The relationship between obstructive sleep apnea syndrome and insomnia: implications for treatment

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THE RELATIONSHIP BETWEEN OBSTRUCTIVE SLEEP APNEA SYNDROME AND INSOMNIA: IMPLICATIONS FOR TREATMENT

A Dissertation

Submitted to the Graduate Faculty of theLouisiana State University andAgricultural and Mechanical Collegein partial fulfillment of therequirements for the degree ofDoctor of Philosophy

in
The Department of Psychology

by
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Obstructive sleep apnea syndrome (OSAS) is a sleep-disordered breathing condition that causes disrupted sleep. Although OSAS is most often associated with daytime hypersomnolence, a number of OSAS patients complain of insomnia, i.e., disorders of initiating or maintaining sleep. If the insomnia in patients with OSAS is secondary to the medical condition, then it would be expected to abate with the successful treatment of OSAS. If, however, the insomnia is primarily of a psychological nature, thus considered primary or psychophysiological insomnia, little to no change in insomnia symptoms would be expected after the treatment of OSAS.

The present study examined the relations between OSAS and insomnia and attempted to determine if insomnia is secondary to OSAS by studying changes in insomnia over the course of treatment for OSAS with continuous positive airway pressure (CPAP). Forty-one individuals participated in the baseline assessment and a smaller sample of participants (n = 15) were followed over the course of OSAS treatment. All participants underwent a nocturnal diagnostic polysomnography (PSG) and self-monitored insomnia, daily functioning, sleep hygiene practices, and somatic and cognitive pre-sleep arousal for a one-week period prior to, and after, CPAP treatment. Baseline and post-treatment analyses provided evidence suggesting that OSAS and insomnia are independent sleep disorders. There was no association between sleep apnea severity and any self-reported measure of insomnia. Furthermore, there were no significant changes in insomnia after successful treatment of OSAS with CPAP. There were however, significant reductions in both somatic and cognitive arousal, constructs known to be closely associated with insomnia, after CPAP treatment. It is
possible that somatic and cognitive arousal decreased as a function of decreased worry and anxiety about the sleep disruption typically caused by OSAS.

Another potential explanation of why insomnia did not improve after CPAP treatment involves the significant correlation ($r = .59$) between CPAP interference and sleep-onset insomnia. These results suggest that a longer adjustment time to the CPAP stimulus might be necessary before definitive conclusions are reached regarding the proposed lack of association between OSAS and insomnia. Implications for the assessment and treatment of comorbid OSAS and insomnia are discussed.
Introduction

Obstructive sleep apnea syndrome (OSAS) is a serious medical condition characterized by repetitive partial or complete obstruction of the upper airway during sleep (Bassiri & Guilleminault, 2000). OSAS is the most common condition evaluated at sleep disorder centers and has higher rates of morbidity and mortality than any other sleep disorder. OSAS is associated with hypertension, increased risk for congestive heart failure, coronary artery disease, and myocardial infarction (Chervin & Guilleminault, 1996). OSAS also presents serious immediate health consequences, such as excessive daytime sleepiness (EDS), which itself can impair cognitive functioning and alertness (Bennett, Barbour, Langford, Stradling, & Davies, 1999; Lichstein, Riedel, Lester, & Aguillard, 1999). An often-cited illustration of the potential short-term impact of the EDS associated with sleep apnea, involves the 7 times higher rate of automobile accidents involving persons with OSAS (Findley, Unverzagt, & Suratt, 1988). A recent replication study of sleepiness-related accidents in sleep apnea patients concluded that those with moderate to severe sleep apnea syndrome had a 15-fold risk increase of motor vehicle accidents compared to normal controls (Horstmann, Hess, Bassetti, Grugger, & Mathis, 2000). In addition to the potentially serious health consequences of OSAS, undiagnosed OSAS bears a sizable economic cost for the United States of America. Cost data from the year prior to the diagnosis of sleep-disordered breathing revealed that patients with undiagnosed sleep apnea required $2720 in medical costs compared to $1384 in age and gender matched controls (Kapur et al., 1999).

