The Impact of Trait Anxiety on Slow-Wave Sleep Processes.

Kristi Hulse Fuller

_Louisiana State University and Agricultural & Mechanical College_

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The impact of trait anxiety on slow-wave sleep processes

Fuller, Kristi Hulse, Ph.D.
The Louisiana State University and Agricultural and Mechanical Col., 1994
THE IMPACT OF TRAIT ANXIETY ON SLOW-WAVE SLEEP PROCESSES

A Dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
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Doctor in Philosophy

in

The Department of Psychology

by
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M.A. Louisiana State University, 1991
August, 1994
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<tr>
<td>EEG</td>
<td>Electroencephalography</td>
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<tr>
<td>EDA</td>
<td>Electrodermal Activity</td>
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<td>EDS</td>
<td>Electrodermal Storming</td>
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<td>EMG</td>
<td>Electromyography</td>
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<td>EOG</td>
<td>Electrooculography</td>
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<td>GAD</td>
<td>Generalized Anxiety Disorder</td>
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<tr>
<td>HVD</td>
<td>High Voltage Delta</td>
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<tr>
<td>NDD</td>
<td>Major Depressive Disorder</td>
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<td>NREM</td>
<td>Non-Rapid Eye Movement</td>
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<td>PD</td>
<td>Panic Disorder</td>
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<tr>
<td>PSWQ</td>
<td>Penn State Worry Questionnaire</td>
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<tr>
<td>PTSD</td>
<td>Post-Traumatic Stress Disorder</td>
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<td>REM</td>
<td>Rapid Eye Movement</td>
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<td>SS</td>
<td>Synchronized Sleep</td>
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<tr>
<td>STAI</td>
<td>State-Trait Anxiety Inventory</td>
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<td>SWS</td>
<td>Slow-Wave Sleep</td>
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<td>W</td>
<td>Awake</td>
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ABSTRACT

The sleep of high anxiety/worry and low anxiety/worry subjects were studied by means of nocturnal polysomnography. Fifteen high anxiety adult subjects were chosen to represent an analog Generalized Anxiety Disorder (GAD) population, and were selected from among individuals scoring within the upper 20th percentile on the trait portion of the State-Trait Anxiety Inventory (STAI) and the Penn State Worry Questionnaire (PSWQ). Fifteen low anxiety subjects were selected from among individuals scoring within the lower 50th percentile on those measures, and were matched with high anxiety subjects for age, sex, race, and Beck Depression Inventory (BDI) scores. Analysis of polysomnography variables revealed that high anxiety subjects took longer to fall asleep, had a smaller percentage of deep (slow-wave) sleep, and a larger percentage of light transitional sleep (stage 1 NREM). High anxiety subjects also had more stage 1 NREM periods, more micro-arousals during the first half of the sleep period, and a lower REM density relative to low anxiety subjects. In addition, high anxiety subjects had more electrodermal storming when SWS and REM sleep variables were co-varied. Results indicated that the pattern of disruption of sleep depth and continuity was very similar to that documented in clinical anxiety disorder patients, and was distinct from that typically
shown in depressed patients. A major implication is that anxiety and worry are associated with disrupted sleep, independent of whether or not a diagnosed anxiety disorder is present. Current results indicate that these factors are associated with the sleep disruption observed in anxiety disorders, independent of the presence of panic, previous exposure to trauma, or a disease process per se.
INTRODUCTION

Research investigating the impact of clinical anxiety disorders and experimental stress paradigms upon EEG sleep profiles suggest a characteristic disturbance of sleep in which disruption of the deeper stages of sleep appears to be primary. Prior to reviewing this literature, a general description of the methods and variables typically utilized in the area of sleep research, as well as a brief discussion of the general factors influencing sleep architecture, will be provided.

Research Procedures and Terminology

Experimental studies of human sleep have characteristically used a procedure referred to as polysomnography. Polysomnography is an electrophysiological recording procedure which utilizes three standardized measures to characterize sleep: electroencephalography (EEG), electrooculography (EOG), and electromyography (EMG). A standardized scoring procedure for polysomnography, developed by Rechtschaffen and Kales in 1968, has been consistently used for both research and clinical purposes. Because the variables discussed throughout the sleep literature make reference to terms defined by this scoring procedure, a basic outline of the scoring system and the definitions of standard the terms will be provided. According to this
scoring procedure, sleep stages are delineated primarily by EEG characteristics including relative amounts of alpha and delta waves, as well as distinctive wave formations such as K-complexes and sleep spindles.

A waking EEG is characterized by beta and alpha activity. Beta activity, which consists of low voltage, irregular, high frequency waves, represents a state of arousal and alertness. Alpha is associated with a state of relaxed wakefulness, with the eyes closed, and also is often present in small amounts during transitional sleep (Stage 1 NREM), the lightest stage of sleep. Alpha activity is represented by a relatively synchronous, low frequency wave (8-12 Hz) of low to moderate voltage. Delta activity, which is characteristic of the deepest most restorative sleep, consists of low frequency (1-4 Hz), synchronous, high voltage activity (Carlson, 1986). Theta activity is not specific to a particular stage of sleep and may be seen during stages 1 and 2 NREM and REM sleep. It is characterized by low frequency (4 to 7 Hz) low to moderate voltage that may be regular or irregular.

In addition to these characteristic wave forms, distinctive wave patterns, which include K-complexes and sleep spindles, are used in sleep scoring. K-complexes are defined as high voltage EEG wave forms having a well delineated negative sharp wave which is immediately followed by a positive component, with the total duration
of the complex lasting at least 0.5 seconds (about 3 to 4 Hz). Sleep spindles are defined by a burst of synchronous 12-14 Hz activity of low to moderate voltage, and must also be of at least a 0.5 second duration (Rechtschaffen & Kales, 1968).

In addition to EEG patterns, electrooculographic (EOG) and electromyographic (EMG) changes are used to define and measure the five stages of sleep (stages 1 - 4 NREM and stage 1 REM) across a normal night. EOG channels provide information about the speed, magnitude and direction of eye movements from electrodes placed near the outer canthus of each eye. EMG recordings provide information regarding the tonicity of muscles on and beneath the chin.

These sources of information (EEG, EOG, EMG) are used to delineate sleep stages in the following manner. EEG recordings immediately prior to sleep onset are characterized by a preponderance of alpha activity and/or low voltage, mixed frequency EEG activity. Although not included within the five sleep stages, this state is frequently referred to as Stage W in research literature. Stage 1 NREM is characterized by low voltage, mixed frequency EEG activity in the 2-7 Hz range in conjunction with a relative decrease in alpha (less than 50% of the standard measurement period, 30 seconds) and the presence of slow movements in the EOG channels. Stage 2 NREM shows
characteristic EEG patterns of K-complexes and sleep spindles on a background of relatively low voltage, mixed frequency EEG activity, while stages 3 and 4 NREM are defined by the occurrence of high voltage low frequency delta waves. REM sleep, which is generally associated with dreaming, occurs when a stage 1 EEG is present in conjunction with markedly decreased EMG activity and the occurrence of rapid eye movements in the EOG channels.

Stages 2-4 NREM are collectively referred to as synchronous sleep (SS) or NREM sleep by researchers. These stages are differentiated by visual ratings of the amounts of high voltage delta (HVD) activity in each EEG epoch: stage 2 NREM contains little HVD (less than 20%), stage 3 NREM an intermediate amount (at least 20% but not more than 50%), and stage 4 NREM is dominated by HVD activity (more than 50%). Stages 3 and 4 NREM are frequently referred to jointly as slow-wave sleep (SWS) or delta sleep.

General Factors Influencing Sleep Architecture

Sleep architecture refers to the orderly progression of stages which occur during an ordinary night's sleep. The five stages of human sleep alternate in a cyclic fashion, with periodic episodes of REM sleep occurring at approximately 90 minute intervals, and with each successive REM episode getting longer. In contrast, SWS
Awake

REM

Stage 1

Stage 2

Stage 3

Stage 4

Figure 1. Polysomnographic recordings illustrating wakefulness and sleep stages 1, 2, 3, 4, and REM. Based on Rechtschaffen & Kales, (1963) A Manual of Standardized Terminology, Techniques, and Scoring System for Sleep Stages of Human Sleep Subjects.
episodes becoming successively shorter as the sleep period progresses. Thus, SWS is generally confined to the first half of the night's sleep, and the majority of REM to the second half (Horne, 1976).

Two primary processes have been found to influence sleep architecture: circadian rhythmicity and homeostasis (Borbely, 1982). Circadian rhythms refer to the cycles of approximately 24 hours which govern the majority of physiological functions in the human body. Light appears to be the most important factor in synchronizing biological cycles, with the suprachiasmatic nucleus of the hypothalamus acting as the main oscillator, or biological clock, within the system (Hauri, 1992). In the context of sleep, homeostasis (the tendency toward stability in the normal physiological state of an organism) relates primarily to SWS. It is expressed by the preservation of deeper stages of sleep in restricted sleep paradigms (Horne & Wilkinson, 1985; Tilley & Wilkinson, 1984) and the preferential rebound of SWS during recovery sleep following sleep restriction (Tilley, Donohoe, & Hensby, 1987).

The duration of SWS is primarily influenced by the length of the prior period of wakefulness. Following sleep deprivation, the changes in SWS from baseline levels during subsequent recovery sleep has been shown to be directly related to outstanding SWS debt (Tilley et al.,
1987). In fact, the remarkable precision of SWS homeostatic control has led researchers to conclude that delta sleep is of considerable biological importance (Feinberg, March, Floyd, Jimison, Bossom-Demitrack & Katz, 1985), though the nature of that function is currently uncertain.

In addition to sleep debt, the timing of sleep exerts an effect upon sleep architecture through the influence of circadian rhythms. A major control variable of the circadian system is body temperature, which has been shown to drive sleep patterns in the absence of light/dark cycles (Czeisler et al, 1980). Sleep onset typically occurs as the body temperature falls, beginning in the early evening hours (5-7 pm) and temperature continues to fall through the mid-morning hours (until 5-7 am). Circadian rhythms appear to exert a particularly strong influence upon REM sleep; the majority of REM occurs during mid morning hours which constitute the low point in the temperature cycle.

In situations where circadian and homeostatic factors exert conflicting influences, the impact of homeostatic drive is more powerful in determining sleep architecture. Although restriction of the sleep period to the first half of the night results in large losses of stage 2 and REM sleep, with only a relatively small loss of SWS (Tilley & Wilkinson, 1984), the response of the sleep system
following this restricted schedule has been found to be relatively unaffected by the REM and stage 2 NREM debt, or by the timing of the subsequent sleep period. Even when recovery sleep occurred during the second half of the night, a period in which circadian factors are believed to be preferentially conducive to REM sleep, the composition of recovery sleep was primarily determined by SWS debt (Tilley, 1985).

In conjunction, these findings strongly support the hypothesis that obtaining a daily quota of SWS acts as the primary drive mechanism of the sleep system. In contrast, the overall amount of REM sleep appears to be related to the length of the sleep period. When the sleep period is extended, absolute amounts of SWS do not increase and remain concentrated during the early portion of the sleep period; the additional sleep is composed primarily of REM and stage 2 (Webb & Agnew, 1970). It has been hypothesized that REM latency is influenced by SWS; that REM onset occurs only after a sufficient amount of the drive for deeper sleep is satisfied (Horne, 1976).

**Functions of Slow-Wave Sleep**

Although substantial research exists suggesting the importance of obtaining a daily quota of SWS, there is no universally accepted theory regarding its purpose. Several theories of the role of SWS have been proposed; it
has been hypothesized to represent a crucial factor in information processing, as well as in cerebral energy conservation and physical and mental restitution.

Researchers supporting the role of SWS in information processing propose a sequential hypothesis in which the information gathered by the brain during the waking period is processed during sleep in two main stages, which correspond with synchronized sleep (SS; SWS) and REM sleep periods. Ambrosini, Sadile, Carnevale, Mattiaccio, and Giuditta (1987a; 1987b; 1991a; 1991b), in a series of studies which investigated the sleep patterns of a group of rats following exposure to a learning paradigm, conclude that synchronized sleep fulfills a primary role in the initial processing of information gathered during the preceding waking period. These conclusions were drawn from observations indicating that the structure of SS was determined by the nature of the previous waking experience (rats exposed to a two way active avoidance task vs. control rats left in home cages), with the average duration of SS episodes markedly increased in rats exposed to the learning paradigm. Rats that effectively learned (L) the task were found to differ from non-learning (NL) rats in the time of appearance and extent of the increments in the average duration of SS episodes followed by wakefulness versus SS episodes followed by paradoxical (REM) sleep. NL rats showed a greater tendency toward SS-
W periods, while L rats demonstrated a greater tendency toward extended SS periods followed by REM sleep. Based upon this information, it was suggested that memory traces lacking adaptive value may be cleared away during SS-W and SS-REM episodes, while the remaining memory traces may be retained and eventually stored again in more integrated form during SS-REM and REM episodes, respectively. These conclusions are problematic in several ways. The failure of these authors to distinguish between high amplitude SS (similar to SWS) and lighter stages of NREM sleep limit the specificity of their findings and may serve to obscure important differences in NREM sleep structure of post-acquisition sleep. In addition, it appears that there is an important uncontrolled variable, stress, which may be reasonably assumed to differ among control, learning, and non-learning rats; and which could have influenced sleep architecture.

Other basic animal research investigators have hypothesized that the decreased levels of mean cerebral metabolism, and markedly lowered metabolism in the thalamic sensory relays and cortex during SWS, supports a role for SWS in cerebral energy conservation (Ramm & Frost, 1986). Other theorists have expanded the energy conservation hypotheses to include restorative functions. SWS has traditionally been linked to general tissue restitution, primarily as a result of the increased
synthesis and release of human growth hormone which occurs during this period (Adams & Oswald, 1977). However, Horne (1983; 1985) reviewed available research regarding SWS and general tissue restoration and concluded that there is little convincing evidence supporting the role of SWS in this process. Rather, he proposed that sleep may actually represent a degenerative period due to the night-time fast. In the absence of evidence for a general tissue restoration hypothesis, it is proposed that SWS more accurately represents a period of cerebral restitution. In both animal and human research, SWS consistently has been found to reflect a degree of lowered cerebral metabolic activity relative to that found in other states of consciousness. In addition, SWS has the appearance of some form of recovery function, as it is the sleep state most highly correlated with the length of prior wakefulness. Horne also concludes that only a portion of sleep seems obligatory, and that this portion is associated with the SWS present during the first few hours of the sleep period.

The theory that SWS represents obligatory and restorative sleep, which is believed to be oriented towards brain restitution, is consistent with evidence indicating that obtaining a daily quota of SWS represents the primary sleep drive. Other periods of sleep, such as REM, represent a less primary drive and are thus
influenced by other factors such as the timing of the sleep period. These secondary sleep functions, which include both REM sleep and the lighter stages of NREM sleep (stages 1 & 2), are believed by many theorists to result from an adaptive, rather than restorative, function of sleep (Webb, 1988). They are proposed to have evolved as an adaptive behavioral responses associated with predator/predator and foraging requirements of the species.

Horne's hypothesis that SWS represents a period of cerebral restitution does not contradict the possibility that SWS is involved in the processing of information gathered during the waking period. Thus, although no firm consensus exists as to the exact function of SWS, the prevailing theories are not inconsistent with one another.

Failure to obtain sufficient amounts of delta sleep have been linked to specific decrements in daytime cognitive performance of healthy subjects. Partial or total sleep deprivation has been shown to produce a significant worsening of performance on vigilance and reaction time tasks (Bonnet, 1985; 1986b; Downey & Bonnet, 1987; Horne, Anderson, & Wilkinson, 1983; Tilley & Wilkinson, 1984). Horne and Wilkinson (1985) attempted to determine if the relationship between cognitive performance and sleep parameters was homogenous or if it had a specific correlation with a certain sleep parameter.
By reducing the last 2 hours of sleep each night during a 6 week period they were able to decrease stage 2 and REM, while leaving SWS unmodified. Under these conditions no deleterious effect upon daytime vigilance was observed. Thus, although it seems clear that daytime performance is affected by sleep deprivation, this decrement does not appear to occur with selective stage 2 NREM or REM sleep loss.

