake variables with the use of Systat MGLH (Wilkinson, 1986). According to Tabachnick and Fidell (1989), the number of cases required for analysis depends on the reliability of the variables. Typical practice in the social sciences is to have 10 cases for each variable used in canonical correlation; however, if reliability is very high then a much lower ratio of cases to variables is acceptable. Within the current study, canonical correlation was used descriptively so more latitude was permitted with respect to the cases-to-variables issue. Polysomnographic and behavioural sleep efficiency measures were not included in the multivariate analyses because they would have violated assumptions regarding within-set multicollinearity.

For the insomniac group on night one, the first canonical correlation was 0.99 (98% of variance); the second canonical correlation was 0.91 (83% of variance). With all five canonical correlations included, $\chi^2(25) = 60.65$, $p = .001$, and with the first canonical correlation removed, $\chi^2(16) = 29.73$, $p = .02$. Subsequent χ^2 tests of the three remaining canonical correlations were not significant (see Table 12). Thus, the first two pairs of canonical variates accounted for the significant relationship between the two sets of variables.

Table 10. Descriptive statistics for polysomnographic and behavioural response data for controls

Variable	Night	$\bar{x}$	sd	range
TRT	1	420.6 min	53.6	168.5
TST	1	376.0 min	60.3	219
Total Time Awake	1	35.2 min	36.3	134
SOL	1	12.1 min	11.1	42.5
Number of Awakenings	1	11.6	8.6	35
BR-TRT (Number Trials)	1	803.9 trials	105.8	291
BR-TST (No Responses)	1	684.5 trials	117.8	403
BR-SOL	1	16.8 min	13.3	43
BRMICRO	1	15.1	7.8	2.0
BRMACRO	1	3.3	1.8	6
TRT	2	434.6 min	52.1	182.5
TST	2	408.9 min	59.2	221
Total Time Awake	2	22.4 min	17.4	58
SOL	2	5.5 min	4.9	13.5
Number of Awakenings	2	8.7	7.7	30
BR-TRT (Number Trials)	2	832.2 trials	140.3	512
BR-TST (No Responses)	2	758.0 trials	177.2	542
BR-SOL	2	8.4 min	6.2	20.5
BRMICRO	2	14.3	8.7	27
BRMACRO	2	2.7	1.2	5
TRT	3	397.3 min	50.7	160.5
TST	3	377.6 min	54.4	180.5
Total Time Awake	3	16.7 min	9.9	28
SOL	3	7.5 min	6.8	22.5
Number of Awakenings	3	6.2	2.9	11
BR-TRT (Number Trials)	3	743.53 trials	90.67	287
BR-TST (No Responses)	3	692.0 trials	102.7	327
BR-SOL	3	12.7 min	6.9	21
BRMICRO	3	10.0	3.9	12
BRMACRO	3	1.7	0.82	2

Note. SOL = sleep onset latency defined as the time from lights out to the first epoch of five consecutive minutes of any sleep stage; BR-TST = number of failed responses following sleep onset; BR-SOL = time from lights out to first of 5 consecutive response failures; BRMICRO = number of microarousals defined as < 5 consecutive responses following initial sleep onset; BRMACRO = number of macroarousals defined as ≥ 5 consecutive responses following initial sleep onset.

Table 11. Pearson correlation matrices for control subjects

Night one

	TRT	TST	TTA	SOL	NA
BR-TRT	.96**	.74**	.09	-.20	.18
BR-TST	.43	.67**	-.55*	-.46	-.66**
BR-SOL	-.20	-.54*	.66**	.84**	.44
BRMICRO	.59*	.36	.28	.07	.12
BRMACRO	.35	.09	.30	-.13	.64**

Night two

	TRT	TST	TTA	SOL	NA
BR-TRT	.86**	.83**	-.27	-.19	-.30
BR-TST	.86**	.89**	-.47	-.23	-.54*
BR-SOL	-.55*	-.59*	.34	.59*	.34
BRMICRO	-.21	-.44	.85**	.15	.80**
BRMACRO	.06	.04	-.02	.05	.15

Night three

	TRT	TST	TTA	SOL	NA
BR-TRT	.95**	.96**	-.37	-.29	-.25
BR-TST	.89**	.93**	-.48	-.40	-.19
BR-SOL	-.28	-.35	-.61*	.66**	-.44
BRMICRO	.17	.19	-.01	-.19	-.01
BRMACRO	-.47	-.41	.03	-.18	.16

Note. TTA = total time awake; SOL = sleep onset latency defined as the time from lights out to the first epoch of five consecutive minutes of any sleep stage; NA = number of awakenings following initial sleep onset; BR-TST = number of failed responses following sleep onset; BR-SOL = time from lights out to first of 5 consecutive response failures; BRMICRO = number of microarousals defined as < 5 consecutive responses following initial sleep onset; BRMACRO = number of macroarousals defined as ≥ 5 consecutive responses following initial sleep onset. * $p < .05$, ** $p < .01$.

For the insomniac group on night two, the first canonical correlation was 0.98 (96% of variance); the second canonical correlation was 0.95 (91% of variance). With all five canonical correlations included, $\chi^2(25) = 64.70$, $p < .001$, and with the first canonical correlation removed, $\chi^2(16) = 35.94$, $p = .003$. Subsequent χ^2 tests of the three remaining canonical correlations were not significant (see Table 12). Again, the first two pairs of canonical variates⁵ accounted for the significant relationship between the two sets of variables.

For the insomniac group on night three, the first canonical correlation was .99 (98% of variance); the second canonical correlation was .97 (94% of variance); and the third was .92 (85% of variance). With all five canonical correlations included, $\chi^2(25) = 77.67$, $p < .001$, with the first canonical correlation removed, $\chi^2(16) = 44.68$, $p < .001$, and with the first and second canonical correlations removed, $\chi^2(9) = 20.37$, $p = .02$. Subsequent χ^2 tests of the two remaining canonical correlations were not significant (see Table 12). The first three pairs of canonical variates, therefore, accounted for the significant relationship between the two sets of variables.

⁵According to Tabachnick and Fidell (1989), canonical variates are linear combinations of variables. One linear combination represents variable set X (e.g., total sleep time, sleep onset latency and number of awakenings) and a second linear combination for variable set Y (e.g., behavioral total sleep time, behavioural sleep onset latency and number of macroarousals). These two combinations form a pair of canonical variates which are derived to maximize the correlation between them (i.e., canonical correlation). While there may be more than one reliable pair of canonical variates, usually only the first few pairs of variates are significant.

Table 12. Canonical correlations, eigenvalues (canonical R squared), and χ^2 p-values for each of the three subject-nights for the insomniac group

Night	Canonical variate pair	Canonical correlation (Rc)	Eigenvalue (Rc ²)	p
1	1	0.99	0.98	.001
	2	0.91	0.83	.019
	3	0.86	0.74	.105
	4	0.55	0.30	.516
	5	0.16	0.03	.639
2	1	0.98	0.96	.001
	2	0.95	0.91	.003
	3	0.86	0.74	.078
	4	0.59	0.35	.384
	5	0.24	0.06	.484
3	1	0.99	0.98	.001
	2	0.97	0.94	.001
	3	0.92	0.85	.016
	4	0.62	0.38	.352
	5	0.19	0.04	.569

With regard to the control group on night one, the first canonical correlation was .99 (98% of variance); the second canonical correlation was .92 (85% of variance); and the third was .89 (79% of variance). With all five canonical correlations included, $\chi^2(25) = 67.47$, $p < .001$, with the first canonical correlation removed, $\chi^2(16) = 32.90$, $p = .008$, and with the first and second canonical correlations removed, $\chi^2(9) = 17.17$, $p = .046$. Subsequent χ^2 tests of the two remaining canonical correlations were not significant (see Table 13). The first three pairs of canonical variates, therefore, accounted for the significant relationship between the two sets of variables.

For the control group on night two, the first canonical correlation was 0.97 (94% of variance); the second canonical correlation was 0.94 (88% of variance). With all five canonical correlations included, $\chi^2(25) = 53.23$, $p < .001$, and with the first canonical

correlation removed, $\chi^2(16) = 28.72$, $p = .026$. Subsequent χ^2 tests of the three remaining canonical correlations were not significant (see Table 13). The first two pairs of canonical variates accounted for the significant relationship between the two sets of variables.

For the control group on night three, the first canonical correlation was .98 (96% of variance). With all five canonical correlations included, $\chi^2(25) = 53.45$, $p = .001$. Subsequent χ^2 tests of the four remaining canonical correlations were not significant (see Table 13). The first pair of canonical variates, therefore, accounted for the significant relationship between the two sets of variables.

Unlike multiple regression, where the squared multiple correlation represents the proportion of dependent variable (DV) variance accounted for by the optimal linear combination of the IVs, the squared canonical correlation does not necessarily signify considerable variance overlap between the two sets of variables (Stevens, 1992), but rather indicates the amount of variance shared by a pair of canonical variates. To determine the amount of variance accounted for by each of the data sets, Stewart and Love (1968) developed the redundancy index, which is equivalent to the average squared multiple correlation obtained separately for each variable in one set (i.e., DV) from the variables in the other set (i.e., IVs). However, using either the redundancy index or calculating the average squared multiple correlations, as from regression analyses, has been criticized by Cramer and Nicewander (1979) because they do not take into account intercorrelations of the variables being predicted. Hence, neither the redundancy index nor the use of average squared multiple correlations are truly multivariate indices of shared variance. As a measure of variance accounted for in canonical correlation, Cramer and Nicewander recommended using the average squared canonical correlation.

Table 13. Canonical correlations, eigenvalues (canonical R squared), and χ^2 p-values for each of the three subject-nights for the control group

Night	Canonical variate pair	Canonical correlation (Rc)	Eigenvalue (Rc ²)	p
1	1	0.99	0.98	.001
	2	0.92	0.85	.008
	3	0.89	0.79	.046
	4	0.60	0.35	.403
	5	0.19	0.03	.582
2	1	0.97	0.94	.001
	2	0.94	0.88	.026
	3	0.76	0.58	.280
	4	0.57	0.32	.485
	5	0.12	0.01	.719
3	1	0.98	0.96	.001
	2	0.93	0.87	.055
	3	0.68	0.45	.519
	4	0.54	0.29	.563
	5	0.08	0.01	.802

For the insomniac group, the average squared canonical correlation for night one was 0.58, indicating that the two sets of variables share 58% of variance. For night two, the average squared canonical correlation was 0.60, suggesting that the two sets of variables share 60% of variance. The average squared canonical correlation for night three was 0.64, indicating that the two sets of variables share 64% of variance. For the control group, the two sets of variables share 60%, 55%, and 52% of the variance during nights one, two, and three, respectively.

Hypotheses Six to Ten - Nights Two and Three Collapsed

For the remainder of the analyses, night one was omitted from statistical procedures and nights two and three were collapsed to produce an average of these two nights. This was done for two reasons: (1) the presence of a first-night effect has been well documented both within this study (see above) and within previous work (Agnew & Webb, 1966; Bonato et al., 1992; Webb, & Campbell, 1979), and it remains common practice for many researchers to omit first-night data from statistical analyses. Second, all of the following analyses had been analyzed using separate nights and it became clear that analyzing the nights separately both increased the amount of variance in the data, and produced results which would be overwhelming to the reader (i.e., 115 tables). Thus, omitting night one from statistical analyses is consonant with research practices; averaging nights two and three both reduces variability and permits an examination of the data which does not overwhelm the reader.

Hypothesis six stated that insomniacs would show shorter response latencies than controls during the sleep onset period and suggested that this may be due to increased cognitive arousal. To examine this, a factorial ANOVA using a critical alpha level of .05 was calculated for each of the two nights using group as the between measure and reaction time during the last 20 responses as the dependent variable. Response failures (i.e., reaction times of 5 seconds) were not included in statistical analyses. Means and standard deviations are presented in Table 14.

Contrary to expectation, the insomniacs showed significantly longer reaction times than the control subjects, $F(1, 976) = 49.76, p = .0001$. Group means and standard deviations are presented in Table 14. Application of the nonparametric Mann-Whitney U test yielded identical results: a z-score of -8.278 with a correction for ties. Reference to Table A of Siegel and Castellan (1988) indicates that a z-score of -8.278 is associated with a probability of less than .0001; that is, $z = -8.278, p < .0001$. To eliminate the

possibility that these results were due to practice effects and monotony (i.e., insomniacs have longer sleep onset latencies and consequently respond much more before the onset of sleep), the tests were repeated using reaction times from the first 20 trials. For this additional analysis, reaction times of 5 seconds (i.e., response failures) were omitted from statistical analyses and the critical alpha level was set at the more conservative .01 level.

Table 14. Means and standard deviations for last 20 responses during sleep onset

Group	Count	Mean	sd
Insomniacs	527	1696.66	811.83
Controls	451	1343.18	743.67

Note. Time in milliseconds. Results are an average of nights 2 and 3.

Again, the insomniacs showed significantly longer reaction times than the control subjects, $F(1, 894)=39.73$, $p = .0001$. Group means and standard deviations are presented in Table 15. Application of the nonparametric Mann-Whitney U test yielded identical results. The Mann-Whitney U test revealed a z-score of -8.147 with a correction for ties. A z-score of -8.147 is associated with a probability of less than .0001; that is, $z = -8.147$, $p < .0001$.

Table 15. Means and standard deviations for first 20 responses during sleep onset

Group	Count	Mean	sd
Insomniacs	508	1381.41	772.19
Controls	388	1062.02	723.60

Note. Time in milliseconds. Results are an average of nights 2 and 3.

Period-Amplitude Analysis of the Electroencephalogram

The use of period-amplitude analysis produced four dependent variables - full-wave, half-wave, first-derivative, and integrated amplitude - per EEG bandwidth

frequency per sleep stage. The five EEG frequency bands were defined as follows: delta 0.5 to 3.99 Hz; theta 4.00 to 7.99 Hz; alpha 8.00 to 11.99 Hz; sigma 12.00 to 15.99 Hz; and beta 16.00 to 30.00 Hz. Recall that the full-wave measure preferentially detects midrange frequencies such as alpha EEG; the half-wave measure preferentially detects the slowest frequency in the ongoing waveform; the first-derivative measure preferentially detects the fastest frequency in the ongoing EEG waveform; and the integrated amplitude measure is conceptually analogous to the FFT power measure. For all MANOVAs, group was used as the between-group independent measure, and sleep stage as the repeated measure. The dependent measures were the PAA incidence and amplitude for each of the five standard frequency bands described above. To compensate for possible violations of repeated measures assumptions (assumption of homogeneity of variance-covariance matrices), Huynh-Feldt epsilon adjustments were used for all univariate tests involving a repeated measures design. Prior to performing the MANOVAs, the complete data set was checked for accuracy of data entry, missing data, outliers (by group), and normality. There were no missing cases in either of the two groups. The following statistical tests were performed to specifically address hypotheses seven and eight. Recall that hypothesis seven stated that period-amplitude analyzed EEG measures would reveal a physiological abnormality in the insomniacs not observed through the application of traditional sleep stage criteria, and that this would be manifested by less delta, theta, and alpha activity, and more beta EEG activity in the insomniacs than in the controls during sleep onset (*hypothesis #7*). Hypothesis eight is based on the findings of Freedman (1986) and Lamarche (1995): the period-amplitude analyzed EEG of insomniacs is hypothesized to show more beta EEG activity and less alpha EEG activity during wakefulness than that of controls.

To evaluate whether the EEG of insomniacs indicated a physiological abnormality not observed through the application of traditional sleep stage criteria (*hypothesis #7*), the

EEG during the sleep onset period (SOP) was period-amplitude analyzed (PAA) using 30-second epochs. First, the last period of wakefulness prior to 15 minutes of stage 2 sleep was identified. Next, epochs not contaminated with muscle and movement artifacts were selected. These epochs consisted of (a maximum of) the last 15 minutes of stage wake up to the first 15 minutes of continuous stage 2 sleep. Due to unequal sample sizes both between groups and between sleep stages, the data for each subject were averaged within subjects and within sleep stages. For the insomniac group, averaging within subjects and stages involved 447, 252, and 708 epochs for sleep stages wake, one, and two sleep, respectively. For the control group, averaging within subjects and stages involved 245, 328, and 702 epochs for sleep stages wake, one, and two, respectively. The following statistical tests were performed to specifically address the hypothesis that insomniacs will show less delta, theta, and alpha EEG, and more beta EEG than controls during the sleep onset period (*hypothesis #7*). For these hypothesized tests, the critical alpha level was set at .05; the critical alpha level for tests involving sigma EEG was set at .01.

C₃-A₂ Period-Amplitude Measures by Sleep Stage

For C₃-A₂, the mixed MANOVA computed on full-wave (FW) incidence revealed a statistically significant main effect for sleep stage (within-subjects factor) and a statistically significant interaction, but no significant main effect for group was found. The results using Wilks' criterion were as follows: $F(5, 24) = 2.40$, $p = .067$ for group; $F(10, 19) = 63.39$, $p < .0001$ for sleep stage; $F(10, 19) = 4.38$, $p = .003$ for the interaction. The means and standard deviations for each group and sleep stage are shown in Table 16. Because of the statistically significant interaction, the univariate Fs for the interaction were examined. As is shown in Table 17, the univariate breakdown showed statistically significant stage by group interactions in the beta, alpha, and delta frequency bands. Due

to these statistically significant interactions the simple effects for group by stage were examined (see Figures 2, 3, 4). The simple effect analysis of stage within the insomniac group for beta full-wave incidence was statistically significant, $F(2, 28) = 130.91$, $p < .00001$, indicating that beta full-wave incidence declines in the insomniac group during the transition from wakefulness through stage one sleep to stage two sleep. Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that all pairwise comparisons were statistically significant (see Table 18). The simple effect analysis of stage within the control group for beta full-wave incidence was statistically significant, $F(2, 28) = 126.58$, $p < .00001$, indicating that beta activity decreases in the control group during the transition from wakefulness to stage two sleep. Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that all pairwise comparisons were statistically significant (see Table 18). The simple effect analysis of group within stage wake was not statistically significant, $F(1, 28) = .98$, $p < .33$, revealing no between group differences in beta full-wave time-in-frequency during stage wake (see Figure 2). A similar analysis of group within stage one was not statistically significant, $F(1, 28) = 1.12$, $p = .29$, indicating no between group differences in beta full-wave incidence during stage one sleep (see Figure 2). The analysis of group within stage two was statistically significant, $F(1, 28) = 5.60$, $p = .025$, with insomniacs showing significantly more beta full-wave incidence than controls during stage two sleep (see Figure 2).

The simple effect analysis of stage within the insomniac group for alpha full-wave incidence was statistically significant, $F(2, 28) = 16.38$, $p = .0009$, indicating that alpha full-wave incidence declines in the insomniac group during the transition from wakefulness through stage one sleep to stage two sleep (see Figure 3). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that all pairwise comparisons were statistically significant (see Table 19). The analysis of stage within the control group for alpha full-wave incidence was also significant, $F(2, 28) = 14.56$, $p =$

.00008, indicating that alpha full-wave incidence decreases in the control group from stage wake to stage two sleep (see Figure 3). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level indicated that stage 2 sleep differed from the other sleep stages (see Table 19). The group within stage effect wake was significant, $F(1, 28) = 7.34$, $p = .01$, revealing significantly more alpha full-wave incidence during stage wake in the insomniac group than in the control group (see Figure 3). The simple effect analysis of group within stage one sleep was not significant, $F(1, 28) = 2.60$, $p = .11$, indicating no between group differences in alpha full-wave incidence during stage one sleep (see Figure 3). The effect of group within stage two sleep was significant, $F(1, 28) = 6.20$, $p = .02$, with insomniacs showing significantly more alpha full-wave incidence during stage two sleep than controls (see Figure 3).

The simple effect analysis of stage within the insomniac group for delta full-wave incidence was statistically significant, $F(2, 28) = 88.79$, $p < .00001$, indicating that delta full-wave incidence increases in the insomniac group during the transition from wakefulness through stage one sleep to stage two sleep (see Figure 4). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that all pairwise comparisons were statistically significant (see Table 20). The analysis of stage within the control group for delta full-wave incidence was significant, $F(2, 28) = 100.39$, $p < .00001$, indicating that delta full-wave incidence increases in the control group during the transition from wakefulness to stage two sleep (see Figure 4). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that all pairwise comparisons were statistically significant (see Table 20). The analysis of group within stage wake was not significant, $F(1, 28) = .37$, $p = .55$ (see Figure 4), indicating no between group differences in delta full-wave incidence during stage wake (see Figure 4). The analysis of group within stage one sleep was statistically significant, $F(1, 28) = 6.23$, $p = .018$, with controls showing significantly more delta full-wave incidence during stage

one sleep than insomniacs (see Figure 4). The analysis of group within stage two sleep was statistically significant, $F(1, 28) = 7.96, p = .009$, with controls showing significantly more delta full-wave incidence during stage two sleep than insomniacs (see Figure 4).

A mixed MANOVA computed on C_3-A_2 half-wave (HW) incidence revealed a statistically significant main effect for group as shown by the significant Wilks' lambda, $F(2, 24) = 3.44, p = .017$, a statistically significant main effect for sleep stage, $F(10, 19) = 49.32, p = .00001$, but the test for an interaction failed to reach statistical significance, $F(10, 19) = 2.28, p = .058$. The means and standard deviations for each group and sleep stage are shown in Table 21. In the univariate breakdown of the main effect for group, both delta and alpha C_3-A_2 half-wave incidence reached statistical significance at a critical alpha level of .05 (see Table 22). As expected, the insomniacs showed less delta EEG activity, but contrary to expectation more alpha C_3-A_2 half-wave incidence was observed in the insomniac group. Concerning the significant sleep stage main effect (within subject measure), the univariate results are shown in Table 23. Statistically significant increases in both theta and delta half-wave incidence were observed during the transition from stage wake through stage one sleep to stage 2 sleep. All remaining C_3-A_2 frequencies showed a significant decrease in half-wave incidence from stage wake to stage 2 sleep (see Table 23).

The C_3-A_2 first-derivative (FD) incidence mixed MANOVA revealed a statistically significant main effect for group and for sleep stage and a statistically significant interaction. The results using Wilks' criterion were as follows: $F(5, 24) = 3.46, p = .017$ for group; $F(10, 19) = 37.16, p < .00001$ for sleep stage; $F(10, 19) = 2.92, p = .021$ for the interaction. The means and standard deviations for each group and sleep stage are shown in Table 24. Because of the statistically significant multivariate interaction, the univariate Fs for the interaction were examined. As is shown in Table 25,

the univariate breakdown showed statistically significant stage by group interactions in the beta, alpha, theta, and delta frequency bands. Due to these significant interactions the simple effects for group by stage were examined (see Figures 5, 6, 7, 8). The simple effect analysis of stage within the insomniac group for beta first-derivative incidence was statistically significant, $F(2, 28) = 23.51$, $p < .00001$ (see Figure 5). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that stage 2 sleep significantly differed from stage wake and stage 1 sleep (see Table 26) indicating that beta first-derivative incidence declines in the insomniac group during the transition from stage one sleep to stage two sleep. The analysis of stage within the control group for beta first-derivative incidence was significant, $F(2, 28) = 49.33$, $p < .00001$, (see Figure 5). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that stage 2 sleep significantly differed from stage wake and stage 1 sleep (see Table 26) indicating that beta activity decreases in the control group during the transition from stage one sleep to stage two sleep. The simple effect analysis of group within stage wake was not statistically significant, $F(1, 28) = .689$, $p < .41$, revealing no between group differences in beta first-derivative time-in-frequency during stage wake (see Figure 5). The analysis of group within stage one was not statistically significant, $F(1, 28) = .011$, $p = .92$, indicating no between group differences in beta first-derivative incidence during stage one sleep (see Figure 5). The analysis of group within stage two was statistically significant, $F(1, 28) = 4.87$, $p = .035$, with insomniacs showing significantly more beta first-derivative incidence than controls during stage two sleep (see Figure 5).