Description and Classification of Obstructive Sleep Apnea Syndrome

An obstructive apnea is characterized by an absence of air exchange at the nose and mouth, for 10 seconds or more, with continued thoracic respiratory effort against a closed or
obstructed airway. In addition to complete cessation (decrease by 70%-100%) of airflow (apneas), individuals with OSAS commonly experience hypopneas, a reduction (decrease of 30%-70%) without cessation in airflow or effort. The exact definition of a hypopnea has been more difficult to determine. Until recently, the standard criteria involved a 10 second or greater reduction of airflow of at least 50% (Parkes, 1985). However, a recent report re-defined hypopneas in an adult patient as abnormal respiratory events lasting at least 10 seconds with at least 30% reduction in thoracoabdominal movement or airflow as compared to baseline, and with at least a 4% oxygen desaturation (Meoli et al., 2001). Apneas and hypopneas typically last between 20 and 30 seconds and are often accompanied by a decrease in arterial oxyhemoglobin saturation (SaO₂) (Shepard, 1989). The extent to which SaO₂ decreases is dependent upon the duration of the respiratory event and the stage of sleep in which the event occurs. Arterial oxyhemoglobin desaturation is almost always greater in REM sleep, as opposed to NREM sleep, typically resulting from a lengthening of the apneic event (Weiss, Launois, & Anand, 2000). There is also a desensitization of medullary respiratory center receptors in REM sleep over and above that, which accompanies NREM sleep.

These sleep-related respiratory events are often accompanied by a central nervous system (CNS) arousal response and an autonomic nervous system (ANS) arousal response, which at a minimum, produce transient arousals from sleep, and if great enough, result in complete awakenings (Strohl, Sullivan, & Saunders, 1984).

Transient arousals from sleep are characterized by bursts of alpha waves lasting between 3 and 14 seconds, which when they occur repeatedly throughout the night, degrade
the overall quality of sleep causing it to be light, discontinuous, and less refreshing or restorative (American Sleep Disorders Association, 1992; Bonnet, 1987; Bonnet, 1989).

The Apnea + Hypopnea Index (A + HI), also referred to as the Respiratory Disturbance Index (RDI), is a measure of the number of sleep-related respiratory events per hour of sleep. The severity of OSAS is frequently determined by an examination of both the A + HI and the minimum oxygen saturation. *Mild* OSAS is considered to have an A + HI of 10 - 15 and a minimum SaO₂ of approximately 89%, compared to *severe* OSAS where the A + HI must be greater than or equal to 30 and the minimum SaO₂ is less than or equal to 80% (Reite, Ruddy, & Nagel, 1997). Moderate OSAS is presumed then, to require an A + HI of 16 - 29 events per hour of sleep, whereas an A + HI of 5 - 9 would be considered to be “borderline”, but is still often diagnosed as OSAS.

The exact prevalence of OSAS is difficult to determine because of the variability in research methodology and procedures used for diagnosing the disorder. Young et al. (1993) reported that OSAS was present in 4% of men and 2% of women between the ages of 30 and 60, when OSAS was defined as an A + HI of at least 5 and included a presenting complaint of EDS. A recent study revealed a 29% (A + HI = 15) to 43% (A + HI = 5) rate of undiagnosed sleep apnea in a sample of older adults (mean age = 69.4 years) with insomnia (Lichstein et al., 1999). Interestingly, this study was concerned with ruling out cases of OSAS and therefore required each subject to undergo a thorough clinical interview before participating in a diagnostic nocturnal polysomnography. The results of this study not only provide an estimate of the prevalence of sleep apnea in an elderly population presenting with a complaint of insomnia, but they highlight the lack of sensitivity clinical interviews may have with respect to diagnosing OSAS.
OSAS is more common in men than women, and increases with age, with up to 24% of elderly individuals having clinically significant sleep apnea syndrome (SAS), which includes both obstructive and central sleep apnea (Ancoli-Israel, Kripke, & Mason, 1987; White, 2000). The gender difference with respect to apnea frequency tends to narrow with age, with women experiencing an average of 26 events per hour versus 34 events per hour for men (Ware, McBrayer, & Scott, 2000).

