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The relation between electrodermal activity in sleep, negative activity affect, and stress in patients referred for nocturnal polysomnography

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ABSTRACT

The study of electrodermal activity (EDA) began over a century ago, and the phenomenon has been linked to many aspects of emotion, arousal and attention. A subset of studies has focused on the occurrence of EDA in sleep (EDAS). These investigations have led to definitive conclusions on when EDA is most likely to occur during the sleep cycle, i.e., slow-wave sleep. Studies have also shown that at least moderate stress tends to increase EDAS, but these studies have fallen short methodologically. The aims of the present study were: (a) to investigate the relation of negative affect and stress to EDAS, and to determine the extent to which these variables are predictive of EDAS; and (b) to explore the utility of EDAS as an index of sympathetic nervous system arousal and of sleep quality. Several additional hypotheses were also tested.

Participants were 70 referrals to a local sleep disorders center. Subjects completed a demographic profile form and self-report measures of mood disturbance, worry and stress and underwent skin potential measurement during an overnight sleep study. Correlation and multiple regression analyses determined that weak relations exist between negative affect, worry and stress and EDAS, and that these variables are largely ineffective in predicting EDAS in a sleep-disordered population. Further, EDAS had no relation to self-reported sleep quality in this sample. Additional analyses found significant associations between apneic and periodic limb movement events and both central (EEG) and autonomic arousals (EDAS), and that a combination of both central and autonomic nervous system arousal variables might provide a better indicator of the amount of sleep disturbance present.

INTRODUCTION

The empirical study of electrical changes in human skin began over 100 years ago. Since then, a wide variety of terms have been used to label this phenomenon including galvanic skin response, psychogalvanic skin response, skin resistance response, skin conductance response, skin potential response and electrodermal activity. Historically, the most widely employed term to describe this phenomenon has been the galvanic skin response (GSR). While the correct usage of GSR is to describe skin conductance and resistance responses only, it has often been misused in the literature and, consequently, its meaning has become ambiguous (Venables & Christie, 1973). Initial research investigating electrical changes of the skin is flawed due to the fact that investigators neglected to make their specific recording methodology clear to the reader, and instead reported their results under the term GSR (Hassett, 1978). Today, electrodermal activity (EDA) is the preferred term to label this phenomenon, as it is a generic reference that subsumes all methods of measuring the electrical activity of the skin (Fowles, 1986).

EDA is the most frequently recorded psychophysiological measure in history (Stern, Ray, & Davis, 1980). Since research on EDA first began, this response system has been linked to many aspects of emotion, arousal, and attention (Dawson, Schell, & Filion, 2000; Hugdahl, 1995). EDA has been utilized to investigate numerous areas such as orienting and attention, learning and conditioning, psychopathology, personality disorders, individual differences, brain asymmetry and laterality, cognitive functions, as well as neuropsychology (Hugdahl, 1995). Boucsein (1992) states that "EDA measures are highly applicable to emotions and stress research. This is because EDA is mediated solely by the sympathetic branch of the autonomic nervous system and is therefore not

subjected to peripheral parasympathetic influences as most other autonomic variables are" (p. 373).

Anatomy and Physiology of the Skin: Apocrine versus Eccrine Sweat Glands

Skin is a selective barrier that prevents entry of foreign matter into the body as well as facilitates the passage of materials in the bloodstream to the exterior of the body. It also regulates the water balance and core body temperature (Dawson et al., 2000). Sweating, which is the physiological function that underlies EDA, can be either thermoregulatory (cooling) or an indication of emotional arousal (Andreassi, 1995). The total number of sweat glands on the human body ranges from 2-5 million (Fowles, 1986; Kuno, 1956).

There are two types of sweat glands: apocrine and eccrine. The apocrine sweat glands are typically found in the genital area and armpits and tend to be large and open into hair follicles (Boucsein, 1992). While the exact function of apocrine glands is not well understood, there is some evidence to suggest that in some mammals, apocrine sweat glands produce a thick secretion that serves as a sexual scent hormone (pheromone). Apocrine sweat glands do not play a role in EDA, and are consequently largely irrelevant to the field of psychophysiology (Boucsein, 1993).

The eccrine sweat glands are more numerous than apocrine glands and secrete a thinner, watery sweat. They are known to respond to sensory stimuli and are more responsive to psychological arousal, and are thus more important to the psychophysiologist (Stern, Ray, & Davis, 1980). Eccrine glands are tubular structures with secretory ducts that open as a small pore on the epidermis. Depending on the degree of sympathetic activation, sweat rises toward the surface of the skin and often overflows onto the surface. Initially, it was thought that EDA was solely determined by how much

sweat was present on the skin, however, changes in EDA occur with even a slight rise or decrease of sweat within the glands (Stern, Ray, & Davis, 1980).

The primary function of most eccrine sweat glands is thermoregulation (Dawson et al., 2000). The eccrine sweat glands are well distributed over most of the skin, but are especially dense on palmar and plantar surfaces of the hands and feet (Andreassi, 1995). Eccrine glands on the palm and foot surfaces respond weakly to heat and strongly to psychological and sensory stimuli, whereas the opposite is true of the eccrine glands found on the forehead, neck and back of the hands (Andreassi, 1995). Although all eccrine glands are responsive to emotional stimuli, sweating on the palmar and plantar surfaces tends to be especially apparent due to high gland density in these areas (Dawson et al., 2000).

The eccrine glands are innervated by the sympathetic branch of the autonomic nervous system (ANS), though acetylcholine is the chemical transmitter at the postganglionic synapse (Stern, Ray, & Davis, 1980). Sweating can be inhibited upon administration of atropine, a cholinergic blocker (Fowles, 1986).

There are several theories that have been proposed to explain the biological significance of eccrine emotional sweating. Most of them try to explain this phenomenon in evolutionary terms. These include improvement in grip (Darrow, 1936, as cited in Hassett, 1978), greater tactile sensitivity, and increased resistance to abrasion or cutting of the skin (Hassett, 1978).

Recording of Electrodermal Activity: Skin Conductance, Resistance, and Potential

There are two methods for objectively measuring the electrical activity of the skin. The first, known as the exosomatic technique, was used initially by Féré in 1888. Féré

first noted that the electrical properties of the skin change in response to emotional situations. Specifically, he found that the skin becomes a better conductor of electricity upon the presentation of external stimuli; that is, sensory or emotional stimulation produced a decrease in skin resistance promoted by the increased hydration of the skin. The exosomatic technique entails passing a small electrical current through the skin from an external source and measuring the resistance to the passage of the current. The exosomatic method generates skin conductance and skin resistance values. There is much evidence to suggest that skin conductance is a superior measurement to resistance, as conductance measurements provide a directly proportional representation of the number of sweat glands involved in a response and values obtained tend to be more normally distributed (Hassett, 1978).

The second method, known as the endosomatic technique, was first used by Tarchanoff in 1889 and involves measuring electrical activity at the skin's surface with no externally imposed current. The voltage changes of the skin itself are measured. This method yields measurements of skin potential, which reflects the electrical activity of the sweat glands themselves (Dawson et al., 2000). Surwillo (1990) notes that both skin conductance and skin potential are reflections of the same process, i.e., eccrine sweating, and thus the two terms have often been used interchangeably in the existing literature despite the fact that they measure two different stages of the process.

There are two types of skin potential activity: skin potential response (SPR) and skin potential level. The SPR is a rapid fluctuation of skin potential, while level values fluctuate gradually as a background to the SPR. That is, skin potential level refers to the measurement of skin potential over lengthy periods of time (tonic activity) and SPR

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refers to brief duration changes (phasic activity); tonic measures reflect relatively long-term changes, while phasic measures reflect more transient responses to stimuli. Spontaneous skin potential responses are eccrine sweat responses that are not attributable to a specific external stimulus. Spontaneous SPR activity increases under conditions of emotional stress and cognitive problem-solving (Hassett, 1978). SPRs may be uniphasic negative, biphasic (negative - positive), triphasic (negative - positive - negative), or less commonly, uniphasic positive (Venables & Christie, 1973).

Skin conductance is used more often than skin potential, especially in research that seeks to relate EDA to behavior (Andreassi, 1995). Both recording techniques have advantages and disadvantages, however. A major advantage of endosomatic recording is that it is regarded as more physiological, since there is no external current that can influence the skin. Additionally, endosomatic recording is especially useful in lengthy recording sessions as electrode polarization is prevented, recordings are not affected by variations in contact area, and it can be measured by using simpler ordinary AC bioamplifiers (Boucsein, 1993). According to Boucsein (1993) "endosomatic recording should be revitalized, not for general application but in basic research, as an aid to gaining a better theoretical understanding of electrodermal phenomena" (p. 32).

Silver-silver chloride (Ag/AgCl) electrodes are optimal for measuring both skin potential and conductance. There is no effect of electrode size on skin potential measurement (Fowles et al., 1981; Venables & Christie, 1973, 1980). Unipolar electrode placement is used in the measurement of skin potential (Stern, Ray, & Davis, 1980), and both level and response values are reported as changes in millivolts (mV) or microvolts (µv) (Venables & Christie, 1980). Standard measurement of skin potential involves an

electrode at both an active and inactive site. The active electrode can be placed at either the hypothenar eminence or on a finger site. The inactive reference is best placed over a lightly abraded site on the volar surface of the forearm (i.e., 2/3 of the distance from the wrist to the elbow) (Venables & Christie, 1980).

Historically, many research investigations have used the non-dominant hand in recording, because it tends to have less cuts and calluses, and it leaves the dominant hand free for other tasks, if needed. There is no definitive evidence that EDA recorded from one hand gives significantly different results when compared to EDA values obtained from the opposite hand (Dawson et al., 2000; Freixa i Baqué & deBons, 1983).

Neural Mechanisms/Pathways and Electrodermal Activity

Surprisingly, the exact physiology of electrodermal activity is not completely understood, though it is a well-established fact that sweat gland activity is reflected as changes in skin conductance and potential (Surwillo, 1990). Animal research has provided much of the information on the brain and EDA. For example, Block (1965) showed that the reticular formation acts as a "pacemaker" for synchronization of EDA in the four paws of a cat (as cited in Ba M'Hamed, Sequeira, & Roy, 1993). Ba M'Hamed et al. (1993) found that electrical stimulation of the central tegmental field evokes skin potential responses during wakefulness and sleep in cats.

The specific neural mechanisms and pathways involved in the production of EDA are both numerous and complex. There are three possible independent pathways that have been proposed as being responsible for the generation of EDA. The first pathway involves ipsilateral hypothalamic influences controlled by the limbic structures; the amygdala is thought to have a facilitory influence, while the hippocampus has an

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inhibitory influence. EDA associated with the amygdala and hippocampus is likely due to emotional and thermoregulatory processes, respectively. The second pathway involved in EDA consists of the premotor basal ganglia combined with premotor cortical areas. It is thought that EDA mediated by the premotor cortex is associated with situations that require fine motor control, while orienting and attention seem to be associated with prefrontal cortical activity. The third and final pathway involved in the production of EDA originates from the reticular formation of the brainstem. Specifically, it is thought that the ventromedial reticular formation has inhibitory effects, while portions of the midbrain and diencephalic reticular formation have excitatory effects on EDA. This type of EDA is thought to be associated with gross motor movements and increased muscle tone (Boucsein, 1992; Dawson et al., 2000).

Important Considerations in Electrodermal Measurement

Environmental and Demographic Considerations. There are several external and internal factors that can be possible sources of variance in EDA measurement; these include both environmental conditions and direct influences inherent in physiological factors such as age, gender, and race.

It is well-established that climatic conditions (i.e., ambient temperature and possibly other meteorological variables) can affect EDA. In a review of studies on this topic, Boucsein (1992) concluded that there is an established dependency of EDA upon the room temperature as well as the seasonal temperature. Boucsein (1992) suggests that these variables should be recorded with an ambient room temperature as close to 23° Celsius (73° Fahrenheit) as possible; Venables and Christie (1973) provide 20-30° C (68-86° F) as an acceptable range. Less is known about the relation between EDA and

meteorological variables, as experimental manipulation of these variables is extremely difficult. Furthermore, most of the studies that have explored these variables have used exosomatic measures. For example, Waters, Koresko, Rossie, and Hackley (1979) investigated the relation between skin conductance and temperature, humidity and air pressure and found that these variables effectively predicted EDA, but the percentage of explained variance was somewhat low (6 - 9%). While air pressure does not appear to influence EDA significantly, relative humidity seems to have more of an effect. It has been suggested that meteorological variables be recorded as controls if clear climatic changes are expected during the course of an experiment (Boucsein, 1992).