Table 16. Means and standard deviations for C₃-A₂ full-wave incidence

GROUP	STAGE	Delta	Theta	Alpha	Sigma	Beta
Insomniacs	0	4.41 (6.73)	17.53 (7.90)	30.39 (11.46)	14.52 (2.72)	22.27 (6.97)
Insomniacs	1	8.65 (4.95)	31.49 (8.20)	23.71 (4.50)	12.59 (2.57)	15.88 (5.74)
Insomniacs	2	23.86 (7.77)	41.38 (5.89)	17.13 (2.69)	8.07 (2.49)	6.39 (4.27)
Controls	0	5.78 (5.54)	18.21 (8.27)	21.33 (6.04)	15.27 (3.93)	24.76 (6.79)
Controls	1	14.02 (6.71)	33.71 (6.60)	21.20 (3.98)	11.47 (2.31)	13.67 (5.62)
Controls	2	31.14 (6.28)	42.11 (2.33)	14.84 (2.35)	6.80 (1.75)	3.64 (1.43)

Note. Standard deviation in parentheses.

Table 17. Univariate F ratios for group by sleep stage interaction for C₃-A₂ full-wave incidence

EEG Frequency	F	p
Delta	3.19	.048
Theta	0.23	.793
Alpha	4.07	.022
Sigma	1.55	.220
Beta	6.07	.004

Note. Analyses are based on 2 and 56 degrees of freedom.

Figure 2. Plot of group x sleep stage interaction for C3-A2 beta full-wave incidence. Arrow indicates significant between group differences at $p < .05$. See also Table 18.

Table 18. Post-hoc Tukey HSD tests for C₃-A₂ beta full-wave incidence

Sleep Stage	Insomniacs			Controls		
	$\bar{x}$			$\bar{x}$		
	22.27	15.88	6.39	24.76	13.67	3.64
	ST0	ST1	ST2	ST0	ST1	ST2
Insom ST0		*	*			
Insom ST1	*		*			
Insom ST2	*	*				
Control ST0					*	*
Control ST1				*		*
Control ST2				*	*	

Note. Tukey HSD Test for specific sleep stages by group. Asteriks indicate a statistically significant difference at $p < .05$.

Figure 3. Plot of group x sleep stage interaction for C3-A2 alpha full-wave incidence. Arrows indicate significant between group differences at $p < .05$. See also Table 19.

Table 19. Post-hoc Tukey HSD tests for C₃-A₂ alpha full-wave incidence

Sleep Stage		Insomniacs			Controls		
		$\bar{x}$			$\bar{x}$		
		30.39	23.71	17.13	21.33	21.20	14.84
		ST0	ST1	ST2	ST0	ST1	ST2
Insom	ST0		*	*			
Insom	ST1	*		*			
Insom	ST2	*	*				
Control	ST0						*
Control	ST1						*
Control	ST2				*	*	

Note. Tukey HSD Test for specific sleep stages by group. Asteriks indicate a statistically significant difference at $p < .05$.

Figure 4. Plot of group x sleep stage interaction for C3-A2 delta full-wave incidence. Arrows indicate significant between group differences at $p < .05$. See also Table 20.

Table 20. Post-hoc Tukey HSD tests for C_3 - A_2 delta full-wave incidence

Sleep Stage		Insomniacs			Controls		
		$\bar{x}$			$\bar{x}$		
		4.41	8.65	23.86	5.78	14.03	31.14
		ST0	ST1	ST2	ST0	ST1	ST2
Insom	ST0		*	*			
Insom	ST1	*		*			
Insom	ST2	*	*				
Control	ST0					*	*
Control	ST1				*		*
Control	ST2				*	*	

Note. Tukey HSD Test for specific sleep stages by group. Asteriks indicate a statistically significant difference at $p < .05$.

Table 21. Means and standard deviations for C₃-A₂ half-wave incidence

GROUP	STAGE	Delta	Theta	Alpha	Sigma	Beta
Insomniacs	0	8.78 (9.16)	19.66 (5.30)	29.60 (9.89)	12.16 (2.11)	17.47 (4.59)
Insomniacs	1	17.17 (6.40)	30.01 (4.85)	19.76 (4.26)	9.71 (1.69)	13.33 (2.69)
Insomniacs	2	33.97 (6.67)	32.55 (3.24)	13.83 (2.02)	7.75 (1.91)	7.13 (1.99)
Controls	0	11.93 (8.81)	19.63 (5.68)	19.75 (5.01)	12.39 (2.27)	19.73 (5.49)
Controls	1	23.48 (7.54)	29.97 (3.13)	16.34 (3.05)	8.74 (1.71)	12.71 (3.84)
Controls	2	39.99 (4.99)	32.04 (2.02)	11.80 (1.97)	7.13 (1.34)	5.83 (0.90)

Note. Standard deviation in parentheses.

Table 22. Mean C₃-A₂ half-wave incidence and univariate F ratios for each EEG frequency between groups

Frequency	Insomniacs	Controls	F	p
Delta	19.97	21.06	5.42	.027
Theta	27.40	27.22	0.02	.873
Alpha	21.06	15.96	14.54	.001
Sigma	9.88	9.42	0.82	.371
Beta	12.64	12.76	0.01	.916

Table 23. Mean C₃-A₂ half-wave incidence collapsed across groups and univariate F ratios for each EEG frequency within sleep stages

Frequency	Stage 0	Stage 1	Stage 2	F	p
Delta	<u>10.36</u>	<u>20.33</u>	<u>36.98</u>	199.84	.00001
Theta	<u>19.64</u>	<u>30.00</u>	<u>32.30</u>	122.81	.00001
Alpha	<u>24.68</u>	<u>18.05</u>	<u>12.81</u>	54.47	.00001
Sigma	<u>12.28</u>	<u>9.23</u>	<u>7.44</u>	74.56	.00001
Beta	<u>18.60</u>	<u>13.02</u>	<u>6.48</u>	174.53	.00001

Note. Significance levels are based on Huynh-Feldt epsilon adjustments. Univariate *F* tests have 2 and 56 degrees of freedom prior to epsilon adjustments. Means underlined on the same line are significantly different ($p < .05$) according to Tukey HSD analysis.

The simple effect analysis of stage within the insomniac group for alpha first-derivative incidence was statistically significant, $F(2, 28) = 5.46$, $p = .03$ (see Figure 6). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that stage 2 sleep significantly differed from stage wake and stage 1 sleep (see Table 27) indicating an increase in alpha first-derivative incidence in the insomniac group during the transition from stage wake to stage two sleep. The analysis of stage within the control group for alpha first-derivative incidence was statistically significant, $F(2, 28) = 15.72$, $p = .00003$ (see Figure 6). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that stage 2 sleep significantly differed from stage wake and stage 1 sleep (see Table 27) indicating that alpha activity increases in the control group during the transition from stage wake to stage two sleep. The simple effect analysis of group within stage wake was not statistically significant, $F(1, 28) = .80$, $p < .38$, revealing no between group differences in alpha first-derivative time-in-frequency during stage wake (see Figure 6). The analysis of group within stage one was not statistically significant, $F(1, 28) = .60$, $p = .45$, indicating no between group differences in alpha first-derivative

Table 24. Means and standard deviations for C₃-A₂ first-derivative incidence

GROUP	STAGE	Delta	Theta	Alpha	Sigma	Beta
Insomniacs	0	0.64 (1.89)	6.60 (7.76)	21.11 (10.36)	13.14 (4.39)	31.16 (8.45)
Insomniacs	1	1.24 (2.51)	12.88 (10.88)	20.32 (10.48)	13.23 (4.70)	30.80 (9.48)
Insomniacs	2	2.15 (1.97)	22.06 (10.30)	25.79 (8.66)	16.57 (5.33)	20.28 (6.66)
Controls	0	0.30 (0.50)	7.27 (6.04)	17.93 (8.45)	13.58 (4.77)	33.54 (7.20)
Controls	1	1.43 (1.71)	17.59 (8.20)	22.78 (6.54)	15.85 (1.94)	30.45 (8.60)
Controls	2	3.64 (2.44)	29.30 (7.25)	28.95 (3.27)	18.42 (2.74)	15.39 (5.41)

Note. Standard deviation in parentheses.

Table 25. Univariate F ratios for group by sleep stage interaction for C₃-A₂ first-derivative incidence

EEG Frequency	F	p
Delta	3.92	.025
Theta	3.52	.036
Alpha	3.41	.040
Sigma	1.42	.249
Beta	3.82	.028

Note. Analyses are based on 2 and 56 degrees of freedom.

incidence during stage one sleep (see Figure 6). The analysis of group within stage two sleep was not statistically significant, $F(1, 28) = 1.75$, $p = .20$, indicating no between group differences in alpha first-derivative time-in-frequency during stage two sleep (see Figure 6).

The simple effect analysis of stage within the insomniac group for theta first-derivative incidence was statistically significant, $F(2, 28) = 29.72$, $p < .00001$ (see Figure 7). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed all pairwise comparisons were significantly different (see Table 28) indicating that theta first-derivative incidence increases in the insomniac group during the transition from stage wake to stage two sleep. The analysis of stage within the control group for theta first-derivative incidence was significant, $F(2, 28) = 112.69$, $p < .00001$ (see Figure 7). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that all pairwise comparisons significantly differed (see Table 28) indicating that theta activity increases in the control group during the transition from stage wake through stage one sleep to stage two sleep. The simple effect analysis of group within stage wake was not statistically significant, $F(1, 28) = .07$, $p < .79$, revealing no between group differences in theta first-derivative time-in-frequency during stage wake (see Figure 7). The analysis of group within stage one was not significant, $F(1, 28) = 1.79$, $p = .19$, indicating no between group differences in theta first-derivative incidence during stage one sleep (see Figure 7). The analysis of group within stage two sleep was significant, $F(1, 28) = 4.94$, $p = .034$, with insomniacs showing less theta first-derivative incidence than controls during stage two sleep (see Figure 7).

Figure 5. Plot of group $\times$ sleep stage interaction for C3-A2 beta first-derivative incidence. Arrow indicates significant between group difference at $p < .05$. See also Table 26.

Table 26. Post-hoc Tukey HSD tests for C₃-A₂ beta first-derivative incidence

Sleep Stage		Insomniacs			Controls		
		$\bar{x}$			$\bar{x}$		
		31.16 ST0	30.79 ST1	20.28 ST2	33.54 ST0	30.45 ST1	15.39 ST2
Insom	ST0			*			
Insom	ST1			*			
Insom	ST2	*	*				
Control	ST0						*
Control	ST1						*
Control	ST2				*	*	

Note. Tukey HSD Test for specific sleep stages by group. Asteriks indicate a statistically significant difference at $p < .05$.

The simple effect analysis of stage within the insomniac group for delta first-derivative incidence was statistically significant, $F(2, 28) = 7.04$, $p = .015$ (see Figure 8). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that stage two sleep significantly differed from stage wake and stage one indicating that delta first-derivative incidence increased in the insomniac group during the transition from stage wake to stage two sleep. The analysis of stage within the control group for delta first-derivative incidence was significant, $F(2, 28) = 20.33$, $p < .00001$ (see Figure 8). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that stage two sleep significantly differed from the other stages (see Table 29). This indicates that delta activity increases in the control group from stage wake and stage one sleep to stage two sleep. The simple effect analysis of group within stage wake was not significant, $F(1, 28) = .45$, $p < .51$, revealing no between group differences in delta first-derivative time-in-frequency during stage wake (see Figure 8). The analysis of group within stage one was not significant, $F(1, 28) = .06$, $p = .81$, indicating no between group differences in delta first-derivative incidence during stage one sleep (see Figure 8). Similarly, the analysis of group within stage two sleep failed to reach statistical significance, $F(1, 28) = 3.37$, $p = .077$ (see Figure 8).

Figure 6. Plot of group x sleep stage interaction for C3-A2 alpha first-derivative incidence. See also Table 27.

Table 27. Post-hoc Tukey HSD tests for C₃-A₂ alpha first-derivative incidence

Sleep Stage	Insomniacs			Controls		
	$\bar{x}$			$\bar{x}$		
	21.11	20.32	25.79	17.93	22.79	28.95
	ST0	ST1	ST2	ST0	ST1	ST2
Insom ST0			*			
Insom ST1			*			
Insom ST2	*	*				
Control ST0						*
Control ST1						*
Control ST2				*	*	

Note. Tukey HSD Test for specific sleep stages by group. Asteriks indicate a statistically significant difference at $p < .05$.

Figure 7. Plot of group x sleep stage interaction for C3-A2 theta first-derivative incidence. Arrow indicates significant between group difference at $p < .05$. See also Table 28.

Table 28. Post-hoc Tukey HSD tests for C₃-A₂ theta first-derivative incidence

Sleep Stage		Insomniacs			Controls		
		$\bar{x}$			$\bar{x}$		
		6.60	12.88	22.06	7.27	17.59	29.29
		ST0	ST1	ST2	ST0	ST1	ST2
Insom	ST0		*	*			
Insom	ST1	*		*			
Insom	ST2	*	*				
Control	ST0					*	*
Control	ST1				*		*
Control	ST2				*	*	

Note. Tukey HSD Test for specific sleep stages by group. Asteriks indicate a statistically significant difference at $p < .05$.

A mixed MANOVA performed on C_3 - A_2 integrated amplitude revealed a statistically significant main effect for group, for sleep stage, and a statistically significant interaction. The results using Wilks' criterion were as follows: $F(5, 24) = 3.73$, $p = .012$ for group; $F(10, 19) = 25.18$, $p < .00001$ for sleep stage; $F(10, 19) = 2.76$, $p = .027$ for the interaction. The means and standard deviations for each group and sleep stage are shown in Table 30. Because of the statistically significant multivariate interaction, the univariate F s for the interaction were examined. As is shown in Table 31, the univariate breakdown only showed a difference in the alpha frequency band. Due to this statistically significant interaction the simple effect for group by stage was examined (see Figure 9). The simple effect analysis of stage within the insomniac group for alpha integrated amplitude was statistically significant, $F(2, 28) = 14.65$, $p = .0015$ (see Figure 9). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that stage wake significantly differed from stage 1 and stage 2 sleep (see Table 32) indicating that alpha integrated amplitude declines in the insomniac group during the transition from stage wake to stage one and stage two sleep. A similar analysis of stage within the control group for alpha integrated amplitude was not significant, $F(2, 28) = .3.30$, $p = .053$ (see Figure 9). The simple effect analysis of group within stage wake was significant, $F(1, 28) = 13.65$, $p = .0009$, revealing significantly greater alpha amplitude within the insomniac group than in the control group during stage wake (see Figure 9). The analysis of group within stage one sleep was significant, $F(1, 28) = 8.72$, $p = .006$, indicating greater alpha amplitude within the insomniac group during stage one sleep (see Figure 9). A similar analysis of group within stage two sleep failed to reach statistical significance, $F(1, 28) = 3.36$, $p = .077$, indicating no group differences in alpha integrated amplitude during stage two sleep (see Figure 9).

Figure 8. Plot of group x sleep stage interaction for C3-A2 delta first-derivative incidence. See also Table 29.

Table 29. Post-hoc Tukey HSD tests for C₃-A₂ delta first-derivative incidence

Sleep Stage	Insomniacs			Controls		
	$\bar{x}$			$\bar{x}$		
	0.63 ST0	1.24 ST1	2.15 ST2	0.29 ST0	1.43 ST1	3.64 ST2
Insom ST0			*			
Insom ST1			*			
Insom ST2	*	*				
Control ST0						*
Control ST1						*
Control ST2				*	*	

Note. Tukey HSD Test for specific sleep stages by group. Asteriks indicate a statistically significant difference at $p < .05$.

Table 30. Means and standard deviations for C_3-A_2 integrated amplitude

GROUP	STAGE	Delta	Theta	Alpha	Sigma	Beta
Insomniacs	0	278.88 (524.36)	333.30 (181.96)	506.34 (293.69)	148.06 (83.89)	159.31 (109.28)
Insomniacs	1	479.23 (302.27)	480.34 (165.09)	247.51 (107.50)	94.64 (40.20)	101.51 (88.50)
Insomniacs	2	2176.89 (748.35)	911.35 (271.28)	245.49 (81.16)	111.92 (46.45)	53.12 (35.13)
Controls	0	318.51 (289.61)	213.72 (109.69)	196.67 (138.19)	101.40 (61.33)	139.06 (114.19)
Controls	1	705.65 (432.06)	448.66 (158.72)	149.42 (70.62)	57.05 (22.34)	55.65 (31.97)
Controls	2	2482.02 (705.62)	849.36 (299.30)	189.68 (85.50)	96.44 (49.64)	35.96 (14.29)

Note. Standard deviation in parentheses.

Table 31. Univariate F ratios for group by sleep stage interaction for C_3-A_2 integrated amplitude

EEG Frequency	F	p
Delta	0.69	.505
Theta	0.67	.513
Alpha	10.67	.0001
Sigma	1.22	.301
Beta	0.40	.669

Note. Analyses are based on 2 and 56 degrees of freedom.

Figure 9. Plot of group x sleep stage interaction for C3-A2 alpha integrated amplitude. Arrows indicate significant between group differences at $p < .05$. See also Table 32.

Table 32. Post-hoc Tukey HSD tests for C₃-A₂ alpha integrated amplitude

Sleep Stage		Insomniacs			Controls		
		$\bar{x}$			$\bar{x}$		
		506.34	247.51	245.49	196.67	149.42	189.68
		ST0	ST1	ST2	ST0	ST1	ST2
Insom	ST0		*	*			
Insom	ST1	*					
Insom	ST2	*					
Control	ST0						
Control	ST1						
Control	ST2						

Note. Tukey HSD Test for specific sleep stages by group. Asteriks indicate a statistically significant difference at $p < .05$.

O₁-A₂ Period-Amplitude Measures by Sleep Stage

The O₁-A₂ full-wave (FW) incidence mixed MANOVA for night one revealed a statistically significant main effect for sleep stage, but neither a statistically significant main effect for group nor a significant interaction. The results using Wilks' criterion were as follows: $F(5, 24) = 2.18$, $p = .09$ for group; $F(10, 19) = 52.58$, $p < .00001$ for sleep stage; $F(10, 19) = 1.57$, $p = .19$ for the interaction. The means and standard deviations for each group and sleep stage are shown in Table 33. The univariate breakdown for the significant sleep stage main effect (within subject measure) indicated that all EEG bands reached statistical significance (see Table 34). Statistically significant increases in both theta and delta O₁-A₂ full-wave incidence were observed during the transition from stage wake through stage one sleep to stage 2 sleep. All remaining O₁-A₂ frequencies showed a significant decrease in full-wave incidence from stage wake to stage 2 sleep (see Figure 10).

Table 33. Means and standard deviations for O₁-A₂ full-wave incidence

GROUP	STAGE	Delta	Theta	Alpha	Sigma	Beta
Insomniacs	0	1.85 (2.85)	13.97 (8.13)	42.69 (16.34)	13.78 (3.19)	19.80 (7.38)
Insomniacs	1	6.04 (4.58)	30.54 (6.32)	28.06 (6.18)	13.13 (2.67)	15.49 (4.74)
Insomniacs	2	21.33 (7.89)	44.06 (5.90)	17.81 (2.92)	7.54 (2.25)	6.30 (3.98)
Controls	0	1.93 (1.73)	12.81 (7.34)	28.77 (9.01)	16.86 (3.67)	26.28 (6.79)
Controls	1	7.74 (5.32)	31.57 (8.90)	24.18 (3.97)	13.41 (2.89)	15.87 (6.06)
Controls	2	24.57 (8.77)	44.80 (3.71)	16.60 (3.71)	7.09 (2.32)	4.87 (2.53)

Note. Standard deviation in parentheses.

Table 34. Mean O₁-A₂ full-wave incidence collapsed across groups and univariate *F* ratios for each EEG frequency within sleep stages

Frequency	Stage 0	Stage 1	Stage 2	<i>F</i>	<i>p</i>
Delta	<u>1.89</u>	<u>6.89</u>	<u>22.95</u>	139.69	.00001
Theta	<u>13.39</u>	<u>31.05</u>	<u>44.43</u>	252.23	.00001
Alpha	<u>35.73</u>	<u>26.12</u>	<u>17.21</u>	47.34	.00001
Sigma	<u>15.32</u>	<u>13.27</u>	<u>7.32</u>	94.76	.00001
Beta	<u>23.04</u>	<u>15.68</u>	<u>5.58</u>	166.06	.00001

Note. Significance levels are based on Huynh-Feldt epsilon adjustments. Univariate *F* tests have 2 and 56 degrees of freedom prior to epsilon adjustments. Means underlined on the same line are significantly different ($p < .05$) according to Tukey HSD analysis.

Figure 10. Plot of sleep stage main effect for O1-A2 full-wave incidence. Solid lines indicate EEG frequencies which increase in incidence from stage wake to stage two sleep. See also Table 34.

A mixed MANOVA computed on O_1 - A_2 half-wave (HW) incidence revealed a statistically significant main effect for group, $F(2, 24) = 4.35$, $p = .006$, and a main effect for sleep stage as shown by the significant Wilks' lambda, $F(10, 19) = 52.49$, $p = .00001$. A statistically significant interaction was also found, $F(10, 19) = 2.49$, $p = .042$. Because of the statistically significant multivariate interaction, the univariate Fs for the interaction were examined. The means and standard deviations for each group and sleep stage are shown in Table 35. As is shown in Table 36, the univariate breakdown showed statistically significant interactions in the alpha, sigma, and beta frequency bands. Due to these significant interactions the simple effect for group by stage was examined (see Figure 11). The simple effect analysis of stage within the insomniac group for O_1 - A_2 beta half-wave incidence was significant, $F(2, 28) = 39.33$, $p < .00001$ (see Figure 11). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that all pairwise comparisons significantly differed (see Table 37). This indicated that beta half-wave incidence declines in the insomniac group during the transition from stage wake through stage one sleep to stage two sleep. The analysis of stage within the control group for O_1 - A_2 beta half-wave incidence was significant, $F(2, 28) = 184.09$, $p < .00001$ (see Figure 11), and post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that all pairwise comparisons significantly differed (see Table 37). This indicated that beta half-wave incidence declines in the insomniac group during the transition from stage wake through stage one sleep to stage two sleep. The simple effect analysis of group within stage wake was significant, $F(1, 28) = 7.08$, $p = .01$, revealing significantly greater beta half-wave incidence within the control group than within the insomniac group during stage wake (see Figure 11). A similar analysis of group within stage one sleep was not significant, $F(1, 28) = .034$, $p = .86$, indicating no group differences in beta half-wave incidence during stage one sleep (see Figure 11). The analysis of group within stage two sleep was not significant, $F(1, 28) = 1.72$, $p = .20$,

indicating no group differences in beta half-wave incidence during stage two sleep (see Figure 11).