While OSAS involves an obstruction of the upper airway, central sleep apnea syndrome (CSAS) is characterized by a loss of respiratory effort (White, 2000). A central apnea is defined, as a period of at least 10 seconds without airflow, during which there is no respiratory effort. Although OSAS and CSAS are often conceptualized as mutually exclusive, in reality most patients present with a combination of obstructive and central apneas; in fact, it is rare to diagnose a “pure” case of CSAS (White, 2000).

Clinical Signs and Symptoms of OSAS

The signs and symptoms of OSAS can be categorized according to symptoms during sleep and daytime symptoms. Symptoms that may occur during sleep include snoring, abnormal motor activity, nocturnal sleep disruption (insomnia), choking, esophageal reflux, nocturia and/or heavy sweating. Patients may not be aware of, or may deny some of the symptoms; therefore, corroboration from family or bed partners is often helpful (Guilleminault, 1989).

Daytime symptoms of OSAS may include excessive daytime sleepiness, cognitive impairments, impaired memory and/or concentration, hypnagogic hallucinations, changes in personality, sexual problems, headaches and/or loss of hearing. The vast majority of patients will not present with all symptoms, but frequently several will be present. A clinical
assessment including an evaluation of obesity and fat distribution, an examination of the oronasomaxillofacial region and other potential aggravating factors such as alcohol use, CNS depressant drugs and/or partial sleep deprivation will aid in making the diagnosis. Ultimately an objective evaluation via nocturnal polysomnography (PSG) is the best clinical method for diagnosing the presence of OSAS. During the PSG, the type, frequency, and duration of apneas and hypopneas are monitored. Numerous other symptoms can also be monitored; however, they must be chosen carefully due to the increased risk of disrupting the patient’s sleep in the laboratory.

**Treatment of OSAS**

Nasal continuous positive airway pressure (CPAP) has become the major non-surgical, long-term treatment for OSAS. CPAP acts as an air-pressure splint, which maintains the patency of the upper airway by providing continuous pressure to the interior of the airway thus, preventing it from collapsing (Grunstein & Sullivan, 2000). The efficacy of CPAP has been established in a number of studies with mild to severe forms of OSAS (Monasterio et al., 2001; Montserrat et al., 2001).

Although CPAP is the treatment of choice for OSAS, patient compliance with CPAP remains an important focus of the treatment. Numerous definitions of compliance have been proposed (Grunstein & Sullivan, 2000). For the purposes of this study, compliance will be defined as the degree to which patients who accept CPAP treatment for OSAS report actually using it. Few studies have examined the number of patients who accept CPAP as a treatment recommendation. Those studies that have been conducted found that 70% - 76% of patients offered a CPAP trial night or the opportunity to take a CPAP machine home accepted the recommendation (Grunstein & Sullivan, 2000).
Daily compliance with CPAP is most often measured via self-report estimates of daily usage (Hui et al., 2001; Monasterio et al., 2001; Montserrat et al., 2001), although CPAP machines that contain built-in usage clocks to record daily use are now available and ideal for improving the reliability of the data. Comparisons of subjective (patient self-report) and objective (built-in time clock) measures of compliance consistently show that patients overestimate their average use of CPAP by approximately 1 hour per night (Rauscher, Formanek, Popp, & Zwick, 1993; Kribbs et al., 1993). Use of CPAP for more than 4 hours per night on more than 70% of the nights and/or symptomatic improvement is generally considered a treatment success (Grunstein & Sullivan, 2000; Kribbs et al., 1993).