In regard to specific subject characteristics, gender and age have received the most research attention. There are established differences between males and females in regard to sweating as well as EDA. Significant differences have been found using both exosomatic and endosomatic recording techniques (Boucsein, 1992). While women have greater sweat gland density, they tend to have more delayed and less prolific sweating than males (Morimoto, 1978). These gender-related differences can be due to endocrine influences (Venables & Christie, 1973) and the menstrual cycle (Edelberg, 1972). For example, progesterone has the ability to decrease palmar eccrine sweat output (Venables & Christie, 1973). There is a reduction in palmar sweat count during the luteal phase of the menstrual cycle (MacKinnon, 1954a) as well as after the administration of progesterone (MacKinnon & Harrison, 1961). A number of studies have determined that there are differences in both electrodermal activity and reactivity between the sexes. Specifically, most research indicates that females display higher tonic EDA, while males tend to have greater electrodermal reactivity under conditions of stimulation (Boucsein,

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1992). One study that investigated sex differences in EDA during sleep found that males had significantly more skin potential responses during sleep than females (Zepelin & McDonald, 1987).

For the most part, old age commonly brings a decrease in skin conductance level and an increase in skin resistance level, but the causes are not well understood (Boucsein, 1992). Research indicates a reduction of palmar sweat counts with age, which has been found for males, females, African-Americans and Caucasians (MacKinnon, 1954b; Juniper & Dykman, 1967). In a study of skin potential activity in 132 male subjects, Surwillo (1965) found that older males had fewer SPRs and found a significant negative correlation between age and skin potential level. In a study of males and females aged 20-79, Eisdorfer, Doerr, and Follette (1980) demonstrated that among older subjects, sex differences in skin conductance were not apparent. Although older subjects had significantly lower resting skin conductance levels, there were no differences between skin conductance responses between the younger and older subjects. Zepelin and McDonald (1987) attempted to assess whether these age differences were apparent during sleep by recording the nocturnal spontaneous skin potential responses of 36 men and women from two age groups (i.e., 18-23 and 57-71 years); they found no significant difference in skin potential responses between the younger and older subjects.

In comparison to research on gender and age variables, racial differences and heritability factors in EDA have received much less research attention. While little is known about heritability, there are established racial differences in EDA. Caucasians show increased skin potential reactivity when compared to African-American subjects (Janes, Hesselbrock, & Stern, 1978; Janes, Worland, & Stern, 1976), as well as lower

skin resistance levels and higher skin conductance levels during resting conditions (Boucsein, 1992). These differences have been attributed to differing amounts of sweat glands dependent on the darkness of the skin; both African-American males and females have fewer active sweat glands than same-sex Caucasians of the same age range (Juniper & Dykman, 1967). Additionally, it has been found that Japanese subjects have more eccrine sweat glands on their extremities compared to individuals of European descent (Venables & Christie, 1973). Consequently, Boucsein (1992) suggests carefully controlling for ethnic differences among subjects in EDA experiments.

Finally, although body fat and the activity of the sympathetic nervous system have been reported to be linked (Peterson et al., 1988), Krohnholm, Alenen, and Hyyppa (1993) found no relation between Body Mass Index and sympathetic activity in their large community sample of 199 individuals.

Asymmetry and Laterality. Factors such as stimulus complexity, emotional significance of stimuli, stimulus duration, gender, handedness, and skin thickness are important variables that have been found to influence daytime EDA lateralization (Boucsein, 1992). However, Roman (1989, 1992) concluded that electrodermal asymmetry is unrelated to sex and/or handedness.

While studies investigating the factors associated with electrodermal asymmetry and lateralization during the waking state are numerous, research investigating asymmetry and laterality during sleep is scant. Freixa i Baqué and deBons (1983) found that electrodermal asymmetry (i.e., R ≠ L) during sleep is significantly higher than asymmetry during the waking state. Ware et al. (1979) examined electrodermal activity (skin resistance responses) from the left and right hands as well as on the penis in normals

during a four-night study. They found that most skin resistance responses occurred during groups of responses (i.e., storms), and all of the subjects stormed on at least one night of the study, with subjects averaging 2-3 storms per night. While storming did not begin and end simultaneously across sites, the various sites tended to overlap.

Electrodermal Activity and Behavior

There are scores of papers reporting the use of EDA as markers of autonomic arousal in emotion and other related contexts (Öhman, Esteves, Flykt, & Soares, 1993). There are three basic experimental paradigms that have been used to investigate the relation of EDA and psychological states and processes: (a) the presentation of a continuous, chronic stimulus or situation (i.e., performing an ongoing task); (b) the study of individual differences in which EDA is assumed to be a relatively stable trait; and (c) the presentation of discrete, novel stimuli. The latter paradigm is probably the most frequently studied and involves presentation of discrete stimuli and measuring the elicitation and habituation of the orienting response (Dawson et al., 2000).

An electrodermal response can be elicited by almost any type of novel stimulus in the environment or by the omission of a stimulus that is expected (Siddle, 1991). Often, this paradigm involves a repetitive presentation of a simple, discrete stimulus with varying intervals of time in between. Research indicates that, typically, the electrodermal response rapidly declines in amplitude with stimulus repetition and then disappears completely; that is, stimulus repetition results in a less active and reactive response system (Dawson et al., 2000), i.e., habituation. The electrodermal response is thought to be the most reliable and easily accessible index of the orienting (attentional shift) response (Öhman, Esteves, Flykt, & Soares, 1993).

Electrodermal Activity and Emotion. Physiological arousal is a central component in emotion (Öhman, Esteves, Flykt, & Soares, 1993). Much experimental evidence exists that suggests that EDA is an index of the "affective value of stimuli for a subject" (Andreassi, 1995), and it has been shown that different emotions produce different physiological responses and varying degrees of EDA. For example, Ax (1953) was the first to demonstrate that fear and anger evoked different patterns of ANS responses. Ax induced fear (anxiety) and anger in normals, and showed that anger was characterized by greater increases in diastolic blood pressure, greater decreases in heart rate, greater increases in muscle tension, and greater increases in the number of skin conductance responses than fear. In contrast, the emotion of fear (anxiety) showed greater increases in the skin conductance level, muscle tension peaks and respiration rate than anger.

Since Ax's publication, numerous studies have focused on studying EDA in relation to emotion. Differential electrodermal responses have been demonstrated with various types of music (Zimny & Weidenfeller, 1963), differing interpretations of observed violence (Geen & Rakosky, 1973) and in comparison of erotic verses neutral stimuli (Hamrick, 1974). Additionally, Lanzetta, Cartwright-Smith, and Kleck (1976) showed that exaggerating, as opposed to concealing, facial expressions associated with the anticipation of electric shock resulted in phasic skin conductance changes of a greater magnitude as well as higher subjective pain ratings. It has also been demonstrated that in experiments that require subjects to generate facial expressions portraying positive (happiness and surprise) and negative emotions (anger, sadness, disgust), EDA is more responsive to negative emotions.

Additionally, experimental evidence indicates that EDA is closely related to emotional arousal processes and that EDA specifically reflects the arousal aspect of emotion (Öhman, Esteves, Flykt, & Soares, 1993). For example, in an investigation of physiological responses to pictorial stimuli, Greenwald, Cook, and Lang (1989) had subjects examine pictures that varied in rated arousal and valence (i.e., liking); skin conductance responses were highly related to the arousal value, and were unrelated to their valence level. Additionally, research focusing on facial recognition in prosapagnosic subjects indicates that while these patients lack the ability to correctly identify familiar faces, they exhibit more frequent and larger EDA responses to familiar faces than new faces (Bauer, 1984; Tranel & Damasio, 1985).

Electrodermal Activity and Motor Performance. Electrodermal activity and motor performance has been extensively studied in reaction time experiments. An early and frequently cited study by Freeman (1940) found that brief reaction times were related to moderate levels of skin conductance, while lengthier reaction times were associated with either very high or low skin conductance. Unfortunately, subsequent studies failed to replicate Freeman's finding, and instead found a linear relationship between the two variables; that is, short and long reaction times are associated with high and low levels of skin conductance, respectively (Schlosberg & Kling, 1959; Andreassi, 1966). Additionally, research on spontaneous EDA (skin potential and conductance) indicates shorter reaction times occur when spontaneous responses are numerous (Andreassi, 1995; Baugher, 1975; Surwillo, 1990; Surwillo & Quilter, 1965).

Electrodermal Activity and Mental Activity. Considerable research has focused on the study of EDA during conditions involving information acquisition and learning.

Both verbal and visual material that is upsetting or sexually arousing, consistently evoke electrodermal responses of a greater magnitude than neutral information (Hamrick, 1974; Manning & Melchiori, 1974).

EDA has also been a popular dependent variable in problem solving experiments (e.g., mental arithmetic). This line of research has consistently indicated that electrodermal levels are higher when subjects are engaged in problem solving than in resting conditions, as well as when computational problems are difficult or the subject is under significant pressure to complete the task (Schnore, 1959; Surwillo, 1990). Verbal learning experiments indicate that, in general, more successful learning is associated with a greater amount of EDA and that higher levels of EDA are associated with increased alertness and effort under conditions that involve novel information (Andreassi, 1995).

Thus, it is obvious that a wide variety of stimuli can elicit EDA. Electrodermal responses can be elicited by novel stimuli, emotional stimuli, threatening stimuli, and attention-getting stimuli (Fowles, 1986).

Electrodermal Activity and Psychopathology

The literature on psychophysiological response in affective disorders is relatively small (Zahn, 1986), and only a few new findings have added to the literature since the 1960s (Boucsein, 1993). The bulk of the studies of EDA and psychopathology focus on EDA in anxiety, depression, schizophrenia, and psychopathy. Results in some of these areas have been inconsistent. While a complete review of this area is beyond the scope of this paper, a broad summary of the major findings will be given.

Anxiety. Studies done by Lader and Wing (1964, 1966) are the most frequently cited studies attempting to differentiate EDA among anxiety and control groups

(Boucsein, 1992). These studies found that skin conductance levels and responses were higher among anxious patients compared to controls. Others have also found that anxious subjects have elevated levels of tonic EDA (e.g., Chattopadhyay & Biswas, 1983; Raskin, 1975). Both elevated phasic and tonic EDA levels have been attributed to an "autonomic overarousal" in these patients. A second noteworthy finding is that anxious subjects do not display the expected habituation to stimuli; that is, it has been found that they tend to have slowed, or even failure of, habituation to stimuli (Lader, 1967, 1975; Lader & Wing, 1964, 1966). However, some studies have not confirmed this finding (e.g., Hart, 1974). Boucsein (1992) noted that the conflicting findings might be a result of the differing methods of diagnosis used in the existing research.

Finally, a subset of research has focused on EDA in phobic subjects. The main finding of these studies is that phobic individuals tend to have elevated electrodermal responses to specific fear-relevant stimuli (Boucsein, 1992).

Depression. Hugdahl (1995) cautions against drawing firm conclusions regarding EDA among depressed patients, as many studies have included results from individuals taking antidepressant medication, and many antidepressant drugs are anticholinergic. Much of the literature suggests low baseline activity among depressives, but the data are mixed. While a number of studies have indicated that EDA is reduced in individuals with depression (Dawson, Schell, & Cantania, 1977; Iacono, Lykken, Peloquin, Lumry, Valentine, & Tuason, 1983; Lader & Wing, 1969; Mirkin & Coppen, 1980; Storrie, Doerr, & Johnson, 1981; Ward & Doerr, 1986; Ward, Doerr, & Storrie, 1983; Williams, Iacono, & Remick, 1985), there are also many studies which have not found this effect (e.g., Albus, Engel, Muller, Zander, & Ackenheil, 1982; Lapierre & Butter, 1980; Toone,

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Cooke, & Lader, 1981). The conflicting findings may also be due to the fact that many of the studies conducted have assessed the effects of depressed mood on EDA in a mixed sample of psychiatric patients (Zahn, 1986).