The simple effect analysis of stage within the insomniac group for O_1 - A_2 sigma half-wave incidence was statistically significant, $F(2, 28) = 27.81$, $p < .00001$ (see Figure 12). Post-hoc multiple comparisons using Tukey's HSD at the .01 alpha level revealed that stage 2 sleep differed from the other sleep stages (see Table 38). This indicated that sigma half-wave incidence declines in the insomniac group during the transition from stage wake through stage one sleep to stage two sleep. A similar analysis of stage within the control group for sigma half-wave incidence was significant, $F(2, 28) = 105.94$, $p < .00001$ (see Figure 12). Post-hoc multiple comparisons using Tukey's HSD at the .01 alpha level revealed that all pairwise comparisons significantly differed (see Table 38), indicating that sigma half-wave incidence declines in the control group during the transition from stage wake through stage one sleep to stage two sleep. The simple effect analysis of group within stage wake was significant, $F(1, 28) = 12.90$, $p = .001$, revealing significantly greater sigma half-wave incidence within the control group than within the insomniac group during stage wake (see Figure 12). A similar analysis of group within stage one sleep was not significant, $F(1, 28) = .51$, $p = .48$, indicating no group differences in sigma half-wave incidence during stage one sleep (see Figure 12). The analysis of group within stage two sleep was not significant, $F(1, 28) = .16$, $p = .70$, indicating no group differences in sigma activity during stage two sleep (see Figure 12).

The simple effect analysis of stage within the insomniac group for O_1 - A_2 alpha half-wave incidence was statistically significant, $F(2, 28) = 38.76$, $p = .00001$ (see Figure 13). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that all pairwise comparisons significantly differed (see Table 39). This indicated that O_1 - A_2 alpha half-wave incidence declines in the insomniac group during the transition from stage wake through stage one sleep to stage two sleep. The analysis of stage within

the control group for O_1 - A_2 alpha half-wave incidence was significant, $F(2, 28) = 31.42$, $p = .00003$ (see Figure 13). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that all pairwise comparisons significantly differed (see Table 39), indicating that O_1 - A_2 alpha half-wave incidence decreases during the transition from stage wake through stage one sleep to stage two sleep. The simple effect analysis of group within stage wake was significant, $F(1, 28) = 7.39$, $p = .001$, revealing significantly greater alpha half-wave incidence within the insomniac group than in the control group during stage wake (see Figure 13). A similar analysis of group within stage one sleep was significant, $F(1, 28) = 4.1$, $p = .05$, indicating greater alpha half-wave incidence within the insomniac group during stage one sleep (see Figure 13). The analysis of group within stage two sleep failed to reach statistical significance, $F(1, 28) = 2.74$, $p = .11$, indicating no group differences in alpha half-wave incidence during stage two sleep (see Figure 13).

Table 35. Means and standard deviations for O₁-A₂ half-wave incidence

GROUP	STAGE	Delta	Theta	Alpha	Sigma	Beta
Insomniacs	0	4.63 (4.90)	16.45 (6.74)	40.36 (15.06)	12.02 (2.15)	16.27 (4.59)
Insomniacs	1	12.57 (5.73)	30.14 (3.28)	23.70 (5.96)	10.27 (1.60)	13.52 (3.11)
Insomniacs	2	29.59 (7.35)	35.79 (3.91)	14.91 (2.05)	7.08 (1.66)	7.42 (2.65)
Controls	0	4.94 (3.67)	15.24 (6.71)	28.12 (8.75)	15.68 (3.31)	20.63 (4.38)
Controls	1	15.65 (7.14)	29.68 (4.80)	19.92 (4.08)	10.74 (1.98)	13.75 (3.62)
Controls	2	33.25 (7.61)	35.88 (2.78)	13.58 (2.33)	6.83 (1.72)	6.40 (1.39)

Note. Standard deviation in parentheses.

Table 36. Univariate F ratios for group by sleep stage interaction for O₁-A₂ half-wave incidence

EEG Frequency	F	p
Delta	0.88	.419
Theta	0.16	.849
Alpha	5.45	.007
Sigma	10.49	.0001
Beta	9.92	.0002

Note. Analyses are based on 2 and 56 degrees of freedom.

Figure 11. Plot of group x sleep stage interaction for O1-A2 beta half-wave incidence. Arrow indicates significant between group difference at $p < .05$. See also Table 37.

Table 37. Post-hoc Tukey HSD tests for O₁-A₂ beta half-wave incidence

Sleep Stage		Insomniacs			Controls		
		$\bar{x}$			$\bar{x}$		
		16.27	13.52	7.41	20.63	13.75	6.40
		ST0	ST1	ST2	ST0	ST1	ST2
Insom	ST0		*	*			
Insom	ST1	*		*			
Insom	ST2	*	*				
Control	ST0					*	*
Control	ST1				*		*
Control	ST2				*	*	

Note. Tukey HSD Test for specific sleep stages by group. Asteriks indicate a statistically significant difference at $p < .05$.

Figure 12. Plot of group x sleep stage interaction for O1-A2 sigma half-wave incidence. Arrow indicates significant between group difference at $p < .01$. See also Table 38.

Table 38. Post-hoc Tukey HSD tests for O₁-A₂ sigma half-wave incidence

Sleep Stage	Insomniacs			Controls		
	$\bar{x}$			$\bar{x}$		
	12.02	10.27	7.07	15.68	10.74	6.83
	ST0	ST1	ST2	ST0	ST1	ST2
Insom ST0			*			
Insom ST1			*			
Insom ST2	*	*				
Control ST0					*	*
Control ST1				*		*
Control ST2				*	*	

Note. Tukey HSD Test for specific sleep stages by group. Asteriks indicate a statistically significant difference at $p < .01$.

Figure 13. Plot of group x sleep stage interaction for O1-A2 alpha half-wave incidence. Arrows indicate significant between group differences at $p < .05$. See also Table 39.

Table 39. Post-hoc Tukey HSD tests for O₁-A₂ alpha half-wave incidence

Sleep Stage		Insomniacs			Controls		
		$\bar{x}$			$\bar{x}$		
		40.35	23.70	14.91	28.12	19.92	13.57
		ST0	ST1	ST2	ST0	ST1	ST2
Insom	ST0		*	*			
Insom	ST1	*		*			
Insom	ST2	*	*				
Control	ST0					*	*
Control	ST1				*		
Control	ST2				*	*	

Note. Tukey HSD Test for specific sleep stages by group. Asteriks indicate a statistically significant difference at $p < .05$.

A mixed MANOVA performed on O_1 - A_2 first-derivative revealed a statistically significant main effect for group, and a statistically significant main effect for sleep stage, but no significant interaction. The results using Wilks' criterion were as follows: $F(5, 24) = 6.87$, $p = .0004$ for group; $F(10, 19) = 38.68$, $p < .00001$ for sleep stage; $F(10, 19) = 1.59$, $p = .18$ for the interaction. The means and standard deviations for each group and sleep stage are shown in Table 40. Interestingly, the univariate breakdown showed no between group differences in any of the EEG frequency bands (see Table 41). This appears to be an instance where the multivariate analysis is more powerful than its univariate counterparts. Concerning the significant sleep stage main effect (within subject measure), the univariate results are shown in Table 42. Statistically significant increases in both theta and delta first-derivative incidence were observed during the transition from stage wake through stage one sleep to stage 2 sleep. A significant decrease in O_1 - A_2 beta first-derivative incidence from stage wake to stage 2 sleep was found (see Table 42).

Table 40. Means and standard deviations for O_1 - A_2 first-derivative incidence

GROUP	STAGE	Delta	Theta	Alpha	Sigma	Beta
Insomniacs	0	1.54 (5.29)	7.65 (9.76)	31.89 (14.12)	14.01 (3.26)	27.55 (8.72)
Insomniacs	1	2.62 (6.99)	16.70 (9.93)	27.00 (9.19)	14.19 (3.15)	26.61 (9.72)
Insomniacs	2	3.76 (3.47)	43.92 (47.14)	27.00 (3.79)	14.30 (2.36)	16.79 (6.46)
Controls	0	0.62 (1.58)	9.08 (8.92)	23.58 (10.81)	16.82 (4.39)	28.30 (7.29)
Controls	1	2.34 (2.09)	22.59 (10.24)	26.76 (4.94)	15.63 (2.21)	23.14 (7.70)
Controls	2	5.06 (3.11)	36.83 (9.81)	26.33 (2.73)	14.21 (3.79)	13.09 (5.27)

Note. Standard deviation in parentheses.

Table 41. Mean O_1 - A_2 first-derivative incidence and univariate F ratios for each EEG frequency between groups

Frequency	Insomniacs	Controls	F	p
Delta	2.64	2.68	0.0005	.982
Theta	22.76	22.83	0.0002	.987
Alpha	23.63	25.56	1.68	.205
Sigma	14.17	15.56	2.84	.103
Beta	23.65	21.51	0.75	.393

Table 42. Mean O_1 - A_2 first-derivative incidence collapsed across groups and univariate F ratios for each EEG frequency within sleep stages

Frequency	Stage 0	Stage 1	Stage 2	F	p
Delta	<u>1.08</u>	<u>2.48</u>	<u>4.41</u>	24.92	.00001
Theta	<u>8.37</u>	<u>19.64</u>	<u>40.38</u>	21.33	.00006
Alpha	27.74	26.88	26.67	0.20	.82044
Sigma	15.41	14.91	14.26	1.18	.31479
Beta	<u>27.92</u>	<u>24.88</u>	<u>14.94</u>	71.53	.00001

Note. Significance levels are based on Huynh-Feldt epsilon adjustments. Univariate F tests have 2 and 56 degrees of freedom prior to epsilon adjustments. Means underlined on the same line are significantly different ($p < .05$) according to Tukey HSD analysis.

A mixed MANOVA computed on O_1 - A_2 integrated amplitude (power) revealed a statistically significant main effect for group, a statistically significant main effect for sleep stage, but no significant interaction. The results using Wilks' criterion were as follows: $F(5, 24) = 3.08$, $p = .027$ for group; $F(10, 19) = 10.94$, $p < .00001$ for sleep stage; $F(10, 19) = 1.61$, $p = .178$ for the interaction. The means and standard deviations for each group and sleep stage are shown in Table 43. The univariate breakdown for the

significant group main effect showed only a difference in the alpha frequency band with the insomniacs showing significantly more O_1 - A_2 alpha integrated amplitude than the controls (see Table 44). With regard to the significant sleep stage main effect (within subject measure), the univariate results are shown in Table 45. Statistically significant increases in both theta and delta integrated amplitude were observed during the transition from stage wake through stage one sleep to stage 2 sleep. All remaining EEG bands showed a statistically significant decrease in O_1 - A_2 integrated amplitude from stage wake through stage 1 sleep to stage 2 sleep (see Table 45).

Table 43. Means and standard deviations for O_1 - A_2 integrated amplitude

GROUP	STAGE	Delta	Theta	Alpha	Sigma	Beta
Insomniacs	0	90.86 (69.73)	246.45 (225.89)	977.21 (766.94)	144.41 (66.57)	150.47 (83.38)
Insomniacs	1	350.43 (335.26)	390.43 (238.29)	314.79 (182.98)	79.35 (32.07)	81.71 (49.46)
Insomniacs	2	1300.99 (721.67)	778.84 (298.12)	216.58 (83.51)	70.54 (31.25)	39.04 (13.27)
Controls	0	177.83 (179.23)	131.55 (72.86)	412.33 (410.52)	149.80 (110.92)	157.19 (152.19)
Controls	1	245.18 (155.59)	299.42 (112.03)	175.81 (122.11)	64.78 (33.87)	51.93 (32.42)
Controls	2	1583.74 (1648.77)	652.03 (242.43)	164.47 (67.40)	60.61 (33.99)	28.25 (13.16)

Note. Standard deviation in parentheses.

Table 44. Mean O_1 - A_2 integrated amplitude and univariate F ratios for each EEG frequency between groups

Frequency	Insomniacs	Controls	F	p
Delta	580.76	668.92	0.32	.574
Theta	471.90	361.00	3.15	.087
Alpha	502.86	250.87	6.78	.014
Sigma	98.10	91.73	0.14	.708
Beta	90.41	79.13	0.40	.531

Table 45. Mean O_1 - A_2 integrated amplitude collapsed across groups and univariate F ratios for each EEG frequency within sleep stages

Frequency	Stage 0	Stage 1	Stage 2	F	p
Delta	<u>134.35</u>	297.80	<u>1442.37</u>	26.14	.00002
Theta	<u>189.00</u>	<u>344.92</u>	<u>715.44</u>	89.79	.00001
Alpha	<u>694.77</u>	245.30	<u>190.53</u>	23.24	.00004
Sigma	<u>147.11</u>	72.07	<u>65.58</u>	29.87	.00001
Beta	<u>153.83</u>	66.83	<u>33.65</u>	23.45	.00003

Note. Significance levels are based on Huynh-Feldt epsilon adjustments. Univariate F tests have 2 and 56 degrees of freedom prior to epsilon adjustments. Means underlined on the same line are significantly different ($p < .05$) according to Tukey HSD analysis.

Hypothesis eight stated that the period-amplitude analyzed EEG of insomniacs would show more beta EEG activity and less alpha EEG activity during wakefulness than that of controls. The results of the analyses performed above can also be used to address this issue. Contrary to expectation, beta activity during wakefulness was found to be significantly higher in control subjects, and alpha activity during stage wake was found to

be significantly greater within the insomniac group than within the control group. Little difference in beta EEG during stage wake was observed between the two groups: only O_1-A_2 beta half-wave incidence was found to be significantly different between the two groups with controls showing more than insomniacs (see above). Concerning alpha activity, C_3-A_2 alpha full-wave incidence, C_3-A_2 alpha integrated amplitude, O_1-A_2 alpha half-wave incidence, and O_1-A_2 alpha amplitude during stage wake were all found to be significantly greater within the insomniac group than within the control group (see above). In addition, a significant main effect was observed for C_3-A_2 alpha half-wave incidence with insomniacs showing more activity than the controls (see above).⁶

⁶These analyses were originally performed on the data from separate nights and the results were virtually identical.

Table 46 Summary table of PAA Sleep Stage Analyses

Insomniacs have more C_3-A_2 beta full-wave than controls during stage 2 sleep.
Insomniacs have more C_3-A_2 alpha full-wave than controls during stage wake.
Controls have more C_3-A_2 delta full-wave than insomniacs during stage one sleep.
Controls have more C_3-A_2 delta full-wave than insomniacs during stage two sleep.
Insomniacs have more C_3-A_2 alpha half-wave than controls during the SOP*.
Controls have more C_3-A_2 delta half-wave than insomniacs during the SOP.
Insomniacs have more C_3-A_2 beta first-derivative than controls during stage two sleep.
Controls have more C_3-A_2 theta first-derivative than insomniacs during stage two sleep.
Insomniacs have more C_3-A_2 alpha amplitude than controls during stage wake.
Insomniacs have more C_3-A_2 alpha amplitude than controls during stage one sleep.
Controls have more O_1-A_2 beta half-wave than insomniacs during stage wake.
Controls have more O_1-A_2 sigma half-wave than insomniacs during stage wake.
Insomniacs show more O_1-A_2 alpha half-wave than controls during stage wake.
Insomniacs have more O_1-A_2 alpha half-wave than controls during stage one sleep.
Insomniacs have more O_1-A_2 alpha amplitude than controls during the SOP.

Note. *SOP = sleep onset period.

Period-Amplitude Measures by Reaction Time

As mentioned earlier, it is accepted practice within the sleep research community to both define sleep as changes in EEG, and to measure sleep by examining EEG alterations; however, important information is obtained by examining brain electrical activity associated with changes in arousal as defined by behavioural response latency. Using this approach, behavioural response latency was used to measure arousal and to examine changes in brain electrical activity during the sleep onset period. Ten seconds of pretone EEG data was sampled prior to each stimulus onset beginning with the last 20 artifact-free responses and ending with the first five consecutive response failures. This

approximated the last 8 - 10 minutes of wakefulness and the first 2 - 2.5 minutes of stage 1 sleep and/or early stage 2 sleep. Typically, 20 responses per subject per night, and five nonresponses per subject per night were obtained to produce a total of 1,264 10-second samples of EEG data. The samples were rank-ordered within subject, averaged, and then placed within five response bins where RT bin 1 contained the fastest responses and RT bin 5 contained only response failures (see Ogilvie, 1991). To make the results comparable to those of Ogilvie et al. (1991), the data were collapsed across the two nights. For the insomniac group, an average of 32.53 responses per subject were obtained for bins 1 - 4, and an average of 12.3 non-responses for bin 5. For the control group, an average of 28.0 responses were obtained for bins 1 - 4, and an average of 10.8 non-responses for bin 5. The data were analyzed using mixed MANOVAs with response latency (bins 1-5) as the within-subjects IV and group as the between-subjects IV. The C_3-A_2 and O_1-A_2 EEG data were saved to an ASCII text file and subsequently subjected to a period-amplitude analysis algorithm written in turbo pascal (see Appendix N). Consequently, the analyses reported below, while based on the same PAA algorithm (Hoffmann et al., 1979) to that reported above for the sleep stage analyses, should be considered to represent relative EEG values rather than absolute EEG values.

The following analyses were computed to specifically examine the hypothesis that chronic psychophysiological insomniacs will show less EEG synchronization than controls - specifically less delta amplitude and incidence, less theta amplitude and incidence, less alpha amplitude and incidence, and more beta EEG amplitude and incidence - as a result of heightened arousal during the sleep onset period (*hypothesis #9*). Another purpose of this analysis is to replicate the finding reported by Ogilvie et al. (1991) of a significant increase in FFT power across all EEG frequencies during sleep onset (*hypothesis #10*) or the transition from slow responding (i.e., slow reaction time or RT) to response cessation (i.e., behavioural sleep). The present study, however, used both

normal and insomniac sleepers and period-amplitude analysis (PAA), a time domain approach, to avoid confounding amplitude and incidence as is the case when using the FFT method. Recall that recent research has reported significant multivariate overlap in shared variance between amplitude analysis and FFT power measures and has indicated that the two signal transformation techniques are generally comparable (Armitage et al., 1995).

The C_3-A_2 full-wave incidence mixed MANOVA revealed a statistically significant main effect for group, a significant main effect for reaction time, and a significant interaction. The results using Wilks' criterion were as follows: $F(5, 24) = 3.21, p = .023$ for group; $F(20, 9) = 13.62, p = .0002$ for reaction time bins; $F(20, 9) = 2.98, p = .047$ for the interaction. Because of the significant multivariate interaction, the univariate Fs for the interaction were examined. The means and standard deviations for each group and RT Bin are shown in Table 47. As is shown in Table 48, the univariate breakdown showed a significant interaction in the alpha frequency band. Due to this significant interaction the simple effect for group by RT Bin was examined (see Figure 14). The simple effect analysis of RT Bin within the insomniac group for C_3-A_2 alpha full-wave incidence was significant, $F(4, 56) = 16.06, p < .00001$ (see Figure 14). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that RT Bin 5 was significantly different from all other RT Bins (see Table 49). This indicated that within the insomniac group C_3-A_2 alpha full-wave incidence takes a dramatic drop at behavioural sleep (RT Bin 5). The simple effect analysis of RT Bin within the control group for C_3-A_2 alpha full-wave incidence was significant, $F(4, 56) = 24.96, p < .00001$ (see Figure 14). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that RT Bin 5 was significantly different from all other RT Bins (see Table 49). In addition, RT Bin 1 was significantly different from RT Bins 3 and 4; RT Bin 2 was significantly different from RT Bin 4. This indicated that, within the control group, C_3-A_2

alpha full-wave incidence decreases from RT Bin 1 to RT Bin 5. The simple effect analysis of group within RT Bin 1 was significant, $F(1, 28) = 7.08$, $p = .013$, revealing significantly greater C_3 - A_2 alpha full-wave incidence within the insomniac group than in the control group during RT Bin 1 (see Figure 14). A similar analysis of group within RT Bin 2 was significant, $F(1, 28) = 10.66$, $p = .003$, revealing significantly greater C_3 - A_2 alpha full-wave incidence within the insomniac group than in the control group during RT Bin 2 (see Figure 14). The analysis of group within RT Bin 3 was significant, $F(1, 28) = 19.89$, $p = .0001$, revealing significantly greater C_3 - A_2 alpha full-wave incidence within the insomniac group than in the control group during RT Bin 3 (see Figure 14). The analysis of group within RT Bin 4 was significant, $F(1, 28) = 7.46$, $p = .01$, revealing significantly greater C_3 - A_2 alpha full-wave incidence within the insomniac group than in the control group during RT Bin 4 (see Figure 14). The analysis of group within RT Bin 5 was significant, $F(1, 28) = 11.91$, $p = .002$, revealing significantly greater C_3 - A_2 alpha full-wave incidence within the insomniac group than in the control group during RT Bin 5 (see Figure 14).

The C_3 - A_2 half-wave incidence mixed MANOVA revealed a statistically significant main effect for group, a statistically significant main effect for reaction time, and a statistically significant interaction. The results using Wilks' criterion were as follows: $F(5, 24) = 4.84$, $p = .003$ for group; $F(20, 9) = 7.67$, $p = .002$ for reaction time bins; $F(20, 9) = 2.93$, $p = .05$ for the interaction. Because of the statistically significant multivariate interaction, the univariate Fs for the interaction were examined. The means and standard deviations for each group and RT Bin are shown in Table 50. As is shown in Table 51, the univariate breakdown showed a significant interaction in the beta frequency band. Due to this significant interaction the simple effect for group by RT bin was examined (see Figure 15). The simple effect analysis of RT Bin within the insomniac group for C_3 - A_2 beta half-wave incidence was not significant, $F(4, 56) = 1.74$, $p = .174$.

(see Figure 15), indicating no difference in C_3 - A_2 beta half-wave incidence during the transition from wakefulness (RT Bin 1) through increasing drowsiness to behavioural sleep (RT Bin 5). A similar analysis of RT Bin within the control group for C_3 - A_2 beta half-wave incidence was significant, $F(4, 56) = 10.82$, $p < .00003$ (see Figure 15). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed numerous differences between RT Bins (see Table 52) indicating that within the control group C_3 - A_2 beta half-wave incidence decreases from wakefulness (RT Bin 1) through drowsiness to behavioural sleep (RT Bin 5). The simple effect analysis of group within RT Bin 1 did not reach statistical significance, $F(1, 28) = 1.62$, $p = .21$, revealing no group differences in C_3 - A_2 beta half-wave incidence within RT Bin 1 (see Figure 15). A similar analysis of group within RT Bin 2 also did not reach statistical significance, $F(1, 28) = 0.34$, $p = .56$, revealing no group differences in C_3 - A_2 beta half-wave incidence within RT Bin 2 (see Figure 15). The analysis of group within RT Bin 3 was not significant, $F(1, 28) = 0.57$, $p = .46$, indicating no group differences in C_3 - A_2 beta half-wave incidence within RT Bin 3 (see Figure 15). The analysis of group within RT Bin 4 was statistically significant, $F(1, 28) = 10.40$, $p = .003$, revealing significantly greater C_3 - A_2 beta half-wave incidence within the insomniac group than in the control group during RT Bin 4 (see Figure 15). The analysis of group within RT Bin 5 was significant, $F(1, 28) = 4.23$, $p = .05$, revealing significantly greater C_3 - A_2 beta half-wave incidence within the insomniac group than in the control group during RT Bin 5 (see Figure 15).