In addition, specific decrements in SWS have been found to be correlated with slowed reaction times in normal subjects. When a group of subjects with slow versus fast reaction times were compared on baseline sleep EEG measures, subjects with slow reaction times were shown to have significantly less delta sleep than subjects with fast reaction times (Jurado, Luna-Villegas, & Buela-Casal, 1989), leading researchers to suggest that attention after awakening is related specifically to the amount of delta sleep during the night. Healthy young subjects deprived of delta sleep by periodic awakenings have also been shown to have significant decreases in morning vigilance and nap latency (Bonnet, 1987), as well as significant increases in subjective ratings of fatigue and decreases in mood ratings suggestive of a blunting of emotional responses. These subjects reported feeling more unhappy, less friendly, less angry, and less clear-thinking following SWS deprivation. In summary, considerable research
evidence suggests that SWS represents the primary drive in the sleep system; and reduction of SWS has been demonstrated to have observable effects on behavior and mood during the subsequent waking period. This is of particular interest given that disruption of sleep architecture and reduction of SWS has been documented to occur in the presence of environmental stressors, and to constitute a prominent symptom in the clinical presentation of many psychiatric disorders.
Impact of Stress on Sleep Profiles

The effect of various stress paradigms on sleep architecture has received some direct research attention in both animal and human studies. Experimental designs used in these studies have typically differed along two continua: whether they involve acute or chronic stress and whether they involve physical or psychological stress.

Transient Stressors

The effects of acute situational anxiety on polysomnographic measures of human sleep have been documented across several experimental situations, with mixed findings. The pre-sleep viewing of a stressful film has been associated with an increased number of arousals from REM sleep and an elevated frequency of REMs (REM density) during REM periods. The disruption appeared to be specific to REM sleep; neither increased frequency of NREM arousals nor disruption or reduction of SWS were noted (Baekland, Koulack & Lasky, 1968). However, this early evidence of disruption of REM processes by transient stressors has not received subsequent research support.

For example, significant changes in sleep parameters were not found among eighteen skydivers undergoing the voluntary stress of a parachute jump. Neither novice nor experienced groups showed disruption of SWS or REM
processes during either the sleep period preceding or following the jump, suggesting that the anticipatory anxiety and physiological arousal associated with the jump did not significantly effect sleep in these individuals (Beaumaster, Knowles, & MacLean, 1978). However, these subjects represent a rather unique group in that they voluntarily sought the arousal associated with the parachute jump, a situation with little similarity to that of individuals experiencing clinical anxiety disorders or undergoing stressful life events.

Very little sleep research utilizing experimental paradigms involving exposure to transient stressors has been published, which may be due to the failure of these paradigms to produce large enough effects on subsequent sleep profiles to reach statistical significance. On the other hand, this trend may reflect the difficulty of doing ethical research on the effects of transient stressors that are of sufficient intensity to produce statistically significant differences. However, stronger and more consistent effects have been shown in subjects involved in ongoing stressful situations.

**Ongoing or Chronic Stressors**

Significant decreases in slow-wave sleep (in both animal and human subjects), as well as disrupted sleep continuity, have been documented in experimental paradigms
involving ongoing stressors of a more chronic nature. Decreases in slow-wave sleep (specifically stage 4 sleep) have been documented in a sample of medical students undergoing exams (Lester, Burch, & Dossett, 1967). In this investigation, 53 healthy subjects spent 10 nights in the sleep laboratory. During this 10 day period, sleep was measured for 3 consecutive nights surrounding 2 independent events (1) a laboratory manipulation designed to represent a mild acute stressor and (2) a day of medical exams. In addition, throughout data acquisition subjects were interviewed regarding significant events during the preceding day. Thus, days containing fortuitous real-life stress events were identified. These real-life stressors and the time period surrounding medical school exams were associated with the highest level of self-reported anxiety among subjects, and subject sleep profiles indicated a decrease in the percentage of stage 4 NREM sleep and an overall increase in awakenings as self-reported daytime stress increased. In addition, anecdotal information gathered during data acquisition suggested that subjects undergoing stressors in their personal lives, which were of a more severe and chronic nature than those represented by exams or the experimental manipulation, showed a more severe disruption of sleep EEG patterns, which was characterized by a complete absence of SWS. However, no significant effect on sleep parameters
were found for the time period surrounding the laboratory stressor, and the percentage of stage REM sleep showed no systematic relationship to self-reported stress in any of the experimental conditions.

Disruption of sleep parameters, most notably SWS processes and sleep continuity, also has been observed among both animal and human populations during periods of ongoing physical and psychological stress. The effects of chronic pain on sleep in the rat were investigated by Landis, Levine, and Robinson, 1989. The chronic pain condition, which involved adjuvant arthritis, was found to result in an increased sleep latency as well as general increases in the percentage of time spent awake and in light sleep. In addition, a significant decrease from baseline in the amount of high amplitude NREM sleep (SWS) was noted in animals following initiation of the chronic pain condition. Decreases in the amount of time spent in deep SWS have also been documented in animal models of chronic anxiety (Marczynski & Urbancic, 1988). Rats that underwent prenatal exposure to diazepam, which produced enduring postnatal deficits of brain benzodiazepine receptors, showed both a reduction of time spent in SWS and an inability to habituate to novel environments such as the radial arm maze. The behavior of "high anxiety" rats in the maze was characterized by numerous fecal deposits and more frequent than normal errors in working
memory, factors which are attributed to increased anxiety secondary to decreased benzodiazepine receptors. Although these behaviors within the maze environment suggest the presence of an animal model of anxiety, the observed decrements in SWS among "high anxiety" rats may actually represent a non-specific effect of the experimental manipulation upon GABA systems associated with SWS, rather than an "anxiety" effect.

Severe disruption of SWS processes have also been documented in human populations who have undergone cardiac surgery. These patients, who were considered to be undergoing extreme physical and psychological stress, displayed a nearly complete suppression of EEG indications of sleep until the second post-operative night (Anch, Orr, & Karacan, 1976). As recovery progressed, SWS remained suppressed longer than REM sleep. This pattern appears particularly noteworthy given the preeminence of the homeostatic drive for SWS, which would predict a preferential rebound effect for lost SWS (Tilley et al., 1987). Problematic confounds exist in interpreting data obtained from this population of critically ill hospital patients. For example, it is not clear to what extent these research subjects were receiving medications, such as benzodiazepines, which may have contributed to the suppression of SWS. In addition, "recovery" of these patients is not clearly defined. Physical recovery may
have progressed, but investigators did not document psychological recovery. Patient stress levels may have remained quite high, thereby contributing to the continued suppression of SWS.

**Acute Physical and Sensory Stressors**

In contrast to findings regarding chronic anxiety or physical stressors, experimental manipulations involving acute physical or sensory stressors have resulted in increased duration of SWS during the sleep period following the stressor, and increased stability of SWS processes as reflected by a decreased number of stage changes. Subjects who were exposed to an acute sensory stress in the form of increased daytime noise load showed increases in amount of SWS and in stage 4 NREM stability during the subsequent nights sleep (Fruhstorfer, Fruhstorfer, & Grass, 1984; Fruhstorfer, Fruhstorfer, Grass, Milerski, Sturm, Weismann, & Wiesel 1985; Fruhstorfer, Pritsch, & Fruhstorfer, 1988). These authors interpreted increases in SWS as indicative of an intensified need for recovery caused by increased central nervous system strain during waking.

The effects of acute physical stressors appear to be similar to those of sensory stressors. Physical stress in the form of extreme physical exercise has been reported to enhance subsequent SWS among physically fit subjects.
When the sleep of athletes completing a 92-km marathon was measured on 4 successive nights, significant increases in total sleep time and slow-wave sleep were found during the three nights following the race, with the most profound increase in stage 4 NREM sleep occurring during the sleep period immediately following the event (Shapiro, Bortz, Mitchell, Bartel, & Jooste, 1981).

Increases in NREM sleep were also observed during an experiment designed to measure the effect of an acute physical stressor on the sleep of infants (Emde, Harmon, Metcalf, Koenig, & Wagonfeld, 1971). These authors investigated the effect of routine circumcision on subsequent sleep. Circumcisions, which were performed without anesthesia in a newborn nursery, were typically followed by a prolonged NREM sleep period. A group of circumcised infants showed increases in NREM sleep from pre-operative baseline levels, which ranged from 41 to 121%, during the night following the operation. A control group of infants, monitored during this same time period, failed to show similar changes from baseline recordings.

Post-circumcision effects on NREM sleep were also reflected in a shorter latency to the first NREM period, an increased number of NREM periods, and an increased number of extended NREM periods. These authors interpret this increased tendency toward NREM sleep as fulfilling a
drive for conservation/withdrawal following exposure to an acute stressor.

**Summary of the Effect of Stress Paradigms upon SWS**

When stress is viewed as a broad unitary construct, overall findings for its effect upon SWS appear somewhat weak and inconsistent. However, when stressors are categorized along an acute vs. chronic continuum, a more cohesive pattern is evident. In general, acute physical or sensory stressors appear to be followed by an increased tendency toward SWS, while ongoing, chronic physical or psychological stress (anxiety) is associated with disrupted SWS. Thus, individuals with psychiatric disorders entailing emotional stress may be viewed as experiencing chronic psychological stress, and would therefore be expected to experience more consistent disruption of sleep processes than individuals exposed to transient stressors.

**Polysomnography Profiles of Psychiatric Patients**

Disruption of sleep architecture is a robust research finding for many psychiatric patient populations. Historically, it is the sleep of patients with affective disorders that has received the primary focus of research attention. Several abnormalities consistently have been documented in this population, including decreased sleep
continuity, diminished SWS, and altered REM sleep. Altered REM sleep findings include shortened latency to the first REM period, increased amount of REM sleep early in the night, and increased REM density relative to matched controls (Benca, Obermeyer, Thisted, & Gillin, 1992; Mendlewicz & Kerkhofs, 1991; Reynolds & Kupfer, 1987). Of these REM abnormalities, increased REM density appears to be the most consistent finding among depressed populations across age ranges (Lauer, Riemann, Wiegand, & Berber, 1991).

In contrast to the attention focused on sleep in affective disorder populations, very little research has been focused on sleep disturbances in anxiety disorder patients. When available research is considered, the most consistent findings across anxiety disorder groups are disturbed sleep continuity and reduced SWS. However, these findings lack specificity and are often noted in other psychiatric disorders as well, including depression, schizophrenia, alcoholism, dementia, and insomnia (Benca et al., 1992).

With the publication of the revised edition of the diagnostic criteria by the American Psychiatric Association in 1980 (DSM-III) and 1987 (DSM-III-R) anxiety disorders have been recognized as independent from depression, and have received increasing research attention. Recent research investigating sleep
disturbances in anxiety disorder patients has focused on delineating characteristic sleep profiles of individuals with clinical anxiety disorders such as Post-Traumatic Stress Disorder (PTSD), Panic Disorder (PD), and Generalized Anxiety Disorder (GAD). In addition, there has been a recent increase in interest in EEG sleep parameters as promising biological markers for differentiating primary anxiety vs. depressive disturbances, and in determining the viability of an independent diagnostic category of mixed anxiety and depression (Akiskal, Lemmi, Dickson, King, Yerevanian, & Van Valkenberg, 1984; Benca et al., 1992). A summary of available research investigating sleep EEG profiles in anxiety disorder patients will now be provided.

**Post-Traumatic Stress Disorder**

Sleep disturbance in individuals diagnosed with PTSD represents the most robust research findings pertaining to sleep in the anxiety disorders. Typical sleep EEG findings for individuals with PTSD include decreased sleep efficiency (decreased proportion of time asleep while in bed; increased sleep latency plus time awake after sleep onset), increased frequency of awakenings, decreased amounts of deep restorative sleep (slow wave sleep), increased amounts of light transitional sleep (stage 1 NREM), increased REM latency, and increased indices of
autonomic arousal and motor activity (Astrom, Lunde, Ortmann, Boysen, & Trojaborg, 1989; Glaubman, Mikulincer, Porat, Wasserman, & Birger, 1990; Kramer & Kinney, 1988; Mellman, Kulich-Bell, Kumar, & Nolan, 1993; van Kammen, Christiansen, van Kammen, & Reynolds, 1990). Individuals with PTSD also frequently report the reexperiencing of traumatic situations in the form of trauma related dreams. These dreams are unusual in the fact that they occur in the early portion of the sleep period during non-REM sleep and are accompanied by great autonomic arousal (Kramer & Kinney, 1988; van der Kolk, Blitz, Burr, Winthrop, & Sherry, 1984). In addition, individuals with PTSD have been found to show an atypical pattern of nocturnal arousals: There is an increased tendency toward arousal during the early portion of the sleep period, a time that is normally characterized by deeper stages of sleep and decreased tendency for arousal among control subjects (Fuller, Waters & Scott, 1993; Kramer & Kinney, 1988).

**Panic Disorder**

The available sleep data from individuals diagnosed with panic disorder is sparse and is plagued by methodological confounds which limit its value in clarifying the nature of sleep disturbances specific to anxiety. In particular, a high incidence of comorbid
Depressive disorders exist in many of the subjects used in investigations of sleep in panic disorder (PD).

Individuals with PD but without concomitant depression have not been found to differ consistently from normal controls on measures of sleep architecture or continuity (Dube, Jones, Bell, Davies, Ross & Sitram, 1985). In studies which have found significant differences, lower sleep efficiency (as reflected by increased sleep onset latency plus decreased total sleep time) has been noted in comparison to normal controls (Mellman & Uhde, 1989). However, methodological problems limit the usefulness of this data: In the study by Mellman and Uhde (1989), eight of the twelve PD patients reported a previous history of major depression, and six reported significant depression at the time of the investigation.

Sleep EEG data from a relatively homogenous sample of nine individuals diagnosed as PD with agoraphobia, who were free from depressive symptoms at the time of polysomnography, demonstrated increased movement time, decreased REM latency, and reduced REM density relative to normal controls (Uhde, Roy-Byrne, Gillin, Mendelson, Boulenger, Wittone, & Post, 1984). However, observed REM latencies were twice the length typically reported in affective disorder samples, and were not statistically different from previously published age norms (Williams et
al., 1974). Additional data obtained from patients participating in the study by Uhde and colleagues indicated significant negative correlations between measures of global anxiety with both total sleep time and REM percentage. In addition, the frequency of panic attacks was found to be negatively correlated with percentage of SWS. Thus, patients with the highest self-report of global anxiety reported the most frequent panic attacks and had the shortest total sleep time and lowest percentages of SWS and REM sleep.

A recent investigation (Stein, Enns, & Kryger, 1993) sought to alleviate the problematic confounds of co-morbidity with depressive disorders and lack of adequate control populations. These researchers compared the sleep of sixteen patients with PD, who did not currently meet criteria for major depression, to sixteen, healthy, age matched, control subjects. PD patients showed a moderate reduction in total sleep time and delta sleep relative to controls, as well as an increased number of stage 1 NREM episodes. In addition, a significant negative correlation was observed between Speilberger Trait anxiety scores and percentage of SWS. No other measures of anxiety or depression correlated significantly with any other sleep parameter, including any of the measures of sleep continuity.
Characteristic sleep patterns have been noted among individuals who experience nocturnal panic attacks. These nocturnal attacks have been found to be relatively prevalent, with 71% of non-hospitalized PD patients reporting their occurrence (Roy-Byrne, Mellman, & Uhde, 1988). Nocturnal panic attacks have been demonstrated to occur during non-REM sleep and to be associated with markedly increased autonomic arousal. In addition, a tendency toward increased REM latencies has been documented on nights in which panic occurs (Mellman & Uhde, 1989).

The frequency of co-morbid depression among individuals with PD has led some researchers to include "mixed" groups of PD and depressed individuals (Granhaus, Rabin, Harel, Greden, Feinberg, & Herman, 1986). Subjects with both PD and depression were found to show increased time to sleep onset as well as disrupted sleep continuity and decreased sleep efficiency relative to individuals with only depressive symptoms. In addition, individuals in this mixed group have been found to demonstrate REM latency times which are significantly longer than those shown by groups diagnosed as major depression without concomitant anxiety disturbances, and are not significantly different from REM latency times among normal individuals (Grundhaus et al., 1986).
Given the current lack of a specific knowledge base regarding the effect of pure anxiety disorders on sleep profiles, inclusion of this "mixed" grouping appears somewhat premature. Mixed samples have been found to have a longer duration of illness, increased severity of symptoms, increased impairment of functioning, and a higher incidence of co-morbid psychopathology (Dube, Jones, Bell, Davies, Ross, & Sitaram, 1985), thus suggesting that differences among mixed groups may actually represent other non-specific factors.