Electrodermal heterogeneity between specific subgroups of depressed patients has been shown. For example, Lader and Wing (1969) found that an agitated depressed group of subjects had significantly higher skin conductance levels and responses than controls, and that a retarded depressive subgroup had significantly lower values than the same control group. Some later attempts to replicate this finding, however, have been unsuccessful (Dawson et al., 1977; Lapierre & Butter, 1980; Toone et al., 1981). However, Iaconco et al. (1983) demonstrated that both unipolar and bipolar depressive subjects displayed reduced EDA (i.e., smaller electrodermal responses) to tones presented.

Schizophrenia. Hugdahl (1995) comments that the most robust finding among studies that have investigated EDA among schizophrenic subjects is that many of these subjects tend to be non-responders when presented with novel stimuli. That is, they either fail to show an orienting response or they have unusually fast habituation to a stimulus (e.g., Bernstein et al., 1982). Approximately 50% of schizophrenics fall into this non-responder group, while the remaining 50% are normally responsive or hyper-responsive, i.e., larger than normal orienting responses and prolonged habituation. Dawson (1990) states that non-responders tend to be more chronically psychotic, while hyper-responsivity is a possible indicator of psychotic episodes. Interestingly, Öhman et al. (1989) demonstrated that non-responders tend to have poorer treatment outcome and more serious symptomatology.

A subset of research has focused on bilateral EDA asymmetry among schizophrenic patients. In general, these studies have confirmed the results of Gruzelier (1973) which indicate that electrodermally responsive schizophrenics have higher right-hand phasic and tonic EDA measurements, while non-responders show elevated left-hand tonic levels. It is not known whether this bilateral electrodermal asymmetry is related to specific diagnostic subgroups and/or therapeutic outcome (Boucsein, 1992).

Antisocial Personality Disorder/Psychopathy. The main finding of studies focusing on EDA and psychopathy is that antisocial individuals tend to show electrodermal hyporeactivity (i.e., reduced EDA) (Boucsein, 1992). Specifically, antisocial subjects fail to show elevated skin conductance responses when anticipating an aversive event (Hare, 1978a; Raine & Venables, 1984) and show a prolonged electrodermal response recovery time as well (Hare, 1978b; Loeb & Mednick, 1977; Siddle, 1977). Lyyken (1957) initially hypothesized that this hyporeactivity may be due to reduced anxiety and arousal in threatening situations. Boucsein (1992) notes that results of these studies should be interpreted with caution, as it is difficult to compare results across studies on this population due to the extreme variety of diagnostic methods used.

Electrodermal Activity During Sleep

Electrodermal activity is more frequent during sleep than during wake in humans (Johnson & Lubin, 1966; McDonald Shallenberger, Koresko, & Kinzy, 1976). The earliest reported observation of nocturnal electrodermal fluctuations is an unpublished doctoral dissertation by Titelbaum (1938). This study measured skin resistance during wakefulness, during natural sleep, and during sleep that followed extended periods of

wakefulness. Sleep recordings revealed that spontaneous skin resistance fluctuations occurred repeatedly during periods of time lasting from 1/2 to 40 minutes. Jung (1954) was also among the first to report on spontaneous EDA during sleep. In his study of 10 normals and 10 epileptic patients, Jung recorded EDA during wake, drowsiness, and sleep. Jung noted that during drowsiness and sleep slowing of some autonomic responses occurs (e.g., pulse rate and respiration), but that EDA shows only minor variability during different states of consciousness.

To date, much of the research that has been conducted to study electrodermal activity in sleep (EDAS) has involved studying the phenomenon in normals. It is a well-established fact that spontaneous EDA, which is generally observable in both skin potential and skin resistance recordings, is most abundant during stage 4 sleep (slow wave sleep) and tends to decrease dramatically during rapid eye movement (REM) sleep (Asahina, 1962; Broughton, Poiré, & Tassinari, 1965; Burch, 1965; Freixa i Baqué et al., 1983; Hori, Miyasita, & Niimi, 1970; Johnson & Lubin, 1966; Koumans, Tursky & Solomon, 1968; Lester, Burch, & Dossett, 1967; McDonald et al., 1976; Niimi, Wantanabe, & Hori, 1968). Because the amount of sustained EDA during slow wave sleep (SWS) tends to be extremely prevalent, Burch (1965) initially coined the term "storm" to describe this unique activity: "We call them GSR storms because they occur in stage 4 sleep and in such profusion that I think the term 'storm' describes it as well as any" (p. 172). A GSR storm has been arbitrarily defined as a minimum of 5 GSRs/min for at least 10 consecutive minutes of sleep (Lester, Burch, & Dossett, 1967).

Interestingly, newborns show a different pattern of EDAS, in that they have more electrodermal responses during REM than during non-REM (NREM) sleep (Curzi-

Cascalova, Dreyfus-Briasac, 1968). At approximately the same time as the initial occurrence of sleep spindles in newborns is an increase in NREM sleep electrodermal responses. By 9-12 weeks of age, infants show a predominance of electrodermal responses during NREM sleep identical to that of adults.

Among normal adults, EDA is not distributed evenly throughout the night, however (Freixa i Baqué et al., Meriaux, & Spreux, 1983; Koumans, Tursky, & Solomon, 1968). Freixa i Baqué et al. (1983) found that spontaneous skin potential responses are less frequent (i.e., 60%) during the first full sleep cycle compared to the latter three cycles. However, at least one study has found that skin potential responses were less frequent in the latter half of sleep, especially after the third full sleep cycle (Hori, Miyasita, & Niimi, 1970).

Studies investigating the ability of EDAS to predict particular sleep stages have found that EDA is useful to differentiate between wake and sleep (Koumans, Tursky, & Solomon, 1968; Hori, 1982), but is not useful in identifying particular stages of sleep (Koumans, Tursky, & Solomon, 1968). However, in a study of students and surgical patients, Johns, Cornell, and Masterton (1969) found that skin resistance EDAS was effective in determining time to sleep onset, disturbance during the night, and a rough estimate of SWS present. Further, in an attempt to determine whether skin potential EDAS activity could be an indicator of arousal, Hori, Miyasita, and Niimi (1970) studied both the skin potential level and the skin potential responses in 15 males during sleep. The skin potential level measured from the palmar surface of the finger, but not on the dorsal surface of the hand, was found to be significantly correlated with stages of sleep. Skin potential responses on both the dorsal hand and finger were significantly correlated

with sleep stage; however, the amplitude of SPR was not correlated with sleep stages.

The relative lack of EDA during REM sleep has spawned some researchers to search for specific conditions under which EDA does occur during REM. EDA consistently decreases during the six minutes preceding a REM period and can be used to detect REM-phase onset (Koumans, Tursky, & Solomon, 1968). An early study by Broughton, Poire, and Tassinari (1965) found that when EDA does occur in REM sleep, it is often accompanied by bursts of eye movement activity (REM). Kushniruk, Rustenburg, and Ogilvie (1985) studied this further by waking subjects 3-4 times during REM sleep under three conditions: EDA with REM, EDA without REM, and neither REM nor EDA. It was found that dream recall during EDA with REM was more "bizarre" than in the other conditions; that is, dream bizarreness was associated with phasic REM-related electrodermal activity. Although Kushniruk et al. (1985) did not elaborate on what constituted bizarreness, it is interesting to note that individuals with anxiety disorders often report disturbed dream content (Kramer, 2000).

Ogilvie, Belicki, and Nagy (1978) recorded electrodermal responses among 10 subjects during 7 consecutive nights in the sleep laboratory. On the night in which subjects were instructed to increase involvement in their dreams, there were significant increases in electrodermal response measurements. Ogilvie et al. concluded that the elevated electrodermal activity resulted from the mildly stressful verbal instructions. Finally, Hauri and Van de Castle (1973) also examined the relation between dream content and physiological arousal in 15 male volunteers. They found a highly significant association between dream emotionality (as rated by independent raters) and skin potential fluctuations during the final minute of 15 minute REM periods.

The "first night effect" refers to the reliable phenomenon that individuals sleep differently during their first nocturnal polysomnography (PSG) when compared to subsequent nights. The unfamiliar surroundings and equipment is presumed to generate moderate levels of anxiety and cause a fairly reliable sleep pattern (Ware, 1988). That is, first night PSGs tend to result in increased awakenings, both a delayed sleep onset and REM sleep onset latency, and a reduced amount of REM and SWS (Agnew, Webb, & Williams, 1966; Rechtschaffen & Verdone, 1964). Interestingly, several studies have demonstrated that nocturnal EDA shows the same effect. For example, McDonald et al. (1976) performed a study on 21 male undergraduates to determine if there are differences in EDA between night one in a laboratory and subsequent nights. The authors found that storming rates for spontaneous skin potential responses significantly decreased from night one to night two in the laboratory. Further, on night two only, storming rate and amount of Stage 3-4 sleep were significantly negatively correlated, suggesting a relation between storming and "poor" sleep, i.e., SWS time. They concluded that the night in lab is a stressor itself and on the second night subjects show adaptation. Additionally, Johnson and Lubin (1966) reported similar decreases in spontaneous skin potential responses over three night periods and hypothesized that a habituation effect was responsible. Although Freixa i Baqué et al. (1983) showed that there was no significant difference in storming (i.e., skin potential responses) among the three nights of their study, all subjects underwent an initial habituation night, which was not included in the final data analyses.

Several studies have attempted to investigate the effect of sleep deprivation on EDA, and results have been mixed. Increased EDA (i.e., increased skin conductance and

decreased resistance levels) has been demonstrated in subjects after 60-264 hours of sleep deprivation (Johnson, Slye, & Dement, 1965; Malmo & Surwillo, 1960; Naitoh, Pasnau, & Kollar, 1971). Contrary to these findings, Ax and Luby (1961) found decreases in skin conductance levels throughout a 123 hour sleep deprivation period, and Kollar, Slater, Palmer, Docter, and Mandell (1966) found that 120 hours of sleep loss resulted in no significant change in EDA. Thus, research suggests that prolonged sleep deprivation may be associated with increases in EDA, but the literature is inconclusive.

Finally, Krohnholm, Alenen, and Hyyppa (1993) measured afternoon skin conductance level and responses among 199 males and females between the ages of 35-55 and found that the latent variable of "sympathetic activity" was significantly associated with nocturnal motor activity. Although Krohnholm et al. did not assess EDA during sleep, it has been demonstrated that nocturnal motor activity is associated with both anxiety (Uhde, 2000) and poor sleep (Monroe, 1967). Although Ware (1988) posited that individuals with EDA during sleep are not generally thought to have better or worse sleep than those without EDA during sleep, these results lend support to the hypothesis that increased EDA may be associated with anxiety as well as poor sleep.

Nocturnal Electrodermal Activity among Subgroups. Several studies have investigated EDAS among specific subgroups of individuals (i.e., subjects with anxiety disorders, sleep disorders etc.) in an effort to ascertain whether EDA differs among these groups and normals.

It has been demonstrated that subjects with high anxiety/worry show an increased prominence of storming activity when SWS and REM sleep variables are controlled for (Fuller, Waters, Binks, & Anderson, 1997). Interestingly, however, a study of chronic

PTSD patients by Fuller, Waters, and Scott (1994) found that although PTSD subjects had significantly higher scores on self-report measures of depression and anxiety (i.e., Beck Depression Inventory & State Trait Anxiety Inventory), they showed less storming activity than controls; however, they also had significantly less SWS than the controls. Fuller et al. explained these results by referring to information provided in the Lester, Burch, and Dosset (1967) study. Specifically, Lester et al. provided anecdotal information that suggested that severe stressors tend to be associated with an absence of SWS and a substantial reduction in electrodermal activity across all stages of sleep. Lester et al. and others (e.g., Wyatt, Stern, Fram, Tursky, & Grinspoon, 1970) have suggested that the relation of severe stress and electrodermal activity needs further study.