Table 47. Reaction time means and standard deviations for C₃-A₂ full-wave incidence

Group	RT	Beta	Sigma	Alpha	Theta	Delta
Insomnia	1	21.48 (10.75)	11.36 (2.64)	26.63 (11.63)	24.98 (4.78)	15.53 (10.84)
Insomnia	2	21.92 (12.88)	10.84 (3.43)	28.10 (11.30)	24.01 (5.81)	15.12 (14.79)
Insomnia	3	17.94 (10.50)	9.40 (2.81)	29.38 (11.89)	27.00 (5.02)	16.29 (13.08)
Insomnia	4	17.82 (10.94)	9.00 (2.58)	23.51 (11.54)	28.17 (5.83)	21.49 (14.97)
Insomnia	5	13.67 (7.9)	7.14 (2.59)	12.84 (4.20)	26.96 (8.69)	36.27 (16.18)
Control	1	22.26 (9.53)	10.31 (2.49)	17.88 (5.20)	26.63 (5.35)	19.85 (12.02)
Control	2	18.37 (9.34)	9.38 (2.74)	17.42 (5.74)	27.32 (5.46)	27.51 (14.46)
Control	3	15.21 (8.28)	7.82 (1.69)	14.36 (5.35)	30.39 (5.71)	32.21 (11.91)
Control	4	14.22 (6.35)	7.08 (1.48)	14.03 (5.67)	29.47 (4.57)	35.19 (13.04)
Control	5	11.79 (13.57)	5.55 (5.15)	8.25 (2.98)	26.43 (5.42)	47.89 (16.78)

Note. Standard deviation in parentheses.

Table 48. Univariate F ratios for group by RT Bin interaction for C₃-A₂ full-wave incidence

EEG Frequency	F	p
Delta	1.97	.10
Theta	1.13	.34
Alpha	4.19	.003
Sigma	0.14	.97
Beta	0.49	.74

Note. Analyses are based on 4 and 112 degrees of freedom.

Figure 14. Plot of group $\times$ RT Bin interaction for C3-A2 alpha full-wave incidence. Arrows indicate significant between group differences at $p < .05$. See also Table 49.

Table 49. Post-hoc Tukey HSD tests for C₃-A₂ alpha full-wave incidence

RT Bin	Insomniacs					Controls				
	$\bar{x}$					$\bar{x}$				
	26.63	28.10	29.37	23.51	12.84	17.87	17.41	14.36	14.03	8.25
	RT1	RT2	RT3	RT4	RT5	RT1	RT2	RT3	RT4	RT5
Ins 1					*					
Ins 2					*					
Ins 3					*					
Ins 4					*					
Ins 5	*	*	*	*						
Ctl 1										*
Ctl 2										*
Ctl 3										*
Ctl 4										*
Ctl 5						*	*	*	*	

Note. Tukey HSD Test for RT Bin by group. Asteriks indicate a statistically significant difference at $p < .05$.

Table 50. Reaction time means and standard deviations for C₃-A₂ half-wave incidence

Group	RT	Beta	Sigma	Alpha	Theta	Delta
Insomnia	1	16.80 (7.35)	9.76 (7.51)	19.81 (6.20)	29.77 (5.09)	23.89 (12.27)
Insomnia	2	18.97 (8.07)	7.76 (2.41)	21.54 (7.14)	29.19 (4.62)	22.54 (15.45)
Insomnia	3	16.47 (7.33)	7.08 (2.25)	20.53 (7.93)	30.18 (4.91)	25.73 (13.78)
Insomnia	4	16.43 (7.60)	6.29 (2.21)	16.77 (6.46)	29.69 (5.29)	30.82 (15.39)
Insomnia	5	14.02 (7.89)	8.00 (8.21)	12.96 (6.70)	22.56 (6.81)	40.57 (11.34)
Control	1	20.17 (7.15)	9.05 (8.27)	15.06 (4.19)	25.79 (4.64)	29.93 (12.72)
Control	2	17.42 (6.40)	6.00 (1.65)	13.29 (3.80)	26.20 (4.18)	37.10 (13.21)
Control	3	14.64 (5.94)	5.43 (1.51)	11.55 (2.81)	25.21 (5.23)	43.17 (11.24)
Control	4	13.58 (4.89)	4.85 (0.88)	10.80 (3.10)	24.31 (5.01)	46.46 (11.15)
Control	5	8.52 (6.70)	5.03 (7.45)	9.10 (7.14)	18.45 (5.05)	58.38 (13.82)

Note. Standard deviation in parentheses.

Table 51. Group by reaction time interaction for C₃-A₂ half-wave incidence and univariate F ratios for each EEG frequency within RT Bins

Frequency	F	p
Delta	1.64	.17
Theta	0.54	.71
Alpha	1.50	.21
Sigma	0.18	.94
Beta	2.92	.03

Note. Analyses are based on 4 and 112 degrees of freedom.

Figure 15. Plot of group $\times$ RT Bin interaction for C3-A2 beta half-wave incidence. Arrows indicate significant between group differences at $p < .05$. See also Table 52.

Table 52. Post-hoc Tukey HSD tests for C_3 - A_2 beta half-wave incidence

RT Bin	Insomniacs					Controls				
	$\bar{x}$					$\bar{x}$				
	16.80	18.97	16.48	16.42	14.02	20.17	17.41	14.64	13.58	8.52
	RT1	RT2	RT3	RT4	RT5	RT1	RT2	RT3	RT4	RT5
Ins 1										
Ins 2										
Ins 3										
Ins 4										
Ins 5										
Ctl 1								*	*	*
Ctl 2										*
Ctl 3						*				*
Ctl 4						*				
Ctl 5						*	*	*		

Note. Tukey HSD Test for RT Bin by group. Asteriks indicate a statistically significant difference at $p < .05$.

The C_3 - A_2 first-derivative incidence mixed MANOVA revealed a statistically significant main effect for reaction time, but no significant main effect for group, and no significant interaction. The results using Wilks' criterion were as follows: $F(20, 9) = 7.08$, $p = .002$ for reaction time bins; $F(5, 24) = 0.62$, $p = .68$ for group; $F(20, 9) = 1.74$, $p = .20$ for the interaction. The means and standard deviations for each group and RT Bin are shown in Table 53. The univariate breakdown for the significant RT Bin main effect revealed significant differences across RT Bins for the beta, theta, and delta frequency bands (see Figure 16 and Table 54). There is a dramatic decrease in beta C_3 - A_2 first-derivative incidence, and an increase in theta and delta C_3 - A_2 first-derivative incidence at behavioural sleep (RT Bin 5).

A problem with multicollinearity (variables are highly correlated showing correlations of 0.90 and above) and singularity (variables are perfectly correlated prohibiting matrix inversion) was revealed with C_3 - A_2 amplitude measures. Examination of univariate correlations indicated 50 cases of multicollinearity. However, deleting the offending variables, as suggested by Tabachnick & Fidell (1989), was not considered an option so five univariate split-plot ANOVAs were used in the place of a mixed MANOVA. Consequently, the critical alpha level was set at .01 to compensate for the increased number of ANOVAs.

Mixed ANOVAs were computed on C_3 - A_2 integrated amplitude measures obtained from five EEG frequencies. Group was used as the between independent variable, reaction time bin (RT) was used as the within-subjects (repeated) independent variable, and the dependent variables were beta, sigma, alpha, theta, and delta C_3 - A_2 integrated amplitude. Means and standard deviations are presented in Table 55 and ANOVA results are shown in Table 56. Note that there were neither significant group main effects nor significant within-subjects main effects when using an alpha critical level of .01; however, there is a significant interaction for the alpha band so the simple

effect for this frequency band will be interpreted (see Table 56).

The simple effect analysis of RT Bin within the insomniac group for C_3 - A_2 alpha integrated amplitude was significant, $F(4, 56) = 5.80$, $p = .009$ (see Figure 17), indicating that C_3 - A_2 alpha integrated amplitude changed during the transition from wakefulness (RT Bin 1) through increasing drowsiness to behavioural sleep (RT Bin 5). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that RT Bin 1 was significantly different from RT Bins 2, 3, and 4. Furthermore, RT Bins 3 and 4 were significantly different from RT Bin 5 (see Table 57). A similar analysis of RT Bin within the control group for C_3 - A_2 alpha integrated amplitude was not statistically significant when using Huynh-Feldt epsilon adjustments, $F(4, 56) = 3.27$, $p = .091$ (see Figure 17). The simple effect analysis of group within RT Bin 1 was not significant, $F(1, 28) = 2.63$, $p = .12$, revealing no group differences in C_3 - A_2 alpha integrated amplitude within RT Bin 1 (see Figure 17). The analysis of group within RT Bin 2 failed to reach statistical significance when using the more stringent critical alpha level of .01, $F(1, 28) = 5.07$, $p = .03$, revealing no group differences in C_3 - A_2 alpha integrated amplitude within RT Bin 2 (see Figure 17). A similar analysis of group within RT Bin 3 was significant, $F(1, 28) = 9.54$, $p = .004$, indicating significantly more C_3 - A_2 alpha integrated amplitude within the insomniac group than within the control group during RT Bin 3 (see Figure 17). The analysis of group within RT Bin 4 was significant, $F(1, 28) = 9.04$, $p = .005$, indicating significantly more C_3 - A_2 alpha integrated amplitude within the insomniac group than within the control group during RT Bin 4 (see Figure 17). The analysis of group within RT Bin 5 was not significant, $F(1, 28) = 2.53$, $p = .12$, revealing no group differences in C_3 - A_2 alpha integrated amplitude within RT Bin 5 (see Figure 17).

Table 53. Reaction time means and standard deviations for C₃-A₂ first-derivative incidence

Group	RT	Beta	Sigma	Alpha	Theta	Delta
Insomnia	1	69.58 (19.35)	13.29 (11.79)	10.66 (6.12)	5.31 (4.92)	1.13 (2.57)
Insomnia	2	69.60 (21.30)	9.20 (5.20)	9.47 (6.73)	7.96 (6.77)	3.77 (6.07)
Insomnia	3	67.24 (21.95)	10.49 (5.87)	10.04 (6.61)	8.11 (7.88)	4.13 (6.92)
Insomnia	4	69.55 (20.65)	9.51 (5.16)	10.59 (7.00)	8.04 (7.20)	2.31 (3.36)
Insomnia	5	61.87 (20.44)	10.48 (4.44)	11.33 (6.04)	11.18 (7.10)	5.14 (7.56)
Control	1	69.23 (24.15)	11.38 (6.55)	11.84 (14.08)	5.52 (6.14)	1.30 (3.12)
Control	2	67.75 (19.36)	9.48 (2.68)	8.97 (3.69)	8.23 (7.22)	5.57 (11.71)
Control	3	63.07 (17.20)	10.69 (1.97)	11.10 (4.25)	9.90 (7.85)	5.23 (7.91)
Control	4	61.94 (19.71)	10.05 (2.88)	9.80 (4.00)	10.34 (8.51)	7.88 (10.92)
Control	5	41.93 (21.43)	10.80 (3.21)	15.79 (11.88)	18.30 (11.23)	13.24 (10.10)

Note. Standard deviation in parentheses.

Figure 16. Plot of RT Bin main effect for C3-A2 first-derivative incidence. See also Table 54.

Table 54. Mean reaction time C₃-A₁, first-derivative incidence and univariate F ratios for each EEG frequency within RT Bins

Frequency	RT1	RT2	RT3	RT4	RT5	F	p
Delta	<u>1.22</u>	<u>4.67</u>	<u>4.68</u>	<u>5.09</u>	<u>9.19</u>	8.22	.00009
Theta	<u>5.41</u>	<u>8.10</u>	<u>9.00</u>	<u>9.19</u>	<u>14.74</u>	15.83	.00001
Alpha	11.25	9.22	10.57	10.19	13.56	1.99	.14446
Sigma	12.33	9.34	10.59	9.78	10.64	1.78	.14756
Beta	<u>69.40</u>	68.67	65.15	65.74	<u>51.90</u>	9.05	.00034

Note. Significance levels are based on Huynh-Feldt epsilon adjustments. Univariate *F* tests have 4 and 112 degrees of freedom prior to epsilon adjustments. Means underlined on the same line are significantly different ($p < .01$) according to Tukey HSD analysis.

Table 55. Reaction time means and standard deviations for C₃-A₂ integrated amplitude

Group	RT	Beta	Sigma	Alpha	Theta	Delta
Insomnia	1	126496.98 (281882.79)	76794.53 (83959.50)	130952.32 (123564.79)	429790.29 (330163.30)	1049193.02 (512635.16)
Insomnia	2	117611.25 (187588.45)	142824.15 (190086.17)	340394.30 (359992.32)	758203.82 (841879.92)	1310335.58 (1501883.09)
Insomnia	3	115716.15 (175741.93)	134595.17 (167257.77)	372910.49 (329053.61)	696000.09 (677914.95)	1153705.52 (1162423.92)
Insomnia	4	127371.30 (196225.29)	133217.40 (171424.02)	385345.60 (367059.92)	651756.10 (686430.95)	931905.38 (938187.62)
Insomnia	5	88668.51 (165889.24)	99636.34 (160531.61)	229045.56 (322503.36)	510358.83 (673040.86)	817847.28 (1124054.11)
Control	1	96278.35 (169257.26)	127455.19 (188303.02)	285830.23 (348593.76)	424590.96 (845613.86)	1381670.77 (1610546.59)
Control	2	100937.27 (197454.36)	66828.56 (67991.96)	120032.59 (118995.46)	340480.43 (275211.37)	798173.44 (512832.50)
Control	3	86436.27 (163439.57)	55736.40 (56978.18)	101691.18 (85678.06)	306587.84 (248718.15)	687441.13 (521822.99)
Control	4	81831.28 (146115.84)	50787.46 (49350.58)	94261.54 (76565.00)	289353.69 (249858.95)	582354.59 (404583.54)
Control	5	86484.04 (174713.70)	52511.70 (59045.17)	92234.66 (83224.44)	318032.96 (234484.16)	698064.99 (383617.96)

Note. Standard deviation in parentheses.

Table 56. Univariate F ratios and probabilities for C₃-A₂ integrated amplitude

Frequency	Group	p	RT	p	Interaction	p
Delta	0.67	.42	2.43	.052	1.90	.115
Theta	2.94	.097	1.14	.339	2.83	.027
Alpha	4.40	.045	1.05	.387	7.99	.001*
Sigma	1.49	.233	0.47	.759	2.90	.025
Beta	0.22	.641	0.14	.967	0.10	.980

Note. Degrees of freedom are 1, 28 for between group analyses; degrees of freedom for within measures are 4, 112. Critical alpha level set at .01.

Figure 17. Plot of group x RT bin interaction for C3-A2 alpha integrated amplitude. Arrows indicate significant between group differences at $p < .01$. See also Table 57.

Table 57. Post-hoc Tukey HSD tests for C₃-A₂ alpha integrated amplitude

RT Bin	Insomniacs					Controls				
	$\bar{x}$					$\bar{x}$				
	RT1	RT2	RT3	RT4	RT5	RT1	RT2	RT3	RT4	RT5
Ins 1		*	*	*						
Ins 2	*									
Ins 3	*				*					
Ins 4	*				*					
Ins 5			*	*						
Ctl 1										
Ctl 2										
Ctl 3										
Ctl 4										
Ctl 5										

Note. Tukey HSD Test for RT Bin by group. Asteriks indicate a statistically significant difference at $p < .01$.

O₁-A₂ Period-Amplitude Measures by Reaction Time

The O₁-A₂ full-wave incidence mixed MANOVA revealed a statistically significant main effect for group, and a significant main effect for reaction time, but no significant interaction. The results using Wilks' criterion were as follows: $F(5, 24) = 2.90, p = .034$ for group; $F(20, 9) = 18.19, p = .00005$ for reaction time bins; $F(20, 9) = 1.33, p = .34$ for the interaction. The means and standard deviations for each group and RT Bin are shown in Table 58. The univariate breakdowns for the group main effect revealed significant differences in the alpha and delta EEG bands (see Table 59). The insomniacs showed significantly more alpha EEG than the controls, but significantly less delta EEG than the controls. Concerning the reaction time main effect, all of the univariate analyses were statistically significant (see Table 60). Not surprisingly, there is a significant decrease in beta, sigma, and alpha EEG from the fastest bin (RT Bin 1) to behavioural sleep (RT Bin 5), and a significant increase in theta and delta EEG from the fastest bin to behavioural sleep (see Figure 18).

Table 58. Reaction time means and standard deviations for O₁-A₂ full-wave incidence

Group	RT	Beta	Sigma	Alpha	Theta	Delta
Insomnia	1	17.64 (9.35)	10.34 (2.67)	33.73 (15.08)	24.77 (6.96)	13.52 (9.68)
Insomnia	2	16.68 (9.39)	9.62 (3.73)	36.50 (16.87)	22.37 (5.06)	14.82 (13.78)
Insomnia	3	15.70 (9.17)	9.88 (3.13)	32.95 (15.45)	24.99 (6.40)	16.49 (14.32)
Insomnia	4	14.05 (8.25)	8.81 (2.96)	30.41 (13.51)	27.61 (6.30)	19.12 (10.92)
Insomnia	5	10.66 (5.96)	6.50 (2.02)	14.05 (4.15)	32.62 (6.88)	36.17 (13.75)
Control	1	22.32 (10.10)	13.66 (3.46)	26.16 (10.14)	23.76 (6.72)	14.64 (9.32)
Control	2	17.54 (7.81)	11.17 (4.34)	24.12 (10.76)	26.23 (5.52)	20.93 (12.14)
Control	3	13.92 (6.21)	9.55 (3.10)	20.19 (10.29)	28.79 (3.71)	27.54 (12.92)
Control	4	13.32 (6.37)	8.48 (2.91)	17.88 (7.92)	30.13 (3.26)	30.19 (11.40)
Control	5	6.88 (3.87)	4.90 (2.19)	11.87 (7.90)	29.85 (4.67)	46.74 (13.03)

Note. Standard deviation in parentheses.

Table 59. Mean O₁-A₂ full-wave incidence and univariate F ratios for each EEG frequency between groups

Frequency	Insomniacs	Controls	F	p
Delta	20.02	28.01	4.36	.046
Theta	26.47	27.75	0.72	.40
Alpha	29.53	20.04	7.18	.01
Sigma	9.03	9.55	0.31	.58
Beta	14.94	14.80	0.003	.95

Table 60. Mean O₁-A₂ full-wave incidence and univariate F ratios for each EEG frequency within RT Bins

Frequency	RT1	RT2	RT3	RT4	RT5	F	p
Delta	<u>14.08</u>	17.87	<u>22.01</u>	<u>24.56</u>	<u>41.45</u>	66.11	.00001
Theta	<u>24.26</u>	<u>24.30</u>	<u>26.89</u>	<u>28.87</u>	<u>31.23</u>	14.21	.00001
Alpha	<u>29.94</u>	<u>31.31</u>	<u>26.56</u>	<u>24.14</u>	<u>12.96</u>	26.14	.00001
Sigma	<u>12.00</u>	<u>10.39</u>	<u>9.71</u>	<u>8.64</u>	<u>5.70</u>	42.81	.00001
Beta	<u>19.98</u>	<u>17.11</u>	<u>14.81</u>	<u>13.68</u>	<u>8.77</u>	20.30	.00001

Note. Significance levels are based on Huynh-Feldt epsilon adjustments. Univariate *F* tests have 4 and 112 degrees of freedom prior to epsilon adjustments. Means underlined on the same line are significantly different ($p < .05$) according to Tukey HSD analysis.

Figure 18. Plot of O1-A2 full-wave incidence within RT bin. Frequencies represented by dashed lines show a significant decrease from RT Bin 1 to RT Bin 5.

The O_1 - A_2 half-wave incidence mixed MANOVA revealed a statistically significant main effect for reaction time, but neither a significant main effect for group nor a significant interaction. The results using Wilks' criterion were as follows: $F(20, 9) = 7.78$, $p = .002$ for reaction time bins; $F(5, 24) = 1.65$, $p = .18$ for group; $F(20, 9) = 1.79$, $p = .18$ for the interaction. The means and standard deviations for each group and RT Bin are shown in Table 61. With the exception of sigma EEG, all of the univariate breakdowns for the RT main effect were significant (see Table 62). There is a significant decrease in beta, alpha, and theta O_1 - A_2 half-wave incidence from the fastest bin to behavioural sleep, and a significant increase in delta O_1 - A_2 half-wave incidence from bin 1 to bin 5 (see Figure 19).

Table 61. Reaction time means and standard deviations for O_1 - A_2 half-wave incidence

Group	RT	Beta	Sigma	Alpha	Theta	Delta
Insomnia	1	20.68 (14.35)	8.24 (2.72)	25.29 (10.96)	29.90 (5.11)	15.89 (9.20)
Insomnia	2	15.35 (7.38)	8.06 (2.54)	27.54 (10.73)	29.01 (5.88)	20.03 (14.57)
Insomnia	3	14.38 (7.17)	7.77 (2.79)	25.14 (10.70)	29.58 (5.18)	23.13 (15.30)
Insomnia	4	13.66 (7.01)	6.61 (1.89)	22.46 (9.51)	29.71 (3.35)	27.57 (12.30)
Insomnia	5	13.13 (12.08)	10.37 (15.46)	11.39 (2.77)	23.58 (10.69)	37.68 (15.80)
Control	1	21.23 (8.91)	8.94 (2.58)	23.55 (8.78)	26.27 (4.05)	20.34 (13.22)
Control	2	15.51 (5.66)	7.71 (2.36)	20.53 (10.06)	26.58 (3.65)	29.67 (14.94)
Control	3	13.14 (4.66)	6.60 (2.16)	16.77 (7.88)	27.08 (3.34)	36.41 (13.36)
Control	4	12.31 (4.92)	5.79 (1.83)	15.60 (6.70)	26.01 (3.48)	40.28 (12.39)
Control	5	11.42 (15.20)	10.18 (17.00)	8.65 (3.76)	20.14 (8.71)	46.09 (21.39)

Note. Standard deviation in parentheses.

Table 62. Mean reaction time O₁-A₂ half-wave incidence and univariate F ratios for each EEG frequency within RT Bins

Frequency	RT1	RT2	RT3	RT4	RT5	F	p
Delta	<u>18.11</u>	<u>24.85</u>	<u>29.77</u>	<u>33.92</u>	<u>41.88</u>	16.24	.00001
Theta	<u>28.08</u>	<u>27.79</u>	<u>28.32</u>	<u>27.85</u>	<u>21.86</u>	10.32	.00016
Alpha	<u>24.42</u>	<u>24.03</u>	<u>20.95</u>	<u>19.03</u>	<u>10.02</u>	37.13	.00001
Sigma	8.59	7.89	7.19	6.20	10.27	1.16	.28941
Beta	<u>20.95</u>	15.43	<u>13.76</u>	<u>12.98</u>	<u>12.28</u>	5.24	.00760

Note. Significance levels are based on Huynh-Feldt epsilon adjustments. Univariate *F* tests have 4 and 112 degrees of freedom prior to epsilon adjustments. Means underlined on the same line are significantly different ($p < .01$) according to Tukey HSD analysis.