**Generalized Anxiety Disorder**

As recently as 1983 a paucity of sleep data existed on the complaint of generalized anxiety (Rosa, Bonnet, & Kramer, 1983), reflecting its residual status prior to the recent publication of DSM-III-R. Recent studies addressing sleep EEG findings among psychiatric patients diagnosed with generalized anxiety disorder (GAD) have typically compared these subjects to patient groups with affective disturbances such as dysthymia or major depression and/or normal control subjects.

A recent study which investigated sleep patterns among psychiatric outpatients with the DSM-III-R diagnosis of GAD found these individuals to have decreased sleep efficiency, decreased percentage of SWS, and increased awake time after sleep onset relative to healthy controls.
(Arriaga & Paiva, 1990). However, when patients with GAD were compared with patients diagnosed with primary dysthymia, they did not consistently differ on measures of SWS. Anxious subjects were found to differ from dysthymic subjects in that they had less total sleep time and showed a predominance of awakenings and stage 2 NREM sleep following initial sleep onset. Thus, the authors concluded that although overall reduction of SWS lacked specificity, it appeared to have different physiopathologic significance in these two psychiatric groups: Among subjects with GAD it seemed to represent an epiphenomenon resulting from reduction and fragmentation of sleep, while it was not associated with similar features of insomnia among dysthymic subjects.

In an uncontrolled investigation which compared outpatients diagnosed with GAD versus major depressive disorder (MDD) (Reynolds, Shaw, Newton, Coble, & Kupfer, 1983), both psychiatric groups demonstrated a pattern of disrupted sleep when compared to previously published age norms (Williams, Karacan & Hursch, 1974). Both sets of subjects showed decreased percentage of SWS and decreased sleep efficiency, as well as problems initiating and maintaining sleep which were evidenced by an increased number of awakenings and increased sleep onset latency. When compared directly to individuals with major depression, GAD subjects were found to experience an
increased amount of intermittent wakefulness, increased REM latency, and decreased REM percentage and density; measures of SWS and sleep continuity were not found to differ between groups. However, the usefulness of the results from this investigation is limited by poor subject selection: Individuals in the anxious group were also significantly depressed, and six of the ten anxious subjects also had concomitant personality or affective disorders. This prevalence of co-morbidity among the anxiety subjects limits the extent to which observed polysomnography profiles may be considered to reflect specific anxiety processes.

Similar results were obtained when a group of inpatients with GAD were compared with inpatients with MDD and normal control subjects. GAD patients were found to have increased sleep latency and decreased total sleep time relative to control subjects (Papadimitriou, Kerkhofs, Kempenaers, & Mendlewicz, 1988). In addition, these GAD subjects showed significantly longer REM latency and lower percentage of REM sleep than MDD subjects. However, contrary to other published research findings (Arriaga & Paiva, 1990; Reynolds et al., 1983), this sample of GAD patients showed fewer awakenings during the sleep period than MDD subjects. Although differences were not statistically significant, GAD subjects did show a
lower mean percentage of SWS relative to both MDD and control groups.

When GAD subjects were compared with a mixed subject group diagnosed with both MDD and GAD, as well as subjects with MDD without concomitant anxiety (Papadimitriou, Linkowski, Kerkhofs, Kempenaers, & Mendlewicz, 1988); no significant differences in sleep EEG were found between the mixed group and the GAD group. The mixed group showed longer REM latency than the MDD group, with values falling close to those of GAD patients. No differences were noted in percentage of SWS or other measures of sleep architecture between groups. However, no control group was available for comparison.

**Mixed Anxiety and Depression**

In addition to differences noted in individuals with clinical anxiety disorders, differences in EEG sleep profiles have been noted among affective disorder patients as a function of anxiety level (Akiskal, Lemmi, Dickson, King, Yerevanian, & van Valkenburg, 1984). A group of dysthymic patients with concomitant "anxiety neurosis" showed significant differences in sleep variables when compared to general dysthymic patients and a control group of medically ill patients. Subjects in the anxious dysthymic group included individuals with generalized anxiety disorder and/or panic disorder in conjunction with
chronic dysphoric symptoms. Insomnia was found to be a prominent presenting complaint among the anxious dysthymics, while "pure" dysthymics reported a high incidence of hypersomnia. In addition, anxious depressives showed higher degrees of arousal as measured by a greater number of awakenings and stage shifts than the other two groups, while dysthymics showed significantly decreased REM latency.

A recent investigation sought to clarify the effect of situational anxiety on the sleep of depressed individuals by examining the effect of severe stress on the polysomnography profiles of individuals with the diagnosis of endogenous MDD without co-morbid anxiety disorders (Monroe, Simmons, & Thase, 1992). Persons who had experienced severe stressors shortly before the onset of depression were found to have essentially normal REM latency values, while patients without such stress showed reduced REM latency values. When the effects of severe events were statistically removed, and the influence of increased "life stress" both before and after onset of depression was assessed, the relationship continued to hold: Individuals with high stress levels showed essentially normal REM latency values. This data supports a hypothesis that stress may influence REM latency through mechanisms other than those which are thought to determine etiologic differences. The authors suggest that life
stress prolongs REM latency, thereby approximating normal values among depressed individuals who would theoretically have abnormally short REM latencies. Unfortunately, reporting of sleep EEG findings in this study was confined to REM latency values, thus limiting the usefulness of results in determining the possible mechanism(s) by which stressors extend REM latency times. In addition, no control group of non-depressed individuals was available for comparison, thus leaving the effect of increased life stress upon REM latency in individuals without depressive disorders unclear.

**Summary of Psychiatric Findings**

In summary, polysomnographic findings across anxiety populations have been somewhat less consistent than results obtained from individuals diagnosed with endogenous depression. General findings include decreased SWS and increased REM latency, as well as decreases in total sleep time, sleep efficiency, and sleep continuity measures. Although overall reduction of SWS and disruption of sleep continuity have not been shown to be specific to anxiety disorder populations, multiple research findings suggest that the total amount of SWS, as well as sleep continuity and duration, are correlated with measures of clinical severity in anxiety populations, but are not similarly correlated in affective disorder
populations (Arriaga, Rosado, & Paiva, 1990; Mellman, Thomas, & Uhde, 1989; Papdimitrou, Linkowsky, & Kerkhofs, 1988).

The relation between anxiety and the disruption of SWS and sleep continuity is further strengthened by research findings among non-psychiatric individuals with high levels of generalized anxiety. When depression was held statistically constant in this population, a significant positive relationship was noted between anxiety ratings and number of awakenings, and a significant negative correlation was found between level of anxiety and amount of SWS (Rosa et al., 1983). In contrast, significant correlations were not present between these sleep parameters and depression ratings. In addition, these high anxiety individuals showed disruptions of sleep similar to those documented in clinical anxiety disorders, with a marked tendency toward less sleep and lighter sleep.

Additional evidence suggesting that a specific disruption of sleep continuity is characteristic of anxiety populations is provided by findings of an atypical arousal pattern among individuals with PTSD. However, this phenomenon has not been investigated in other anxiety disorder groups, and it is unclear to what extent it may be limited specifically to individuals previously exposed to traumatic situations. In addition, no formal scoring
criteria for arousals or awakenings was available prior to the standards published in 1992 (Bonnet et al., 1992), and this lack of methodological consistency limits the reliability of previous research results.

The area of polysomnographic research among anxiety disorder populations contains several other problematic confounds, including the frequent presence of co-morbid pathology in anxiety disorder populations (Sanderson, DiNardo, Rapee, & Barlow, 1990) and the long-standing nature of the psychiatric disturbance in many such research subjects (Akiskal et al., 1984; Reynolds et al., 1983). These factors call into question the assumption that it is primarily the anxiety level of the subjects which accounts for polysomnographic changes noted among these clinical groups, and necessitate further research on individuals with "pure" anxiety states which are not complicated by comorbid psychopathology.
THE PRESENT STUDY

Population of Interest

The purpose of the investigation was to clarify the nature of sleep disturbance associated with "pure" anxiety states. In order to investigate the impact of anxiety levels on sleep in individuals without concomitant psychopathology, a sample of individuals with an analogue of generalized anxiety disorder (GAD) was selected from among undergraduate students at Louisiana State University. Generalized Anxiety Disorder has been characterized as a relatively pure instance of high trait anxiety (Rapee, 1991). Age of onset is generally early and insidious, many individuals report the disorder as lifelong (Rapee, 1985). It has been characterized as the most "normal" anxiety disorder, and it is virtually impossible to define a cutoff point quantitatively distinguishing GAD from normal anxiety (Hoehn-Saric & MacLeod, 1992). There does not appear to be a clear qualitative demarcation distinguishing GADs from non-psychiatric populations with high levels of trait anxiety. In fact, cognitive differences between GAD subjects and normals have been found to be much the same as between normals who are high and normals who are low on measures of trait anxiety (Eysenck & Mathews, 1987). The tendency to worry, which is the central feature of GAD as defined by DSM-III-R, also has been found to be highly correlated
with trait anxiety (Borkovec, Robinson, Pruzinske, & Depree, 1983). In light of the similarities between individuals with high trait anxiety and those with GAD, an analog population of individuals with high trait anxiety was chosen as one that could be appropriately compared to normal individuals with low trait anxiety.

There is typically a long time between the onset of GAD (mid-teens) and presentation to a clinical facility, with an average period of almost 25 years (Rapee, 1991). During this time there is ample opportunity for the development of secondary psychopathology, such as depression, as a consequence of demoralization resulting from chronic anxiety (Akiskal et al., 1984; Barlow, 1992). Thus, by identifying a relatively young subject pool high in trait anxiety prior to their presentation to a clinical facility it was hoped that the confounding co-morbidity present in the psychiatric literature would be avoided.

The trait portion of the State-Trait Anxiety Inventory (STAI Appendix A) and the Penn State Worry Questionnaire (PSWQ Appendix B) were used as screening measures to identify this subject pool. The STAI is a frequently used, brief, self-report measure of state and trait anxiety which has been shown to be of adequate reliability and validity. Alpha coefficients for the trait anxiety component of the instrument range from .90 to .92 (Speilberger, 1983). Items for the trait portion
of the scale, which was utilized in the present investigation, were originally selected because they were highly correlated with the Manifest Anxiety Scale (Taylor, 1951), were stable over time, and were not much affected by situational stress (Taylor & Arnow, 1988). In general, the assumptions of the STAI have been confirmed. The STAI-state has been shown to be influenced by situational factors, while the STAI-trait has shown stable properties. STAI-trait test-retest reliability coefficients have been found to range from .76 to .86 across a 20 day delay (Speilberger, 1983).

The second selection instrument, the PSWQ (Appendix B), is a recently developed, brief self-report measure which has been shown to possess high internal consistency (alpha coefficient = .94) and a test-retest reliability coefficient of .92 (Meyer, Miller, Metzger, & Borkovec, 1990). The measure has also been shown to be stable over periods of 2 weeks (r = .75) and 4 weeks (r = .74), and to significantly discriminate college samples who met all, some, or none of the DSM-III-R criteria for generalized anxiety disorder. The PSWQ has also shown discriminant validity in that it successfully identified GAD from all other anxiety disorders (Brown, Antony, & Barlow, 1992).

These two selection measures were chosen for their relative brevity and sensitivity to factors associated with GAD. The STAI-trait was included due to its
widespread use as a research tool and its previous success in differentiating college samples into high versus low trait anxiety samples. Subjects selected by virtue of STAI-trait scores have shown significant differences in self-reported sleep patterns as well as measures of whole blood serotonin content (Davis, Dunlop, Shea, Brittain, & Hendrie, 1985). The PSWQ was included for its emphasis on cognitive factors associated with GAD. With the advent of DSM-III-R, emphasis of diagnostic criteria for GAD has shifted toward the cognitive component of the disorder, worry. Current trends also suggest that pathological worry (worry perceived as uncontrollable) will become an essential feature of the DSM-IV criteria (Barlow & DiNardo, 1991). Thus, it was anticipated that through inclusion of these two measures, a sample would be identified with the characteristics of high trait anxiety, as defined in previous research, as well as the cognitive component (worry) which most reliably distinguishes GAD as a diagnostic entity apart from other anxiety disorders.

High anxiety subjects were chosen from among individuals scoring within the top 20th percentile of the screening sample on both the STAI-trait and PSWQ; low anxiety subjects were chosen from among individuals scoring within the bottom 50th percentile on these measures.
Subjects were also administered the Beck Depression Inventory (BDI Appendix C; Beck, 1978; Beck, Ward, Mendelson, Mock & Erbaugh, 1961) during initial screening procedures. The BDI is the most frequently used self-report method for assessing level of depression (Shaw, Vallis, & McCabe, 1985). It has been shown to have adequate internal consistency (Beck & Steer, 1987; Reynolds & Gould, 1981), with alpha coefficients of .86 and .81 for psychiatric and non-psychiatric populations, respectively. The BDI has also been shown to have concurrent validity with clinician's ratings of depression (Beck et al., 1961; Brumberry, Oliver, & McClure, 1978). Subjects within the high and low anxiety groups were matched for BDI score (within 2 points) in order to avoid a difference in level of depression between groups. In addition, any subject scoring a BDI of 15 or higher was excluded from further participation, as this level has been reported to be indicative of clinically significant depressive symptoms (Beck & Steer, 1987).

After reading and signing a General Informed Consent (Appendix D) subjects were also screened orally to rule out the presence several psychiatric disorders which have been found to affect sleep, including panic disorder, PTSD, obsessive compulsive disorder, major depression, dysthymia, mania/hypomania, psychosis, and substance abuse. The format for this oral screening (Appendix E)
was based on DSM-III-R diagnostic criteria. The purpose of the screening was not formal diagnosis, but to identify and exclude subjects who may have met diagnostic criteria for previously listed psychiatric disorders. Thus, subjects were excluded if they reported significant psychiatric symptoms, even if sufficient information was not present to formulate a definite diagnosis.

Any subjects who reported significant symptoms suggestive of psychiatric disorders other than GAD were excluded from further participation. Subjects were also screened for the presence of confounding sleep disturbances by means of the Sleep Disorders Inventory (SDI; Appendix F). Subjects were excluded who endorsed items consistent with the presence of sleep apnea, periodic limb movements of sleep, restless leg syndrome, or various parasomnias (e.g. sleep walking). Subjects who reported relying upon alcohol or hypnotic medications to fall or stay asleep and individuals with irregular sleep-wake cycles (e.g. shift workers) were also excluded from participation. Other exclusion criteria included previous history of psychiatric treatment and regular use of illegal drugs or prescription medications known to affect sleep.

Variables of Interest

Measures of sleep architecture and quality, including sleep onset latency, REM latency, percentage REM,
percentage Stage 1 NREM, percentage SWS, total stage changes, and time awake after sleep onset were of interest to the present investigation. High anxiety individuals were expected to show greater disruption of sleep continuity, increased sleep latencies, more light transitional sleep, and reduction of SWS.

In addition to these traditional sleep parameters, micro-arousals (spontaneous transient arousals) during the first and second halves of the sleep period, number of stage 1 NREM episodes, and electrodermal activity during sleep were assessed in an attempt to maximize the sensitivity of polysomnographic recordings. Current researchers encourage the study of sleep micro-structure to increase the discriminative power of sleep EEG findings (Arriaga & Paiva, 1990), and it was hoped that inclusion of these additional parameters would aid in clarifying the nature of SWS attenuation and sleep continuity disturbance previously documented among anxiety subjects.

In addition, prior research has lacked standardization of scoring for arousals. The present study utilized recently published standardized criteria for arousals (Bonnet et al., 1992) to provide a more reliable index of these events. These micro-arousals are of considerable clinical importance, and have been shown to be equally as disruptive of sleep quality and restorativeness as full awakenings or movement arousals.
Periodic micro-arousals have been linked to suppression of SWS, as well as degraded mood, increased sleepiness, and decline of performance on vigilance tasks during the subsequent waking period (Bonnet, 1985; 1986a; 1986b; 1987).