Satoh and Harada (1973) studied skin potential changes on the palm and forearm among 15 patients with bruxism and found that skin potential changes on the forearm were frequently associated with K-complexes as well as phasic vasoconstrictions of the fingertip; the K-complex is considered to be an electroencephalogric (EEG) index of the arousal reaction (Roth, Shaw, & Green, 1956; Johnson & Lubin, 1967). Further, Satoh and Harada found spontaneous episodes of tooth-grinding were often accompanied by skin potential changes on the forearm (i.e., 54%) and less frequently on the hand (29%). In general, these changes in sympathetic activity preceded the onset of tooth-grinding. Research has shown that sleep bruxism is related to anxiety and stress (Lavigne & Manzini, 2000), which lends further support to the hypothesis that EDA during sleep may be related these variables.

Ware, Karacan, Salis, Thornby, and Hirshkowitz (1984) studied 60 impotent males, of whom 42 had "abnormal" nocturnal penile tumescence. Ware et al. found that the

organically impotent patients (i.e., 42 males with abnormal nocturnal penile tumescence) had more electrodermal activity during REM sleep and less during stage 2 sleep than the group with normal erectile capability. However, there were no significant differences between the normal and abnormal nocturnal penile tumescence groups on State Trait Anxiety Inventory scores or Minnesota Multiphasic Personality Inventory profiles. Ware et al. state that "sleep-related electrodermal activity may change in certain other clinical conditions possibly including that of depression because of the associated changes in NREM/REM sleep structure" (p. 253).

Finally, Wyatt, Stern, Fram, Tursky, and Grinspoon (1970) studied spontaneous skin potential fluctuations during the sleep of 8 unmedicated schizophrenics and 6 normals. While the normals showed a typical EDAS pattern during sleep (i.e., decreases during REM and excesses during Stages 3 and 4), the schizophrenic subjects showed a pattern opposite to this. That is, the spontaneous skin potential rate was significantly higher during REM compared to the controls. Further, the schizophrenic subjects had the greatest number of skin potential fluctuations during waking and REM sleep and the fewest during SWS.

Presleep Activity/Stress and Electrodermal Activity in Sleep. Bursch (1965) was among the first to suggest that nocturnal electrodermal activity is related to presleep arousal/anxiety levels; however, Johnson and Lubin (1966) disagreed with this hypothesis based on the findings of their study. They conducted daytime and nocturnal PSGs on 12 epileptic subjects and 17 normals and found that there was no significant relation between waking electrodermal response rates and EDAS. These authors concluded that EDA during sleep could not be interpreted in the same way as EDA

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during wakefulness and that "the increase of spontaneous electrodermal responses during sleep, especially slow wave sleep, and the decrease during the 'activated' period of REM sleep cast doubt upon the very common acceptance of spontaneous EDA as an indicator of arousal, vigilance, or alertness " (p. 16). These authors concluded that storming is essentially a by-product of the release of cortical inhibitory mechanisms, which are at their greatest during SWS.

Subsequent research on nocturnal EDA resulted in a much different conclusion, however. For example, McDonald et al. (1976) reported significant correlations between spontaneous response rates during wakefulness and EDAS. Furthermore, in one of the most cited publications on stress and nocturnal EDA, Lester, Burch, and Dossett (1967) conducted an investigation on various intensity stressors and EEG and skin resistance measures. A total of 53 subjects were studied for a total of 336 nights. Among this group were five medical students who underwent 10-14 nocturnal PSGs each. Each subject spent two habituation nights in the laboratory, and then three nights before, during and after a time-interval anticipation test (mild stressor in which subjects had to anticipate and prevent the occurrence of a repetitive tone), three nights clustered around an important medical school examination (strong stressor), and two nights after the end of the semester.

Lester et al. found that stressful experiences (e.g., first night in lab, medical school exams) were associated with increased GSR activity and decreased SWS. Presleep stress was found to increase the frequency of nocturnal GSRs during all sleep stages, as well as decrease the overall percentage of SWS. Additionally, Lester et al. commented on the

fact that the REM periods that did contain clusters of EDA (12% of 1207) were more likely to occur on high stress nights.

Lester et al.'s study provides us with much informative data; however, the methodology used was flawed and, consequently, this study leaves many questions unanswered. One blatant methodological error is that the results were based mostly on descriptive statistics, and the use of comparative statistical analyses was minimal. Additionally, there were no quantifiable measures of stress, as each time subjects reported to the sleep laboratory they underwent an unstructured "presleep examination", during which time they were interviewed for "significant events" of the preceding day. Related, is the fact that Lester et al. assumed that the medical students were in a high stress state during examination time. Thus, while this study provides a basis for the hypothesis that stress affects EDA during sleep, it cannot be considered conclusive.

Several research projects since the Lester et al. study have sought to investigate more thoroughly the type of presleep arousal that affects EDAS. For example, Hauri (1968) investigated nocturnal EDA after three differing presleep conditions in 15 male subjects: six hours of strenuous exercise, six hours of studying, and six hours of relaxing activities. Hauri demonstrated that the number of rapid fluctuations in skin potential was increased only in the exercise condition, and only during delta sleep that occurred during the first two hours of the sleep period. Thus, even lengthy strenuous physical exercise seems to have a minimal and brief effect on subsequent EDA.

Two unpublished doctoral dissertations have focussed on psychophysiological activation and sleep. Ware (1976) varied psychophysiological arousal in six subjects during three conditions: low arousal (i.e., estimating 20-second time intervals), high

arousal (i.e., working on the Raven Advanced Progressive Matrixes), and a control condition (i.e., reading and watching television). While Ware determined that presleep psychophysiological arousal was not a good predictor of REM or SWS, it was indicative of the soundness of subsequent sleep as measured by both objective and subjective measures. In a more direct investigation of EDA during sleep, Koresko (1976) manipulated presleep stress by sounding a brief series of very loud (i.e., 109 dB) marine air horns to 10 subjects and a quieter 54 dB horn presented to a control group. Koresko found that the experimental group experienced more electrodermal activity during Stage 3 sleep when compared to controls. Koresko concluded that his study provided "some minimal support" for the role of psychological factors in electrodermal activity during sleep.

In a series of studies, McDonald et al. (1976) concluded that electrodermal storming is "highly related to some presleep variables and is not a mere by-product of the presence of Stage 3-4 sleep" (p.132). Specifically, these authors found that electrodermal storming during sleep was significantly positively correlated with EDA during presleep wakefulness and that those subjects who remained alert and did not doze during the awake recording subsequently showed higher electrodermal fluctuations during sleep.

Although the stress-EDAS link is fairly well-established, much of the research in this area is not very well controlled (e.g., controls for sex, race, and age) or designed (e.g., small N) and little has been done with valid and reliable assessment measures of negative affect and stress. While some researchers have included self-report stress questionnaires in their investigations, a thorough assessment of how the various affective states (i.e., depression, anxiety, anger, worry, daily stress etc.) relate to EDAS has not been done.

Because existing data on the relation between EDAS, negative affect and stress is minimal, it is difficult to draw firm conclusions on specific predictors of nocturnal EDA. Several studies have attempted to relate EDAS with various self-report questionnaires. For example, McDonald et al. (1976) obtained Minnesota Multiphasic Personality Inventory profiles from their sample of 21 male undergraduates; it was found that stormers scored significantly higher on the Psychasthenia Scale (Pt), Welsh's A scale, and the Barron's Ego-Strength (Es) Scale, but not on the Taylor Manifest Anxiety Scale. Additionally, McDonald et al. found that dream reports were significantly more common when subjects were awakened during storming (54%) verses nonstorming (15%). Koresko (1976), however, had 20 male subjects complete the State Trait Anxiety Inventory and found that scores were not significantly correlated with spontaneous electrodermal fluctuations in skin resistance and skin potential during sleep. Ogilvie, Belicki, and Nagy (1978) had subjects complete the Rotter Internal-External Locus of Control Scale, and it was found that internally controlled subjects had greater electrodermal response activation and variability when compared to the externally controlled subjects in the study; internally controlled subjects tend to credit themselves with the ability to control reinforcing events in their environments, while externally controlled subjects tend to perceive reinforcing events as controlled by factors external to themselves. Additionally, Thompson and Mueller (1984) found that introverts had higher skin potential EDAS rate when compared to extraverts, but only during the second half of the night. Finally, in their rather large community sample of 199 individuals, Kronholm, Alanen, and Hyyppa (1993) found that the latent variables of "sympathetic activity" (i.e., tonic skin-conductance level, stimulus-unrelated rate of skin conductance response, skin

conductance response dishabituation) and "psychological distress" (i.e., Beck Depression Inventory, Brief Symptom Inventory general severity index, psychic anxiety score of the Karolinska scales of personality) were not significantly correlated.

Sleep Apnea Syndrome and Autonomic Arousal

Sleep apnea syndrome is a medical condition involving repeated cessations of airflow during sleep. There are three types of sleep apnea: obstructive, central, and mixed. An obstructive apnea is characterized by an absence of air exchange at the nose and mouth, for ten seconds or more, with continued thoracic and respiratory effort against a closed or obstructed airway. A central apnea occurs when there is total cessation of both airflow and respiratory effort, for ten seconds or more, with a normally patent airway. Obstructive or central apneas are rarely seen in isolation and, typically, one tends to predominate. Most patients diagnosed with sleep apnea primarily have obstructive apneas. In a mixed apnea, there is a central respiratory pause that precedes an obstructed attempt at breathing. In addition to complete cessation of airflow (apneas), individuals with sleep apnea syndrome commonly experience hypopneas, a ten second or greater reduction of airflow of at least 50% (Bassiri & Guilleminault, 2000; White, 2000). Apneas and hypopneas are usually accompanied by a decrease in arterial oxyhemoglobin saturation (SaO_2), and the extent to which SaO_2 decreases (oxygen desaturation) is highly contingent upon the duration of the respiratory event (Weiss, Launois, & Anand, 2000). Most important for the diagnosis of sleep apnea syndrome is the Apnea-Hypopnea Index (AHI), which identifies the number of problematic respiratory events per hour of sleep. The AHI is derived by dividing the total number of respiratory events during sleep by total minutes of sleep time, and multiplying this number by 60. Although many adults

experience isolated apneic events during sleep, an AHI of 5 or more events per hour is the accepted criterion for a formal diagnosis of sleep apnea syndrome (Bassiri & Guilleminault, 2000).

Sleep apnea syndrome is especially problematic because it decreases the overall quality and restorativeness of sleep. Individuals with obstructive and central sleep apnea show considerable overlap in waking symptomology (i.e., signs of daytime hypersomnolence) because both impair sleep continuity and, thus, sleep quality (restorativeness). They also both act to decrease the deepest and most restorative sleep (slow wave sleep), increase light/transitional sleep (stage 1 NREM sleep), and cause both complete awakenings and transient arousals (the latter being far more frequent and damaging to sleep quality and restorativeness) (Guilleminault, Van Den Hoed, & Mitler, 1978).

In general, normal sleep tends to be associated with an increase in parasympathetic activation and a decline in sympathetic outflow (Hirshkowitz, Moore, & Minhoto, 1997). It has been demonstrated, however, that sleep apnea syndrome is associated with increased ANS/Sympathetic Nervous System/endocrine activity (Dyken, 2000; Eisenberg, Zimlichman, & Lavie, 1990; Schnall, Shlitner, Sheffy, Kedar, & Lavie, 1999; Waldhorne, 1987). Sympathetic activity is especially heightened both during and after acute apneic events (Hedner, Ejnell, Sellgren, Hedner, & Wallin, 1988; Morgan, Crabtree, Palta, & Skatrud, 1995).

Both obstructive and central events result in a temporary increase in systemic and pulmonary arterial blood pressures. While prevalence tends to vary between studies, more than 40% of sleep apnea patients have been diagnosed with daytime systemic

hypertension (Bassiri & Guilleminault, 2000) and 15-20% have pulmonary hypertension (Kessler et al., 1996). During apneic events, there is a marked increase in systemic blood pressure which typically peaks at the end of the apnea immediately after the resumption of air movement. Recurrent apneas result in a cumulative and prolonged increase in blood pressure (Fletcher, 2000b). Research has suggested that hypoxia, probably combined with repeated arousals, is the main contributor to blood pressure elevations among those with sleep apnea (Fletcher, 2000a).