Figure 19. Plot of O1-A2 half-wave incidence within RT bin. Frequencies represented by dashed lines show a significant decrease from RT Bin 1 to RT Bin 5.

The O_1 - A_2 first-derivative incidence mixed MANOVA revealed a statistically significant main effect for reaction time, and a significant interaction, but no significant main effect for group. The results using Wilks' criterion were as follows: $F(5, 24) = 2.21$, $p = .09$ for group; $F(20, 9) = 4.04$, $p = .02$ for reaction time bins; $F(20, 9) = 5.41$, $p = .006$ for the interaction. The means and standard deviations for each group and RT Bin are shown in Table 63. Interestingly, the univariate breakdown for the interaction did not show any statistically significant differences in any of the EEG frequencies (see Table 64). This appears to be another instance where the multivariate approach is more powerful than its univariate counterparts.

Table 63. Reaction time means and standard deviations for O_1 - A_2 first-derivative incidence

Group	RT	Beta	Sigma	Alpha	Theta	Delta
Insomnia	1	61.23 (19.15)	10.77 (3.67)	12.15 (5.31)	12.99 (16.79)	2.83 (4.59)
Insomnia	2	60.38 (20.33)	9.53 (3.53)	12.53 (6.34)	11.16 (7.59)	6.39 (8.63)
Insomnia	3	57.58 (23.92)	10.80 (3.78)	16.58 (15.36)	9.39 (7.19)	5.65 (8.33)
Insomnia	4	59.14 (18.09)	11.92 (2.88)	13.57 (5.52)	11.07 (6.93)	4.29 (6.79)
Insomnia	5	46.54 (18.32)	13.37 (5.86)	14.83 (4.27)	18.08 (8.24)	6.93 (6.99)
Control	1	71.71 (12.76)	11.08 (3.55)	8.62 (3.04)	8.05 (12.47)	0.54 (0.89)
Control	2	68.97 (7.86)	12.02 (3.02)	11.30 (2.75)	6.72 (3.27)	1.00 (2.03)
Control	3	59.63 (15.22)	12.31 (2.44)	16.57 (14.55)	9.09 (5.09)	2.31 (3.06)
Control	4	59.90 (9.07)	12.34 (3.54)	12.89 (3.39)	11.22 (4.24)	3.65 (3.59)
Control	5	54.16 (19.97)	13.81 (9.33)	13.09 (3.59)	13.39 (8.88)	5.56 (6.56)

Note. Standard deviation in parentheses.

Table 64. Group by reaction time interaction for O_1 - A_2 first-derivative incidence and univariate F ratios for each EEG frequency within RT Bins

Frequency	F	p
Delta	0.97	.42
Theta	0.61	.66
Alpha	0.25	.91
Sigma	0.36	.83
Beta	0.57	.69

Note. Analyses are based on 4 and 112 degrees of freedom.

A multicollinearity and singularity problem was also revealed with O_1 - A_2 amplitude measures. An examination of the bivariate correlations indicated 26 cases of multicollinearity. Again, deleting the offending variables was not considered an option so five univariate split-plot ANOVAs were used in the place of a mixed MANOVA. The critical alpha level was set at .01 to compensate for the increased number of ANOVAs.

Mixed ANOVAs were computed on O_1 - A_2 integrated amplitude measures obtained from five EEG frequencies. Group was used as the between independent variable, reaction time bin (RT) was used as the within-subjects (repeated) independent variable, and the dependent variables were beta, sigma, alpha, theta, and delta O_1 - A_2 integrated amplitude. Means and standard deviations are presented in Table 65 and ANOVA results are shown in Table 66. Note that there was a significant interaction for alpha integrated amplitude so the simple effect for this frequency band will be interpreted (see Table 66).

The simple effect analysis of RT Bin within the insomniac group for O_1 - A_2 alpha integrated amplitude was statistically significant, $F(4, 56) = 6.70$, $p = .004$ (see Figure 20), indicating a difference in O_1 - A_2 alpha integrated amplitude values during the

transition from wakefulness (RT Bin 1) through increasing drowsiness to behavioural sleep (RT Bin 5). Post-hoc multiple comparisons using Tukey's HSD at the .05 alpha level revealed that RT Bin 1 was significantly different from RT Bins 3, and 4; RT Bin 5 differed from RT Bins 2, 3, and 4. A similar analysis of RT Bin within the control group for O_1 - A_2 alpha integrated amplitude was not significant, $F(4, 56) = 0.40$, $p = .67$ (see Figure 20) indicating no difference in O_1 - A_2 alpha integrated amplitude during the transition from wakefulness (RT Bin 1) through increasing drowsiness to behavioural sleep (RT Bin 5). The simple effect analysis of group within RT Bin 1 was not significant, $F(1, 28) = 1.00$, $p = .32$, revealing no group differences in O_1 - A_2 alpha integrated amplitude within RT Bin 1 (see Figure 20). The analysis of group within RT Bin 2 was not significant, $F(1, 28) = 2.62$, $p = .12$, revealing no group differences in O_1 - A_2 alpha integrated amplitude within RT Bin 2 (see Figure 20). A similar analysis of group within RT Bin 3 failed to reach statistical significance when using a critical alpha level of .01, $F(1, 28) = 6.77$, $p = .015$, indicating no group differences in O_1 - A_2 alpha integrated amplitude within RT Bin 3 (see Figure 20). The analysis of group within RT Bin 4 was significant, $F(1, 28) = 8.55$, $p = .007$, indicating significantly more O_1 - A_2 alpha integrated amplitude within the insomniac group than within the control group during RT Bin 4 (see Figure 20). The analysis of group within RT Bin 5 was not significant, $F(1, 28) = 0.55$, $p = .46$, revealing no group differences in O_1 - A_2 alpha integrated amplitude within RT Bin 5 (see Figure 20).

RT Bin 1 vs. RT Bin 5

Following this initial analysis of the PAA RT data it was apparent that more information might be obtained through a different examination of the PAA RT data. It appeared that the insomniacs showed less change in EEG measures during the sleep onset period than control subjects. In other words, it is possible that the sleep onset process

might be impaired in insomniacs by maintained arousal not observed within the normal group and this would manifest itself as less change in EEG measures from RT Bin 1 to RT Bin 5 within the insomniac group when compared to the control group. One-way between groups ANOVAs were performed on change scores obtained from subtracting RT Bin 1 data from RT Bin 5 data for each brainwave frequency (delta, theta, alpha, sigma, and beta) for half-wave, full-wave, first-derivative, and integrated amplitude measures. Results of these post-hoc analyses were not considered to be significant unless they were below a critical alpha level of .01. Only one measure, C_3-A_2 beta half-wave incidence reached this level of statistical significance, $F(1, 28)=7.78$, $p = .009$).

Table 65. Reaction time means and standard deviations for O_1-A_2 integrated amplitude

Group	RT	Beta	Sigma	Alpha	Theta	Delta
Insomnia	1	54769.59 (73435.27)	61740.44 (58094.49)	291934.34 (325166.36)	387597.42 (281268.42)	553300.28 (447539.25)
Insomnia	2	63451.19 (72318.48)	72859.24 (60622.22)	360922.47 (331554.95)	412056.88 (288764.88)	584056.22 (579302.54)
Insomnia	3	66728.48 (57974.25)	76320.05 (51279.6)	417208.14 (331892.66)	412611.65 (259678.83)	554470.02 (520018.01)
Insomnia	4	62957.21 (59779.62)	74555.68 (54325.02)	431609.28 (320358.28)	390747.68 (271967.35)	515910.74 (540977.28)
Insomnia	5	44242.14 (57114.68)	47092.30 (48241.47)	210520.88 (234292.72)	260977.24 (239428.10)	359704.56 (492238.77)
Control	1	107967.01 (234556.33)	84757.03 (116326.59)	194115.95 (192747.35)	338100.44 (235517.08)	566884.10 (447539.25)
Control	2	88069.86 (162013.65)	75429.82 (90039.70)	197499.52 (207585.96)	266705.49 (215204.66)	388661.22 (304617.71)
Control	3	76457.56 (133156.59)	66668.19 (83489.06)	167088.20 (168549.01)	222870.13 (178895.67)	383582.04 (261920.07)
Control	4	72673.46 (119237.84)	64729.67 (79218.79)	160602.75 (161697.25)	221069.59 (172312.63)	362213.33 (229413.79)
Control	5	80879.14 (143975.03)	65197.17 (85658.60)	153716.05 (183718.96)	316117.82 (357685.63)	1010651.13 (2383291.28)

Note. Standard deviation in parentheses.

Table 66. F ratios and probabilities for O₁-A₂ integrated amplitude

Frequency	Group	p	RT	p	Interaction	p
Beta	0.55	.47	0.22	.93	0.37	.83
Sigma	0.05	.83	0.69	.60	0.74	.56
Alpha	4.09	.05	4.02	.004	3.85	.006*
Theta	1.68	.21	0.98	.42	3.02	.02
Delta	0.019	.89	0.54	.71	1.78	.14

Note. Degrees of freedom are 1, 28 for between group analyses; degrees of freedom for within measures are 4, 112. Critical alpha level set at .01.

Figure 20. Plot of group x RT bin interaction for alpha O1-A2 integrated amplitude. Arrow indicates significant between group difference at $p < .01$.

Table 67. Summary table of PAA RT Bin Analyses

Insomniacs have more C_3-A_2 alpha full-wave within all RT Bins than controls.

There is a significant group x RT Bin interaction for C_3-A_2 alpha full-wave incidence.

Insomniacs have more C_3-A_2 beta half-wave than controls within RT Bins 4 and 5.

There is a significant group x RT Bin interaction for C_3-A_2 beta half-wave incidence.

Both groups show a decrease in C_3-A_2 beta first-derivative from RT Bin 1 to RT Bin 5.

Both groups show an increase in C_3-A_2 theta first-derivative from RT Bin 1 to RT Bin 5.

Both groups show an increase in C_3-A_2 delta first-derivative from RT Bin 1 to RT Bin 5.

Controls show a decrease in C_3-A_2 alpha amplitude from RT Bin 1 to RT Bin 5.

Insomniacs do not show a decrease in C_3-A_2 alpha amplitude from RT Bin 1 to RT Bin 5.

There is a significant group x RT Bin interaction for C_3-A_2 alpha amplitude.

Insomniacs have more O_1-A_2 alpha full-wave than controls during the SOP.*

Controls have more O_1-A_2 delta full-wave than insomniacs during the SOP.

Both groups show a decrease in O_1-A_2 beta full-wave from RT Bin 1 to RT Bin 5.

Both groups show a decrease in O_1-A_2 sigma full-wave from RT Bin 1 to RT Bin 5.

Both groups show a decrease in O_1-A_2 alpha full-wave from RT Bin 1 to RT Bin 5.

Both groups show an increase in O_1-A_2 theta full-wave from RT Bin 1 to RT Bin 5.

Both groups show an increase in O_1-A_2 delta full-wave from RT Bin 1 to RT Bin 5.

Both groups show a decrease in O_1-A_2 beta half-wave from RT Bin 1 to RT Bin 5.

Both groups show a decrease in O_1-A_2 alpha half-wave from RT Bin 1 to RT Bin 5.

Both groups show a decrease in O_1-A_2 theta half-wave from RT Bin 1 to RT Bin 5.

Both groups show an increase in O_1-A_2 delta half-wave from RT Bin 1 to RT Bin 5.

There is a significant group x RT Bin interaction for O_1-A_2 alpha amplitude.

Note. *SOP = sleep onset period.

Figure 21. The following page shows a 30-second period (one epoch) of polysomnographic data. The top channel (BR Unit) shows the delivery of a tone represented by a square wave. The 2-second duration of the tone indicates that a behavioural response was made during polysomnographically defined sleep. Note the 10-seconds of pretone EEG data on the channels labelled C3 and O1. The time scale is at bottom.

Figure 22. The following page shows a 30-second period (one epoch) of polysomnographic data from the same subject shown in Figure 21. The top channel (BR Unit) shows the delivery of a tone represented by a square wave. The 5-second duration of the tone indicates that no response was made indicating both behavioural and polysomnographic sleep. Note the 10-seconds of pretone EEG data on the channels labelled C3 and O1. The time scale is at bottom.

Discussion

Overview:

The findings within this study, one of the most comprehensive examinations of sleep onset in good sleepers and insomniacs to date, support most of the hypotheses postulated *a priori*. Before proceeding into a detailed discussion of the results, a succinct summary of the 10 hypotheses will be provided. First, the data suggest that, as postulated, defining sleep onset latency simply as 90 seconds of stage 1 sleep is misleading because a number of more conservative definitions correlate more strongly with both subjective and behavioural indices (hypothesis #1). Subjective sleep onset measures were significantly longer in both groups than the 90 second definition of sleep onset, and within the control group, a behavioural response definition of sleep onset latency was significantly longer than the 90 second definition of sleep onset. All of this reveals the inaccuracy of the 90 second definition of sleep onset. Hypothesis two was supported: the behavioural definition of sleep onset is aligned between stages 1 and 2 sleep (see Figure 1) as shown by the lack of any difference between the behavioural, stage 1, and the stage 2 sleep onset definitions (i.e., BR SOL, SOL, S2 SOL, SOL 5). As predicted, insomniacs showed significantly more stage reversals prior to the first 15 minutes of continuous sleep (excluding stage 1 sleep) supporting hypothesis three. Interpretation of the accuracy and validity of the behavioural response system is unambiguous: sleep/wakefulness is accurately assessed in both chronic, psychophysiological insomniacs and in healthy controls by an economical behavioural response system which shows overall concordance with traditional polysomnograms of greater than 93%. This fully supports hypothesis four. The high degree of multivariate overlap between polysomnographic and behavioural sleep/wake measurements, as shown by the significant canonical correlations, confirms hypothesis five. While significant group differences in reaction time were observed during the sleep onset period, the results were in the opposite

direction to those anticipated (hypothesis #6), revealing that insomniacs had significantly longer reaction times than the control subjects.

Period-amplitude analysis (PAA) of sleep stages during the transition from wake to sleep revealed many statistically significant findings. The finding of significantly more delta EEG within the control group confirms the utility of digital analytical techniques to provide information not obtained through the application of a traditional stage scoring schema (hypothesis #7). It is rather simplistic to state that insomniacs took longer to fall asleep; however, much more information is gained by indicating that group differences exist within the delta, alpha, and beta EEG bands during the sleep onset period. These differences support hypothesis seven and will be discussed in detail below. The general finding of a decrease in beta, sigma, and alpha values, and an increase in theta and delta values during the transition from wakefulness to stage two sleep is not surprising and confirms visual stage scoring data. More interesting is the finding that hypothesis eight was not supported: there was no difference between the two groups in C_3-A_2 beta measures during stage wake, and the statistically significant between group difference observed in O_1-A_2 beta half-wave incidence revealed that controls have more of this type of beta activity during wakefulness than insomniacs. The finding that alpha EEG measures during wakefulness were greater in the insomniac group than in the control group was not anticipated and consequently did not support hypothesis eight. These unexpected findings led to important reformulations of both normal and atypical sleep onset.

The analysis of EEG in association with changes in reaction time permitted the examination of hypotheses nine and ten. There is limited support for hypothesis nine: chronic psychophysiological insomniacs have less delta incidence during the sleep onset period than controls, and the C_3-A_2 half-wave incidence measure reveals that insomniacs have more beta incidence in the late RT Bins than controls; however, no group

differences were observed in beta or delta amplitude measures. Contrary to expectation, no group differences were observed in theta activity and the significant group differences in alpha incidence and amplitude were in the direction opposite to those anticipated with insomniacs showing significantly more alpha EEG activity than controls. On the other hand, the finding of significantly more alpha incidence within the insomniac group than within the control group, coupled with no decrease in alpha amplitude from RT Bin 1 to RT Bin 5 within the insomniac group suggests the presence of alpha intrusion within psychophysiological insomnia during the sleep onset process. This important finding implies an impaired sleep onset process within this type of chronic primary insomnia. Hypothesis ten was not supported. While the data revealed a dissociation of amplitude and incidence during the transition from fast responding (RT Bin 1) to response failure (RT Bin 5), the anticipated increase in EEG amplitude across all bandwidth frequencies was not observed. Thus, this PAA analysis extended current knowledge by showing that changes previously attributed to increased amplitude in fast Fourier transform (FFT) analyses are actually due to incidence increases. The findings summarized thus far are discussed in detail on the following pages.

Stage 1 “sleep” or stage 1 “drowsiness”?

According to Hauri and Olmstead (1983), the moment of sleep onset for most insomniacs is best defined as the time from lights out to the beginning of the first 15 minutes of uninterrupted stage 2 sleep. They further suggest that for good sleepers the moment of sleep onset is best defined as the time from lights out to the first epoch of stage 2 sleep. The correlational data do not strongly support Hauri and Olmstead (1983), although the results of hypothesis three (see below) do offer support for their position. Reference to Tables 3 and 4 reveals that the 15 minute definition of sleep onset best correlates with the subjective measure for the insomniac group on night one only. The

five minute definition of sleep onset best correlates with the subjective measure for the control group on night two only. On the other hand, there is support for hypothesis one.

Recall that hypothesis one suggested that a stage 1 sleep definition of sleep onset (i.e., definition 1 as described on page 29) would underestimate and precede sleep onset according to subjective and behavioural measures. There is support for this hypothesis within the data. Specifically, subjective and behavioural indices of sleep onset best correlated with a stage 1 sleep definition on three of six occasions (see Table 3) within the insomniac group, and on 2 of six occasions (see Table 4) within the control group. More conservative definitions of sleep onset best correlated with subjective and behavioural definitions on the remainder of the occasions (see Tables 3 & 4). In addition, the 90 second definition of sleep onset was significantly shorter than subjective sleep onset measures in both groups; within the control group the behavioural response definition of sleep onset latency was significantly longer than the 90 second definition of sleep onset. Thus, a 90 second definition of sleep onset is both misleading and inaccurate because both subjective and behavioural indices suggest that sleep occurs at a later time.

Hypothesis two was supported: as anticipated, the behavioural sleep onset latency aligned itself between stages 1 and 2 sleep for both groups (see Figure 1). No significant differences were observed between a behavioural sleep onset latency (BR SOL) and either the more conservative stage 1 sleep measure of sleep onset latency (SOL) or the early stage 2 sleep measures of sleep onset latency (S2 SOL, SOL 5). The presence of behavioural responding during stage 1 sleep and early stage 2 sleep within the present study is not surprising and has been reported by previous research (Cote & Ogilvie, 1994; MacLean et al., 1992; Ogilvie & Wilkinson, 1988). Indeed, the occurrence of responding during the transition into sleep is consonant with the observations made by early pioneers of sleep research (Davis et al., 1938) who remarked that the definition of sleep onset is dependent on the operational definition used. More recently, Cote and Ogilvie (1994)

observed that the majority of disagreement between the polysomnographic and behavioural response indices occur during transition to and from sleep. They also presented evidence that arousals from sleep occur with equal frequency pre- and post-tone, rather than representing responses in sleep. Furthermore, they found that more points of disagreement occurred early in the night, with more disagreement during the lighter stages of sleep or during transitional periods. All of this suggests that stage 1 sleep is not sleep *per se*, but rather a transitory stage. Interpreted from this perspective, the occurrence of behavioural responding during stage 1 sleep emphasizes the need to conceptualize the process of falling asleep as a period of sleep onset rather than as a paradox. Research which has come to this conclusion (Ogilvie & Wilkinson, 1988) has suggested the use of the term *sleep onset period* to accommodate both fluctuations in behavioural responsiveness to faint auditory stimuli and concomitant alterations in the polysomnogram. Therefore, the results of the present study support the concept that stage 1 sleep is a stage of light to heavy drowsiness rather than sleep - a conclusion made by previous researchers who have subdivided stage one sleep into a period of light to heavy drowsiness.

Broughton (1993) states that stage 1 sleep may be subdivided into substages 1A and 1B. Stage 1A is characterized by a movement of diffuse alpha activity to the anterior region of the head. This alpha activity is often slower than the alpha activity observed during wakefulness (by 0.5 to 1.5 Hz) and becomes fragmented before disappearing. Stage 1B, which is the same as the traditional Rechtschaffen and Kales (1968) stage 1 sleep, is scored when less than 20% of an epoch contains such slow diffuse alpha activity of less than 40 μ V in amplitude. During this substage, the EEG is characterized by medium amplitude mixed frequency theta activity with occasional negative vertex sharp waves. According to Broughton, slow rolling eye movements may occur during either stage 1A or stage 1B sleep. Other researchers have also divided stage 1 sleep into

additional substages. For example, Hori et al. (1994) subdivide stage 1 sleep into six sleep stages (see the introduction for a brief description). What does this reveal about stage 1 sleep? Previous calls for subdividing stage 1 sleep reinforce the notion that stage 1 sleep is not a unitary period, but is truly a period of light to heavy drowsiness. Such a conclusion has implications for studies which have found that insomniacs frequently overestimate sleep onset latency.

In the literature, it is frequently stated that insomniacs overestimate sleep onset latency (Hauri, 1982; Downey & Bonnet, 1992); however, the results of this study suggest that on night one, a 15-minute definition of sleep onset latency best correlates with a subjective estimate of sleep onset latency within the insomniac group. In addition, the 90 SOL (90 seconds of stage 1 sleep) and the SOL (5 continuous minutes of any sleep stage) sleep onset definitions were both significantly shorter than subjective sleep onset latency within the control group, suggesting that control subjects also overestimate sleep onset latency. Consequently, single night polysomnographic studies which conclude that insomniacs overestimate sleep onset latency may be misleading if sleep onset latency is defined using a stage 1 or stage 2 sleep latency definition. Moreover, the conclusion by Carskadon et al. (1976), that insomniacs overestimate sleep onset latency, is somewhat simplistic since sleep onset latency was defined therein as latency to 90 seconds of stage 1 sleep or the first epoch of stage 2 sleep. Bonnet and Moore (1982) have demonstrated that only 50% of individuals reported being asleep during the first epoch of stage 2 sleep (first sleep spindle) thus qualifying the use of the first epoch of stage 2 sleep as a reliable index of sleep onset. The data from the present study - which consist of seven different indices of sleep onset - suggest that the more conservative measures of sleep onset (i.e., beyond a mere 90 seconds of stage 1 sleep) more accurately reflect bona fide sleep. Therefore, the findings within the present study suggest the need for more vigilance in interpreting the results of single night polysomnographic studies which have concluded

that insomniacs overestimate sleep onset latency.

How can stage reversals help characterize the sleep onset period of insomniacs?