It was also hypothesized that individuals with high trait anxiety would show an atypical arousal pattern similar to that evidenced in PTSD subjects, which is characterized by a relatively greater number of arousals during the first half of the sleep period than is seen in normal control subjects. In individuals with normal sleep patterns, spontaneous awakenings from sleep are hypothesized to reflect the rising phase in the underlying circadian rhythm during the sleep period (Gillberg & Akerstedt, 1983). Early portions of the nocturnal sleep period are associated with a rapid cooling of the body's core temperature, a phenomenon which is correlated with high probability of sleep onset, sleep maintenance, and the presence of SWS. As the core temperature rises during the latest portions of the nocturnal sleep period, the probability of arousal becomes much greater and the likelihood of sleep onset and sleep maintenance is reduced, even in conditions of sleep deprivation (Gillberg & Akerstedt, 1983; Webb, 1982). Thus, spontaneous arousals tend to occur predominantly during the second half of the night's sleep while circadian factors are more
favorable to awakenings and the lighter stages of sleep are more prevalent.

Data from previous research investigations (Fuller et al., 1993; Kramer & Kinney, 1988) have demonstrated a pattern of arousals in control subjects which is consistent with this model, and an arousal pattern among PTSD subjects which is not. In contrast to control subjects, who show the expected decreased arousability during the early portion of the sleep period, individuals with PTSD show a constant pattern of arousability across the sleep period, with significantly greater amounts of disruption during the early portions of the night's sleep. Kramer and Kinney (1988) proposed that individuals who have experienced universal trauma, and who are experiencing acute symptoms, thereafter maintain an altered physiology which is reflected in a disrupted sleep rhythm characterized by inappropriately high levels of ANS activity during the early portion of the night's sleep, with resulting interruption of SWS processes. It was of interest whether this pattern is specific to individuals who have been exposed to traumatic stimuli and have subsequently developed PTSD, or whether a similar pattern of arousals would be observed among non-psychiatric subjects with high levels of generalized anxiety.

The number of Stage 1 NREM episodes was also of interest as a measure of sleep continuity and quality.
Brief episodes of Stage 1 NREM appear to reflect decreased continuity of sleep because they are transitional in nature and subjects are easily awakened during these periods. Because Stage 1 NREM episodes may be very brief in nature, their relative number is not necessarily reflected in total Stage 1 NREM percentage (Stein et al, 1993).

In addition, electrodermal measures were used to determine whether there was a greater tendency towards periods of excessive electrodermal activity (EDA) in highly anxious subjects. Previous research examining all night recordings of electrodermal responses have suggested an increased tendency toward EDA during SWS and decreased EDA during REM sleep (Koumans, Tursky, & Solomon, 1968). These periods of increased spontaneous electrodermal activity are referred to as electrodermal "storming." Storming rates have been found to be associated with spontaneous EDA response rates during waking, which have been associated with level of anxiety (Taylor & Arnow, 1988). Storming also appears to be associated with presleep trait variables: For example, individuals with high levels of storming have been shown to score higher on MMPI measures of anxiety and lower on measures of ego strength than individuals with less nocturnal storming (McDonald, Shellenberger, Koresko, & Kinzy, 1976).
This phenomenon has also been associated with increased situational anxiety in a group of medical students studied by Lester et al. (1967). These subjects showed a disruption of sleep architecture during periods of increased daytime stress, characterized by increased awakenings and decreased SWS. In addition, subjects showed elevations of electrodermal activity during periods of situational stress. Electrodermal activity, which is primarily a SWS phenomenon under normal conditions, increased in all EEG stages as daytime stress increased. In contrast, subjects undergoing extreme stressors, who showed suppression of SWS sleep, showed decreased electrodermal activity from baseline conditions; thus suggesting that SWS in a necessary condition for notable electrodermal activity in the sleeping state. It was of interest to the present investigation whether high levels of trait anxiety were associated with elevations of storming activity similar to those observed during periods of situational stress.

Summary and Hypotheses

In summary, the following polysomnography variables were investigated in the current study: sleep onset latency, REM latency, percentage of stage 1 NREM, percentage of SWS, percentage of REM sleep, number of arousals during the first half of the sleep period, number
of arousals in the second half of the sleep period, number of stage changes, time awake after sleep onset, REM density, number of stage 1 NREM episodes, and percentage of epochs containing electrodermal storming. Based on available research findings, the following hypotheses were offered regarding variables related to depth of sleep, sleep continuity, electrodermal activity, and sleep onset:

1. High anxiety subjects were expected to show greater disruption of sleep continuity as reflected by higher frequency of micro-arousals, higher frequency of stage 1 NREM episodes, more time awake after sleep onset, and a greater number of stage changes relative to low anxiety subjects.

2. High anxiety subjects were expected to differ from low anxiety subjects in distribution of arousals across the sleep period, with high anxiety subjects showing a greater frequency of arousals during the first half of the sleep period.

3. High anxiety subjects were expected to spend a greater amount of total sleep time in light, transitional sleep (stage 1 NREM) and less time in deep restorative sleep (SWS) relative to low anxiety subjects.

4. High anxiety subjects were expected to show longer latency to sleep onset relative to low anxiety subjects.
5. High anxiety subjects were expected to show higher levels of electrodermal "storming" relative to low anxiety subjects.

Research findings regarding the relationship between REM sleep variables and anxiety is inconsistent, although these variables have been found to be relevant to depression. They were included in the present investigation in an attempt to clarify their relationship to "pure" anxiety states. Although somewhat inconsistent, available research suggests the following patterns:

6. High anxiety subjects were expected to show longer REM latency, lower REM density, and lower REM percentage relative to low anxiety subjects.
METHOD

Subject Selection

Subjects were recruited from among the undergraduate population at Louisiana State University. A total of 435 subjects underwent preliminary screening which consisted of the trait portion of the State-Trait Anxiety Inventory (STAI-trait) the Penn State Worry Questionnaire (PSWQ) and the Beck Depression Inventory (BDI). The STAI-trait and the PSWQ, which are designed to measure the related constructs of trait anxiety and worry, showed a high positive correlation among the screening sample (r = .683, \( p < .001 \)), which was similar to the correlation noted between these measures (r = .64) by Meyer and colleagues (1990) in their sample of 386 undergraduates. Although significantly correlated, the STAI-Trait and PSWQ shared only 47% of the variance, which is consistent with assertions by Meyer and colleagues that the PSWQ measures the independent construct of worry.

These selection measures also showed low positive correlations with the BDI in the screening sample: The BDI correlated .41 and .31 with the STAI-trait and PSWQ, respectively. These low, positive correlations likely reflect the dysphoria (or negative affect) component shared between generalized anxiety and depression (Clark & Watson, 1991; Watson Clark & Carey, 1988). The characteristics of the initial screening sample, from
which final subjects were selected, are presented in Table 1. STAI-trait scores are expressed in T-scores while PSWQ and BDI scores are expressed as raw scores.

From this sample, 36 individuals were identified as appropriate for screening interviews. High anxiety subjects were chosen from among individuals scoring within the upper 20th percentile on both the STAI-trait and the PSWQ. Low anxiety subjects were chosen from among individuals scoring within the lower 50th percentile on these measures. BDI scores were used as a matching variable to assure that groups did not differ on this measure of depression. Subjects were matched within 2 BDI points across groups, and individuals scoring above 15 were excluded from further participation.

During the course of screening, five subjects met previously defined exclusion criteria and were eliminated from further participation. Subjects scoring within the appropriate ranges on the STAI-trait, PSWQ, and BDI, and who did not meet exclusion criteria, were scheduled to complete one night of polysomnography at the Baton Rouge General Medical Center. Thirty-one subjects underwent polysomnography. Subject groups were equated for age (+/- 3 years), race, and sex to avoid possible confounds. One subject was excluded prior to data analysis due to inability to recruit an age-matched subject in the other group. The final sample consisted of 15 subjects per
Table 1. Characteristics of Screening Sample (n = 435).

<table>
<thead>
<tr>
<th>Measure</th>
<th>Mean</th>
<th>Standard Deviation</th>
</tr>
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<tbody>
<tr>
<td>STAI-trait</td>
<td>52.2</td>
<td>11.0</td>
</tr>
<tr>
<td>PSWQ</td>
<td>46.7</td>
<td>14.9</td>
</tr>
<tr>
<td>BDI</td>
<td>7.5</td>
<td>6.2</td>
</tr>
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</table>
group; each group contained seven males, eight females, thirteen Caucasian and two African-American students. Additional sample characteristics are described in Table 2.

Subjects were instructed not to nap during normal waking hours and to rise by 8:00 am on the day of polysomnography. They were also instructed to abstain from alcoholic beverages 24 hours prior to sleep recordings, and not to consume caffeine after 6pm on the scheduled date of polysomnography. All subjects reported that they had been compliant with these instructions.

Hospital Procedures

Prior to scheduling of polysomnography the procedure was fully explained to subjects (Appendix G). Upon arrival at the sleep laboratory subjects were asked to read and sign an informed consent for polysomnography (Appendix H). A standard polysomnographic hookup, which is fully described in Appendix I, was performed according the montage presented in Appendix J. Subjects slept in a private room in the sleep laboratory of the Baton Rouge General Medical Center. A research assistant was present to monitor them from an adjacent control room throughout the night. All recordings were made on a Grass Electroencephalograph Model 8-16 D. All subjects arrived
Table 2. Characteristics of Final Subject Sample 
(n = 30).

<table>
<thead>
<tr>
<th>Measure</th>
<th>Mean</th>
<th>Standard Deviation</th>
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<tbody>
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<td><strong>High Anxiety Subjects:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
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<td>(9.1)</td>
</tr>
<tr>
<td>STAI-trait</td>
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<td>PSWQ</td>
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<td>(15.7)</td>
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<td>BDI</td>
<td>7.9</td>
<td>(2.6)</td>
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<tr>
<td><strong>Low Anxiety Subjects:</strong></td>
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<td></td>
</tr>
<tr>
<td>Age</td>
<td>24.8</td>
<td>(9.2)</td>
</tr>
<tr>
<td>STAI-trait</td>
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<td>(5.0)</td>
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<tr>
<td>PSWQ</td>
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<td>(8.8)</td>
</tr>
<tr>
<td>BDI</td>
<td>7.1</td>
<td>(2.6)</td>
</tr>
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</table>
at the laboratory prior to 21:00, and hookup procedures were completed by 22:30.

Subjects were awakened by 06:30, the electrodes were removed, and they were instructed to dress for the day. Subjects then completed a sleep diary (Appendix K) in order to determine a subjective rating of his/her sleep quality during the recording period, and were given extra credit vouchers. If a subject had any remaining questions regarding the procedure they were answered at this time.

**Scoring**

Analysis and scoring of the polysomnographic records followed Rechtsaffen and Kales' (1968) standardized method for human subjects. Of the 30 research records, 10 were scored independently by 2 individuals experienced in the scoring of polysomnography records. These scorers were found to have adequate inter-rater reliability; the average percent agreement between independent scorers was 92.7% (standard deviation = 2.4%, range of 88.2% to 95.7% agreement). Scoring of micro-arousals followed the recent standardized guidelines described by Bonnet et al. (1992), which are fully described in Appendix L.
RESULTS

Variables of interest were defined in the following manner: Sleep onset was defined by the number of minutes between lights-out and initial presence of stage 2 NREM sleep. Micro-arousals were defined according to ASDA criteria as presented in Appendix L, and summed separately for the first and second halves of the sleep period. REM latency was determined by the number of minutes between sleep onset and initial presence of REM sleep. REM density was determined by dividing the number of observed rapid eye movements by the total minutes spent in REM sleep. Time awake after sleep onset was defined the number of minutes of wake time following sleep onset. Number of stage 1 NREM epochs was determined by counting the number of independent stage 1 NREM episodes and dividing by the total hours of sleep. Number of stage changes was determined by counting the total number of stage changes and dividing by the total hours of sleep. Percentage of electrodermal storming (EDS) epochs was determined by dividing the total number of epochs which contained storming activity by the total number of sleep epochs. Percentages of stage 1 NREM, SWS, and REM sleep were determined by dividing the total amount of time in each of these stages by the subject's total sleep time. Grand means and standard deviations on these 12 variables of interest are provided in Table 3.
Table 3. Grand means and standard deviations for the total subject sample (n = 30) on the 12 dependent variables.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Grand Mean</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep Onset Latency</td>
<td>35.03 minutes</td>
<td>(26.26)</td>
</tr>
<tr>
<td>Percentage SWS</td>
<td>17.75%</td>
<td>(7.54)</td>
</tr>
<tr>
<td>Number of Stage 1 NREM Periods</td>
<td>4.59/hour sleep</td>
<td>(1.53)</td>
</tr>
<tr>
<td>Arousals (first half)</td>
<td>30.87 total</td>
<td>(16.32)</td>
</tr>
<tr>
<td>REM Density</td>
<td>2.93/minute REM</td>
<td>(1.23)</td>
</tr>
<tr>
<td>Percentage Stage 1 NREM</td>
<td>9.29%</td>
<td>(5.10)</td>
</tr>
<tr>
<td>Percentage REM Sleep</td>
<td>17.79%</td>
<td>(6.04)</td>
</tr>
<tr>
<td>Time Awake After Sleep Onset</td>
<td>30.23 minutes</td>
<td>(25.02)</td>
</tr>
<tr>
<td>Number of Stage Changes</td>
<td>18.15/hour sleep</td>
<td>(5.42)</td>
</tr>
<tr>
<td>Percentage Storming</td>
<td>36.57% sleep epochs</td>
<td>(20.03)</td>
</tr>
<tr>
<td>REM Latency</td>
<td>111.95 minutes</td>
<td>(53.14)</td>
</tr>
<tr>
<td>Arousals (second half)</td>
<td>27.73 total</td>
<td>(11.29)</td>
</tr>
</tbody>
</table>
Among the 12 dependent variables, significant correlations were present between the number of arousals during the first half of the sleep period and five other variables: percentage of SWS ($r = -.50$), percentage of stage 1 NREM sleep ($r = .73$), number of stage changes ($r = .57$), number of arousals during the second half of the sleep period ($r = .56$), and number of stage NREM 1 episodes ($r = .64$). The number of stage 1 NREM episodes was correlated significantly with three additional variables: percentage of SWS ($r = -.47$), time awake after sleep onset ($r = .52$), and number of stage changes ($r = .60$). A significant negative correlation ($r = -.53$) was also present between percentage of REM sleep and REM latency, in which subjects with longer REM latency showed a lower percentage of REM sleep. A complete listing of correlations among dependent variables is provided in Table 4.

The data set was screened for possible outliers within groups. Univariate and Multivariate screenings were performed through separate within group comparisons for high and low anxiety groups. Initial data screening revealed the presence of one variable, number of stage 1 NREM episodes, which contained two values representing univariate outliers. These data points fell more than three standard deviations above the high anxiety group mean for two subjects in the high-anxiety group. To
Table 4. Correlations between dependent variables for entire subject sample (n = 30; * = p < .01, ** = p < .001).