Sleep apnea patients are also thought to be more prone to cardiac arrhythmias. The most common pattern of arrhythmia associated with apneic events is bradycardia during the cessation of breathing, followed by tachycardia at the termination of the event (Bassiri & Guilleminault, 2000). Research has demonstrated that an association between sleep disordered breathing and cardiovascular disease exists (Young & Peppard, 2000). Specifically, there is much evidence that suggests that individuals with sleep apnea are at an increased risk for cardiovascular disease and/or cardiovascular disease complications, even when confounding variables such as obesity, diabetes, and hypercholesterolemia are controlled for (Hedner, 2000).

Finally, studies investigating both plasma and urinary norepinephrine levels, which reflect the degree of sympathetic activity, indicate that individuals with sleep apnea have elevated catacholamine levels. Significant correlations between norepinephrine levels and oxygen saturation (Eisenberg et al., 1990) as well as AHI (Dimsdale, Coy, Ziegler, Ancoli-Israel, & Clausen, 1995) have been demonstrated.

In sum, it is clear that sleep apnea syndrome results in ANS arousal, and these autonomic arousals are partially independent from central nervous system (CNS)

arousals; that is, ANS arousals are not always accompanied by the EEG frequency changes that define central nervous system arousal. These ANS arousals may also contribute to a loss in restfulness and quality of sleep. It would be useful to see if the rate of respiratory event-related EDAS adds to the prediction of sleep quality provided by CNS (EEG) arousals.

Summary and Study Rationale

The study of electrical activity of the skin began over a century ago, and since then much has been learned about this phenomenon. It is clear that EDA is associated with numerous factors such as alertness, attentional shift, effort, negative affect, reaction time, problem solving, successful learning of information, etc. A subset of studies has focused on the occurrence of EDA during sleep. The results of these investigations have led researchers to draw definitive conclusions on when electrodermal storming tends to occur during the sleep cycle. Studies have also provided data that indicates that at least moderate stress tends to increase electrodermal activity during sleep, though, there are currently still many unanswered questions pertaining to this topic.

Specific research endeavors that have shown that EDAS is affected by stress have fallen short in several respects. One major criticism of the existing studies is that they have often utilized poorly quantified or unreliable subjective reports of negative affect and/or stress. Only a few research studies of EDAS have used proven psychometrically sound instruments to specify the exact kinds of negative affect and/or stress that may affect EDA during sleep. Those that did use such reliable quantified measures did not include multiple emotion-specific assessments of presleep negative affects and did not use reliable and valid measures of stress. Further, many studies have failed to include

females, and no study has ever been performed to assess EDAS in a sample that includes patients with common sleep disorders (e.g., sleep apnea, periodic limb movements [PLM] of sleep).

The aims of the present study were: (a) to investigate the relation of negative affect and stress to EDAS, and to determine the extent to which these variables are predictive of EDAS; and (b) to explore the utility of EDAS as an index of sympathetic nervous system arousal and of sleep quality/restorativeness. Additionally, there were several secondary hypotheses tested. The aim of these analyses was to assess for possible relations between apnea and periodic limb movement central (EEG) and autonomic (SPR) arousal variables, and determine the degree to which the respiratory event related EDA and respiratory event arousal indices predicted an objective index of sleep disruption (AHI).

METHOD

<u>Participants</u>

Subjects were recruited from referrals to the Ochsner Clinic of Baton Rouge's Sleep Disorders Center. All patients referred to the sleep laboratory for a diagnostic PSG from November 2001 – May 2002 were asked to participate, except those referred solely for titration of continuous positive airway pressure (CPAP) treatment. Prior to the nocturnal polysomnography, physical examination and medical history information was obtained. In addition, all patients completed a Sleep Disorders Inventory, a self-report questionnaire that assesses for signs and symptoms of all major sleep disorders and sleep hygiene problems. Subjects with psychiatric diagnoses were not excluded, as this would have restricted the range of scores on the self-report measures.

Seventy-eight patients agreed to participate in the study. Eight subjects were excluded from the data analysis for the following reasons: error in measurement by the sleep laboratory technician (5), invalid completion of the questionnaire measures (2), and poor quality recording (1). Therefore, a total of 70 patients were included in the final analyses (41 males, 29 females; mean age = 50.71 ± 11.86). The final sample was composed of 53 Caucasians, 16 African-Americans and 1 Asian-American. See Table 1 for descriptive data regarding sleep disorder severity and sleep architecture obtained during the PSG.

<u>Procedure</u>

Referrals who agreed to participate in this investigation were first asked to read the study consent form (see Appendix A) and were given the opportunity to ask any questions about the investigation. Each subject was asked to complete the following self-report measures: the Demographic Profile Form (see Appendix B), the Profile of Mood

Table 1. Descriptive Data on Sleep Architecture and Sleep Disorder Severity Obtained During the Polysomnography

Polysomnography Variables	Mean	Standard Deviation
% Stage 1 Sleep	13.24	10.02
% Stage 2 Sleep	51.96	13.71
% Slow Wave Sleep	20.38	11.85
% Rapid Eye Movement Sleep	14.43	8.99
Total Sleep Time	4.07	1.83
Apnea-Hypopnea Index	26.86	28.41
Periodic Limb Movement Index	13.43	28.68

States (POMS; McNair, Lorr, & Droppleman, 1992), the Derogatis Stress Profile (DSP; Derogatis, 1980), and the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990).

After completion of the self-report measures, subjects underwent a standard hook-up procedure for a nocturnal polysomnography (see Appendix C), the sleep disorder evaluation for which they were referred. Participation in this investigation involved the addition of two extra surface electrodes to the non-dominant arm and hand. After the subject washed his/her hands with mild soap and water, the additional electrodes were placed on the distal phalange of the middle finger (active site) and on the volar surface of the forearm approximately two-thirds of the distance from wrist to elbow (neutral/inactive site). Due to the fact that the recording equipment has capability to measure only 13 AC channels of EEG, a redundant channel (i.e., C3-A2) was deleted from the standard montage.

Patients slept in a queen-size bed in a hotel-like room, and a Grass Heritage Digital Polysomnograph was used for the recordings. Gold plated cup electrodes were used to monitor electroencephalogram (EEG), submental/chin electromyogram (EMG), electro-oculogram (EOG), electrocardiogram (ECG), and anterior tibialis EMG. Nasal and oral airflow was measured using a Pro-Tech breath sensor (thermistor). Respiratory effort (abdominal and thoracic expansion) was monitored with Med Associates Sleepmate respiratory belts. Subjects' sleep position was monitored on video using an infra-red camera and light source system. If patients evidenced more than 30 apneic events before 2 am., they underwent a split-night study in which the second half of the night was used to titrate CPAP treatment. Otherwise, patients underwent an uninterrupted full-night

diagnostic polysomnography. Following the sleep study, subjects were requested to complete a brief Sleep Diary, which is a modification of one published by Lacks (1987), to obtain a subjective report of sleep quality (see Appendix D).

The PSG data was scored by a licensed polysomnography technologist. Each recorded epoch (30 seconds) of sleep was scored by hand, and respiratory events, sleep stages, and EEG arousals were scored according to Rechtschaffen and Kales (1968) and American Sleep Disorders Association (1992) criteria, respectively. Additionally, skin potential responses were scored to generate three EDAS values: spontaneous skin potential responses per hour of sleep (SSPR) and hourly rates of skin potential responses associated with an apnea/hypopnea (Respiratory Event-SPR; RE-SPR) or periodic limb movements of sleep (PLM-SPR). A computer-generated summary report included the objective variables of interest for this investigation.

Materials/Apparatus: Subjective and Objective Stress Measures

Profile of Mood States (POMS). The POMS is a 65-item adjective checklist that asks the responder to rate adjectives on a 5-point intensity scale (0 = "Not at all"; 4 = "Extremely") according to how they feel at the time of completion. Responses to the POMS generate 6 separate sub-scores: Tension/Anxiety, Depression/Dejection, Anger/Hostility, Vigor/Activity, Fatigue/Inertia, and Confusion/Bewilderment. Additionally, a Total Mood Disturbance (TMD) score can be calculated by summing the scores across all six factors (weighing Vigor/Activity negatively) (McNair, Lorr, & Droppleman, 1992). The POMS-Tension/Anxiety, POMS-Depression/Dejection, POMS-Anger/Hostility, and POMS-Total Mood Disturbance raw scores were used in the initial analyses, but the POMS-TMD was used in the final regression procedures due to

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multicollinearity and singularity issues (See the Primary Analyses section for elaboration).

The revised POMS has been demonstrated to have adequate internal consistency, with reliability values ranging from .84 - .95. Test-retest reliability for the current version was obtained by obtaining intake and pre-treatment scores from 100 psychiatric outpatients; the time range was 3-110 days (mean = 20 days). Reliability estimates for this period ranged from .65 (Vigor) - .74 (Depression). These stability coefficients may appear to be somewhat lower than optimal, but mood states are typically expected to fluctuate with the passage of even short amounts of time (McNair, Lorr, & Droppleman, 1992).

Derogatis Stress Profile (DSP). The DSP is a 77-item self-report inventory based on interactional stress theory, which posits that stress is comprised of three main interactional components: environmental events (vocational environment, domestic environment, health environment), personality mediators (role definition, time pressure, driven behavior, attitude posture, relaxation potential), and emotional responses (hostility, anxiety, depression). Respondents are requested to rate items on a 5-point Likert scale format (0 = not at all true of me; 4 = extremely true of me). The DSP yields two total scores: the Total Stress Score and the Subjective Stress Score (Derogatis, 1980). The Total Stress Score was used in the statistical analyses.

Test-retest and internal consistency coefficients for the 11 dimensions as well as for the Total Stress Score have yielded adequate values (11 dimensions - test-retest: .79 - .93; internal consistency: .72 - .92; overall test-retest: .90). Convergent validity has also been established by generating significant correlations between the DSP and portions of other stress scales such as the Jenkins Activity Survey, the Life Experience Survey, and the

Daily Hassles Scale. High correlations between the DSP Emotional Response measures and heart rate reactivity to stress have also been shown in both laboratory and field environments (Derogatis & Fleming, 1997).

Penn State Worry Questionnaire (PSWQ). The PSWQ is a 16-item self-report measure to quantify worry. The PSWQ is psychometrically sound in that it has high internal consistency (coefficient alphas = .86 - .95) and test-retest reliability (2 weeks: r = .75; 4 weeks: r = .74 - .93; 8 - 10 weeks: r = .92), as well as adequate convergent and discriminant validity (Brown, Anthony, & Barlow, 1992; Meyer, Miller, Metzger, & Borkovec, 1990; Molina & Borkovec, 1994). The PSWQ also effectively distinguishes between those with generalized anxiety disorder from individuals diagnosed with other anxiety disorders (Brown et al., 1992). Raw scores were used in all of the analyses.

EDAS Measures: Skin Potential. Skin potential was recorded using Rochester Electro-Medical Bio-Potential silver/silver chloride electrodes and a sodium chloride electrode jelly solution. Two-sided adhesive collar disks were used to optimize the attachment. An hourly rate of spontaneous skin potential responses (SSPR) and skin potential responses associated with respiratory events (RE-SPR) and periodic limb movements (PLM-SPR) served as the objective stress variables.