Insomniacs showed significantly more stage reversals prior to the first 15 minutes of continuous sleep (excluding stage 1 sleep) which supported hypothesis three. This strongly suggests that insomniacs fail to remain locked within sleep-related processes as they begin, and further shows that any examination of insomnia must take into account the number of stage reversals during the sleep onset period to get an accurate description of the sleep onset process. Given the greater number of stage reversals during sleep onset in the insomniac group than in the control group and the known relationship between time-since-last-awakening (length of sleep) and the perception of sleep (Bonnet & Arand, 1994; Bonnet & Moore, 1982), the data herein support Hauri and Olmstead's (1983) position that the experience of being asleep occurs later for insomniacs than it does for good sleepers. It is unlikely that the greater number of stage reversals within the insomniac group were due to greater hypersensitivity to auditory stimuli because Haynes et al. (1985) found no difference in the auditory awakening thresholds of insomniacs and good sleepers. The present data indicate that insomniacs have more intervening periods of stage wake and stage 1 sleep during the sleep onset period which may explain why insomniacs overestimate sleep onset latency. Thus, the tendency for insomniacs to overestimate sleep onset latency may, in part, be accounted for by a combination of a lighter sleep and a greater frequency of stage reversals than occurs in good sleepers during the sleep onset period.

Do polysomnographic and behavioural measures of sleep agree?

The results of this study provide impressive evidence indicating very high agreement between polysomnographic and behavioural data for the identification of sleep

and wakefulness. The multivariate canonical correlations evaluate the relationship between the two sets of variables while taking into account the intercorrelations of the variables. This statistical approach simultaneously enters all variables into a mathematical equation rather than examining the relationship between a single pair of variables, as in the univariate correlation. Significant multivariate overlap between the two sets of variables was found for both groups on each night, thus confirming hypothesis five. For the insomniac group, the average of the significant canonical correlations was 0.96; for the control group, the average of the significant canonical correlations was 0.90. This is compelling evidence indicating that the set of behavioural sleep/wake measures are strongly associated with the set of polysomnographic sleep/wake indices and account for a substantial portion of variance in the set of polysomnographic parameters. In other words, a clinician or researcher can record sleep using such a behavioural device, and can remain confident that the majority of the information captured by a polysomnogram to evaluate sleep/wakefulness will also be captured by the behavioural device.

The overall percent concordance between the polysomnographic and behavioural data exceeded 90% for both the insomniac and control groups, thus supporting hypothesis four. As expected, the percent agreement between behavioural sleep/wake and traditional sleep stage scoring increased from stage 1 sleep, through wakefulness, stage 2 sleep, REM sleep, stage 3 sleep, to stage 4 sleep. Again, the pattern of agreement was identical for both the insomniac and control groups. These results are exactly as predicted and are consistent with previously published data (Cote & Ogilvie, 1994; MacLean et al., 1992; Kemp & MacLean, 1994); however, the present study is unique in that it extends previous findings into a clinical sample of chronic psychophysiological insomniacs and has used a more sophisticated statistical procedure to compare these fundamental measurement systems.

The pattern of agreement/disagreement between behavioural and polysomnographic measures is essentially identical in both control and insomniac subjects. This further validates sleep/wake discriminations made in earlier studies (Bonato & Ogilvie, 1989; Cote & Ogilvie, 1994). With regard to the concordance rates reported herein, the lower percent agreement found with stage 1 sleep is again not surprising since this traditional stage of drowsiness is not subjectively experienced as sleep by most. Indeed, Walker (1972, cited in Agnew and Webb, 1972) had 54 subjects remain in bed for 45 minutes in the early afternoon. In this study, Walker compared phenomenological sleep/wake experience with the EEG. It was reported that following session one, 56 percent (10 of 18) and 33 percent (5 of 15) of subjects reported being awake although they had reached stage 1 sleep and stage 2 sleep, respectively. Only 13 percent (2 of 15 subjects) reported being awake even though they had reached slow wave sleep. Following session two the next day, 58 percent (7 of 12) and 10 percent (2 of 20) of subjects reported being awake even though they had achieved stage 1 sleep and stage 2 sleep, respectively. What does all of this tell us about behavioural and polysomnographic sleep/wake indices?

An entire body of literature dating back to the 1930s, whether using phenomenological, behavioural, or polysomnographic indices of sleep/wakefulness, has shown time and again that the majority of normal, healthy sleepers will not perceive sleep during the initial period of stage 1 sleep (Bonnet & Moore, 1982; Johnson, 1973; Kamiya, 1961; Loomis et al., 1937; Ogilvie & Wilkinson, 1984, 1988). This body of research has also shown that the subjective perception of sleep does not occur until stage 2 sleep. Bonnet and Moore (1982) found that 50% of normal sleepers will perceive sleep five minutes into stage 2 sleep - the point they termed the sleep onset threshold. Johnson considered sleep to begin during stage 2 sleep because the sleep spindles observed therein remain unique to sleep. Therefore, high agreement is found between behavioural and

polysomnographic measures for the identification of sleep and wakefulness with most disagreement occurring either early in the night or during transitional periods such as stage 1 sleep (drowsiness). Attempts to reconcile periods of disagreement is futile and it is appropriate to conclude that sleep onset is a transitional process involving waxing and waning of the arousal continuum rather than a discrete, instantaneous process analogous to the opening or closing of an electrical circuit.

Concerning the univariate correlations reported herein (see Tables 9 and 11), statistically significant correlations were observed between all comparable pairs of polysomnographic and behavioural measures across the two subject groups lending further support to hypothesis five. This represents further evidence supporting the use of a behavioural system to accurately record fundamental sleep/wake parameters outside of the sleep laboratory. Just as importantly, however, this study demonstrates that the behavioural response system may be successfully used in a clinical population of chronic psychophysiological insomniacs. The behavioural response system could, therefore, be used for accurate and reliable in-home diagnosis of individuals reporting insomnia, or for monitoring the progress of therapeutic interventions for insomniacs.

According to a recent American Sleep Disorders Association (ASDA) practice parameters report on the use of polysomnography in evaluating insomnia (ASDA, 1995), polysomnography is only recommended when the cause of insomnia is not clear, when therapeutic intervention is unsuccessful, or for screening suspected cases of PLMs or sleep apnea. Using the behavioural response device for home-monitoring of insomnia is an inexpensive, easy-to-use, accurate, and valid alternative to ambulatory polysomnography. Such an alternative provides important information concerning fundamental sleep parameters, and would provide a valuable adjunct to traditional sleep/wake logs. Experience shows that given clear and concise instructions, most individuals easily tolerate the apparatus although some insomniacs, particularly those

who habitually wear earplugs during the night, may not feel comfortable with this device.

To summarize the comparison between polysomnographic and behavioural indices of sleep, the behavioural response device has been shown to be an accurate discriminator of sleep/wakefulness in a clinical sample of chronic psychophysiological insomniacs and in a sample of healthy controls, when cross-validated against EEG indices. Percent concordance, univariate, and multivariate statistics have all indicated a strong association between the two measurement techniques. Responses observed during stage 1 sleep do not indicate error with the behavioural system, but suggest the need for revision of traditional sleep stage scoring (Rechtschaffen & Kales, 1968) to incorporate a transitory stage 1 drowsiness period rather than stage 1 sleep. It is not suggested that alternative behavioural measures are better than polysomnography or could replace polysomnography, but rather behavioural measures provide valuable information which is both theoretically and clinically relevant.

Do insomniacs have shorter response latencies than controls?

It was anticipated that insomniacs would show shorter response latencies than controls during the sleep onset period (recall hypothesis #6), and it was suggested that such a finding might be the result of an increased arousal level within the insomniac group. While significant group differences in reaction time were observed during the sleep onset period, the results were in the opposite direction to those anticipated revealing that insomniacs had significantly longer reaction times than the control subjects. Instructions were identical for all subjects so this could not be a source for the statistically significant group main effect. To ensure that this difference was not due to a practice/monotony effect (i.e., insomniacs responded much more before falling asleep because they had much longer sleep onset latencies), the analysis was repeated using reaction times immediately following lights-out, and a more conservative critical alpha

level of .01. This post-hoc analysis revealed identical results. Due to positively skewed distributions, nonparametric analyses were also used and yielded identical results. Two possible explanations exist for this group difference: (1) insomniacs are physiologically hyperaroused and reaction time differences reflect Yerkes-Dodson's law (inverted-U) or (2) the auditory stimulation distracts insomniacs from cognitive rumination and is therefore beneficial.

It is possible that insomniacs are physiologically hyperaroused as suggested by Monroe (1967) and Lamarche (1995) among others. In this scenario, the Yerkes-Dodson law would apply suggesting that the reduced performance (longer response latencies) observed in the insomniac group is due to a very high level of arousal. Research has found that if a warning signal is given to subjects just before the presentation of a signal, those subjects who have the greatest slowing of heart rate during the warning period have the fastest reaction times (Lacey & Lacey, 1970). This finding was interpreted to support the optimal-arousal theory. However, within the current study the explanation of reaction time differences based on Yerkes-Dodson's law seems unlikely given the finding reported below (interpretation of hypothesis #8) that there was either no difference between the two groups in beta activity during wakefulness or that controls had significantly more beta EEG during wakefulness. An alternative, more parsimonious explanation is that insomniacs respond more slowly because the auditory stimulation serves as a distractor from cognitive rumination experienced during the sleep onset process.

A survey of 296 insomniacs conducted by Lichstein and Rosenthal (1980) found that intrusive cognitions are far more prevalent than somatic factors in creating insomnia. In the present study, the Behavioural Response Postsleep Questionnaire inquired about a "racing mind" while falling asleep during the preceding night. Insomniacs reported a "racing mind" during 20 of 45 recording nights. It may be speculated that insomniacs

responded more slowly because the auditory stimuli distracted them from cognitive ruminations and were thus perceived as beneficial. In this case, the behavioural response device would function much like a white noise device. An anecdotal report from one male insomniac indicated that he found the auditory stimuli calming because they took his mind off of his troubles. Additional research is required to examine the therapeutic aspect of the behavioural response device perhaps within a social psychological misattribution paradigm.

What is learned by using period-amplitude analysis during sleep stages?

A number of significant group and stage differences are found through the application of period-amplitude analysis of the EEG during the transition from wake to sleep; however, before proceeding with a discussion dealing with quantitative EEG analysis, it is important to recall the differences between the period-amplitude analysis (PAA) algorithm used herein, and the more commonly used fast Fourier transform (FFT) method. Using PAA is known to produce a different bias for each method of analysis (i.e., full-wave, half-wave, first-derivative, and integrated amplitude) than the FFT. Relative to the FFT method, which over-represents the lower frequencies due to a bias towards amplitude or power, PAA is more sensitive to the faster frequencies, such as beta, sigma, and alpha, when using the first-derivative method of analysis.

In contrast, the use of the half-wave analysis will bias the PAA results in favour of the slowest frequency in the ongoing waveform, and the full-wave analysis preferentially detects the mid-range components in the ongoing EEG. Note that each of these dependent variables are recorded as incidence or the amount of time occupied by each bandwidth frequency (i.e., time-in-frequency) during a period of 30 seconds (one epoch). To record the amplitude of a waveform within a particular frequency band, the integrated amplitude measure is used. These various components or DVs obtained from

PAA offer a number of advantages over FFT analysis. First, it is clearly evident that the PAA method provides a detailed focus on specific frequency bands of interest and provides the researcher with a separate measurement for both amplitude and incidence. Unlike the PAA method, the FFT technique confounds amplitude and incidence and is particularly sensitive to power (amplitude) changes within frequency bands. According to Ktonas & Gosalia (1981), there are an infinite number of morphologically distinct EEG epochs which will provide identical FFT power values. Thus, the FFT method (power spectral analysis) may not discriminate between two EEG epochs where, for example, one epoch (30 seconds) may have a few delta waves of high amplitude, and another may have many more delta waves but of lower amplitude. In other words, the two samples of EEG data may appear radically different yet provide identical power spectral results because the FFT method confounds wave amplitude and incidence.

Another advantage of PAA over the FFT method is that no assumption regarding stationarity is required when using the PAA method. Consequently, there is no need to separate the EEG signal into discrete mini-epochs and then average the mini-epochs within traditional 30-second epochs to obtain power spectral results for each epoch. Finally, the results of a recent study by Armitage et al. (1995), which compared the PAA and FFT techniques, found that the PAA method could account for more variance within the FFT data than vice versa. The authors concluded that PAA provides more information than the FFT method and they questioned the appropriateness of applying power spectral analysis to clinical samples.

The following pages discuss the results of period-amplitude analyzed EEG obtained from the C_3-A_2 and O_1-A_2 sites. The C_3-A_2 site was used because it is the textbook site for stage scoring recommended by Rechtschaffen and Kales (1968) and the O_1-A_2 site was used because it is an appropriate site to examine the disappearance of alpha activity associated with sleep onset.

The general trend revealed by the quantitative EEG analysis is not surprising and confirms visual stage scoring. Both the C_3-A_2 and O_1-A_2 sites reveal an increase in theta and delta incidence and amplitude values and a decrease in beta, sigma, and alpha incidence and amplitude values during the transition from wakefulness to stage 2 sleep. The only exception to this is C_3-A_2 alpha first-derivative incidence which showed an increase from stage wake and stage 1 sleep to stage 2 sleep. The explanation for this is quite simple. The first-derivative analysis preferentially detects the fastest frequency in the ongoing waveform (see previous paragraph) which becomes the alpha band during stage 2 sleep. The C_3-A_2 first-derivative values dramatically decreased within the beta band during the transition from stage wake to stage 2 sleep, but increased within both the theta and delta EEG bands. Thus, the increase in C_3-A_2 alpha first-derivative during the transition from stage wake to stage 2 sleep is partly a result of a movement away from faster frequency beta EEG to the slower EEG bands, such as alpha, theta, and delta, and is picked up as an increase within the alpha measure because the first-derivative algorithm preferentially detects the fastest frequency within the EEG waveform which is within the alpha EEG band during stage 2 sleep.

Although PAA should confirm visual stage scoring during the sleep onset period, the use of PAA reveals much more information than simple confirmation. The ability of the PAA method to uncover information not obtained through the application of a traditional stage scoring schema reinforces the utility of digital analytical techniques (hypothesis #7). Beyond simply concluding that insomniacs took longer to fall asleep, had more stage reversals, and poorer sleep efficiency, the PAA results provide a close examination of EEG activity during sleep onset. It was anticipated that insomniacs would show less delta, theta and alpha EEG activity, and more beta EEG than controls during the sleep onset period (*hypothesis #7*) and the results generally support this hypothesis. Consideration of the C_3-A_2 and O_1-A_2 sites indicate that they both show identical or

similar information. The PAA results reveal that insomniacs have significantly more C_3-A_2 beta first-derivative and beta full-wave incidence during stage 2 sleep than controls which supports hypothesis seven, but neither the C_3-A_2 nor O_1-A_2 sites reveal group differences in beta first-derivative or beta full-wave incidence during stage wake thus failing to support hypothesis eight. The O_1-A_2 site indicated greater beta half-wave incidence within the control group than within the insomniac group and this finding also failed to support hypothesis eight. This shows that during stage wake there is no difference between the two groups in beta incidence at the C_3-A_2 site and greater beta activity within the control group at the O_1-A_2 site; however, the greater levels of beta activity within the insomniac group during stage 2 sleep indicates a greater level of activation within the insomniac group during this sleep stage and this finding supports hypothesis seven.

Concerning the alpha activity, both the C_3-A_2 and O_1-A_2 sites show significantly more alpha activity which is contrary to expectation and fails to support either hypothesis seven or eight. There is more C_3-A_2 alpha full-wave incidence within the insomniac group during stage wake and stage 2 sleep, more C_3-A_2 alpha integrated amplitude and more O_1-A_2 alpha half-wave incidence within the insomniac group during stage wake and stage 1 sleep, and more C_3-A_2 alpha half-wave incidence and more O_1-A_2 alpha integrated amplitude within the insomniac group when collapsed across sleep stages. All of this indicates that there is more alpha EEG activity within the insomniac group during the sleep onset period than within the control group, suggesting the presence of alpha intrusion as an indicator of an impaired sleep onset process within the insomniac group.

Regarding theta activity during the sleep onset period, only the C_3-A_2 theta first-derivative measure indicates any difference in theta activity between the two groups. The difference was in the predicted direction (hypothesis #7) with controls showing more C_3-A_2 theta first-derivative incidence than insomniacs. The delta measures also show

differences in the predicted direction (hypothesis #7) with the controls having more C_3-A_2 delta full-wave incidence during stages 1 and 2 sleep than insomniacs, and more C_3-A_2 delta half-wave incidence within the control group than within the insomniac group when collapsed across sleep stages. Thus, it is apparent that when the results of the PAA analyses are combined, they provide important information concerning the sleep onset process within chronic psychophysiological insomnia.

The data reported herein suggest that the sleep onset process is substantially different in insomniac subjects. In the control subjects, sleep onset is characterized by a sharp increase in delta activity to levels greater than those seen in insomniacs during stage 2 sleep, and a dramatic decrease in beta activity to levels lower than those observed in insomniacs during stage 2 sleep. Furthermore, the greater levels of alpha activity recorded in the insomniac group suggests alpha intrusion impairs sleep onset in insomniacs. These findings are consistent with previous research. Merica and Gaillard (1992) reported greater CNS activation during sleep within their insomniac sample than within their control sample and Lamarche (1995) reported more delta EEG activity within the control group than within the insomniac group.

Contrary to what was observed by Freedman (1986), Jacobs, Benson, and Friedman (1993), and Lamarche (1995), there is no evidence in the present study to suggest that insomniacs have more beta EEG during wakefulness. Thus, the beta measures recorded during wakefulness within the present study do not provide any support for a physiological-hyperactivity theory of insomnia. Concerning the alpha EEG measures, the alpha measures are greater within the insomniac group during sleep onset than within the control group. Coupled with the findings of greater beta activity during stage 2 sleep within the insomniac group, this suggests that insomniacs suffer from an impaired sleep onset process characterized by alpha intrusion and elevated beta activity during stage 2 sleep. This suggests that insomniacs have a higher level of cortical arousal

which impairs the sleep onset process. Note that all of the previous EEG analyses used the FFT method to decompose and represent power within the five primary frequency bands whereas the present study used PAA. The inherent limitation of spectral analysis to confound amplitude and incidence may account for these inconsistencies. Thus, this PAA analysis once again advances our understanding of both normal and insomniac sleep onset processes.

Arousal changes and electroencephalographic alterations

The examination of electroencephalographic activity associated with reaction time is independent of categorical sleep stage scoring criteria, and thus provides important and meaningful information concerning the process of sleep onset. It was hypothesized that chronic psychophysiological insomniacs would show less delta, theta, and alpha amplitude and incidence, and more beta EEG amplitude and incidence than controls during the sleep onset period (*hypothesis #9*). To examine this hypothesis, a total of 1,264 10-second samples of pretone EEG data were carefully selected during the sleep onset period and subjected to a period-amplitude algorithm.

The results of this study indicate limited support for hypothesis nine: chronic psychophysiological insomniacs did show more C_3-A_2 beta half-wave incidence than control subjects during RT Bins 4 and 5, and less O_1-A_2 delta full-wave incidence during the sleep onset period, but there was no difference in the amplitude measures. As is the case with the sleep stage analyses, results within the alpha EEG band were in the opposite direction to that predicted with insomniacs showing more C_3-A_2 and O_1-A_2 alpha incidence during the sleep onset period than control subjects. Furthermore, control subjects show a decrease in C_3-A_2 alpha amplitude from RT Bin 1 to RT Bin 5 whereas insomniac subjects do not show this decrease. Both C_3-A_2 and O_1-A_2 alpha amplitude is greater during RT Bin 4 within the insomniac group than within the control group.

Consideration of the trends within the data again suggest different processes in sleep onset regulation within the two groups.

Examination of the trends for mean values within C_3-A_2 and O_1-A_2 incidence indicate that the controls consistently end up at a higher level of delta incidence and at a lower level of beta incidence than insomniacs within RT Bin 5, even if the controls show similar or lower delta values and similar or greater beta values within RT Bin 1. Moreover, the controls consistently show a quasi-linear drop in alpha and beta measures from RT Bin 1 through RT Bin 5 whereas the insomniacs show a curvilinear pattern with values increasing then decreasing (see Figures 14, 15, 17, 18, 21). This shows a definite difference between the two groups in the pattern of EEG activity across the RT Bins which, in turn, is strong evidence suggesting that insomniacs have an impaired sleep generating system characterized by greater alpha activity, and to a certain extent during RT Bins 4 and 5, beta activity. Although the trend from RT Bin 1 through RT Bin 5 initially appears to support hypothesis nine, the results of change score analyses were largely unsupported. Change scores were used to examine the amount of change from RT Bin 1 to RT Bin 5, but virtually all analyses were nonsignificant when using a conservative alpha level of .01. Nevertheless, the patterns of activity within RT Bins 1 through 5 remain radically different between the two groups, suggesting a different sleep generating system is functioning within the two groups. The control group is characterized by an efficient sleep generating system with consistent quasi-linear change from RT Bin 1 to RT Bin 5, whereas the insomniac group demonstrates a curvilinear system indicating alpha intrusion and the delayed change in EEG patterns suggesting an impaired ability to initiate sleep onset.

Concerning the validation of EEG indices against a reaction time measure, as predicted there was a general decrease in the faster frequency EEG components and an increase in the slower frequencies associated with lengthening reaction time. Both groups

show a decrease in C_3-A_2 beta first-derivative incidence from RT Bin 1 through RT Bin 5, and an increase in C_3-A_2 theta and delta first-derivative incidence from RT Bin 1 through RT Bin 5. Across the behaviourally-defined sleep onset period (i.e., from RT Bin 1 through RT Bin 5), the O_1-A_2 site for both groups shows a decrease in beta, sigma, and alpha full-wave incidence, a decrease in beta and alpha half-wave incidence, an increase in theta and delta full-wave incidence, and an increase in delta half-wave incidence. Although these changes in EEG incidence are in the anticipated direction, the results provide strong cross validation of EEG measures against changes in arousal associated with increasing reaction times. However, it is particularly noteworthy that while a slowing in the EEG was observed, there was no accompanying increase in EEG amplitude. This suggests that any measure of sleepiness would best measure EEG incidence independent of EEG amplitude.

The traditional understanding of sleep onset is that, on average, the EEG activity both slows and increases in amplitude. Thus, any measurement of sleep by EEG activity alone must take into account both EEG incidence and EEG amplitude. Feinberg (1988) has stated the importance of both incidence and amplitude in the study of EEG. While it is generally understood that a slowing in EEG frequency is associated with migration towards sleep, increasing EEG amplitude is an indicator of sleep intensity. Specifically, higher amplitude delta EEG activity indicates greater neuronal recruitment which, in turn, indicates greater sleep intensity. While there is no increase in theta and delta EEG amplitude from RT Bin 1 through RT Bin 5, theta and delta EEG amplitude increase from stage wake to stage 2 sleep (see PAA analysis of sleep stages above). The explanation for this difference between the PAA stage score and PAA RT measures lies, in part, in the period of time under examination. The evaluation of pretone EEG consisted of five 10-second intervals of noncontinuous data during the first two minutes of sleep (approximately), whereas the sleep stage analyses used continuous 30-second epochs for

up to the first 15 minutes of sleep. Thus, the pretone data is focused on the earliest aspects of sleep onset and offers much more precision than the analysis of traditional sleep stages. Therefore, the dissociation of EEG amplitude and incidence observed herein suggests that the neuronal activity in the brain begins to slow down before any significant increase in EEG amplitude is observed. This finding expands the traditional belief that increased neuronal synchronization is consistently associated with increased amplitude (i.e., Ogilvie et al., 1991).