<table>
<thead>
<tr>
<th></th>
<th>NREM1%</th>
<th>SWS%</th>
<th>REM%</th>
<th>Onset</th>
<th>REML</th>
<th>AR1</th>
<th>AR2</th>
<th>TimeW</th>
<th>#Chgs</th>
<th>Storm</th>
<th>#NREM1</th>
<th>REMD</th>
</tr>
</thead>
<tbody>
<tr>
<td>NREM1%</td>
<td>1.0</td>
<td>0.43</td>
<td>0.28</td>
<td>0.03</td>
<td>0.37**</td>
<td>0.33</td>
<td>0.17</td>
<td>0.73**</td>
<td>0.22</td>
<td>0.26</td>
<td>0.46</td>
<td>0.09</td>
</tr>
<tr>
<td>SWS%</td>
<td>1.0</td>
<td>0.16</td>
<td>0.35</td>
<td>0.05</td>
<td>0.50*</td>
<td>0.06</td>
<td>0.40</td>
<td>0.16</td>
<td>0.13</td>
<td>0.47*</td>
<td>0.09</td>
<td></td>
</tr>
<tr>
<td>REM%</td>
<td>1.0</td>
<td>0.22</td>
<td>0.53*</td>
<td>0.02</td>
<td>0.08</td>
<td>0.32</td>
<td>0.06</td>
<td>0.04</td>
<td>0.26</td>
<td>0.19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Onset</td>
<td>1.0</td>
<td>0.04</td>
<td>0.14</td>
<td>0.21</td>
<td>0.36</td>
<td>0.15</td>
<td>0.08</td>
<td>0.45</td>
<td>0.29</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>REML</td>
<td>1.0</td>
<td>0.03</td>
<td>0.09</td>
<td>0.34</td>
<td>0.26</td>
<td>0.13</td>
<td>0.15</td>
<td>0.14</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AR 1</td>
<td>1.0</td>
<td>0.56*</td>
<td>0.23</td>
<td>0.57</td>
<td>0.08</td>
<td>0.64**</td>
<td>0.03</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AR 2</td>
<td>1.0</td>
<td>0.04</td>
<td>0.34</td>
<td>0.11</td>
<td>0.42</td>
<td>0.30</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TimeW</td>
<td>1.0</td>
<td>0.44</td>
<td>0.07</td>
<td>0.52*</td>
<td>0.07</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>#Chgs</td>
<td>1.0</td>
<td>0.01</td>
<td>0.60**</td>
<td>0.14</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Storm</td>
<td>1.0</td>
<td>0.00</td>
<td>0.24</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>#NREM1</td>
<td>1.0</td>
<td>0.10</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>REMD</td>
<td>1.0</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**KEY**

NREM1% = percentage stage 1 NREM
SWS% = percentage SWS
REM% = percentage REM
Onset = sleep onset latency
TimeW = time awake after sleep onset
REML = REM Latency
Storm = percentage of storming epochs
REMD = REM density.

#Chgs = number of stage changes per hour sleep

#NREM1 = number of stage 1 NREM episodes

AR1 = number of arousals in first half of sleep period

AR2 = number of arousals in second half of sleep period
eliminate the potential impact of extreme values during data analysis, these values were removed and group means were substituted for missing values. No other univariate outliers were identified, and no missing data was present within the data set. No within group multivariate outliers were identified as determined by examination of Mahalanobis's distance ($p < .001$); and evaluation of assumptions of normality, linearity, homogeneity of variance-covariance matrices, and multicollinearity revealed no threat of Type 1 error inflation during multivariate analysis.

A between-groups multivariate analysis of variance (MANOVA) was used for initial analysis, and the 12 dependent variables were entered into the model simultaneously. With the use of Wilks' criterion, the high and low anxiety groups were found to differ significantly on the combined dependent variables, $F(12,17) = 5.34, p<.001$. Means and standard deviations for these variables are provided in Table 5.

Significant differences (Univariate F-tests) between subject group means suggested that high anxiety subjects took longer to fall asleep (longer sleep onset latency) and had lighter sleep (lower SWS percentage and greater percentage Stage 1 NREM sleep) which was more prone to arousal, especially during the first half of the sleep period (more arousals during the first half of the sleep
period and higher number of stage 1 NREM episodes). In addition, high anxiety subjects showed a significantly lower REM density, as well as non-significant trends towards less REM sleep and more time awake after sleep onset. Univariate F-tests for dependent variables are provided in Table 6.

A direct discriminant function analysis (DFA) was then utilized to determine to what extent group membership (high versus low anxiety group) could be determined using objective polysomnography variables as predictors. A significant discriminant function was identified ($p < .001$) which accounted for 79.01% of the variance between groups on the 12 dependant polysomnography variables. When the rule of thumb ($r > .30$) for interpretation suggested by Tabachnick and Fidell (1989) was applied, pooled within groups correlations between discriminating variables and canonical discriminant function (Table 7) indicated that high anxiety subjects could be reliability identified to show the following sleep pattern: They took longer to fall asleep, had less SWS, and more frequent stage 1 NREM episodes.

Results of DFA classification procedure for the total sample of 30 subjects indicated that 29 (96.67%) were classified correctly. All high anxiety subjects and 14 of 15 low anxiety subjects were correctly classified, while one low anxiety subject was classified with the high
anxiety group. The generalizability of this classification procedure is limited due to the fact that the relatively small sample size did not allow for a cross-validation to be performed, possibly leading to an artificially inflated classification rate. However, the primary purpose of the classification procedure in the current investigation was not classification, but rather to determine the consistency of sleep pattern across subjects. Results of the classification procedure did show that the discriminant function reliably distinguished among individuals across the entire subject sample. Thus, a frequent methodological criticism of psychiatric sleep research, that a few subjects account for the majority of the variance between groups (Anch, Browman, Mitler, & Walsh, 1988), does not apply to the current classification.

Arousal patterns were further investigated through a repeated measures analysis of variance to determine if the two groups differed in their pattern of arousals across the sleep period (i.e. first half verses second half of the sleep period). Results indicated that groups did not differ significantly in overall number of arousals \( F(1,28) = 2.09, p = .16 \), but a significant interaction \( F(1,28) = 9.76, p < .01 \) was present between group and distribution of arousals across the first versus second halves of the sleep period.
Table 5. Means and standard deviations for high and low anxiety subjects on polysomnography dependent variables (* = p < .05; ** = p < .01).

<table>
<thead>
<tr>
<th>Variable</th>
<th>High Anxiety Group</th>
<th>Low Anxiety Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>** Sleep Onset Latency</td>
<td>51.03 minutes (13.51)</td>
<td>19.03 minutes (12.13)</td>
</tr>
<tr>
<td>** Percentage SWS</td>
<td>13.51% (7.12)</td>
<td>21.98% (5.34)</td>
</tr>
<tr>
<td>** Number of Stage 1 NREM Periods</td>
<td>5.41/hour sleep (1.32)</td>
<td>3.78/hour sleep (1.29)</td>
</tr>
<tr>
<td>* Arousals (first half)</td>
<td>37.47 total (18.52)</td>
<td>24.27 total (10.72)</td>
</tr>
<tr>
<td>* REM Density</td>
<td>2.43/minute REM (.85)</td>
<td>3.42/minute REM (1.37)</td>
</tr>
<tr>
<td>* Percentage Stage 1 NREM</td>
<td>11.19% (5.93)</td>
<td>7.39% (3.30)</td>
</tr>
<tr>
<td>Percentage REM Sleep</td>
<td>15.86% (5.46)</td>
<td>19.27% (6.16)</td>
</tr>
<tr>
<td>Time Awake After Sleep Onset</td>
<td>37.63 minutes (31.89)</td>
<td>22.83 minutes (12.73)</td>
</tr>
<tr>
<td>Number of Stage Changes</td>
<td>19.10/hour sleep (5.92)</td>
<td>17.21/hour sleep (4.89)</td>
</tr>
<tr>
<td>Percentage Storming</td>
<td>9.85% epochs (21.47)</td>
<td>33.30 epochs (18.63)</td>
</tr>
<tr>
<td>REM Latency</td>
<td>115.23 minutes (54.12)</td>
<td>108.67 minutes (53.83)</td>
</tr>
<tr>
<td>Arousals (second half)</td>
<td>27.47 total (10.95)</td>
<td>28.00 total (11.99)</td>
</tr>
</tbody>
</table>
Table 6. Univariate F-tests with (1,28) degrees of freedom.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Univariate F Value</th>
<th>Univariate Significance of F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep Onset Latency</td>
<td>17.45</td>
<td>.000</td>
</tr>
<tr>
<td>Percentage SWS</td>
<td>13.57</td>
<td>.001</td>
</tr>
<tr>
<td># Stage 1 NREM periods</td>
<td>11.64</td>
<td>.002</td>
</tr>
<tr>
<td>Arousal (first half)</td>
<td>5.70</td>
<td>.024</td>
</tr>
<tr>
<td>REM Density</td>
<td>5.65</td>
<td>.024</td>
</tr>
<tr>
<td>Percentage Stage 1 NREM</td>
<td>4.69</td>
<td>.039</td>
</tr>
<tr>
<td>Percentage REM Sleep</td>
<td>3.31</td>
<td>.079</td>
</tr>
<tr>
<td>Time Awake After Onset</td>
<td>2.79</td>
<td>.106</td>
</tr>
<tr>
<td>Number of Stage Changes</td>
<td>.91</td>
<td>.348</td>
</tr>
<tr>
<td>Percentage Storming</td>
<td>.80</td>
<td>.380</td>
</tr>
<tr>
<td>REM Latency</td>
<td>.11</td>
<td>.741</td>
</tr>
<tr>
<td>Arousal (second half)</td>
<td>.02</td>
<td>.900</td>
</tr>
</tbody>
</table>
Table 7. Pooled within groups correlations between discriminating variables and canonical discriminant function.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>*Sleep Onset Latency</td>
<td>.40683</td>
</tr>
<tr>
<td>*Percentage SWS</td>
<td>-.35865</td>
</tr>
<tr>
<td>*# NREM1 episodes</td>
<td>.33214</td>
</tr>
<tr>
<td>Arousals (first half)</td>
<td>.23260</td>
</tr>
<tr>
<td>REM Density</td>
<td>-.23163</td>
</tr>
<tr>
<td>Percentage Stage 1 NREM</td>
<td>.21092</td>
</tr>
<tr>
<td>Percentage REM Sleep</td>
<td>-.17727</td>
</tr>
<tr>
<td>Time Awake After Sleep Onset</td>
<td>.16253</td>
</tr>
<tr>
<td>Number of Stage Changes</td>
<td>.09286</td>
</tr>
<tr>
<td>Percentage Storming</td>
<td>.08691</td>
</tr>
<tr>
<td>REM Latency</td>
<td>.03244</td>
</tr>
<tr>
<td>Arousals (second half)</td>
<td>-.01239</td>
</tr>
</tbody>
</table>
As illustrated in Figure 2, subjects in the high anxiety group had a higher mean level of arousals during the first half of the sleep period than the low anxiety group, while both groups had a similar mean number of arousals during the second half of the sleep period. During the first half of the sleep period, high anxiety subjects showed a mean of 37.47 arousals compared to the mean of 24.27 for low anxiety subjects. During the second half of the sleep period, the low anxiety subjects had a mean of 28.00 arousals and the high anxiety subjects had a mean of 27.27 arousals.

In order to clarify the interpretation of polysomnography dependent variables, the groups were compared on additional objective sleep parameters and subjective sleep estimates. Evaluation of this additional data revealed that high anxiety and low anxiety groups did not differ in total amount of bed time or total sleep time. Thus, observed differences in dependent variables were not attributable to differences in opportunity for sleep or total amount of sleep obtained.

Estimates of subjective sleep quality were obtained from subjects' responses on the Daily Sleep Diary (Appendix K) which they filled out on the morning following polysomnography. Numerical values for subjective sleep quality were obtained by summing questions seven (Rate the quality of last night's sleep),
Figure 2. Mean number of arousals across the first and second halves of the sleep period for high and low anxiety subjects.
and six (How rested do you feel this morning?) Numerical values for subjective estimates of difficulty falling asleep represent the sum of responses from questions five (How difficult was it for you to fall asleep last night?), eight (What was your level of physical tension when you went to bed last night?), and nine (Rate your level of mental activity when you went to bed last night).

Subjects were asked to rate their sleep during the night of polysomnography in comparison to their sleep at home on a "typical" night. Subjects responded on five-point Likert scales with higher numbers representing poorer perceived sleep quality and increased difficulty falling asleep. The reliability of these subjective measures were evaluated through the use of Chronbach's Alpha. Alpha coefficients were .80 for measures of sleep onset difficulty (questions #6 and #7; Appendix K) and .81 for subjective measures of sleep quality (questions #5, #8, and #9; Appendix K). Thus, these subjective measures appear to be adequately reliable. Subject groups did not differ in either of these subjective measures of sleep; both groups responded with mid-range likert scores, suggesting that the sleep that they obtained in the laboratory was neither markedly good or poor relative to a "typical" nights sleep. Means and standard deviations for these general measures are provided in Table 8.
Table 8. Means and standard deviations for general sleep parameters and subjective estimates.

<table>
<thead>
<tr>
<th>Variable</th>
<th>High Anxiety Group</th>
<th>Low Anxiety Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Time in Bed</td>
<td>7.35 hours (.39)</td>
<td>7.36 hours (.53)</td>
</tr>
<tr>
<td>Total Sleep Time minutes</td>
<td>353.23 minutes (50.06)</td>
<td>371.51 minutes (107.77)</td>
</tr>
<tr>
<td>Subjective Sleep Quality</td>
<td>5.66 (1.59)</td>
<td>5.60 (1.88)</td>
</tr>
<tr>
<td>Subjective Onset Difficulty</td>
<td>8.20 (2.48)</td>
<td>7.47 (2.48)</td>
</tr>
</tbody>
</table>
DISCUSSION

The current investigation found significant differences in several objective polysomnography variables between a group of high anxiety subjects, who were chosen to represent an analog GAD population, and a group of matched control subjects with lower anxiety levels. Discriminant function analysis indicated that the two subject groups could be differentiated by a discriminant function (97%) in which high anxiety subjects were reliability identified as taking longer to fall asleep, having less SWS, and showing more frequent stage 1 NREM episodes. MANOVA and follow-up ANOVA indicated that, in addition, high anxiety subjects showed a significantly higher frequency of arousals during the first half of the sleep period, lower REM density, and a higher percentage of stage 1 NREM sleep.

In general, these results suggest that healthy individuals with high levels of generalized anxiety and worry, and without other psychiatric co-morbidity, experience a pattern of sleep disruption that is very similar to what has been documented in clinical anxiety disorders, and which is quite distinct from what has been observed among individuals experiencing dysthymia and/or major depression. Hypotheses based on research data from studies using generalized anxiety disorder subjects (Arriaga & Paiva, 1990; Papadimitriou et al, 1988;
Reynolds et al., 1983), which predicted greater latency to sleep onset (Hypothesis 4), lighter sleep (Hypothesis 3), and disruption of sleep continuity (Hypothesis 1), were supported. High anxiety subjects showed longer sleep latency and less deep sleep as reflected in less SWS and more light transitional sleep (stage 1 NREM). Disruption of sleep continuity in the high anxiety subjects was indicated both by a greater number of episodes of light transitional sleep throughout the night and by more transient arousals from sleep (micro-arousals) during the first half of the sleep period.

In addition, the pattern of micro-arousals across the sleep period in high anxiety subjects was found to differ from low anxiety subjects in a manner which supported Hypothesis 2, and was consistent with previous research findings in clinical samples suffering from Post-Traumatic Stress Disorder (Fuller, Waters, & Scott, in press; Kramer & Kinney, 1988): The high anxiety subjects, like PTSD subjects, showed a greater arousability during the first half of the sleep period relative to low anxiety subjects and normal controls. This finding suggests that the observed pattern of micro-arousals in individuals with PTSD is likely to be a function of the elevated baseline anxiety (and perhaps worry) prevalent in the PTSD population, and is not specific to individuals who have been previously exposed to a traumatic event.
This shared component of elevated baseline anxiety/worry may also account for other (PSG) findings that were common to both PTSD patients and analog GAD subjects in the present investigation. In addition to increased arousability during the first half of the sleep period, both samples had low sleep efficiency (longer sleep latency and time awake after sleep onset), lower amounts of deep restorative sleep (SWS), and more light transitional sleep (stage 1 NREM) (Astrom et al, 1989; Fuller et al, in press; Glaubman et al, 1990; Kramer & Kinney, 1988; Mellman et al, 1993; van Kammen et al, 1990).

Results of the present investigation are also generally consistent with recent research findings from a sample of panic disorder (PD) patients who were relatively free of co-morbidity (Stein et al., 1993). These panic disorder patients showed a significantly higher number of stage 1 NREM episodes and less SWS relative to matched controls, and their sleep disturbance was most pronounced during the first half of the sleep period. Thus, it appears that the pattern of disrupted sleep continuity and disturbed depth of sleep shown by the analog GAD subjects in the current investigation is consistent with findings among clinical research samples including GAD, PTSD, and PD.
The lack of sleep depth (low percentage of SWS and high percentage of stage 1 NREM) and disruption of sleep continuity observed during the current investigation were also generally consistent with previous research investigating sleep parameters among non-clinical populations with high trait anxiety (Rosa et al, 1983) and in human and animal populations exposed to chronic, ongoing stressors (Anch, Orr and Karacan, 1976; Landis et al., 1989; Lester, et al, 1967; Marczynski & Urbancic, 1988). However, subjects in the current investigation did not show the significant mean increase in electrodermal activity predicted in Hypothesis 5 which has been previously noted among individuals exposed to the ongoing stress of medical school exams (Lester et al, 1967), nor did they show the low levels of electrodermal storming previously observed among individuals with chronic PTSD (Fuller et al, in press).