Insomnia and OSAS

Although OSAS more typically produces a disorder of excessive somnolence, a number of cases of insomnia associated with a polysomnographic finding of sleep apnea have been reported (George, 2000; Guilleminault, Eldridge, & Dement, 1973). The disordered breathing events often result in transient arousals or complete awakenings from sleep, potentially producing disorders of initiating and maintaining sleep. Sleep fragmentation is typically not perceived by classic OSAS patients, however OSAS patients with insomnia are more aware of sleep fragmentation and complain of recurrent awakenings and sleep maintenance difficulties (Reite, Buysse, Reynolds, & Mendelsohn, 1995). Insomnia tends to be more common in patients diagnosed with central sleep apnea, although mild obstructive and mixed apneas also can result in a complaint of insomnia rather than hypersomnolence (Fredrickson & Krueger, 1989). The nature of the apnea may also dictate the type of insomnia experienced; that is, patients with sleep onset central apneas are more apt to complain about sleep onset insomnia whereas, patients with OSAS may readily fall asleep but
repeatedly awaken during the night, thus experiencing sleep maintenance insomnia. Polysomnographic findings tend to confirm the apneic patient’s subjective complaint. In patients with OSAS, reductions in total sleep time and sleep efficiency have been noted in association with prolonged latency to initial sleep onset and increased wakefulness after initial sleep onset (Fredrickson & Krueger, 1989). Furthermore, the percentage of light and transitional stage 1 sleep is typically elevated due to the number of arousals and awakenings.

Prevalence rates of the co-occurrence of SAS and insomnia vary widely. A large national cooperative study conducted in 1982 revealed that 31% of participants had a sleep onset or sleep maintenance insomnia complaint, but only 6.2% of this group also received a diagnosis of sleep apnea (Coleman, Roffwarg, & Kennedy, 1982). Therefore, it was assumed that OSAS was associated with insomnia far less often than with excessive daytime sleepiness. Despite the paucity of research in this area, recent studies have indicated that OSAS with insomnia may be more common than was once assumed. In a group of 45 older adults recruited for the treatment of late-life insomnia, 40% were diagnosed with co-morbid OSAS (A + HI ≥ 10), and an additional 24% were identified as having an A + HI between 5 and 9 (Stone, Morin, Hart, Remsberg, & Mercer, 1994). Krakow et al. (2001) studied sleep disturbance in posttraumatic stress disorder and found that insomnia and OSAS co-occurred in 50% of the participants studied. Preliminary follow up data indicated that 85% of the participants who were treated with CPAP reported moderate to marked improvement in insomnia.

There are a number of plausible hypotheses as to why some patients with OSAS may also experience insomnia. The following list highlights the most likely explanations for the co-occurrence of the two disorders:
1. Insomnia may be a direct function of OSAS. Lichstein, Wilson, and Johnson (2000) describe two types of secondary insomnia, which suggest that a medical condition (e.g., OSAS) is directly responsible for sleep difficulties: (1) *Absolute Secondary Insomnia* refers to the conventional definition of secondary insomnia -- insomnia based on psychiatric or medical (including pain or discomfort) disturbance, and; (2) *Partial Secondary Insomnia*, which asserts that the primary disorder affects sleep but does not account for 100% of the variability in the insomnia.

2. OSAS patients wake up, realize they aren’t breathing and are afraid to go back to sleep. This scenario is more typically found in patients with CSAS.

3. Nonrestorative sleep (from frequent awakenings due to OSAS) leads patients to develop poor sleep hygiene habits (e.g., napping, staying in bed longer), which maintains or exacerbates the insomnia.

4. Impaired daytime functioning due to hypersomnolence or fatigue contributes to increased stress and worry at night. Stress also increases as a function of worrying about not breathing or being frustrated about not sleeping. This worry and frustration serves to perpetuate the insomnia by inhibiting sleep.

5. Finally, patients may develop conditioned psychophysiological insomnia, which is where cues, such as the bedroom, bedtime, and attempting to sleep become associated with respiratory pause-induced awakenings and thus produce sleep-initiating arousal. This arousal may be in the form of either somatic (physiological) arousal and/or cognitive (mental) arousal.

   The purposes of this study were to determine if the insomnia experienced by some patients with OSAS is of a primary or secondary nature, by examining whether insomnia
improved as a function of treatment of OSAS with CPAP. Theory and empirical evidence from the sleep disorders literature informed the following hypotheses for this study.