Skin potential responses were scored if they were $\geq$ 50 microvolts (peak to trough) and $\geq$ 1.5 seconds from start of a negative limb to baseline (uniphasic negative; i.e., no positive limb) or to baseline after the positive limb, if biphasic. Triphasic SPRs were scored if they were $\geq$ 1.5 seconds from the start of a negative limb to the return to baseline after the second negative limb (See Figure 1 for illustration of types of skin potential responses). SPRs can also be monophasic positive, with no negative limb

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monophasic/uniphasic

biphasic
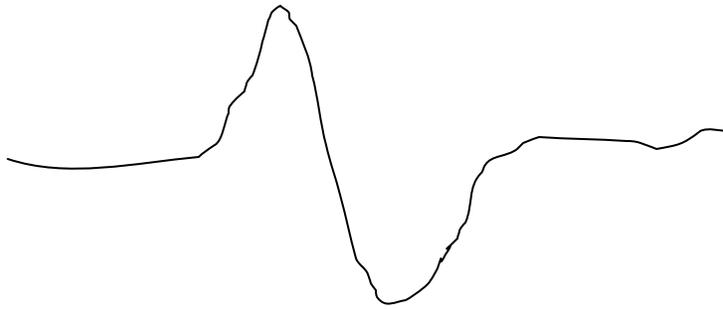
triphasic
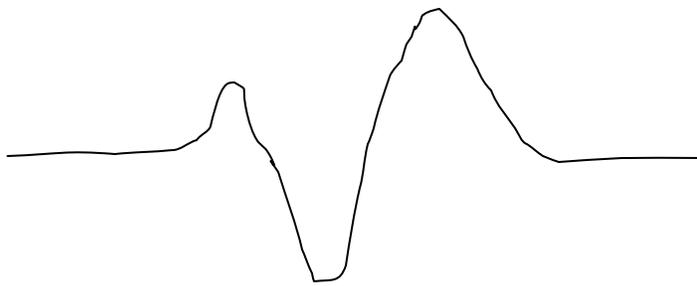
Figure 1. Different Types of Skin Potential Responses

(Boucsein, 1993). Therefore, uniphasic and biphasic SPRs with initial negative limbs were also scored. A SPR was scored as a RE-SPR or PLM-SPR if it occurred during or began from 1-5 seconds after a respiratory event or a PLM. If a SPR occurred simultaneously with a "K"-complex, an indicator of arousal, it was scored as a SSPR.

Experimental Hypotheses

Preliminary Analyses. To determine if it will be necessary to control for specific subject factors of gender, age, race, medication use and study type in the primary analyses.

Hypothesis 1. Negative affect and stress variables will be positively correlated with each of the three EDAS variables: SPR per hour during all sleep stages (SSPR), SPR per hour during SWS (SPR-SWS), and SPR per hour during REM sleep (SPR-REM).

Hypothesis 2. Negative affect and stress variables will be significant predictors of SSPR, SPR-SWS, and SPR-REM.

Hypothesis 3. Subjective ratings of sleep quality and restfulness (i.e., sleep diary data; 1 = more restful/better sleep quality, 5 = poor restfulness/sleep quality) obtained the morning after the sleep study will be positively correlated with the following objective variables: spontaneous transient arousals per hour asleep, respiratory event arousals per hour asleep (RE-arousals), periodic limb movement arousals per hour asleep (PLM-arousals), total arousals per hour asleep, SSPR, RE-SPR, and PLM-SPR.

Hypothesis 4. Objective sleep quality and restfulness, as expressed by six arousal variables (spontaneous transient arousals, RE-arousals, PLM-arousals, SSPR, RE-SPR, and PLM-SPR), will be significant predictors of an overall subjective sleep quality score (sum of the restfulness and quality scores from the sleep diary).

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Additional Hypotheses. Apnea severity and central (EEG) and autonomic (SPR) arousal variables will be positively correlated with one another and both the RE-SPR and RE-arousal indices will be significant predictors of sleep disruption (i.e., AHI). There will also be a positive correlation between the periodic limb movement central (EEG) and autonomic variables (SPR).

RESULTS

All statistical analyses were conducted using SPSS (Statistical Package for the Social Sciences, version 11.0 for Windows). Transformation of the physiological variables was necessary in order to reduce skewness, reduce the number of outliers, and improve the normality, linearity, and homoscedasticity of residuals. See Table 2 for pre-transformed means and standard deviations for independent and dependent variables.

Logarithmic transformations were used on the following stress and sleep variables: SSPR, RE-SPR, transient arousals, RE-arousals, and total arousals. These transformed values were used in all subsequent analyses. Kurtosis and skewness were not sufficiently reduced by either logarithmic or square-root transformations for the PLM-SPR and PLM-arousal variables, as these variables had a large number of subjects that exhibited no responses. Thus, PLM variables were dummy coded (0 = absent; 1 = present) in all of the regression analyses (see Cohen & Cohen, 1983).

Study type (i.e., full-night and split-night) was also dummy coded to control for the two study types in the regression analyses. This was done because the two PSG groups differ in an important way: split-night subjects received CPAP treatment during the second half of the night, a procedure that can seriously affect sleep architecture. Altered sleep architecture is also likely to affect EDAS, as EDAS is most likely to occur during SWS and is least common during REM sleep. Finally, CPAP is a novel stimulus (Grunstein & Sullivan, 2000) that could affect EDAS. Therefore, the pre-CPAP (first) half of the night provided the data for split-night subjects.

Due to the exploratory nature of this investigation, statistical (forward selection) regression was chosen as the optimal regression procedure. A criterion of .15 was used

Table 2. Pre-transformed Means and Standard Deviations (SD) of Independent and Dependent Stress and Sleep Quality Variables

	Variable	Mean	SD
Subjective Stress Variables	POMS – Total Mood Disturbance	36.61	37.98
	Penn State Worry Questionnaire	45.21	14.05
	DSP – Total Stress Score	46.68	8.92
Objective Stress Variables	Overall SSPR Index	53.68	66.67
	SWS/SPR Index (N=67)	110.73	137.54
	REM/SPR Index (N=62)	31.27	55.21
	RE-SPR Index	14.70	28.81
	PLM-SPR Index	2.79	8.16
Subjective Sleep Quality Variable	Sleep Quality/Restfulness Ratings	6.13	2.02
Objective Sleep Quality Variables	Spontaneous Transient Arousals	13.13	7.23
	RE-arousals	12.19	17.46
	PLM-arousals	1.45	3.92
	Total arousals	26.78	20.38

for entry of predictors in the regression analyses, as several authors suggest using more liberal criteria for entry in this statistical procedure (Bendel & Afifi, 1977; Tabachnick & Fidell, 2001).

Preliminary Analyses

Preliminary Analysis 1. Because results from daytime studies suggest that certain demographic variables (i.e., sex, age, race) may affect EDAS, t-test comparisons of SSPR were conducted to determine if it would be necessary to control for these variables in the subsequent analyses. Group comparisons revealed no significant difference in the SSPR index between male and female patients, younger and older patients (using the upper and lower third ages of the subject pool), or between Caucasian and African-American patients. See Table 3 for means, standard deviations, t-values, and significance levels for the group comparisons.

Preliminary Analysis 2. Prescription and over-the-counter medications such as antidepressants and antihistamines have anticholinergic effects that can affect EDAS. Therefore, a t-test comparison was conducted in order to establish if there was a significant difference in the SSPR index among individuals taking anticholinergic medications and those who were not. The group comparison was not significant (See Table 3).

Preliminary Analysis 3. A Multivariate Analysis of Variance (MANOVA) was conducted on the variables of interest to assess for significant differences in the split- and full-night subjects. The overall MANOVA was significant (F [15, 40] = 3.13; p < .01). Subsequent univariate analyses indicated that the two groups were significantly different on the following variables: Derogatis Stress Profile, subjective sleep quality, RE-SPR

Table 3. Means, Standard Deviations (SD), t-values, and Significance Levels for the Preliminary Analyses Group Comparisons of Spontaneous Skin Potential Responses

Variable	Sub-group	N	Mean	SD	t	Significance level
Sex	Male	41	1.35	.60		
	Female	29	1.55	.49	-1.45	n.s.
Age	Younger (N=22)	22	1.49	.59		
	Older (N=23)	23	1.31	.57	1.04	n.s.
Race	Caucasian	53	1.42	1.42		
	African-American	16	1.43	1.43	-.09	n.s.
Anticholinergic Medication	Yes	11	1.50	.53		
	No	59	1.42	.57	.41	n.s.

Note. n.s. = not significant (p < .05).

index, RE-arousal index, and total arousal index. The PSG related variables (RE-SPR, RE-arousal, and total arousal) indicated greater arousal for the split-night (more severe apnea) subjects. See Table 4 for Means, standard deviations, F, and p-values.

Primary Analyses

Hypotheses 1 and 2. To assess for multicollinearity between predictor variables, Pearson product-moment correlations were conducted for the six subjective questionnaire measures: POMS-Anxiety, POMS-Depression, POMS-Anger, POMS-Total Mood Disturbance, Derogatis Stress Profile, and Penn State Worry Questionnaire. Subjective self-report variables were all significantly correlated with one another; r-values ranged from .56 to .94. See Table 5 for bivariate correlations between the subjective measures.

Although there is no specific rule as to what constitutes multicollinearity, Tabachnick and Fidell (2001) and Licht (1995) identify independent variables with r-values of .80 and higher as significantly problematic. Because multicollinearity, as well as singularity (i.e., redundancy), between independent variables can pose statistical problems, the bivariate correlations were inspected and the following three variables were selected as the least likely to violate this assumption of multiple regression analysis, while still testing the related hypotheses: POMS-Total Mood Disturbance, DSP, and PSWQ (see Table 5).

Pearson product-moment correlations (two-tailed) were computed to assess for associations between the self-report measures and SSPR, SPR-SWS, and SPR-REM. None of the correlations were significant. See Table 6 for r-values between the subjective and objective stress variables.

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Table 4. Means, Standard Deviations (SD), F, and p‑values for Split‑and Full‑night Comparisons

		Mean	SD	F	p-value
Penn State Worry Questionnaire	split‑night	42.97	14.75		
	full‑night	47.22	13.27	1.61	n.s.
POMS-TMD	split‑night	32.55	33.33		
	full‑night	40.33	41.93	.72	n.s.
Derogatis Stress Profile	split‑night	44.24	9.33		
	full‑night	49.92	8.02	5.00	.03
Sleep Quality	split‑night	5.55	1.84		
	full‑night	6.67	2.06	5.66	.02
SSPR index	split‑night	1.34	.61		
	full‑night	1.51	.51	1.61	n.s.
SPR-SWS index	split‑night	1.47	.85		
	full‑night	1.69	.69	1.35	n.s.
SPR-REM index	split‑night	.80	.69		
	full‑night	1.23	.56	7.07	n.s.
RE-SPR index	split‑night	1.05	.62		
	full‑night	.51	.42	19.19	.01
PLM-SPR index	split‑night	.25	.46		
	full‑night	.25	.38	.00	n.s.
Transient Arousal index	split‑night	1.09	.23		
	full‑night	1.10	.20	.05	n.s.
RE-arousal index	split‑night	1.19	.39		
	full‑night	.55	.30	59.90	.01
PLM arousal index	split‑night	.19	.33		
	full‑night	.23	.31	.35	n.s.
Total arousal index	split‑night	1.49	.27		
	full‑night	1.24	.19	20.04	.01

Note. n.s. = not significant (p < .05).

Table 5. Pearson Product-moment Correlations between Questionnaire Scales

	POMS-anxiety	POMS-depression	POMS-anger	POMS-TMD	PSWQ	DSP
POMS-Anxiety	--	.85*	.65*	.88*	.62*	.60*
POMS-Depression		--	.78*	.94*	.57*	.58*
POMS-Anger			--	.82*	.58*	.56*
POMS-TMD				--	.61*	.63*
PSWQ					--	.73*
DSP						--

Note. All of the correlations were significant at p < .01.

Table 6. Pearson Product-moment Correlations for Subjective and Objective Stress Variables

	SSPR	SPR-SWS	SPR-REM
POMS-Total Mood Disturbance	.00	-.04	-.15
	(.97)	(.75)	(.24)
Penn State Worry Questionnaire	.12	.05	-.06
	(.32)	(.67)	(.67)
Derogatis Stress Profile	.16	.08	.11
	(.20)	(.52)	(.40)

Note: Significance levels appear in parentheses below the correlation coefficients. None of the correlations were significant at p < .05.

Three separate regression analyses were conducted to determine the degree to which study type, negative affect, worry, and stress predicted: (a) SSPR during all sleep stages, (b) SPR-SWS, and (c) SPR-REM. The latter two analyses were done because individuals who have little or no SWS sometimes have EDAS in other stages, including REM sleep. None of the independent variables predicted the overall SSPR and SPR-SWS indices. Study type, POMS-TMD, and DSP predicted the SPR-REM index ($R = .418$; $R^2 = .175$; adjusted $R^2 = .131$), accounting for 17.5% (13.1% adjusted) of the total variance. The study type entered first, accounting for 10.5% (9% adjusted) of the variance. The regression coefficients (i.e., beta weights) revealed that mood disturbance and stress showed opposite relations to EDAS. That is, as POMS-TMD increased, SPR-REM decreased, but the relation between subjective stress and SPR-REM was positive. As DSP increased SPR-REM increased. See Table 7 for more detailed results of the regression analysis.