Thus, results of the current investigation failed to directly clarify the apparently complex relationship between chronic anxiety states, situational stressors, and electrodermal activity during sleep. However, hints about this relationship were provided when data from the current study were subjected to an additional analysis in which SWS percentage, REM percentage, and REM density were co-varied to remove their theorized influence on electrodermal storming (EDS).
In the context of normal sleep, EDS activity occurs primarily during SWS (Koumans et al., 1968). Therefore, it was hypothesized that individual differences in the amount of SWS may have affected the "window of opportunity" for EDS activity. Previous research has also shown that EDS is rare in REM sleep (Koumans et al., 1968; McDonald et al., 1976), probably due to inhibited thermoregulation during REM sleep. Therefore, the impact of individual differences in the prominence of REM sleep (as measured by the percentage of REM and REM density) were also seen as possible confounding variables effecting the "window of opportunity" for electrodermal activity.

In order to control for the hypothesized effects of these variables on EDS activity, the percentage of SWS, REM sleep percentage, and REM density were entered as co-variates in a hierarchial MANOVA framework. When these variables were co-varied, stepwise analyses indicated that the percentage of storming assumed a significant value (Roy-Bargmann Stepdown F (1,25) = 7.61, p < .01), with high anxiety subjects showing a significantly greater percentage of storming epochs. These findings indicate that the relation between electrodermal storming and anxiety is complex, as it is affected by the degree of both SWS and REM sleep.

The present findings, that high anxiety subjects had more electrodermal storming and less SWS than low anxiety
subjects, is consistent with previous research findings which indicate that, among healthy young adults, anxiety states both decrease the amount of SWS observed during polysomnography and increase storming activity (Lester et al., 1967). However, the relationship between anxiety, EDS, and REM sleep variables among subjects in the current investigation is less clear. Although the mean percentage of REM sleep the high anxiety subjects was smaller than for the low anxiety groups, the groups were not statistically different on this measure. High anxiety subjects did show a significantly lower REM density, which is suggestive of less sleep satiation (Aserinsky, 1969; 1971; 1973). These findings suggest that, for high anxiety subjects as a group, REM sleep was somewhat less prominent in their sleep architecture. Given its importance as a co-variate, REM percentage, though not significantly lower for high anxiety subjects, still exerted influences on EDS. Thus, the results of the current investigation provide possible clues to the relation between anxiety and nocturnal electrodermal storming activity, an apparently complex interaction between EDS activity, SWS percentage, REM percentage, and REM density. However, the exact relationship between trait anxiety and EDS activity requires further investigation.
In attempting to clarify the impact of trait anxiety upon sleep architecture, the pattern of correlations between dependent variables in the current investigation is of theoretical interest. The number of arousals during the first half of the sleep period was positively correlated with several variables, including the number of stage 1 NREM periods, percentage of stage 1 NREM sleep, and number of stage changes. It appeared that increased early arousals were associated with more frequent overall stage changes, and specifically with shifts into light, transitional sleep; and that such shifts were associated with overall increases in percentage of the sleep period spent in light sleep. In contrast, both the number of arousals during the first half of the sleep period and the number of shifts into light, transitional sleep were negatively correlated with the percentage of the sleep period spent in deep, restorative sleep (SWS).

These patterns strongly suggest a relation between repeated arousals during the early portions of the sleep period and less sleep depth (more light sleep and less SWS). However, due to the correlational nature of findings, it can not be determined to what extent either increased early arousability or decreased SWS is the causal element in this relation, or whether a third underlying mechanism is accounting for the observed relations. However, as these variables also were found to
differ between high and low anxiety/worry subjects who were otherwise equivalent, the anxiety/worry variable appears to have explanatory power. Anxiety level may account for observed polysomnography (PSG) findings indirectly through the reduction of sleep depth (less SWS, more stage 1 NREM), permitting arousals by external stimuli which would not arouse individuals in deeper stages of sleep. Conversely, anxiety level may have acted to increase arousability directly, especially during the first half of the sleep period, via the direct inhibition SWS and a congruent increase in stage 1 NREM.

Previous research has indicated that increased arousals may represent the causal factor in observed PSG findings; micro-arousals produced by external stimuli during the nocturnal sleep period have been demonstrated to result in suppression of SWS, as well as degraded mood and increased sleepiness upon awakening (Bonnet, 1985; 1986a; 1986b; 1987). However, further research is needed to clarify possible causal links between reduced SWS and increased arousability during the first half of the sleep period, and to determine to what extent relations between these variables may be specific to anxious samples. In answering these questions it would be important to investigate whether similar relations are present in groups of individuals defined by other variables associated with SWS decrements; such as age, depression,
or schizophrenia (Benca et al., 1992; Williams et al., 1974), or whether they are specific to anxious individuals.

The current investigation failed to find significant differences between high and low anxiety groups in REM latency or percentage of REM sleep (Hypothesis 6), variables which have been highly inconsistent in anxiety disorder samples, but which have been observed to differ consistently between depressed patients and controls. REM sleep abnormalities which typically have been observed in depressed samples include shortened latency to the first REM period and increased REM percentage, especially during early portions of the sleep period (Benca et al., 1992; Reynolds & Kupfer, 1987). Failure of the current investigation to find reliable evidence of differences in these variables between "pure" samples of healthy young adults with high versus low trait anxiety suggest that previous inconsistent research findings may have been a reflection of co-morbidity confounds among psychiatric subjects; specifically, depression in anxiety disorder patients acting to decrease REM latency and increase REM percentage and density.

However, Hypothesis 6 was supported in that a significant difference between groups in REM density was found. Research findings in depressed samples have also consistently found increased REM density relative to
matched controls (Benca, Obermeyer, Thisted, & Gillin, 1992; Mendlewicz & Kerkhofs, 1991; Reynolds & Kupfer, 1987). REM density appears to represent the most robust biological marker, across age ranges, differentiating the sleep of depressed individuals relative to anxious and normal control subjects (Lauer Riemann, Wiegand, & Berber, 1991). Differences in REM density were observed in the current investigation, with high anxiety subjects showing a lower REM density. This result is consistent with previous research findings in panic disorder patients (Uhde, et al., 1984), and is opposite of the pattern which characterizes depressed individuals.

REM density has been hypothesized to be an indication of sleep satiation, with greater REM density indicating higher levels of satiation. In studies of healthy young adults permitted to sleep to their fullest capacity, REM density has been found to increase throughout the sleep period (Aserinsky, 1968; 1971; 1973), reaching its maximal capacity at 7.5 to 10 hours. Furthermore, when subjects were studied continuously across a 54 hour period, prior sleep was found to be positively correlated with REM density, whereas the length of previous waking period was negatively correlated (Aserinsky, 1973). When viewed in this context, the significantly lower REM density found in the high anxiety group may be considered an indication that these subjects have not reached a level of sleep
satiety equal to that of the less anxious subject group. This explanation is consistent with research indicating that more anxious individuals tend both to report preferring to sleep for longer periods of time (McCann & Stewin, 1987) and to show evidence of increased daytime sleepiness (Roehrs, Timms, Zwghuizen-Doorenbos, Buzenski, & Roth, 1990) relative to less anxious control subjects. Thus, the observed differences in REM density between groups appears to reflect decreased sleep satiation in high anxiety subjects, which is likely a function of the decreased sleep depth and continuity also documented in polysomnography findings.

The present investigation failed to find significant differences between high and low anxiety groups in overall number of stage changes or in total time awake after sleep onset. These measures appear to have been less sensitive than other measures included in the analysis. The number of stage changes, which reflected both shifts into lighter and deeper stages of sleep, appeared to be less sensitive to differences in anxiety levels than a measure that represented only shifts into light, transitional sleep (number of stage 1 NREM episodes). In a similar manner, the number of waking epochs appeared to be less sensitive to differences in anxiety level than the prevalence of micro-arousals early in the sleep period and the number of stage 1 NREM episodes. This suggests that it was the
disruption of sleep micro-structure, rather than the time spent consciously awake, that was influenced by anxiety. The finding that the two groups did not differ in subjective self-report measures of sleep quality was unexpected given the significant differences found between the groups in objective PSG measures of sleep depth and continuity. This contradiction between objective and subjective measures appears to have resulted from the method used to assess subjective sleep quality, in which subjects were asked to compare how they slept in the laboratory to a "typical" night of sleep for them. Subjects who felt that they "typically" slept badly may have given subjective estimates of a bad night in the lab which, although non restorative, was no worse than usual. These scores would not differ from scores of subjects who typically slept well and also slept well in the lab.

The fact that subjects spent only one night in the sleep laboratory creates a possible confound when comparing the results of the current study with previous research findings. The first night in the sleep laboratory has been shown to be associated with increased sleep latency, decreased REM, and decreased stage 4 sleep in normal individuals (Williams, Karacan, & Hursch, 1974). This effect is lessened with subsequent nights spent in the laboratory setting, and has resulted in the common research practice of allowing an adaptation night in the
sleep laboratory prior to data collection. This proved unfeasible in the present investigation due to limited resources and need to maximize sample size.

Control subjects showed indications of a possible mild first night effect in that they showed slightly less REM sleep and slightly more stage 1 NREM sleep than predicted by normative values for their age range (Williams et al, 1974). However, polysomnography results indicated that control subjects' percentage of SWS, sleep onset latency, and REM latency fell within expected ranges despite the absence of an adaption night. Thus, the current data do not suggest the presence of a noteworthy first night effect among control subjects. Due to the lack of a relevant comparison group, it is difficult to determine to what extent a first night effect may have been present among high anxiety subjects. However, because the sleep of high and low anxiety subjects was examined under identical experimental conditions the absence of an adaption night is not problematic in interpreting the current results. Furthermore, previous research findings regarding possible differences in response to the first night spent in the sleep laboratory for anxious subjects do not suggest that these individuals show a more disrupted sleep pattern under novel conditions than do normal control subjects. Rather, available research has suggested that clinical anxiety disorder
samples (including PTSD and GAD) as well as non-clinical samples of highly anxious subjects, show few changes from the first to the second nights spent in a sleep laboratory (Reynolds et al., 1983; Rosa et al., 1983; van Kammen et al., 1990), and may actually show less habituation to the lab environment than control subjects (van Kammen et al., 1990). These latter findings actually suggests a possible second night confound when comparing anxious and normal control subjects. Thus, the possible presence of a first night effect does not appear to represent a confound in the interpretation of observed differences in polysomnography variables between the high and low anxiety groups in this study.

Summary and Implications

Results of the current investigation suggest that a characteristic pattern of sleep disturbance is present in highly anxious and worried individuals without comorbidity and other confounds prevalent in the psychiatric literature. Objective polysomnographic findings indicate that the sleep pattern of these individuals is consistent with patterns documented in various anxiety disorder samples, and is clearly distinct from that shown in individuals with depressive disorders. High anxiety subjects were reliably identified as taking longer to fall asleep, having less SWS, and showing more frequent stage 1
NREM episodes than low anxiety subjects. The high anxiety subject group also showed a significantly higher frequency of arousals during the first half of the sleep period, lower REM density, and a higher percentage of stage 1 NREM sleep than the low anxiety subject group. In addition being statistically different, this observed sleep disruption in highly anxious subjects appears to represent a clinically significant phenomenon. In contrast to low anxiety control subjects, who fell within normal ranges, high anxiety subjects showed clinically significant sleep onset insomnia and decreased sleep depth. Although no significant differences in mean EDS levels were present during initial analyses, when SWS and REM variables were entered as co-variates, results suggested that high anxiety subjects showed relatively higher levels of EDS activity than low anxiety subjects.

Disruption of sleep continuity and diminished SWS has been documented in both anxious and depressed samples (Benca et al., 1992). However, the relation between anxiety level and REM sleep variables observed during the current investigation is distinct from patterns documented in depressed samples. Current results revealed that typical REM findings in depressed samples, which include increased REM density, decreased REM latency and increased REM percentage, are absent among individuals with "pure" trait anxiety. In fact, anxious and worried subjects
showed less REM density than low anxiety control subjects, findings opposite that of depressed subjects.

Decreased REM density in a group of subjects with "pure" trait anxiety suggests that, although disruption of sleep depth and continuity are not unique to anxiety disorder populations, decreased SWS in anxious individuals may be associated with a specific unique decrement in sleep satiation. In this context, it is particularly noteworthy that the disruption of sleep in anxious individuals in the current investigation appeared to occur primarily during the initial portion of the sleep period, a time which is characterized by a prominence of deep sleep and low arousability in normal subjects. This portion of the sleep period has also been shown to be significantly disrupted in clinical anxiety disorders, and has been specifically associated with episodes of nocturnal panic among PD subjects (Mellman & Uhde, 1989) and traumatic nightmares among individuals diagnosed with PTSD (Kramer & Kinney, 1988; van der Kolk et al., 1984).

This pattern suggests that the sleep of anxious individuals may be particularly vulnerable to disruption during early portions of the sleep period. Thus, future researchers should give specific attention to the initial half of the sleep period. In addition, further investigation of this initial disruption may provide clues regarding the relations between, and possible underlying
mechanisms accounting for, objective polysomnography findings and other symptoms, such as the hyper-vigilance and psychophysiological insomnia frequently observed in anxiety disorder populations.

In conclusion, results of the current investigation indicate that anxiety and worry affect are associated with disrupted sleep, independent of whether or not a diagnosed anxiety disorder is present. Current results indicate that these factors are associated with a pattern very similar to the sleep disruption observed in anxiety disorders, independent of the presence of panic, previous exposure to trauma, or a disease process per se. The relatively strong findings in the present study, in comparison to the often inconsistent findings in clinical anxiety disorder samples, suggest the need to concentrate on controlling for co-morbidity and other confounds frequently present in psychiatric subject samples. In addition to attempting to recruit relatively "clean" clinical subjects, future investigations of sleep profiles in psychiatric samples should avoid premature inclusion of "mixed" diagnostic groups until such time as the specific impact of trait anxiety upon sleep is understood. Prior to such understanding, future research which attempts to clarify the possible causal links between reduced sleep depth, disrupted sleep continuity, and anxiety/worry is needed. Replication of the reduced REM density finding
among high anxiety subjects, as well as the apparently complex relationship between anxiety level, EDS, SWS, REM variables are also indicated in order to evaluate the robustness of these findings and to establish to what extent they may be specific to anxious and worried individuals.
REFERENCES


Fuller, K., Waters, W., & Scott, O. An investigation of slow wave sleep processes among individuals with chronic post traumatic stress disorder. Journal of Anxiety Disorders, in press.


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101 - 102

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APPENDIX B

PENN STATE WORRY QUESTIONNAIRE

Please mark the following items with the number (1-5) which best indicates how well the statement describes you.

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not typical of me at all</td>
<td>Very typical of me</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. _____ If I do not have enough time to do everything, I do not worry about it.
2. _____ My worries overwhelm me.
3. _____ I do not tend to worry about things.
4. _____ Many situations make me worry.
5. _____ I know I should not worry about things, but I just cannot help it.
6. _____ When I am under pressure I worry a lot.
7. _____ I am always worrying about something.
8. _____ I find it easy to dismiss worrisome thoughts.
9. _____ As soon as I finish one task, I start to worry about everything else I have to do.
10. _____ I never worry about anything.
11. _____ When there is nothing more I can do about a concern, I do not worry about it anymore.
12. _____ I have been a worrier all of my life.
13. _____ I notice that I have been worrying about things.
14. _____ Once I start worrying, I cannot stop.
15. _____ I worry all of the time.
16. _____ I worry about projects until they are done.
APPENDIX D

GENERAL INFORMED CONSENT

I hereby agree to participate in a research project designed to study the sleep patterns of undergraduates. I understand that I will be asked to complete questionnaires and answer interview questions regarding my sleep practices and mood, as well as my use of medications and other substances which are known to influence sleep. I understand that my participation is voluntary and that I am able to discontinue at any time without penalty. I further understand that my responses are confidential and that my name will in no way be associated with the data from the study. I acknowledge that I have been given an opportunity to ask questions regarding study and understand that I may ask questions following the completion of the study.