**Hypotheses**

1. It was hypothesized that the diagnostic $A + HI$ and measures of sleep hygiene and pre-sleep arousal would be significantly correlated with measures of insomnia and daily functioning.

2. It was hypothesized that diagnostic $A + HI$ and pre-sleep arousal, as measured in the first half of the baseline week, would predict significant unique variance in insomnia (SOL, WASO), as measured in the last half of the baseline week.

3. For those participants who were followed over the course of treatment, it was hypothesized that all variables of interest would significantly change from baseline (pre-CPAP) to post-treatment (post-CPAP).
   
   (a) There would be a significant decrease from the diagnostic $A + HI$ to the titration $A + HI$. These results would suggest that the CPAP treatment was effective in reducing the apneic events associated with OSAS.

   (b) Patient-reported insomnia would significantly decrease after treatment of OSAS with CPAP, thus suggesting that the insomnia was secondary to the medical condition (i.e., OSAS).

   (c) Self-reported sleep hygiene habits would significantly improve from pre-CPAP to post-CPAP as a result of the specific sleep hygiene counseling routinely provided after the diagnostic PSG. It is expected that this change would be smaller in size compared to the other variables, given the small to moderate impact sleep hygiene education has been found to have on behaviors.
(d) Pre-sleep arousal was hypothesized to significantly decrease after CPAP treatment as a function, in part, of less stress and worry about the sleep and related daytime symptoms caused by OSAS.

4. It was hypothesized that greater CPAP compliance would be correlated with greater improvement in insomnia, thus reinforcing a relation between OSAS and insomnia.

5. It was hypothesized that there would be a significant correlation between changes in A + HI and changes in insomnia. Such findings would provide strong evidence for the existence of partial secondary insomnia in patients with OSAS. If a significant relationship between change in OSAS and change in insomnia was identified, then it was hypothesized that a hierarchical multiple regression would reveal the following results:
   
   (a) A + HI change would account for statistically significant variance in explaining subjective changes in insomnia.

   (b) Changes in pre-sleep arousal would account for statistically significant variance in changes in insomnia above and beyond that of the OSAS treatment effects alone (i.e., change in A + HI).

   (c) Changes in sleep hygiene would not account for additional statistically significant variance in changes in insomnia, since sleep hygiene treatment provided in isolation, does not typically improve insomnia symptoms.

6. For those participants still experiencing insomnia after treatment for OSAS with CPAP, it was hypothesized that continued insomnia would be associated with poor CPAP compliance and direct interference of the CPAP with the ability to initiate or maintain sleep.
Method

Participants

Sixty-nine participants were recruited from referrals to the Ochsner Clinic Foundation of Baton Rouge's Sleep Disorders Center (OCFBR-SDC) for an evaluation of sleep apnea syndrome. Only patients who met the criteria for OSAS as determined from their nocturnal diagnostic polysomnography (PSG), and indicated symptoms consistent with insomnia were included in the study. That is, patients must have evidenced a minimum A + HI of 5 and reported a period of one month, of three or more nights per week with either sleep onset difficulty (30 minutes or greater) and/or sleep maintenance difficulty (one or more nocturnal awakenings of at least 30 minutes in duration or three or more awakenings of a least 10 minutes in duration each). Of the 69 participants recruited for the study, 23 were excluded because they did not meet the diagnostic criteria for OSAS, and 5 participants changed their minds about participating before the study period was initiated. Therefore, 41 individuals met the inclusion criteria and agreed to participate in this study. A subset of these participants (n = 29) were prescribed and accepted CPAP treatment for OSAS and agreed to participate in the post-CPAP treatment phase of the research. Of these 29 participants, 15 individuals completed the 2-week post-treatment phase of the study and returned the self-monitoring packets. The remaining 14 participants were contacted on several occasions and encouraged to return their completed study materials, but they were never received.