According to Ogilvie et al. (1991), increased EEG synchrony occurs in all bandwidth frequencies as behavioural sleep onset approaches (i.e., longer response latencies will be associated with increased pretone EEG synchrony). It was thus hypothesized (*hypothesis #10*) that there would be a significant increase in PAA integrated amplitude across all EEG frequencies during the transition from slow responding to response cessation (i.e., from slow reaction time to behavioural sleep). Although the data reveal a dissociation of amplitude and incidence during the transition from fast responding (RT Bin 1) to response failure (RT Bin 5), EEG integrated amplitude does not increase across *all* bandwidth frequencies. Consequently, the present study does not provide unequivocal support for hypothesis ten.

As mentioned earlier, PAA does not confound amplitude and incidence as is the case with the FFT method of analysis. Historically, increased neuronal synchronization has been associated with increased amplitude because synchrony has largely been based on the FFT technique. The present study has used the PAA signal transformation technique to separately examine EEG incidence from EEG amplitude - a task not possible with the Fourier method. This has extended previous investigations into electroencephalographic correlates of sleep onset by showing that during the very early part of the sleep onset period EEG incidence increases without any concomitant increase in EEG amplitude. This enhances previous investigations into sleep onset by suggesting

the presence of an additional component - the dissociation of amplitude and incidence - for the detection of drowsiness and sleep onset. Therefore, the use of a reaction time measure indicates that EEG incidence changes profoundly as one enters sleep, while EEG integrated amplitude remains largely unaltered, suggesting that the former is an excellent index of sleepiness and sleep onset.⁷ This is further evidence of the unique contributions of the period-amplitude analysis technique.

Arousal changes within insomniacs

Monroe (1967) compared good and poor sleepers and found elevated autonomic measures in the group of poor sleepers. This classical study established the physiological-hyperactivity hypothesis of insomnia which was subsequently examined by a number of studies (Borkovec, 1982). According to Borkovec (1982), findings of elevated physiological activity in insomniacs have not been consistent. With respect to EEG measures, past findings of elevated beta EEG in insomniacs (Freeman, 1986; Jacobs, Benson, & Friedman, 1993; Lamarche, 1995) have not been supported within the present study. Thus, within the current study, PAA beta EEG measures during wakefulness fail to support a physiological hyperactivity explanation for insomnia. The elevated levels of alpha activity can be interpreted two ways: (1) the greater alpha EEG incidence levels in insomniacs indicate a higher level of cortical arousal and an impaired ability to initiate sleep or (2) the higher alpha levels observed in the insomniac group could be a byproduct of the much longer sleep onset latency in this group than in the control group. With regard to the former interpretation, the presence of alpha EEG can be seen to indicate that while control subjects descend down the arousal continuum into sleep, the insomniacs remain closer to wakefulness and at a higher level of alertness. Concerning the latter interpretation, reaction time measures were used precisely because they more accurately

⁷Incidentally, statistical tests performed on the control group only yielded identical results.

gauge arousal level than discrete 30-second epochs. Thus, the time into sleep onset should not represent a confounding variable because the EEG samples were selected based on each individual's arousal level as indicated by reaction time. In other words, the EEG samples for each individual were neither selected using time into recording nor sleep stage scores, but rather based on reaction time to the auditory stimulus and consequently represent an accurate measurement of each individual's EEG in association with arousal level. The results of the current study indicate that sleep onset in insomniacs is characterized by a higher level of cortical arousal than in the control group. Insomniacs show lighter sleep as indicated by significantly less delta EEG, significantly more beta EEG during stage 2 sleep, and significantly more alpha EEG during the sleep onset period than the control group. From a clinical perspective, this suggests that substantial information beyond mere sleep stage scoring can be obtained from the EEG using computerized techniques, such as behaviourally categorized period amplitude analysis, to analyze the sleep onset period. An additional conclusion can be made about the sleep onset process in insomniacs: the lower delta levels and higher alpha levels in the insomniac group either causes, or is caused by, more frequent sleep stage reversals than observed in good sleepers.

Interpreting EEG changes in the sleep onset period in insomniacs

Although much remains unknown or uncertain about the neurophysiological origin of particular EEG bandwidth frequencies (Pedley & Traub, 1990), all electroencephalographers would agree that "the cerebral cortex generates all brain waves recorded by conventional scalp EEG" (p. 130). Research has not identified any single sleep center in the brain, but several sleep initiating areas in the forebrain and brainstem are connected to the cortex via ascending and descending projections. Research suggests that the solitary tract nucleus in the medulla, the serotonergic raphé nuclei, the

nonspecific thalamic nuclei, and the anterior hypothalamus-preoptic area and basal forebrain are some of the areas involved in cortical synchrony and behavioural sleep (Jones, 1994).

Delta EEG activity is predominant during slow wave sleep which is the time during which the putative restorative function of sleep occurs. According to the restorative function of sleep hypothesis, sleep serves to restore biochemical or physiological processes that are depleted during prior wakefulness (Oswald, 1980). Neurochemical research (Jasper & Tessier, 1971) has found that the presence of delta activity in the cortex may reflect an absence of cholinergic input - an input which is high during REM sleep and wakefulness. Jouvet (1972) identified serotonin (5-HT) as a principal neurotransmitter involved in the regulation of slow wave sleep (delta EEG), but his original serotonergic theory of sleep has since been modified to suggest that serotonin may not directly induce sleep, but rather modulate it through its effects on other sleep inducing elements within the anterior hypothalamus and the suprachiasmatic nuclei (Berger, 1990). The belief that serotonin may play a role in sleep induction is so widespread that many insomniacs have been consuming tryptophan (an amino acid precursor of serotonin found in many foods) in an attempt to increase serotonin levels and improve sleep (Hauri, 1990). Within the current study, the presence of greater delta activity in the control group during the sleep onset period, implies that therapeutic interventions for insomniacs should address increasing the amount of delta EEG to enable insomniacs to reach a deeper, more restorative, and uninterrupted sleep more quickly.

At one time, the theta frequency band was subsumed within the delta range but early researchers considered an intermediate frequency band to be necessary (Niedermeyer, 1993). As a result, the term "theta" was chosen to describe EEG within the 4 to < 8 Hz range because of the presumed thalamic origin of this brain wave frequency. The hippocampus has generally been implicated in the genesis of the theta rhythm

because of its 3 - 8 Hz electrical activity which has been recorded during arousal, purposeful or exploratory activity, and during REM sleep in the rat (Pedley & Traub, 1990). With regard to theta activity during sleep onset, theta EEG increases as sleep approaches and is most evident during stage 1 sleep, and during REM sleep. Greater theta activity within the control group during stage 2 sleep adds further support to the finding that control subjects reach a deeper sleep relative to insomniacs.

Concerning the alpha EEG rhythm, no neurophysiological alpha rhythm theory has yet found general acceptance (Niedermeyer, 1993), although the theory of a thalamic pacemaker for this frequency band was proposed by Andersen and Andersson (1968). Alpha EEG activity is predominant in the occipital cortex during relaxed, eyes closed wakefulness and its greater presence within the insomniac group relative to the control group suggests that sleep onset is impaired within the insomniac group. In a more extreme form, a clinical phenomenon known as alpha-delta sleep is associated with light, interrupted, and nonrestorative sleep (Fredrickson & Krueger, 1994). Thus, the greater level of alpha activity within the insomniac group during sleep onset suggests the potential for a less restorative sleep susceptible to more interruption relative to the sleep of normals. The findings concerning beta EEG also imply that the sleep of insomniacs suffers from an impaired sleep onset process consisting of lighter sleep.

Beta EEG, described as low voltage, desynchronized EEG, represents an active and alert mind (Niedermeyer, 1993) and is influenced by subcortical regions, such as the ascending reticular activating system within the brainstem (Jones, 1994). The insomniacs show more beta EEG during late RT Bins and during stage 2 sleep, which in combination with the delta, theta, and alpha findings discussed above, confirms that insomniacs have lighter initial sleep. What are some possible implications of these findings for the treatment and evaluation of insomnia?

The differences in these four bandwidth frequencies (delta, theta, alpha, beta)

between the two groups, provides direction for the treatment of insomniacs. The goal of any therapy would be to narrow the EEG differences between insomniacs and controls: increase delta EEG within insomniacs, reduce alpha and beta EEG. Therapies which increase the body's need for sleep, such as the partial sleep deprivation therapy known as sleep restriction therapy (Glovinsky & Spielman, 1991), will increase the propensity for delta EEG and decrease the occurrence of beta EEG. The greater the propensity for delta EEG, the better the quality of sleep. When sleep restriction therapy is coupled with additional nonpharmacological therapies, such as stimulus control which helps break the association of the bedroom with anxiety about sleep (Bootzin, Epstein, & Wood, 1991), and bright light therapy which helps regulate the timing of sleep (Allen, 1991), these therapies provide an avenue with which to steer insomniacs towards the goal of achieving a more rapid descent into deeper sleep consisting of greater delta EEG, and less alpha and beta EEG.⁸ Period-amplitude analysis of the EEG before and after such treatments might allow one to check for fundamental reversals of EEG patterns now known to be associated with chronic psychophysiological insomnia.

An apparent implication for the behavioural response system with regard to insomnia would involve a detailed examination of the sleep disorder known as sleep state misperception. Recall that sleep state misperception is characterized by a polysomnogram indicative of normal sleep, but a persistent subjective complaint of chronic insomnia. A behavioural response system should be used to examine this sleep disorder because a behavioural response system is an active, cued behavioural response measure of sleep/wakefulness which requires subject participation not needed in passive electrographic indices. Two theories have been suggested to explain sleep state misperception (Hauri, 1989): (1) the EEG does not reflect an active and alert mind even

⁸The American Sleep Disorders Association (ASDA, 1995) does not recommend pharmacological treatments (i.e., benzodiazepine administration) as a long-term therapy for the treatment of chronic primary insomnia.

though the EEG indicates bona fide sleep, and (2) that there is an underlying physiological abnormality not detected by current technology (i.e., a Type II error). From a theoretical standpoint, an active, cued behavioural response measure of sleep state misperception combined with period-amplitude analysis would test the hypothesis that the EEG does not reflect an active and alert mind which would be associated with sustained behavioural responding during EEG-defined sleep. Furthermore, the use of period-amplitude analysis alone would test the hypothesis that current visual sleep stage scoring technology fails to detect an underlying physiological abnormality. This would be the most appropriate means to definitively examine this sleep disorder precisely because the behavioural response system, unlike the polysomnogram, is not a passive measure.

Conclusion:

This study provides new and important information about alterations in EEG which occur very early in the sleep onset process. The use of period-amplitude analysis in conjunction with a reaction time measure has detected changing patterns in the EEG at sleep onset and helped to increase our understanding of the sleep onset process: brain frequencies begin to slow down before there is an increase in amplitude and this change in EEG frequency, rather than amplitude, is an excellent index of sleepiness and sleep onset. In addition, this study reveals that the sleep onset process is fundamentally different between insomniacs and good sleepers: insomniacs show more alpha EEG activity, more beta EEG activity during stage 2 sleep, lower amounts of delta EEG, and show more stage reversals than good sleepers during the sleep onset process. Thus, sleep onset in insomniacs is of a lighter nature and vastly different from sleep onset in controls. The results of the present study replicate and extend past results which have shown the validity and accuracy of the behavioural response system for the discrimination of sleep and wakefulness. Finally, it must be emphasized that responses observed during stage 1

sleep do not indicate error with the behavioural system, but suggest the need for a long overdue revision of traditional sleep stage scoring (Rechtschaffen & Kales, 1968) to incorporate a transitory stage 1 drowsiness period rather than continuing to call stage 1 “sleep.”

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Appendix A

Information on the Behavioural Response System

In addition to the 14 electrodes attached to you tonight we would like you to use a behavioural response system to monitor your sleep/wakefulness. To do this, you will be required to have an earphone in one ear, and a button (switch) in your right hand. If you know you have any hearing problems please let us know right now.

This behavioural response system is just a computer that will produce a very quiet tone in the earphone at random intervals ranging from 10 to 30 seconds throughout the night. The table on the next page indicates the relative loudness of the tone. Whenever you hear this tone you should turn the tone off by depressing the button with your thumb; however, do **not** try to respond as quickly as possible to each tone. The tone will be 5 seconds in duration unless you press the button in your palm to turn it off. It is **very important** that you try to get a normal night's sleep - if you hear the tone, turn it off by pressing the button but do not try to respond as quickly as possible - this device is only a way for us to determine whether you are asleep or awake. If you have any questions please do not hesitate to ask.

Loudness of Some Sounds

Appendix B**Initial Subject Assessment Questionnaire**

If you answer “Yes” to any of the following statements check the appropriate box?

- ☐ (1) I have problems with my hearing.
- ☐ (2) I am left-handed.
- ☐ (3) I have a history of epilepsy.
- ☐ (4) I may require medical intervention, such as insulin, during the night.
- ☐ (5) I have been told that I stop breathing during the night.
- ☐ (6) I have been told that my legs move and kick during the night.
- ☐ (7) I frequently wake up with morning headaches.
- ☐ (8) I have a history of sleepwalking.
- ☐ (9) I have a history of sleeptalking.
- ☐ (10) I have a history of violent behaviour at night.
- ☐ (11) I have a history of heart problems.
- ☐ (12) I am currently involved in shiftwork.
- ☐ (13) I am currently taking prescription drugs.
- ☐ (14) I nap habitually.
- ☐ (15) I have a history of alcohol and/or drug abuse.

Please let us know if you answered “Yes” to any of the statements listed above.

Appendix C

Informed Consent Form

Code: _____ Sex: F / M Date: _____

Investigators: Richard Bonato, M.A. (722-6521 x6248)

Supervisors: Dr. R. Hoffmann, Dr. R. Ogilvie, and Dr. D. Radonjic

I, the undersigned, agree to participate in this research project concerning sleep. I understand that I will be required to read a brief set of instructions, to complete a sleep history questionnaire, as well as other presleep and postsleep questionnaires. In addition, I will spend a total of three consecutive nights in the sleep laboratory while wearing 14 electrodes during my sleep, a respiratory belt, and a device to measure oral/nasal airflow. During these three nights I agree to use a behavioural response measure of sleep/wakefulness which requires me to press a small thumb-button when any faint tones are heard from an earphone. I will be instructed simply to fall asleep while many tones are presented via an earphone.

I understand that all material will be kept strictly confidential and will be used for research purposes only. I also understand that my participation is entirely voluntary and that I may choose to terminate my participation at any time. I may leave blank any questions that I choose not to answer, and I may request that the behavioural response system be removed from my being at any time. Further, participants may ask questions at any time.

Name: _____ Date: _____

Signature: _____

Appendix D

BDI

Code: _____ Sex: F / M Date: _____

Instructions: On this questionnaire are groups of statements. Please read each group of statements carefully. Then pick out the one statement in each group which best describes the way you have been feeling during the PAST WEEK, INCLUDING TODAY. Circle the number beside the statement you picked. If several statements in the group seem to apply equally well, circle each one. *Be sure to read all of the statements in each group before making your choice.*

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

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

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

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

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

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

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

- 8) 0 I don't feel I am any worse than anybody else.
1 I am critical of myself for my weaknesses or mistakes.
2 I blame myself all the time for my faults.
3 I blame myself for everything bad that happens.
- 9) 0 I don't have any thoughts of killing myself.
1 I have thoughts of killing myself, but I would not carry them out.
2 I would like to kill myself.
3 I would kill myself if I had the chance.
- 10) 0 I don't cry anymore than usual.
1 I cry more now than I used to.
2 I cry all the time now.
3 I used to be able to cry, but now I can't cry even though I want to.
- 11) 0 I am no more irritated now than I ever am.
1 I get annoyed or irritated more easily than I used to.
2 I feel irritated all the time now.
3 I don't get irritated at all by the things that used to irritate me.
- 12) 0 I have not lost interest in other people.
1 I am less interested in other people than I used to be.
2 I have lost most of my interest in other people.
3 I have lost all of my interest in other people.
- 13) 0 I make decisions about as well as I ever could.
1 I put off making decisions more than I used to.
2 I have greater difficulty in making decisions than before.
3 I can't make decisions at all anymore.
- 14) 0 I don't feel I look any worse than I used to.
1 I am worried that I am looking old or unattractive.
2 I feel that there are permanent changes in my appearance that make me look unattractive.
3 I believe that I look ugly.
- 15) 0 I can work about as well as before.
1 It takes an extra effort to get started at doing something.
2 I have to push myself very hard to do anything.
3 I can't do any work at all.
- 16) 0 I can sleep as well as usual.
1 I don't sleep as well as I used to.
2 I wake up 1-2 hours earlier than usual and find it hard to go back to sleep.
3 I wake up several hours earlier than I used to and cannot get back to sleep.

- 17) 0 I don't get more tired than usual.
1 I get tired more easily than I used to.
2 I get tired from doing almost anything.
3 I am too tired to do anything.
- 18) 0 My appetite is no worse than usual.
1 My appetite is not as good as it used to be.
2 My appetite is much worse now.
3 I have no appetite at all anymore.
- 19) 0 I haven't lost much weight, if any, lately.
1 I have lost more than 5 pounds.
2 I have lost more than 10 pounds.
3 I have lost more than 15 pounds.
- 20) 0 I am no more worried about my health than usual.
1 I am worried about physical problems such as aches and pains; or upset stomach; or constipation.
2 I am very worried about physical problems and it's hard to think of much else.
3 I am so worried about my physical problems, that I cannot think about anything else.
- 21) 0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

Appendix E

BAI

Code: _____ Sex: F / M Date: _____

Instructions: On this questionnaire are statements. Please read each statement carefully and use the scale to rate how much you have been bothered by each item during the PAST WEEK, INCLUDING TODAY. Circle the number below the statement.

Scale

	0	1	2	3
	not at all	rarely	somewhat	severely
1) I was bothered by numbness or tingling.				
	0	1	2	3
	not at all	rarely	somewhat	severely
2) I felt hot.				
	0	1	2	3
	not at all	rarely	somewhat	severely
3) I had wobbliness in my legs.				
	0	1	2	3
	not at all	rarely	somewhat	severely
4) I was unable to relax.				
	0	1	2	3
	not at all	rarely	somewhat	severely
5) I feared the worst was going to happen.				
	0	1	2	3
	not at all	rarely	somewhat	severely
6) I felt dizzy or lightheaded.				
	0	1	2	3
	not at all	rarely	somewhat	severely

7) My heart was pounding or racing.

0	1	2	3
not at all	rarely	somewhat	severely

8) I felt unsteady.

0	1	2	3
not at all	rarely	somewhat	severely

9) I felt terrified.

0	1	2	3
not at all	rarely	somewhat	severely

10) I felt nervous.

0	1	2	3
not at all	rarely	somewhat	severely

11) I had feelings of choking.

0	1	2	3
not at all	rarely	somewhat	severely

12) My hands were trembling.

0	1	2	3
not at all	rarely	somewhat	severely

13) I felt shaky.

0	1	2	3
not at all	rarely	somewhat	severely

14) I feared losing control.

0	1	2	3
not at all	rarely	somewhat	severely

15) I had difficulty breathing.

0	1	2	3
not at all	rarely	somewhat	severely

16) I feared dying.

0	1	2	3
not at all	rarely	somewhat	severely

17) I felt scared.

0	1	2	3
not at all	rarely	somewhat	severely

18) I had indigestion or discomfort in my abdomen.

0	1	2	3
not at all	rarely	somewhat	severely

19) I felt faint.

0	1	2	3
not at all	rarely	somewhat	severely

20) My face felt flushed.

0	1	2	3
not at all	rarely	somewhat	severely

21) I was sweating (not due to heat).

0	1	2	3
not at all	rarely	somewhat	severely

Appendix F

MMPI Critical Items Set

Code: _____ Sex: F / M Date: _____

Instructions: This inventory consists of numbered statements. Read each statement and decide whether it is *true as applied to you* or *false as applied to you*.

Indicate your answers by circling either *T (true)* or *F (false)* at the end of each statement.

Remember to give **your own** opinion of yourself.

Please respond to every statement.