_________________________________  __________________________
Your Name                                   Date

_________________________________
Researcher
APPENDIX E
SCREENING INTERVIEW

Subject Number:

1. Previous Psychiatric History:
Have you ever had treatment or been hospitalized for anxiety, depression, alcohol abuse or other emotional or psychological problems?  Y  N

If Yes to 1- When did these problems occur?

____________________________________________________________________________________

2. Panic Disorder:
Have you had times when you felt a sudden rush of intense fear or discomfort in which you felt physical symptoms such as shortness of breath, rapid heartbeat, trembling, shaking, choking sensations, nausea, dizziness, or chest pain; and/or were concerned that you would loose your mind or die?  Y  N

If Yes to 2
Have these feelings come "out of the blue" or unexpectedly? I mean that they were not associated with a particular situation and did not occur only while you were ill or under the influence of a substance such as caffeine.  Y  N

How many times has this happened during the past month?

____________________________________________________________________________________

3. Post-Traumatic Stress Disorder:
Have you experienced any extremely stressful, life threatening or traumatic event such as serious physical injury, assault, or seeing someone badly hurt or killed in the past which still troubles you?  Y  N

If Yes to 3
Do you reexperience this event through "flashback" episodes or nightmares?  Y  N.

Do you have intrusive thoughts about this event or experience extreme anxiety in situations which remind you of this event?  Y  N

4. Social/Simple Phobia
4a. Are you unusually afraid of objects or situations the average person is not disturbed by, such as heights, air travel, or certain animals?  Y  N
If yes to 4a
What objects or situations cause you anxiety?

Does this situation or object always make you very anxious, and do you feel that your fear is excessive and somewhat unreasonable? Y N

Do you avoid situations which may evoke this fear? Y N

Do your fears and/or your avoidance associated with this object or situation interfere with your daily life? Y N

4b. Are you uncomfortable doing certain things most people do; like eating in a restaurant, using a public restroom or speaking in a group of people because you are afraid of embarrassing yourself? Y N

If yes to 4b
What type of activities make you very uncomfortable?

Does this discomfort interfere with your social life or your ability to successfully perform your job or schoolwork? Y N

5. Obsessive-Compulsive Disorder
5a. Are you bothered by recurrent thoughts, impulses, or images that you can't stop from coming into your mind, and which you feel are intrusive and senseless? This is not the same as worrying about things that might happen. I mean things like a parent having repeated impulses to kill a loved child, or a religious persons having recurrent blasphemous thoughts. Y N

If Yes to 5a-What kind of thoughts trouble you?

Do you try to ignore these thoughts or do something to keep them out of your mind? Y N

5b. Do you often repeat some act over and over again that doesn't seem to make sense and that you find distressing; such as washing something over and over, counting things or checking something repeatedly? Y N
If Yes to 5b-What are these actions? Are they related to certain situations?

6. **Major Depression:**
Did you ever have a period of time, which lasted 2 weeks or longer, when you felt depressed, sad, hopeless, or lost interest in almost all of your usual activities? Y N

If yes to 6
During this time did you notice changes in your appetite, sleep or energy level? (if yes) What kinds of changes did you notice?

How about changes in the way you felt about yourself and the future such as feelings of low self esteem, worthlessness, or hopelessness? (if yes) Tell me about these feelings

During this time did you notice any differences in your ability to think or make decisions? (if yes) Tell me about these changes

Have you been feeling this way nearly every day for the last two weeks? Y N

7. **Dysthymia:**
In the last two years have you felt down, blue, depressed or have lost interest in things that usually give you pleasure? Y N

If yes to 7
Have you felt this way more days than not for most of the day during the past two years? Y N
What is the longest period during the past two years in which you did not feel down or depressed? 

8. **Mania/Hypomania**
Did you ever have a period of time, at least several days long, when you felt extremely good or high- a feeling very different from just being in a "good mood?" I am talking about things like feeling that you possessed special powers, having a decreased need for sleep, racing thoughts, or feeling a pressure to keep talking such that others were not able to keep up with you in conversation. Y N
If yes to 8
Tell me about this time; did you do anything that caused negative consequences such as unrestrained spending or sexual activity?

Did you spend a lot more time than usual doing things like studying, working, or social activities?

9. Psychosis:
Has there ever been a period of time when you had strange experiences such as hearing voices or seeing visions which other people could not see or hear? Y N

Have there ever been a time when people had trouble understanding you because your speech was mixed up or because you didn't make sense in the way that you were talking? Y N

Have you ever had the feeling that something odd was going on around you, that people were doing things to test you or antagonize you or hurt you so that you felt you had constantly be on guard? Y N

If yes to 10- When did this happen, explain

Did you have difficulty performing your usual activities, maintaining your social relationships, and/or taking care of yourself during this time?

10. Substance Abuse
How much do you usually drink?
Do you take recreational or illegal drugs such as marijuana, LSD, Cocaine? Y N

If subject reports alcohol or drug use
How often and in what situations do you typically use drugs/alcohol
Has the use of drugs or alcohol ever interfered with your school or work performance, or interfered in your relationships with friends and family? Y  N If yes, how has it interfered?
APPENDIX F

SLEEP DISORDERS INVENTORY
INSTRUCTIONS

The Sleep Disorders Inventory is a questionnaire that is designed to obtain information that will assist in the description and diagnosis of a patient's sleep disorder. It also provides information that is helpful in determining the causes and the correct treatment of a patient's sleep disorder.

Please answer these questions to the best of your ability. It is understood that some of your answers will be approximations, not exact. Such estimates are acceptable. Also, a few of the questions may require information from a person who has observed your sleep. If you have access to such information, feel free to use it.

If you are asked to give a YES/NO answer, and you are not certain of your response, give the answer that is closest to the truth as best you can determine. If you are asked to give a numerical answer, such as the number of minutes it takes you to fall asleep, and you are not certain, give an approximate answer that is closest to the truth as best you can determine.

Please answer ALL of the questions. If a YES/NO item does not apply to you, answer NO. If you encounter an item that calls for a numerical response and it does not apply to you, or you cannot give an estimate, the number 0 will indicate that it does not apply or that you cannot estimate.

Before completing the SDI, please provide the following information:

NAME: ___________________________ DATE: ___________________

SOCIAL SECURITY NUMBER: ________________ CLINIC NUMBER: ________

THE SLEEP DISORDERS INVENTORY BEGINS ON THE NEXT PAGE.
SLEEP DISORDERS INVENTORY

* Please CIRCLE the appropriate response for each of the YES_NO questions. Please FILL IN THE BLANK with the correct number for each question that is followed by a blank.

1. Do you have difficulty falling asleep at night?...............................YES__NO
   *** If your answer is NO, go to the next item. If your answer is YES, go to (a).
   (a) How many NIGHTS per WEEK do you usually have difficulty?..............
   (b) On nights when you have difficulty falling asleep, how many MINUTES does it usually take you to fall asleep?..............................
   (c) On nights when you have this problem, how many HOURS do you sleep?........

2. How many MINUTES does it usually take you to fall asleep on your BEST nights?...

3. Do you wake up during the night and have difficulty falling back to sleep......YES__NO
   *** If your answer is NO, go to the next item. If your answer is YES, go to (a).
   (a) About how many NIGHTS does this happen each WEEK?.........................
   (b) On the average, how many TIMES do you wake up each NIGHT?..................
   (c) How many MINUTES does it take you to fall asleep after awakening?...........
   (d) On nights when you have this problem, how many HOURS do you sleep?...........

4. Do you wake up in the morning before your scheduled wake time, and are unable to return to sleep?..............................YES__NO
   *** If your answer is NO, go to the next item. If your answer is YES, go to (a).
   (a) How many TIMES per WEEK do you have this problem?...........................
   (b) On nights when you have this problem, how many HOURS do you sleep?...........

5. Do you wake up during the night frequently, but fall asleep soon afterwards?....YES__NO
   *** If your answer is NO, go to the next item. If your answer is YES, go to (a).
   (a) How many TIMES a NIGHT do you wake up?........................................
   (b) How many NIGHTS each WEEK do you have this problem?.........................

6. How many HOURS do you sleep on your BEST nights?..............................

7. Does your bed partner disturb your sleep at night?..............................YES__NO
   *** If your answer is NO, go to the next item. If your answer is YES, go to (a).
   (a) Is this a reason for your insomnia SOME of the time?...........................
   (b) Is this the reason for your insomnia MOST of the time?.........................

8. Is your sleep disturbed by environmental factors such as traffic, neighbors or family members? 

*** If your answer is NO, go to the next item. If your answer is YES, go to (a).

(a) Is this a reason for your insomnia SOME of the time? 

(b) Is this the reason for your insomnia MOST of the time? 

9. Is your bedroom dark enough for sleep at night? 

*** If your answer is YES, go to the next item. If your answer is NO, go to (a).

(a) Is this a reason for your insomnia SOME of the time? 

(b) Is this the reason for your insomnia MOST of the time? 

10. Is your bedroom temperature comfortable enough for sleep at night? 

*** If your answer is YES, go to the next item. If your answer is NO, go to (a).

(a) Is this a reason for your insomnia SOME of the time? 

(b) Is this the reason for your insomnia MOST of the time? 

11. How many NIGHTMARES have you had in the last MONTH? 

   (a) How many TIMES in the last MONTH have nightmares awakened you? 

   (b) How INTENSE are your nightmares (Rate 1, 2, 3, 4, or 5; where 1=mild, 3=frightening, 5=terrifying)? 

12. Do you often move vigorously during your sleep, especially while dreaming, and sometimes even hurt your partner by accident, or fall out of bed? 

13. Have you ever awakened from a deep sleep sweating, your heart beating fast or pounding, with a feeling of fear but with no memory of a dream? 

*** If your answer is NO, go to the next item. If your answer is YES, go to (a).

(a) How many TIMES in the last MONTH has this happened? 

(b) How INTENSE was this experience (Rate 1, 2, 3, 4, or 5; where 1=mild, 3=frightening, 5=terrifying)? 

14. How many NIGHTS per MONTH do you:

   ____ Snore loudly
   ____ Thrash about while asleep (but are not dreaming)
   ____ Gasp or snort while asleep
   ____ Stop breathing while asleep
   ____ Wake up in the night and feel unable to breathe

15. Do you often awaken with headaches? 

   YES NO
SLEEP DISORDERS INVENTORY

16. Do you sleep enough hours but have trouble waking up, feeling unrefreshed? .......YES NO
17. Do you sleep enough hours but fall asleep involuntarily during the day, but only when relatively unstimulated? .................................................................YES NO
18. Do you sleep enough hours but fall asleep involuntarily during the day, even when doing something very important, like driving? ..............................YES NO
19. Do you sleep enough hours but still have trouble functioning during the day? ....YES NO
20. Do you ever wake up because your legs jerk repeatedly while you are asleep? ..............................YES NO
   *** Answer only if YES: How many NIGHTS per MONTH does this occur? ....................
21. Do your legs ever feel restless or odd after you go to bed at night, and it keeps you awake? ..................................................................................................................YES NO
   *** Answer only if YES: How many NIGHTS per MONTH does this occur? ....................
22. Do you walk in your sleep? .......................................................................................YES NO
   *** If your answer is NO, go to the next item. If your answer is YES, go to (a).
   (a) How many TIMES a MONTH does this happen? .............................................
   (b) About how many MINUTES pass by while you are sleepwalking? ..............
23. Do you talk in your sleep? .......................................................................................YES NO
24. Do you grind your teeth in your sleep? .................................................................YES NO
25. Do you suddenly sit up in bed, still asleep, then lie down again? .......................YES NO
26. Do you ever wet your bed while asleep? ...............................................................YES NO
27. Do you experience extreme weakness in any of your muscles or even collapse, when you are excited, nervous, emotional, or laughing? ...........................YES NO
28. Do you often see things that are not there, as you are falling asleep or waking up? ..................................................................................................................YES NO
29. Do you often feel paralyzed while falling asleep or waking up? ...........................YES NO
30. Do you ever work the night shift, for example 11 p.m.-7 a.m.? ...........................YES NO
   *** Answer only if YES: How many NIGHTS per MONTH? ....................................
31. Does lack of sleep often cause you to fall asleep at inappropriate times or in the wrong place during the day? .................................................................YES NO
32. Does lack of sleep often cause you to have trouble functioning during the day? ...YES NO
33. Do you ever bring work home and do it after 8:00 p.m.? .......................................YES NO
   *** Answer only if YES: How many NIGHTS per WEEK? ......................................
34. On weekends or your days off, do you sleep more than an hour later than your usual wake up time? .................................................................YES NO
SLEEP DISORDERS INVENTORY

35. How many TIMES per NIGHT do you wake up to use the bathroom?

36. How many NIGHTS per WEEK do you wake up with indigestion or heartburn?

37. Do you eat your last meal or a large snack within 3 hours of bedtime? YES_NO

38. Are you on a weight loss program? YES_NO

39. Do you engage in some kind of regular physical exercise? YES_NO

40. Do you exercise at night, within four hours of bedtime? YES_NO

41. How many GLASSES (bottles/cans) of alcoholic drink do you have each DAY?

42. How many NIGHTS each MONTH do you use alcohol to aid sleep?

43. How many CUPS or GLASSES of caffeinated beverages do you drink in a DAY?

44. How many DAYS a WEEK do you drink caffeinated beverages after 6 p.m.?

45. How many CIGARETTES a DAY do you smoke?

46. Do you take any medications that contain caffeine or stimulants, such as allergy medications, nasal decongestants, or pain killers? YES_NO

*** Answer only if YES: How many MINUTES before trying to sleep do you take it?

47. Is your sleep problem sometimes worse than other times? YES_NO

*** If your answer is NO, go to the next item. If your answer is YES, go to (a).

(a) Is it worse when you are under stress? YES_NO

(b) Is it better when you sleep away from home? YES_NO

48. Was the onset of your sleep problem related to any specific event? YES_NO

*** If your answer is NO, go to the next item. If your answer is YES, go to (a).

(a) Were you under unusual stress at the onset of your sleep problem? YES_NO

(b) Were you very ill or in pain at the onset of your sleep problem? YES_NO

49. Is your sleep problem caused or made worse by physical discomfort or pain:

Never: ___ Rarely: ___ Sometimes: ___ Often: ___ Most or all of the time.

50. When you try to sleep, is it hard for you to turn off your mind? YES_NO

51. When you have sleep problems, do you:

(a) Go to bed earlier on some nights to make up lost sleep? YES_NO

(b) Wake up later on some mornings to make up lost sleep? YES_NO

(c) Get up out of bed and watch television, read, catch up on work, or eat? YES_NO
SLEEP DISORDERS INVENTORY

(d) Take naps during normal waking hours?........................................YES NO

*** If your answer is NO, go to the next item. If your answer is YES, go to (1).

(1) How many TIMES per WEEK do you take naps?....................................

(2) How many MINUTES are your naps, on average?...................................

(3) Do you awaken from your naps refreshed?.........................................YES NO

52. Have you been under more stress than usual recently?..........................YES NO

53. Are you the kind of person who tends to worry a lot?...........................YES NO

54. Does your sleep problem affect your mood during the day?.....................YES NO

55. Have you ever been treated or hospitalized for mental, emotional, drug, or
alcohol problems?..............................................................YES NO

56. Have you ever had any convulsions or significant head injury?.................YES NO

57. How long have you had your sleep problem?....._____Years _____Months _____Weeks

58. Have you previously received non-drug treatment for sleep problems?.........YES NO

59. Have you recently taken any prescription or over-the-counter medication for
sleep problems?............................................................................YES NO

*** If your answer is NO, go to the next item. If your answer is YES, go to (a).

(a) How many NIGHTS a WEEK do you usually take this medication?...............____

(b) How many MONTHS have you been taking sleep medication?......................____

60. Please list which medications you are now taking and the condition for which each is
being taken. Include over-the-counter medications.

<table>
<thead>
<tr>
<th>MEDICATION</th>
<th>CONDITION</th>
<th>MEDICATION</th>
<th>CONDITION</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
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<td>5</td>
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<td>3</td>
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<td>7</td>
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<tr>
<td>4</td>
<td></td>
<td>8</td>
<td></td>
</tr>
</tbody>
</table>

Please list any medications you have recently stopped taking and the condition for which each was being taken. Include over-the-counter medications.