Hypotheses 3 and 4. Pearson product-moment, point-biserial, and phi coefficient correlations were calculated between subjective rating of sleep quality and the following objective PSG variables: spontaneous transient arousals, RE-arousals, PLM-arousals, total arousals, SSPR, RE-SPR, and PLM-SPR. Correlations are reported for full-night subjects because of the significant group difference found in ratings of sleep quality for the two types of PSG groups (see discussion for elaboration). The total arousal index was the only variable that was significantly associated with subjective sleep quality ($r = .36$; $p < .05$). See Table 8 for more detailed results of the correlations between the subjective sleep quality variable, objective arousal variables, and skin potential response indices.

Table 7. Statistical Multiple Regression of Self-report Measures on Electrodermal Activity During REM Sleep

Variable	R	R^2	Adjusted R^2	Unstandardized Beta Coefficients	Standardized Beta Coefficients
Study Type	.325	.105	.090	-.381	-.295
POMS-TMD	.375	.141	.111	-.006	-.339
Derogatis Stress Profile	.418	.175	.131	.018	.246

Table 8. Correlations between Subjective Sleep Quality, Objective Arousal Variables, and EDAS Variables for Full-night Subjects (N = 37)

	SQ	STA	RE-arousals	PLM-arousals[a]	TA	SSPR	RE-SPR	PLM-SPR[a]
Sleep Quality (SQ)	--	.31 (.06)	.07 (.67)	.00 (1.0)	.36* (.03)	.16 (.35)	.06 (.71)	.13 (.46)
Spontaneous Transient Arousals (STA)		--	-.06 (.74)	.19 (.25)	.82** (.01)	-.04 (.81)	-.23 (.17)	.12 (.47)
RE-arousals			--	.22 (.19)	.40* (.02)	.38* (.02)	.61** (.01)	.22 (.19)
PLM-arousals				--	.40** (.01)	.29 (.08)	.09 (.61)	.84**[b] (.01)
Total Arousals (TA)					--	.10 (.56)	-.01 (.96)	.33* (.04)
SSPR						--	.78** (.01)	.19 (.26)
RE-SPR							--	-.01 (1.0)
PLM-SPR								--

Note.   Significance levels appear in parentheses below the correlation coefficients. [a]Point-biserial correlations. [b]Phi coefficient correlation. * = p < .05. ** = p < .01.

A regression analysis was conducted to determine the degree to which objective arousal variables were significant predictors of a subjective sleep quality score (sum of the restfulness and quality scores from the Sleep Diary; 1 = very rested/excellent, 5 = poorly rested/very poor). Predictor variables were: study type, transient arousals, RE-arousals, PLM-arousals, SSPR, RE-SPR, and PLM-SPR. Study type and STA predicted subjective sleep quality (R = .386; R^2 = .149; adj. R^2 = .123) accounting for 14.9% (12.3% adjusted) of the total variance. The study type entered first, accounting for 7.8% (6.4% adjusted) of the total variance. The regression coefficient revealed that as STA increased, subjective sleep quality was rated worse. See Table 9 for more detailed results of the regression analysis.

Additional Analyses. The AHI was logarithmically transformed to reduce skewness, reduce the number of outliers, and improve the normality, linearity, and homoscedasticity of residuals. To assess for possible relations between apnea severity and central (EEG) and autonomic (SPR) arousal variables, Pearson product-moment correlations were conducted between the following variables: Apnea-Hypopnea Index, spontaneous transient arousals, SSPR, and Respiratory Event-SPR. Among the significant correlations, the following relations are most noteworthy: AHI was positively correlated with both RE-SPR (r = .74; p < .01) and RE-arousals (r = .95; p <. 01). See Table 10 for the results of the correlations. A similar relation between PLM-arousals and PLM-SPR (full-night subjects: r = .84; p < .01; all subjects: r = .91; p < .01) also emerged.

A regression analysis was also conducted to determine the degree to which the RE-SPR and RE-arousal indices predicted AHI. The predictor variables accounted for a total

Table 9. Statistical Multiple Regression of Arousal and EDAS Variables on Self-report of Sleep Quality

Variable	R	R^2	Adjusted R^2	Unstandardized Beta Coefficients	Standardized Beta Coefficients
Study Type	.279	.078	.064	-1.12	-.278
STA	.386	.149	.123	2.58	.267

Note. STA = Spontaneous Transient Arousal.

Table 10. Correlations between Apnea-Hypopnea Index and CNS and ANS Arousal Variables

	AHI	RE-arousals	STA	SSPR	RE-SPR
AHI	--	.95** (.01)	-.13 (.27)	-.06 (.61)	.74** (.01)
RE-arousals		--	.02 (.90)	-.13 (.30)	.71** (.01)
STA			--	-.13 (.28)	-.22 (.07)
SSPR				--	.37** (.01)
RE-SPR					--

Note: Significance levels appear in parentheses below the correlation coefficients. AHI = Apnea-Hypopnea Index; STA = Spontaneous Transient Arousals. ** = $p < .01$.

of 91% of the total variance. The RE-arousal index entered first, while the RE-SPR

accounted for an additional 1% of the total variance. See Table 11 for more detailed

results of the regression analysis.

Table 11. Statistical Multiple Regression of CNS and ANS Arousal Variables on Apnea-Hypopnea Index

Variable	R	R^2	Adjusted R^2	Unstandardized Beta Coefficients	Standardized Beta Coefficients
RE-arousals	.950	.903	.902	.835	.852
RE-SPR	.955	.913	.910	.109	.138

DISCUSSION

The aims of this investigation were: (a) to determine the relation of negative affect and stress to EDAS and the extent to which these variables are predictive of EDAS; and (b) to explore the utility of EDAS as an index of sympathetic nervous system arousal and of sleep quality. The study sought to determine if EDAS has the potential to be an index of the effect of presleep stress upon sleep quality, and an index of sympathetic nervous system arousal caused by respiratory events (apneas and hypopneas) that disrupt and cause non-restorative sleep.

The correlational analyses conducted on self-report of negative affect, worry and stress yielded no significant correlations between the questionnaire measures and EDAS. In the related regression analyses, none of the self-report measures reliably predicted overall EDAS or EDA during SWS; however, study type and self-report of mood disturbance and stress reliably predicted EDA during REM sleep, accounting for 18% (13% adjusted) of the total variance. The study type entered first, accounting for 11% (9% adjusted) of the variance. As mood disturbance increased, EDA during REM sleep decreased, while the association between subjective stress and EDA during REM was positive, i.e., as subjective stress increased, EDA during REM also increased.

The fact that the study type was a significant predictor of EDA during REM is understandable since full-night subjects had more of an opportunity for REM sleep than split-night subjects. The finding that subjective report of stress was a contributor was also consistent with the original hypothesis and with previous research that has shown a positive relation between pre-sleep stress and EDAS (e.g., McDonald et al., 1976; Lester, Burch, & Dossett, 1967; Koresko, 1976). This finding is unique, however, because it

occurred only in REM sleep. It may be that EDA in REM occurs when stress is sufficient to reduce SWS.

The negative relation between mood disturbance scores and EDA during REM sleep was contrary to the original hypothesis. There are two possible explanations for this finding. First, because the POMS-Total Mood Disturbance score is a collective combination of 6 subscale scores (generated by combining the five subscales of depression, anxiety, anger, fatigue, and confusion while weighing the vigor subscale negatively) it is a reflection of numerous variables, only three of which reflect negative affect. This potential problem was examined by a post-hoc analysis using the same predictor variables (i.e., POMS, PSWQ and DSP), however, a negative affect score comprised of the negative affect variables of interest (i.e., POMS-Anxiety + POMS-Depression + POMS-Anger) was used in place of the overall mood disturbance score. These additional analyses yielded comparable results to those obtained in the primary analyses, as none of the self-report measures were reliable predictors of either EDA across all sleep stages or during SWS. The POMS-negative affect score and the DSP predicted the SPR-REM index, accounting for 21% of the total variance ($R = .46$; $R^2 = .21$; adj. $R^2 = .17$). As in the original regression, self-report of mood disturbance and stress were negatively and positively associated with EDA during REM sleep, respectively. Therefore, it is unlikely that the additional scores of fatigue, confusion and vigor were the source of this unexpected finding.

A second reason for this finding may be related to the differing physiological effects of anxiety and depression. Specifically, while EDA studies focusing on anxious individuals have generally found a positive relation between these two variables, the

results of daytime EDA studies among depressed individuals have been mixed; that is, some investigations have found reduced activity among depressed subjects, while others have not found this effect (Hugdahl, 1995; Boucsein, 1993). Therefore, the emotions of anxiety and depression have shown differing results in regard to their relation to EDA, which may have been a confound. Because individuals with sleep apnea (84% in this sample) often report significant depressive symptoms (Beutler, Ware, Karacan, & Thornby, 1981; Borak, Cieslicki, Koziej, Matuszewski, & Zielinski, 1996; Kales et al., 1885; Kaplan, 1992; Millman, Fogel, McNamara, & Carlisle, 1989; Mosko et al., 1989; Reynolds et al., 1984), it may be that the POMS-Total Mood Disturbance scores in this sample were largely affected by the tendency of the subjects to report high rates of depressive symptomatology and, thus, a negative relation between mood disturbance and EDAS emerged.

The correlational analyses addressing hypothesis 3 were first conducted among the entire subject sample (N = 70), and it yielded two significant correlations between subjective report of sleep quality and arousal variables (see Appendix E for the complete results of the correlational analyses). There was a significant positive association between subjective sleep quality and spontaneous transient arousals; as transient arousals increased, subjective report of sleep quality was rated as worse. It was expected that decreased sleep quality would be associated with more frequent transient arousals. The second significant correlation was between sleep quality and the Respiratory Event-SPR index. As Respiratory Event-SPR increased, subjects rated their sleep quality ratings as better, which is a finding opposite to the original hypothesis. The most plausible explanation for this finding is that the sleep diary, which was completed the morning

after the sleep study, was capturing the typical dramatic improvement in sleep architecture and the greatly improved sleep quality that individuals receiving CPAP often report (Grunstein & Sullivan, 2000). In fact, the univariate ANOVA conducted as part of the preliminary analyses indicated that the split-and full-night groups differed significantly on the self-reported sleep quality variable with full-night subjects reporting poorer sleep quality than CPAP treated split-night subjects ($p < .05$). Therefore, the analysis addressing hypothesis 3 was re-conducted among the full-night group, and the only significant correlation that emerged was between self-reported sleep quality and total arousals.

In the related regression analysis conducted with the entire subject pool, the study type and spontaneous transient arousal variables predicted subjective sleep quality, accounting for 15% (12% adjusted) of the total variance. As spontaneous transient arousals increased, subjective sleep quality was rated worse. As in the related correlations, the relation between spontaneous transient arousals and sleep quality was expected. To confirm this finding, a post-hoc analysis with the same predictor and criterion variables was conducted among full-night subjects only ($N = 37$), eliminating the potential confounding influence of CPAP treatment. Results were the same as in the original analyses, i.e., the spontaneous transient arousal variable was the only independent variable that significantly predicted sleep quality ($R = .312$; $R^2 = .097$; adj. $R^2 = .071$). Thus, the overall finding of these analyses is that EDAS had a negligible effect on self-report of sleep quality, but transient arousals were significantly related.

One interesting non-hypothesized finding was the significant correlations that emerged between Respiratory Event and PLM-arousals and their respective EDAS

variables. The significant correlations between RE-arousals and RE-SPR and the equally strong correlation between PLM-arousals and PLM-SPR indicate the presence of a very strong relation between respiratory events and periodic limb movements with both central and autonomic nervous system arousal. Other research has demonstrated that sleep apnea syndrome is associated with increased ANS/Sympathetic Nervous System/endocrine activity (e.g., Dyken, 2000; Eisenberg, Zimlichman, & Lavie, 1990; Schnall, Shlitner, Sheffy, Kedar, & Lavie, 1999; Waldhorne, 1987). The present data reveal that skin potential is a valid measurement of sleep disruption associated with apneic events and periodic limb movements, and that a combination of both CNS and ANS arousal variables is a better indicator of the amount of sleep disruption present.