1. I have a good appetite. T or F (2)
2. I wake up fresh and rested most mornings. T or F (3)
3. I am about as able to work as I ever was. T or F (9)
4. I work under a great deal of tension. T or F (13)
5. I am sure I get a raw deal from life. T or F (16)
6. My sex life is satisfactory. T or F (20)
7. At times I have very much wanted to leave home. T or F (21)
8. I am troubled by attacks of nausea and vomiting. T or F (23)
9. When someone does me a wrong I feel I should pay him back if I can, just for the principle of the thing. T or F (28)
10. I am bothered by stomach acid several times per week. T or F (29)
11. I have had very peculiar and strange experiences. T or F (33)
12. I seldom worry about my health. T or F (36)
13. I have never been in trouble because of my sex behaviour. T or F (37)
14. During one period when I was a youngster I engaged in petty thievery. T or F (38)
15. Much of the time my head seems to hurt all over. T or F (44)
16. Once a week or more often, I feel suddenly hot all over, without apparent cause. T or F (47)

17. When I am with people I am bothered by hearing very queer things. T or F (48)
18. I am almost never bothered by pains over the heart or in my chest. T or F (55)
19. As a youngster, I was suspended from school one or more times for skipping class. T or F (56)
20. Parts of my body often have feelings like burning, tingling, crawling, or like "going to sleep." T or F (62)
21. I see things or animals or people around me that others do not see. T or F (66)
22. I hardly ever feel pain in the back of the neck. T or F (68)
23. I am very strongly attracted by members of my own sex. T or F (69)
24. I am troubled by discomfort in the pit of my stomach every day or every few days. T or F (72)

Women, please answer question 25a ONLY

Men, please answer question 25b ONLY

- 25a. I have often wished I were a man. T or F
- 25b. I have often wished I were a woman. T or F (74)
26. Most of the time I feel blue. T or F (76)
27. I am certainly lacking in self-confidence. T or F (86)
28. I usually feel that life is worthwhile. T or F (88)
29. I have very few quarrels with members of my family. T or F (96)
30. At times I have a strong urge to do something harmful or shocking. T or F (97)
31. Someone has it in for me. T or F (110)
32. Often I feel as if there were a tight band about my head. T or F (114)
33. In school I was sometimes sent to the principal for skipping class. T or F (118)
34. My speech is the same as always (not faster or slower, or slurring; no hoarseness) T or F (119)
35. I believe I am being plotted against. T or F (121)

36. I believe I am being followed. T or F (123)
37. I have a great deal of stomach trouble. T or F (125)
38. I have never indulged in any unusual sex practices. T or F (133)
39. At times my thoughts have raced ahead faster than I could speak them. T or F (134)
40. I believe that my home life is as pleasant as that of most people I know. T or F (137)
41. Sometimes I feel as if I must injure either myself or someone else. T or F (139)
42. I certainly feel useless at times. T or F (142)
43. At times I feel like picking a fist fight with someone. T or F (145)
44. Someone has been trying to poison me. T or F (151)
45. I have never had a fit or convulsion. T or F (154)
46. I have had periods in which I carried on activities without knowing later what I had been doing. T or F (156)
47. There is something wrong with my mind. T or F (168)
48. I have never had a fainting spell. T or F (174)
49. I seldom or never have dizzy spells. T or F (175)
50. My memory seems to be all right. T or F (178)
51. I am worried about sex matters. T or F (179)
52. I commonly hear voices without knowing where they come from. T or F (184)
53. I frequently notice my hand shakes when I try to do something. T or F (186)
54. I feel weak all over much of the time. T or F (189)
55. I have very few headaches. T or F (190)
56. I have had attacks in which I could not control my movements or speech but in which I knew what was going on around me. T or F (194)
57. Someone has been trying to rob me. T or F (197)
58. There are persons who are trying to steal my thoughts and ideas. T or F (200)

59. At times it has been impossible for me to keep
from stealing or shoplifting something. T or F (205)
60. I have used alcohol excessively. T or F (215)
61. I get mad easily and then get over it soon. T or F (234)
62. I have periods of such great restlessness that
I cannot sit long in a chair. T or F (238)
63. I believe I am no more nervous than most others. T or F (242)
64. I have few or no pains. T or F (243)
65. My parents and family find more fault with me
than they should. T or F (245)
66. I don't blame anyone for trying to grab everything
he can get in this world. T or F (250)
67. I have had blank spells in which my activities were
interrupted and I did not know what was going on
around me. T or F (251)
68. I can easily make other people afraid of me, and
sometimes do for the fun of it. T or F (269)
69. I have numbness in one or more regions of
my skin. T or F (273)
70. Someone has control over my mind. T or F (275)
71. Most people make friends because friends are likely
to be useful to them. T or F (280)
72. I do not often notice my ears ringing
or buzzing. T or F (281)
73. I am sure I am being talked about. T or F (284)
74. I have very few fears compared to my friends. T or F (287)
75. At one or more times in my life I felt that someone
was making me do things by hypnotizing me. T or F (291)
76. Someone has been trying to influence my mind. T or F (293)
77. I have never been in trouble with the law. T or F (294)
78. I wish I were not bothered by thoughts about sex. T or F (297)
79. Life is a strain for me much of the time. T or F (301)
80. I have never been paralyzed or had any unusual
weakness of any of my muscles. T or F (330)

61. If people had not had it in for me I would have
been much more successful. T or F (331)
82. Peculiar odors come to me at times. T or F (334)
83. I cannot keep my mind on one thing. T or F (335)
84. I feel anxiety about something or someone almost
all the time. T or F (337)
85. Most of the time I wish I were dead. T or F (339)
86. At times I hear so well it bothers me. T or F (341)
87. I have no enemies who really wish to harm me. T or F (347)
88. I have strange and peculiar thoughts. T or F (349)
89. I hear strange things when I am alone. T or F (350)
90. I have been afraid of things or people that I knew
could not hurt me. T or F (352)
91. People say insulting and vulgar things about me. T or F (364)
92. I am often said to be hotheaded. T or F (381)
93. I have sometimes felt that difficulties were piling
up so high that I could not overcome them. T or F (397)
94. I am usually calm and not easily upset. T or F (407)
95. At times I think I am no good at all. T or F (418)
96. I have had some very unusual religious experiences. T or F (420)
97. I worry quite a bit over possible misfortunes. T or F (431)
98. I have used alcohol moderately (or not at all). T or F (460)
99. I have never seen a vision. T or F (464)
100. Except by a doctor's orders, I never take drugs or
sleeping aids. T or F (466)
101. There is something wrong with my sex organs. T or F (519)
102. The future seems hopeless to me. T or F (526)
103. Several times a week I feel as if something
dreadful is about to happen. T or F (543)
104. I feel tired a good deal of the time. T or F (544)
105. Sometimes I am sure that other people can tell what
I am thinking. T or F (551)

Appendix G**General Sleep History Questionnaire (0791)**

NAME _____

DATE _____

The Sleep Disorders Centre is interested in some specifics concerning your sleep pattern and daytime functioning, which may help us pinpoint possible sleep-related disorders. Your careful attention to the questions will greatly assist us in the evaluation of your sleep prior to sleep recordings at the Centre. We recommend that you complete this survey with the help of your spouse or bed partner (if applicable) since some aspects of your sleep may be unknown to you.

NIGHT TIME

1. How many hours of nightly sleep are you currently getting? _____
2. Do you feel that you currently have a problem with sleep? _____
3. Do you currently have a problem:
(check) sleeping too much ___ sleeping too little ___
 falling asleep at night ___ staying asleep at night ___
4. How would you rate this problem?
(check) mild ___ moderate ___ severe ___
5. What is your usual bedtime on weekdays? _____
6. How long does it take to fall asleep once you are in bed? _____
7. How many times do you wake up in a typical night? _____

8. Is it difficult for you to fall back asleep afterward?

(Yes/No) _____

9. What things most often wake you at night?

(check) noise ____ cold ____ heat ____ light ____ child ____
 dream ____ bedpartner's movement ____ different bed/room ____
 body pains ____ other _____

10. Which part(s) of the night do you most frequently awake from?

(check) first 1/3 ____ middle 1/3 ____ last 1/3 ____

11. What thing(s) most often wake you in the morning?

(check) alarm clock ____ bedpartner ____ body discomfort ____
 dream ____ spontaneous awakening ____
 other _____

12. How do you typically feel in the morning?

(check) alert ____ drowsy ____ relaxed ____ tense ____ happy ____
 sad ____ sociable ____ withdrawn ____ strong ____ weak ____

13. Have you or your bedpartner noticed any of the following behaviors during your sleep in the past year?

(check) walking ____ talking ____ frequent urges to urinate ____
 restless sleep ____ rocking movements ____ violent movements ____
 falling out of bed ____ teeth grinding ____ bed-wetting ____
 waking up with hunger ____ sweating ____ shouting/screaming ____
 heart palpitations ____ waking up with frightening images ____
 waking up with terror/anxiety ____ unable to move/speak upon
 awakening ____ twitching of arms/legs ____ large body jerks ____
 waking up with chest/back pains ____ periods of no breathing ____
 loud snoring ____ waking up gasping/choking ____
 other _____

14. Did you have any of the following childhood sleep behaviors?

(check) walking __ talking __ breathing difficulties __
 bed-wetting __ bad dreams __ always tired during the day __
 teeth grinding __ head banging __
 other _____

15. How many dreams do you typically remember each night? _____

16. How would you describe your typical dream?

(check) long __ short __ pleasant __ troubled __
 vivid __ hazy __ exciting __ dull __ black & white __
 color __ observer __ participant __
 other _____

DAYTIME

1. Is there any aspect of your daytime functioning that is impaired by a sleep-related problem?

2. What times of the day do you feel the sleepiest?

3. When are your preferred naps (if applicable)?

4. How long are your naps? _____

5. Do you feel more alert after naps? _____

6. Do you ever have strange imagery during naps? _____
7. Do you frequently fall asleep at work? (Yes/No) _____
If so how often? _____
8. Have you ever had brief attacks of weakness in the arms or legs
and/or complete collapse?

9. What usually triggers these attacks? _____
10. What part of the day do you function best? _____
11. During what part of the day do you function worst? _____

RELATED SLEEP HISTORY

1. Can you describe any sleep problems experienced currently or
in the past by any member of your extended family?

2. Are there any other aspects of your sleep or wake behavior
that would be helpful in evaluating your sleep problem?

Appendix H

NAME _____ DATE _____

2-WEEK SLEEP/WAKE LOG (0791)

Please return this form to the ROH Sleep Disorders Centre after completion, as it will be an important aid in the evaluation of your sleep problem. You should fill out a part of the form each morning based on the previous day and night. The following is a description of the log entries:

BT (**Bed Time**) - the time you went to bed last night
 (e.g., 11:30)

MT (Morning Time) - the time you got up this morning
(e.g., 7:10)

SL (Sleep Latency) - how many minutes it took you to fall asleep last night (e.g., 35)

EMA (Early Morning Awakening) - how many minutes you were awake before getting out of bed this morning (e.g., 15)

QS (Quality of Sleep) - rate how you slept last night
(e.g., 4)

1	2	3	4	5	6	7	8	9
-----	-----	-----	-----	-----	-----	-----	-----	-----
very		well		fair		poor		very
well								poor

- #A** (Number of Awakenings) - how many times you woke up last night (e.g., 2)
- AT** (Awake Time) - total minutes you spent awake after first falling asleep (e.g., 45)
- #N** (Number of Naps) - how many times you fell asleep during the daytime yesterday (e.g., 1)
- NT** (Nap Time) - total minutes you spent asleep during the daytime yesterday (e.g., 75)
- EL** (Energy Level) - Rate your energy level yesterday (e.g., 6)

1	2	3	4	5	6	7	8	9
---- ---- ---- ---- ---- ---- ---- ----								
very		high		fair		low		very
high								low

DRUGS - what medication, alcohol, or caffeine you took yesterday (e.g., 3 coffees, 2 beers, 1 Tylenol, 2 Cokes)

PROBLEMS - any problems or behaviors that may have affected your sleep (e.g., asthma attacks, nightmares, laid off)

Appendix I

Stanford Sleepiness Scale

Code: _____ Sex: F / M Date: _____

Scale values:

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

Test 1 (PM) Time: _____

1	2	3	4	5	6	7
---	---	---	---	---	---	---

Test 2 (AM) Time: _____

1	2	3	4	5	6	7
---	---	---	---	---	---	---

Appendix J

ROH Post-sleep Questionnaire (1293)

Code: _____ Sex: F / M Date: _____

1. When did you go to bed last night? _____

When do you usually go to bed at home? _____

2. When did you get up this morning? _____

When do you usually get up at home? _____

3. **Rate** how sleepy you were when you went to bed last night:

not at all ----- very

4. How long to fall asleep last night? _____

Usually, how long to fall asleep at home? _____

5. **Rate** how difficult it was for you to fall asleep last night:

not at all ----- very

6. How long did you sleep last night? _____

How long do you usually sleep at home? _____

7. How often do you recall waking up last night? _____

8. What caused these awakenings? _____

9. **Rate** your difficulty falling back asleep after an awakening:

not at all ----- very

10. **Rate** how soundly you slept last night:

not at all ----- very

11. Did you get enough sleep last night? _____

12. How did your sleep differ from home? _____

13. **Rate** how difficult it was for you to wake up this morning:

not at all ----- very

14. What woke you up this morning? _____

15. **Rate** how rested you felt after waking up this morning:

not at all ----- very

16. What were your dreams like last night? _____

Appendix K

Behavioural Response Post-sleep Questionnaire

Code: _____ Sex: F / M Date: _____

Please read each question carefully.

If at any time necessary, use reverse.

1) Please rate on the following scale how tired you feel right now:

2) How many times did you wake up last night for less than 2 minutes?

3) How many times did you wake up last night for more than 2 minutes?

4) Keeping questions #2 & #3 in mind, I would say I woke up a total of _____ times during the entire night.

5) Please rate on the following scale how easy/difficult it was for you to use the behavioural response system:

6) If you were to sleep in the sleep laboratory without using the behavioural response system, would your sleep be better, worse, or the same? Please indicate your answer on the following scale:

7) Please rate on the following scale how easy/difficult it was for you to fall asleep with the behavioural response system:

8) Please rate on the following scale how easy/difficult it was for you to resume asleep with the behavioural response system:

9) Using the following scale, please rate the discomfort of the equipment which you wore during the night:

- 1 = very comfortable
- 2 = comfortable
- 3 = somewhat comfortable, somewhat uncomfortable
- 4 = uncomfortable
- 5 = very uncomfortable

- I) Electrodes behind my ears: _____
- II) Electrodes beside my eyes: _____
- III) Electrodes under/on my chin: _____
- IV) Earphone in my left ear: _____
- V) Electrodes on my shoulders: _____
- VI) Electrodes on my scalp: _____
- VII) Electrodes on legs: _____
- VIII) Belt around my abdomen: _____
- IX) Button device in my right hand: _____

If applicable, please rate the following:

- X) Thermocouple under my nose: _____
- XI) Belt around my chest: _____
- XII) Finger probe on left hand: _____

10) Did you have a "racing mind" at any time last night? Y or N
If yes, did it interfere with your sleep? Y or N

Please elaborate: _____

11) Did you have trouble sleeping last night due to your insomnia? Yes or No If yes, would you say your trouble sleeping was caused by an active, alert or "racing mind," a tense and nervous body, or something else? (please be specific and as detailed if possible)

11) If you remember waking up during the night, what was the very first thing that came into your mind? _____

12) Your comments regarding the behavioural response system would be very helpful to us. If you have any additional comments you wish to offer, please write them on the back of this page.

Appendix L
Subject Debriefing for Insomniacs

This behavioural response system is a computer system that records your sleep and wakefulness. By pressing the button you are telling us that you are awake - failure to respond tells us that you are asleep. Previous research has found this system to be very effective in discriminating between sleep and wakefulness. All of those times you pressed the button (and the times you did not) were recorded in the computer and by the end of the night we get a whole bunch of numbers which we can use to get an idea of how long it took you to fall asleep, how many times you woke up (by either pressing the button once, or more than once), or to find out things like your sleep efficiency. This study was required to determine how well the device worked with people who report to a sleep clinic because of a sleep problem. In the future we hope to conduct additional research with other people. If you require additional information, please do not hesitate to contact me, Richard Bonato, at 722-6521 x6248.

Appendix M

Subject Debriefing for Controls

This behavioural response system is a computer system that records your sleep and wakefulness. By pressing the button you are telling us that you are awake - failure to respond tells us that you are asleep. Previous research has found this system to be very effective in discriminating between sleep and wakefulness. All of those times you pressed the button (and the times you did not) were recorded in the computer and by the end of the night we get a whole bunch of numbers which we can use to get an idea of how long it took you to fall asleep, how many times you woke up (by either pressing the button once, or more than once), or to find out things like your sleep efficiency. This study was required to determine how well the device worked with people who report to a sleep clinic because of a sleep problem. In the future we hope to conduct additional research with other people. If you have any questions concerning this questionnaire I may be contacted at 788-2600 x1411 or by mail at :

Richard Bonato
Department of Psychology
Carleton University
Ottawa, Ontario
K1S 5B6

If you were troubled by your responses to any of the questions, counselling is available:
Counselling and Student Life Services, 501 Unicentre, 788-2600 x6600
Peer Counselling Centre, 316 Unicentre, 788-2600 x3581

If you have any questions concerning the ethics of this study you may contact:
Dr. Lise Paquet, Ethics Committee Chair, at 788-2600 x2692.

Other questions concerning this research may be directed to:
Dr. William Jones, Department Chair, at 788-2600 x2648.

Appendix N

Period-Amplitude Analysis Algorithm in Turbo Pascal

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```

program RickDPA;      {program to generate DPA data for Rick's PhD.
                      Data is originally collected and translated
                      into ASCII by Sandman.
                      Output generated will be tab delimited for easy
                      input into Excel, Wingz, etc.}
uses crt, WinDos, Strings;

var
  FirstFile, i,j,k:integer;
  x,y,z:real;
  crit:real;
  fnin,fnout: array[0..80] of Char;
  fin,fout,frep:text;
  epnum:integer;
  dirin,dirout:string;
  ans:char;
  oldfd,oldfz,oldhz,newfd,newfz,newhz,dur,
  channel,variable:integer;
  ptot1,ptot2,ttot:real;

  dtpnts:array[1..2,1..3200] of real;
  datout:array[1..2,1..20] of real;
  lines:array[1..5] of string;

  DirInfo: TSearchRec;
  Path: array[0..fsPathName] of Char;
  Dir: array[0..fsDirectory] of Char;
  Name: array[0..fsFileName] of Char;
  Ext: array[0..fsExtension] of Char;

function evalhalf(num:integer):integer;
begin
  if (num>320) then
    begin
      evalhalf:=0;
      exit;
    end;
  if (num>40) then
    begin
      evalhalf:=5;
      exit;
    end;
end;

```

```

    if (num>20) then
        begin
            evalhalf:=4;
            exit;
        end;
    if (num>13) then
        begin
            evalhalf:=3;
            exit;
        end;
    if (num>10) then
        begin
            evalhalf:=2;
            exit;
        end
    else
        begin
            evalhalf:=1;
            exit;
        end;
end;

function evalfull(num:integer):integer;

begin
    if (num>640) then
        begin
            evalfull:=0;
            exit;
        end;
    if (num>80) then
        begin
            evalfull:=5;
            exit;
        end;
    if (num>40) then
        begin
            evalfull:=4;
            exit;
        end;
    if (num>27) then
        begin
            evalfull:=3;
            exit;
        end;
    if (num>20) then
        begin
            evalfull:=2;
            exit;
        end
    else

```

```

        begin
            evalfull:=1;
            exit;
        end;

    end;

procedure FirstDeriv(a:integer);

var i,j,k,freq:integer;

begin
    newfd:=a;
    dur:=newfd-oldfd+1;
    freq:=evalfull(dur);
    oldfd:=newfd;
    if (freq>0) then
        datout[channel,freq]:=datout[channel,freq]+dur;
    end;

procedure FullWave(a:integer);

var i,j,k,freq:integer;

begin
    newfz:=a;
    dur:=newfz-oldfz;
    freq:=evalfull(dur)+5;
    if (freq > 0) then
        begin
            datout[channel,freq]:=datout[channel,freq]+dur;
            freq:=freq+10;
            for i:=oldfz to newfz do
                datout[channel,freq]:=datout[channel,freq]-(dtpnts[channel,i]*
                    dtpnts[channel,i]);
            oldfz:=newfz;
        end;
    end;

procedure HalfWave(a:integer);

var i,j,k,freq:integer;

begin
    newhz:=a;
    dur:=newhz-oldhz;
    freq:=evalhalf(dur)+10;
    if (freq>0) then
        datout[channel,freq]:=datout[channel,freq]+dur;
    oldhz:=newhz;
end;

```



```

procedure new_criterion;

begin
  clrscr;
  writeln('Old criterion was ',crit:0:2);
  write('Enter new criterion ');
  readln(crit);
end;

procedure new_DirIn;

begin
  clrscr;
  writeln('Old directory was ',DirIn);
  write('Enter new directory ');
  readln(DirIn);
end;

procedure new_DirOut;

begin
  clrscr;
  writeln('Output file directory was ',DirOut);
  write('Enter new output directorty ');
  readln(DirOut);
end;

procedure new_PutOut;

begin
  clrscr;
  writeln('Old Output Filename was ',fnout);
  write('Enter new Output Filename ');
  readln(fnout);
end;

procedure new_GetIn;

begin
  clrscr;
  writeln('Old filename was ',fnin);
  write('Enter new Input filename ');
  readln(fnin);
end;

procedure startup;

begin

repeat {until 'B' for ANS}

```

```

clrscr;
{writeln('Put data out to file ',fnout);
writeln('Output file directory is ',DirOut);
writeln('Get input data from file ',fnin);
writeln('Input file directory is ',DirIn); }
writeln('First file only');
writeln('Criterion is now ',crit:0:2,' microvolts');
writeln('Begin');
writeln('Quit');

repeat {until we get a key typed}
until keypressed;
ans:=readkey;

case ans of
  'i','I':new_DirIn;
  'g','G':new_GetIn;
  'p','P':New_PutOut;
  'o','O':new_DirOut;
  'c','C':new_criterion;
  'q','Q':halt;

end;
until (ans='F') or (ans='f') or (ans='b') or (ans='B'));

end;
begin
  textcolor(14);
  clrscr;
  for i:=1 to 2 do
    for j:=1 to 20 do
      datout[i,j]:=0;
    newfd:=0;
    newfz:=0;
    newhz:=0;
    dirIn:='Empty';
    dirOut:='Empty';
    StrCopy(fnin, 'Empty');
    StrCopy(fnout, 'Default.out');
    crit:=0;

    repeat {Until ans=q}
    STARTUP;

    {----- Starting DPA here -----}

    {chdir(DirIn);}
    FindFirst('*.ex', FaArchive, DirInfo);
    FirstFile := 1;

```

```

assign(fout,fnout);
rewrite(fout);

while (DosError = 0) AND (NOT(ans='q')) AND (NOT(ans='Q')) AND
(FirstFile > 0) do
begin
if (ans='F') OR (ans='f') then FirstFile := 0;

StrCopy(fnin, DirInfo.Name);
assign(fin,fnin);
reset(fin);

for i:=1 to 4 do readln(fin);
{ chdir(DirOut);}
{ chdir(DirOut);}

write(fout, fnin);

writeln('Reading data from ',fnin);
for i:=1 to 4 do readln(fin,lines[i]); {Skip over extra information}
for i:=1 to 3200 do
begin
    readln(fin,x,dtpts[1,i],dtpts[2,i]);    {Read input data here}
end;
writeln;
writeln('Doing DPA');
for j:=1 to 2 do
begin
    oldfd:=1;
    oldfz:=1;
    oldhz:=1;
for i:=3 to 3200 do        {DPA identification}
begin
    if (abs(dtpts[j,i]-dtpts[j,i-1]))>crit then
begin
    if ((dtpts[j,i] > dtpts[j,i-1]) and (dtpts[j,i-1] <=
dtpts[j,i-2] )) then
begin
        channel:=j;
        FirstDeriv(i);
end;
    if (dtpts[j,i]>0) and (dtpts[j,i-1]<0) then
begin
        channel:=j;
        FullWave(i);
end;
    if (dtpts[j,i]*dtpts[j,i-1]) <0 then
begin
        channel:=j;
        HalfWave(i);

```

```

        end;
    end; {end of criterion loop}
    end; {end of 3200 data points}
    end; {end of 2 channels}
writeln;
for j:=1 to 2 do {Calculating percentages here}
    begin
        ttot:=0;
        for i:=1 to 5 do
            ttot:=ttot+datout[j,i];
        for i:=1 to 5 do datout[j,i]:=datout[j,i]/ttot;
        ttot:=0;
        for i:=6 to 10 do
            ttot:=ttot+datout[j,i];
        for i:=6 to 10 do datout[j,i]:=datout[j,i]/ttot;
        ttot:=0;
        for i:=11 to 15 do
            ttot:=ttot+datout[j,i];
        for i:=11 to 15 do datout[j,i]:=datout[j,i]/ttot;
    end;
    writeln('Doing output now');
    for j:=1 to 2 do
        begin
            write(fout, 'Channel ', j:0, chr(9));
            for i:=1 to 20 do
                write(fout, (datout[j,i])*100:0:2, chr(9));
            end;
        {***** OUTPUT TO SCREEN *****}
        writeln;
        writeln('
                                BD          SD          AD          TD          DD');
        for j:=1 to 2 do
            begin
                write('Channel ', j:0, ' ');
                for i:=1 to 5 do write((datout[j,i])*100:8:2);
                writeln;
            end;
        writeln;
        writeln('
                                BF          SF          AF          TF          DF');
        for j:=1 to 2 do
            begin
                write('Channel ', j:0, ' ');
                for i:=6 to 10 do write((datout[j,i])*100:8:2);
                writeln;
            end;
        writeln;
        writeln('
                                BH          SH          AH          TH          DH');
        for j:=1 to 2 do
            begin
                write('Channel ', j:0, ' ');
                for i:=11 to 15 do write((datout[j,i])*100:8:2);
                writeln;
            end;
        end;
    end;
end;

```

```
        end;
        writeln(fout);
        if (FirstFile = 0) then
        begin
            writeln;
            writeln('Hit <CR> to try another');
            readln;
        end
        else
        begin
            delay(2);
        end;

        close(fin);
        FindNext(DirInfo);

    end;
    close(fout);

until (ans='q') or (ans='Q');
end.
```

IMAGE EVALUATION TEST TARGET (QA-3)

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