<table>
<thead>
<tr>
<th>MEDICATION</th>
<th>CONDITION</th>
<th>MEDICATION</th>
<th>CONDITION</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>2</td>
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<td></td>
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<tr>
<td>3</td>
<td></td>
<td>6</td>
<td></td>
</tr>
</tbody>
</table>

*** THANK YOU FOR COMPLETING THIS INVENTORY ***
APPENDIX G

POLYSOMNOGRAPHY DESCRIPTION FOR SUBJECTS

We ask that you report to the hospital approximately two hours before your normal bedtime. There you will find a comfortable bedroom setting in which to spend the night. Each subject will be provided with a private bedroom and bathroom to assure privacy. Subjects will provide their own sleeping attire, and should plan to bring these items with them to the hospital. Please refrain from napping or consumption of alcoholic beverages or medications during the day on which you are scheduled to participate in the sleep study. In addition, do not consume foods or beverages containing caffeine or other stimulants after 6 pm on the evening of the polysomnogram.

During the hookup procedure small gold cups will be placed in various positions on your head, face, and legs. This is a painless and harmless procedure, using a paste that easily washes off. You will be asked to sleep in the bedroom with the electrodes attached to you. The experience of many years of such recordings indicates that individuals have no difficulty sleeping with these recording elements attached to their skin. We will be recording electrical activity of your brain, movement of your eyes, and activity of your sweat glands and muscles.

A research assistant in a nearby room will be watching a television monitor connected to a video camera in the room in which you are sleeping. The purpose of this camera is to determine the position in which you are sleeping during the night. The research assistant will also be monitoring the room over an intercom system. Therefore, you can be heard easily in the event that you should need assistance during the night.

You will be awakened for the day at 6:30 am. Recording equipment will be removed and you will be asked to complete a brief questionnaire asking about how well you slept the night before. Your participation will then be complete, and you will be able to leave the lab at approximately 6:45.

I understand the procedures involved in polysomnography, and agree to participate in this portion of the research study. I will arrive at the Baton Rouge General Medical Center

at______________ pm on the following date______________.

__________________________  ______________________________
Signature of Subject        Signature of Research Assistant

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APPENDIX H
INFORMED CONSENT FOR POLYSOMNOGRAPHY

I, ____________________________, freely and willingly consent to participate in a research project investigating sleep patterns in individuals with high versus low trait anxiety. As a research subject, I will complete psychometric screening measures and participate in one night of polysomnography.

I understand that this procedure is non-invasive and are no foreseeable risks associated with polysomnography. I understand that the results of this study may potentially assist in the further understanding of the sleep disturbances which often accompany anxiety states.

I understand that I may chose to decline to participate in this study at any time without any penalty or jeopardizing, in any way, any future medical treatment in this institution. Finally, I understand that my privacy will be protected and my name will not be used in any manner whatsoever in subsequent use of the results of this study. I understand that results of both polysomnography and psychometric testing will remain strictly confidential; and that I will be assigned a subject number which will be used in subsequent data analysis in the place of my name.

I have read and thoroughly understand this consent form and acknowledge that I have been given a copy of the consent form for my own use.

Subject's Name ____________________________ Date __________

Experimenter ____________________________ Date __________
APPENDIX I
POLYSOMNOGRAPHY HOOK-UP PROCEEDURES

Subjects were allowed to ask any questions prior to electrode placement. The subject then changed into his/her sleeping apparel and sat in a chair placed at bedside for electrode placement. Electrodes were placed on the subject's scalp, face and hand. All EEG, EMG, and EOG electrode placements were based on the system developed at Stanford University School of Medicine and described in Carskadon's widely used manual *A Manual for Polysomnography (PSG) Technicians* (Carskadon, 1980).

Electroencephalogram (EEG) electrode placements were based on a simplified version of the 10-20 system developed by the International Federation of Societies of Electroencephalography and Clinical Neurology (Carskadon, 1980). This system is based on measurements from four standard points on the head: (1) nasion (depression at bridge of the nose), (2) inion (the protrusion of bone in the back of the head), and (3) left and right pre-auricular points (notches just in front and above the lobes of the ears (Hassett, 1978). The following notations are used in designation of electrode placements: F (frontal), C (central), P (parietal), T (temporal), O (occipital), Fp (frontal pole), and z (midline). In addition, odd subscripts refer to the left side of the skull and even
subscripts refer to the right side of the head when referring the electrode placement sites.

The following procedures were used to determine exact electrode placement on individual subjects. The distance between the nasion and inion was be measured using a flexible tape. A grease pencil was used to mark the center point on the skull between these two reference points (Cz). Marks were also placed perpendicular to the tape at distance 10% up from the inion (Oz) and nasion (Fpz). These marks served as landmarks for other measurements. The distance between the left and right pre-auricular points was then measured by placing the tape along the first Cz mark. A mark was placed perpendicular to the tape at the center point on the skull. The intersection of the first and second mark indicated the precise location of Cz (the middle of the top of the head. Marks were also placed 10% up from the left and right pre-auricular points (T3 and T4 respectively). These marks also served as landmarks for other measurements.

The distance between T3 and Cz was measured by placing the tape on the transverse plane through Cz. A mark was then placed perpendicular to the tape at the center point (C3). This same series of measurements was repeated (using points T4 and Cz) in determining the location of C4. The measuring tape was then placed around the circumference of the head passing through Fpz, T3, Oz and T4. Marks were
placed perpendicular to the tape at distances of 5% of the circumference to the left and right of Fpz and Oz. These points represented Fp1, Fp2, O1 and O2 respectively. The second mark, delineating the exact location of C3, was made by placing the measuring tape from O1 to Fp1 through the existing mark for C3. A mark was placed perpendicular to the tape at the center point, thus forming an intersection with the previous C3 mark which pinpoints the exact location of C3. The same procedure was used on the right side of the skull to determine the exact placement of C4. The second marks for O1 and O2 were made by continuing the mark from Oz to intersect with the original O1 and O2 marks.

Grass electrodes (gold plated, 8mm diameter) were placed at the intersection of marks for Fpz, C3, C4, Cz, O1, and O2. The electrode of Fpz served as a ground; it was not referenced to another electrode and did not serve as a recording channel. The electrode for C3 was referenced to an electrode placed on the mastoid process behind the right ear (A2). The electrode for C4 was referenced to an electrode placed on the mastoid process behind the left ear (A1). The C3 and C4 electrodes, along with their respective reference points, served as backup recording channels for each other. The electrode for Cz was referenced to both A1 and A2 electrodes. The O2 electrode was referenced to the Cz electrode. The
electrode at O1 served as a backup in case the O2 electrode lost contact with the scalp during the course of the recording. The signals from electrodes at C3, Cz, C4, O1, and O2 (along with their respective reference electrodes) were fed into a Grass Electroencephalograph Model 8-16 D, with the sensitivity set at 5 microvolts per mm and the high frequency and low frequency filters set at 35 Hz and .3 Hz respectively.

Continuous eye movement recordings (EOG's) were also made with Grass electrodes (gold plated, 8mm diameter) which were attached one centimeter above the right outer canthus (outer corner of the eye) and one centimeter below the left outer canthus. The electrodes for both the right eye (EOG-right) and left eye (EOG-left) were referenced to the electrode placed on the left mastoid process (A1). Sensitivities for these recording channels were set at 15 microvolts per mm. The high frequency filters were set at 35 Hz and low frequency filter at .3 Hz.

Electromyogram (EMG) muscle potential recordings were also recorded continuously using Grass electrodes (gold plated, 8mm diameter) which were attached to muscle areas on (mental muscles) and beneath (submental muscles) the chin and on both legs (right and left anterior tibialis muscles). Sensitivity for the channel recording mental/submental EMG was set at 2 microvolts per mm, with the high frequency filter set at 70 Hz and the low
frequency filter set at 5 Hz. EMG recordings taken from the anterior tibialis muscles of each leg were taken from two electrode sites placed 4 cm apart on each leg. Sensitivities for these recordings were set at 7 microvolts per mm, the high frequency filter was also set to 70 Hz and the low frequency filter to 5 Hz for these channels.

Skin potential was used as a measure of electrodermal activity, and was recorded with Med Associates silver-silver chloride electrodes (20 mm diameter) taped to the palmar surface of the distal phalange of the ring finger of the right hand and to the medial surface at the elbow of the right arm. This channel was recorded using a sensitivity setting of 7 microvolts per mm, with the high frequency filter set to 15 Hz and the low frequency filter set to .1 Hz.

In addition, airflow was monitored via an oral/nasal thermistor in order to rule out the presence of sleep apnea. The sensitivity for this recording channel was set at 7 microvolts per mm and high and low frequency filters were set at 15 Hz and .1 Hz respectively.

All electrode sites were prepared by lightly abrading the skin surface with Omni Prep, a standard hypo-allergenic skin cleanser. Electrodes for EEG and chin EMG were affixed to the skin by placing a one-inch gauze square which has been soaked in collodion (a standard adhesive) over the electrode and drying the gauze using an air
compressor with an electrode applicator attached. These electrodes were removed using collodion remover, a standard hypo-allergenic adhesive remover, in the morning. Electrodes for EOG and leg EMG were taped to the skin. All electrodes were filled through a hole on their top surface with Beckman Electrode Electrolyte using a syringe with a blunt needle. Electrodes for skin potential were attached to the skin using a double sided adhesive collar, and were filled with Beckman Electrode Electrolyte prior to attachment to the skin.

After the electrodes were placed on the subject, they were attached to a terminal box. The impedance was then checked by attaching the terminal box to an impedance meter which has been calibrated to 10 kilohms. The impedance of EEG, EMG, and EOG channels were checked to ensure that all impedance readings were below 10 kilohms. If the impedance is greater than 10 kilohms for any electrode, that electrode was removed from the subject, the site was cleaned again, and the electrode re-attached. Once all impedance readings are below 10 kilohms, the terminal box was connected to the Grass Electroencephalograph Model 8-16 D. Paper speed for all recordings was 10 mm per second, at which one page of polygraph paper was recorded in 30 seconds of electrophysiological activity (one epoch).

After the terminal box was connected to the electroencephalograph, recording channels were checked.
The subject was then asked to move his/her eyes, bite down, move his/her legs, hold his/her breath for ten seconds, and to close his/her eyes and relax so that EEG, EMG and EOG channels could be checked. When all channels were functioning properly, the technician answered any questions which the subject had at this time. The lights were then turned off and the subject allowed to sleep.
## APPENDIX J

### SLEEP PROTOCOL MONTAGE

<table>
<thead>
<tr>
<th>Montage</th>
<th>Channel</th>
<th>Sensitivity</th>
<th>HFL (Hz)</th>
<th>LLF (Hz)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Channels 1-4 are EEG</td>
<td>1</td>
<td>5 uv/mm</td>
<td>35</td>
<td>.3 C3-A2</td>
</tr>
<tr>
<td>channels</td>
<td>2</td>
<td>5 uv/mm</td>
<td>35</td>
<td>.3 C4-A1</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>5 uv/mm</td>
<td>35</td>
<td>.3 CZ-A1/A2</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>5 uv/mm</td>
<td>35</td>
<td>.3 O2-CZ</td>
</tr>
<tr>
<td>Chin EMG</td>
<td>5</td>
<td>2 uv/mm</td>
<td>70</td>
<td>5 F3-FZ</td>
</tr>
<tr>
<td>EOG Left</td>
<td>6</td>
<td>15 uv/mm</td>
<td>15</td>
<td>.1 Fp1-A1</td>
</tr>
<tr>
<td>EOG Right</td>
<td>7</td>
<td>15 uv/mm</td>
<td>15</td>
<td>.1 Fp2-A2</td>
</tr>
<tr>
<td>Oral/Nasal Thermistor</td>
<td>8</td>
<td>7 uv/mm</td>
<td>15</td>
<td>.1 T3-T4</td>
</tr>
<tr>
<td>Skin Response</td>
<td>9</td>
<td>7 uv/mm</td>
<td>15</td>
<td>.1 36-37</td>
</tr>
<tr>
<td>Leg EMG (R)</td>
<td>10</td>
<td>7 uv/mm</td>
<td>70</td>
<td>5 26-27</td>
</tr>
<tr>
<td>Leg EMG (L)</td>
<td>11</td>
<td>7 uv/mm</td>
<td>70</td>
<td>5 28-29</td>
</tr>
</tbody>
</table>

Paper speed is 10 mm/second
APPENDIX K
DAILY SLEEP DIARY

NAME: _______________________ DATE: _________________

PLEASE RESPOND TO THE FOLLOWING QUESTIONS.

1. About what time did you first try to fall asleep last night? __________

2. Approximately how many minutes did it take you to fall asleep? _________

3. Please record the approximate times you awoke last night, and below that, how long it took you to get back to sleep.

   _______ min  _______ min  _______ min  _______ min  _______ min

4. When did you awaken for the day this morning?_________

5. How difficult was it for you to fall asleep last night?

   1  2  3  4  5
   Not Very Difficult
   Extremely

6. How rested do you feel this morning?

   1  2  3  4  5
   Very Rested Poorly Rested

7. Rate the quality of last night's sleep.

   1  2  3  4  5
   Excellent Very Poor

8. What was your level of physical tension when you went to bed last night?

   1  2  3  4  5
   Extremely Tense
   Relaxed

9. Rate your level of mental activity when you went to bed last night.

   1  2  3  4  5
   Very Quiet Very Active
APPENDIX L
MICRO-AROUSAL SCORING PROCEDURES

Scoring of micro-arousals will follow the recent standardized guidelines described by Bonnet et al. (1992). A micro-arousal will be defined as an abrupt shift in EEG frequency, which may include theta, alpha, and/or frequencies greater than 16 Hz but not spindles, subject to the following rules and conditions: (1) Subjects must be asleep, defined as 10 continuous seconds or more of the indications of any stage of sleep before an arousal can be scored. Arousal scoring is independent of Rechtschaffen and Kales epoch scoring (i.e. an arousal can be scored in an epoch of recording which would be classified as wake by Rechtschaffen and Kales criteria). (2) A minimum of 10 continuous seconds of intervening sleep is necessary between independent arousals. (3) The EEG frequency shift must be 3 seconds or greater in duration to be scored as an arousal. (4) Arousals in NREM sleep may occur without concurrent increases in chin EMG amplitude. (5) Arousals in are scored in REM sleep only when accompanied by concurrent increases in chin EMG amplitude. (6) Arousals cannot be scored based on changes in chin EMG amplitude alone. (7) Artifacts, K complexes, or delta waves are not scored as arousals unless accompanied by an EEG frequency shift of at least 3 seconds duration. If such activity
precedes an EEG frequency shift, it is not included in reaching the 3-second duration criteria. When occurring within the EEG frequency shift, such artifacts are included in meeting duration criteria. (8) The occurrence of pen blocking artifact is considered an arousal only if an EEG arousal pattern is contiguous. In this event, the pen blocking artifact may be included in meeting the duration criteria. (9) Non-concurrent, but contiguous, EEG and EMG changes which were individually less than 3 seconds, but together were greater than 3 seconds in duration, are not scored as arousals. (10) Intrusion of alpha activity of less than 3 seconds into NREM sleep at a rate greater than one burst per 10 seconds is not scored as an EEG arousal. (11). Transitions from one stage of sleep to another are not sufficient of themselves to be scored as EEG arousals unless they meet the criteria previously outlined.
VITA

Kristi Hulse Fuller was born in Anchorage, Alaska on November 2, 1965. She was raised in the small town of Chugiak, Alaska, where she graduated from high school in 1984. After briefly attending Baylor University and the University of Alaska, she transferred to the University of Idaho where she earned her bachelor's degree in psychology in 1989. Following graduation, she began graduate studies in clinical psychology at Louisiana State University, where she pursued clinical and research interests in the areas of clinical neuropsychology and sleep disorders. She currently resides in the state of Washington with her husband, Richard D. Fuller. The couple is presently in the process of moving to Portland, Oregon, where she will begin a postdoctoral residency in medial psychology at Oregon Health Sciences University in September of 1994.
Candidate: Kristi Hulse Fuller

Major Field: Psychology

Title of Dissertation: The Impact of Trait Anxiety on Slow-Wave Sleep Processes

Approved:

Major Professor and Chairman

Dean of the Graduate School

EXAMINING COMMITTEE:

June 15, 1994