There are several possible reasons why the hypothesized relations between EDAS and negative affect/stress and sleep quality did not emerge. Two explanations lie in characteristics of the subject sample, 84% of which was comprised of individuals with diagnosable sleep apnea syndrome (AHI > 5 per hour; mean AHI = 26 ± 28). It is well known that individuals with sleep apnea syndrome often have greatly disrupted sleep with high rates of autonomic instability and movement (Bassiri & Guilleminault, 2000; Weiss, Launois, & Anand, 2000). Thus, the hypothesized results could have been obscured by this characteristic poor sleep and other ANS arousal variables (e.g., the strong sympathetic nervous system response to long involuntary respiratory pauses), resulting in non-significant associations between independent and dependent variables. The fact that expected group differences (e.g., sex, age, and race variables) found in the existing literature (see Boucsein, 1992) were not apparent in this sample further supports this hypothesis. In fact, a study of EDAS in individuals with sleep apnea has not been

conducted to test whether or not EDAS in this subgroup differs from the pattern seen in normals. The inclusion of more normal participants would have allowed for valuable group comparisons to be made.

Finally, it is possible that the negative findings of this study were due to methodological problems and flaws. One limitation of this study was the absence of a habituation night prior to the collection of EDAS variables, as the novel conditions of the sleep laboratory have been shown to disrupt sleep and act as a stressor (Agnew, Webb, & Williams, 1966). It may be that the stress of the overnight sleep study increased EDAS frequency and thus obscured state arousal measurements. Related, is that the data was collected during one night of polysomnography, where it would have been optimal to study individuals over a longer period of time as some of the previous research has done (e.g., Lester et al., 1967). Finally, it would have added substantially to the findings if additional ANS arousal variables were measured to see if they showed the same effect.

Conclusions

The results of this investigation determined that weak relations between negative affect, worry and stress and EDAS exist among a sleep-disordered population, and that these variables are largely ineffective in predicting EDAS. Further, EDAS had no relation to self-reported sleep quality among the subjects studied. While the original hypotheses were not supported, additional analyses indicated that significant associations between apneic and periodic limb movement events and both central (EEG) and autonomic (SPR) arousals exist. Though EEG arousals associated with apneic events (Respiratory Event-arousals) account for the majority of the variance in sleep disruption,

a combination of both CNS and ANS arousal variables might provide a better indicator of

the amount of sleep disturbance (i.e., AHI) present.

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OCHSNER CLINIC FOUNDATION INFORMED CONSENT

Study to Investigate Electrodermal Storming Among Sleep Disordered Patients

Principal Investigator: William F. Waters, Ph.D.
Sub-Investigator: Laura E. Lajos, M.A.

You have been invited to participate in a clinical research study.

The doctors at Ochsner study the nature of disease and attempt to develop improved methods of diagnosis and treatment. This is called clinical research. To decide whether or not you should agree to be part of this research study, you should understand enough about its risks and benefits to make an informed judgment. This process is called informed consent.

PURPOSE
The purpose of this study is to determine the relation between stress and electrodermal activity (EDA) (i.e., sweating during sleep), as well as give us a better idea of how EDA may affect sleep quality/restorativeness. You have been asked to participate in this study because you were referred to the Sleep Disorders Center for a polysomnography (PSG), and all referrals are asked to take part.

PROCEDURE
Participation in this investigation involves the completion of several questionnaires and the addition of two extra electrodes to your non-dominant arm and hand. The additional electrodes will be placed on the tip of the middle finger (active site) and on an inactive site on the forearm area (neutral site).

BENEFITS
The study will not benefit the patient directly, but participation will add to our current understanding of EDA during sleep.

RISKS
There are no foreseeable risks to participating in this study, as it only involves completion of several questionnaires and the addition of two extra electrodes to measure sweating during your sleep study.

ALTERNATIVE METHODS/TREATMENTS
This investigation will not evaluate specific treatments for sleep disorders. Because the study does not evaluate treatment, there is no alternative.

PAYMENT FOR PARTICIPATION AND/OR REIMBURSEMENT OF EXPENSES
Your participation will not result in any additional medical costs and you will not be financially compensated. Further, this investigation is being conducted for research purposes only; the principal investigator and/or the co-investigator will not receive any financial compensation.

ADDITIONAL COSTS
There are no known additional costs that will result from the study.

COMPENSATION FOR INJURY
You should understand that in the event of related injury from the research procedures, medical treatment, including hospitalization, if necessary for injuries or illness, is available. This medical treatment and/or hospitalization *is not* provided free of charge. If you feel that you have suffered an injury which may be related to the research procedures, you should contact William F. Waters, Ph.D. at (225) 761-5852 for further information.

CONFIDENTIALITY
The confidentiality of your records will be maintained to the extent consistent with the law. Government agencies, such as the Food and Drug Administration, may review and copy your records to ensure compliance with regulations and protocols.   Additionally, the sponsor and representatives of Ochsner's Clinical Investigations Committee, Office of Research Administration, and/or the Research Compliance Office may review the records to ensure that all regulatory requirements are being met. You will not be identified in any reports or publications resulting from this study.

Complete secrecy cannot be guaranteed. Your personal data may be disclosed if required by law. Your records from this study may be sent by facsimile transmission (FAX machine) or over the Internet. It is possible (although unlikely) that your records could be sent to the wrong person. Records sent by computer will be sent using current Internet security.

CONTACT INFORMATION
You should understand that should you have any questions about the study, you should contact the Responsible Investigator Dr. William F. Waters or his associate Laura E. Lajos by calling (210) 641-6361. You should know that you may take any problem or any question concerning your rights to the Office of Research administration, fifth floor, Brent House, Telephone (504) 842-3562.

If you are interested in psychological treatment of stress (e.g., anxiety, depression, stress management training) please contact the Ochsner Clinic of Baton Rouge's Department of Psychiatry/Social Work at (225) 761-5200 or the Louisiana State University Psychological Services Center at (225) 388-1494 to make an appointment.

SAFEGUARDS

1. Where no assurance can be made concerning the results that may be obtained (since results from a clinical research study cannot be predicted), your physician, acting as Responsible Investigator, will take every precaution consistent with the best medical practice;

2. By signing this consent form you have not waived any of your legal rights or released this institution from liability for negligence;

3. Participation in this study is voluntary. You may revoke your consent and withdraw from this study at any time without any penalty or loss of benefits to which you are otherwise entitled;

4. You should disclose to your doctors all of your past and present diseases and allergies of which you are aware an all drugs and medications which you are presently using.

STATEMENT OF VOLUNTARY AGREEMENT TO PARTICIPATE

The procedure involved, expected duration of participation, alternative methods/treatments, and possible benefits, discomforts, risks, and adverse effects have been explained to me in language I understand as set above by Dr. Waters or Laura Lajos. I have been given the time to ask questions, which have been answered to my satisfaction. I have been given a copy of this consent for my information and records. I voluntarily consent to participate in this investigation.

_____   _____

Patient   **Date**

_____   _____

Person Obtaining Consent   **Date**

_____   _____

Responsible Investigator   **Date**

DEMOGRAPHIC PROFILE FORM

****

Sex: ÿ Male   **Age:** _____
        ÿ Female

Race: ÿ Caucasian
         ÿ African-American
         ÿ Asian
         ÿ Other: _____ (please specify)

Height: ____ feet ____inches   **Weight:** _____ pounds

Handedness: ÿ Right handed
               ÿ Left handed

Highest Level of Education: ÿ Did not complete high-school
                               ÿ Completed high-school
                               ÿ GED
                               ÿ Some college: how many years?_____
                               ÿ College degree
                               ÿ Post-college degree: please specify: _____

Please List any medications (prescription or over-the-counter) that you are currently taking and the condition you are taking it for. Also, place a checkmark next to the medications you plan to take within 2 hours of your sleep study.

Name of Medication	**Condition**
_____	_____
_____	_____
_____	_____
_____	_____

(please continue your list on the back of this form, if necessary)

For Females Only:
When was the first day of your last period? _____(month) _____ (date)
When do you expect your next period to begin? _____(month) _____ (date)
<u>OR</u>
ÿ   Check here if you no longer menstruate and have reached menopause

APPENDIX C

STANDARD HOOK-UP FOR POLYSOMNOGRAPHY

	Sleep Apnea Montage	Narcolepsy Montage
1.	Skin Potential	Skin Potential
2.	C4 - A1	C4 - A1
3.	C7 - (A2 & A2)	C7 - (A2 & A2)
4.	02 - C7	02 - C7
5.	EMG (Chin)	EMG (Chin)
6.	Left Eye - (A2)	Left Eye - (A2)
7.	Right Eye - (A2)	Right Eye - (A2)
8.	Snoring Mike	EKG
9.	Nasal - Oral Flow	-
10.	Chest Effort	-
11.	Abdominal Effort	-
12.	EKG	-
13.	Left & Right Leg EMG	-

APPENDIX D

DAILY SLEEP DIARY

DATE: _____

PLEASE RESPOND TO THESE QUESTIONS SOON AFTER YOU WAKE UP FOR THE DAY.

1. How many minutes did it take you to fall sleep last night? _____

2. How many times did you awaken last night? _____

3. Please estimate the total number of minutes you spent awake last night. _____minutes

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

1	2	3	4	5
Not Very Difficult				Extremely Difficult

5. How rested do you feel this morning?

1	2	3	4	5
Very Rested				Poorly Rested

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

1	2	3	4	5
Excellent				Very Poor

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

1	2	3	4	5
Extremely Relaxed				Extremely Tense

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

1	2	3	4	5
Very Quiet				Very Active

10. How well do you think you were functioning yesterday?

1	2	3	4	5
Very Well				Very Poorly

APPENDIX E

CORRELATIONS BETWEEN SUBJECTIVE SLEEP QUALITY, OBJECTIVE AROUSAL VARIABLES, AND EDAS

VARIABLES

	SQ	STA	RE-arousals	PLM-arousals[a]	TA	SSPR	RE-SPR	PLM-SPR[a]
Sleep Quality (SQ)	--	.27* (.03)	-.17 (.17)	.03 (.80)	.03 (.82)	.07 (.55)	-.26* (.03)	.09 (.46)
Spontaneous Transient Arousals (STA)		--	.02 (.90)	.06 (.63)	.53** (.01)	-.13 (.28)	-.22 (.07)	.02 (.85)
RE-arousals			--	.07 (.58)	.78** (.01)	-.13 (.30)	.71** (.01)	.09 (.48)
PLM-arousals				--	.21 (.08)	.22 (.07)	.05 (.67)	.91**[b] (.01)
Total Arousals (TA)					--	-.22 (.07)	.42** (.01)	.20 (.10)
SSPR						--	.37** (.01)	.17 (.17)
RE-SPR							--	.03 (.80)
PLM-SPR								--

Note.   Significance levels appear in parentheses below the correlation coefficients; N = 70. [a]Point-biserial correlations. [b]Phi coefficient correlation. * = $p < .05$. ** = $p < .01$.

VITA

Laura Elaine Lajos was born in Buffalo, New York, on June 1, 1971. After completing high school at the Buffalo Seminary in 1989, she enrolled at the University of Rochester and was awarded a Bachelor of Arts degree in Psychology in 1993. She went on to receive a Master of Arts degree in Psychology from Boston University. Following graduation, she worked for two years at McLean Hospital, a psychiatric facility in Belmont, Massachusetts. In August 1996, she began graduate school in the Clinical Psychology program at Louisiana State University and was awarded a second Master of Arts degree in Psychology in 1999. She completed her pre-doctoral internship at the South Texas Veterans Health Care System, Audie L. Murphy Division. In September 2002, she will begin a one-year post-doctoral fellowship in Health Psychology and will receive her doctoral degree in Clinical Psychology in December 2002.