Procedure

Prior to the patients’ scheduled PSG, a medical history was obtained and a physical examination was performed by the patient’s OCFBR-SDC physician. In addition, all patients
completed the Sleep Disorders Inventory (SDI; Waters, unpublished), a self-report questionnaire that assesses for signs and symptoms of all major sleep disorders and sleep hygiene problems.

Patients who met the eligibility requirements were approached and asked to participate in the current study, which was described as an investigation of the relations between sleep apnea and insomnia. Participants completed a demographic questionnaire and a brief measure of self-reported insomnia. Participants also received a packet of self-monitoring forms including sleep diaries and measures of sleep hygiene and pre-sleep arousal. It was emphasized that the research was purely descriptive and would not interfere with their course of treatment at the OCFBR-SDC. All participants understood that they might not be eligible to continue in the study if they did not receive a diagnosis of OSAS after completing the PSG. The experimenter contacted all participants after the PSG scoring became available to inform them as to whether or not they were eligible to continue in the study.

All participants underwent a standard hook-up procedure for a nocturnal polysomnography. Patients slept in a queen-size bed in a hotel-like room, and a Grass Heritage Digital Polysomnograph was used for the PSG 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. Participants’ sleep position was monitored on video using an infrared camera and light source system. If participants 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 PSG and were required to come back to the OCFBR-SDC for a full night of CPAP titration, if the diagnostic PSG was positive for OSAS.

The PSG data was scored by a trained polysomnography technologist at the OCFBR-SDC. Each recorded epoch (30 seconds) of sleep was hand scored for respiratory events, sleep stages, and EEG arousals; sleep stages and arousals were scored according to Rechtschaffen and Kales (1968) and American Sleep Disorders Association (1992) criteria, respectively. A computer-generated summary reported the A + HI (diagnostic) and A + HI (titration), which were used as indicators of the presence and severity of OSAS in the current study.

All participants were contacted by telephone the day the scored PSG data became available, and those who meet the eligibility requirements for the study were instructed to begin self-monitoring for a 1-week period. Each morning, participants recorded details about their previous night’s sleep (sleep diary), sleep hygiene habits, and pre-sleep arousal.

Approximately 3-4 weeks later, those participants who took part in the post-treatment phase of the study, returned to OCFBR-SDC to meet with the clinic Director (a clinical psychologist, board certified in Sleep Medicine) to discuss the results of their PSG, and the ensuing treatment recommendations. During this meeting, participants received clinical feedback regarding their sleep hygiene, as reported on the SDI, which included receiving information and a list of “rules” to follow to improve sleep. A sleep hygiene quiz was included with the materials and was used as a manipulation check to ensure that the participants read the information. The session concluded with the participants receiving a post-test packet of self-monitoring forms. The participants were instructed to self-monitor
daily for 2-weeks after their treatment with CPAP commenced. Only data from the second week of self-monitoring was used, to allow time for adjustment to the CPAP stimulus. The post-test packet included all of the self-monitoring forms from the baseline packet plus the sleep hygiene quiz and a form inquiring about CPAP compliance, discomfort, and interference. Additionally, these participants completed the self-report measure of sleep difficulties at the end of the 2-week monitoring period to reassess insomnia. Table 1 provides a list of assessment measures that were utilized during the course of this research.

Table 1.

List of assessment measures and schedule of data collection.

<table>
<thead>
<tr>
<th>Measures</th>
<th>Pre-PSG</th>
<th>Nocturnal PSG</th>
<th>Baseline</th>
<th>Post CPAP</th>
</tr>
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<tbody>
<tr>
<td>Sleep Disorders Inventory</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Insomnia Severity Index</td>
<td>X</td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>A + HI</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A + HI (titration)</td>
<td></td>
<td></td>
<td>X*</td>
<td></td>
</tr>
<tr>
<td>Sleep Diary (SOL, WASO)</td>
<td></td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Sleep Hygiene</td>
<td>X</td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Pre-sleep Arousal</td>
<td>X</td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>CPAP Compliance &amp; Interference</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
</tr>
</tbody>
</table>

* Titration may have occurred during the diagnostic PSG (i.e., split night) or taken place on a separate night solely devoted to CPAP titration.

Participants were instructed to mail back the study packets in the addressed and stamped enveloped provided. In an effort to increase compliance with self-monitoring, participants received regular telephone calls from the experimenter encouraging them to complete the self-monitoring and mail the packet back as soon as possible. Participants who indicated that they had completed the study requirements were debriefed at that time.
Measures

Sleep Disorders Inventory

The Sleep Disorders Inventory (SDI; Waters, unpublished) is a 60-item self-report measure, based on the International Classification of Sleep Disorders (ICSD), designed to assess signs of sleep disorders and dysfunctions. The SDI, like all other sleep inventories, has not been psychometrically validated due to the low baserates of a number of the sleep disorders and the enormous cost associated with performing multiple PSGs. However, it is accepted practice to screen for sleep disorders using ICSD criteria (Speilman, Yang, & Glovinski, 2000), and the SDI assesses all of the major ICSD classifications (i.e., sleep apnea, psychophysiological insomnia, narcolepsy, idiopathic hypersomnia, periodic limb movement disorder, restless legs syndrome, shift work sleep disorder, nightmares, inadequate sleep hygiene, REM sleep behavior disorder, and insufficient sleep syndrome), as well as descriptions of daytime hypersomnolence, insomnia, sleep time allocation, and sleep loss. Any responses that were indicative of a possible sleep disorder were followed up with a brief interview, based on ICSD diagnostic criteria, by the OCBR-SDC Director at the post-PSG interview.

Insomnia Severity Index

The Insomnia Severity Index (ISI; Bastien, Vallieres & Morin, 2001) is a brief self-report instrument used to measure a patient’s perception of his or her insomnia. The ISI is composed of 7 items that evaluate: (a) sleep-onset insomnia, (b) sleep maintenance insomnia, (c) early morning awakening insomnia, (d) satisfaction with current sleep pattern, (e) interference with daily functioning, (f) noticeability of impairment attributed to the sleep problem, and (g) level of distress caused by the sleep problem. Each of these items is rated
over the past 2 weeks on a 5-point Likert scale. The ISI has demonstrated adequate internal consistency (Cronbach’s alpha = .74; average item-total correlation = .54), concurrent validity ($r = .45$ with PSG sleep onset and $r = .37$ with sleep diary sleep onset), and sensitivity to changes in perceived sleep difficulties, i.e., ISI change scores were significantly correlated with change scores from the sleep diary and PSG (Bastien et al., 2001). In the present study, the ISI demonstrated adequate internal consistency (Cronbach’s alpha = .74), but poor concurrent validity with other measures of insomnia ($r (39) = .11$ with PSG sleep onset and $r (39) = .06$ with sleep diary sleep onset latency). Therefore, the results involving the ISI must be interpreted with caution and in the context of other more widely accepted measures of insomnia used in this study.

**Sleep Diary**

A sleep diary (Lacks, 1987) is a daily report of sleep and waking activities. Sleep diaries are widely used in clinical and research settings and have been found to be a reliable estimate of sleep/wake patterns over 24-hour period (Rogers, Caruso, & Aldrich, 1993). The Sleep Diary (Lacks, 1987) test-retest reliability is reported to be high for sleep onset latency ($r = .98$), number of arousals ($r = .88$), and time awake after sleep onset ($r = .84$). The Sleep Diary also significantly correlates with objective data from PSGs for sleep onset latency ($r = .62 - .99$ depending on criteria used for PSG sleep onset scoring) (Lacks, 1987). Although there are many potential sources of bias with self-monitoring (Korotitsch & Nelson-Gray, 1999; Nelson, Boykin, & Hayes, 1982), insomniacs tend to consistently overestimate wakefulness and underestimate total sleep time and number of arousals (Carskadon et al., 1976). Therefore, sleep diaries provide a reliable and valid (although relative) index of sleep disturbance (Schoicket, Bertelson, & Lacks, 1988). The Sleep Diary that was used in the
present study reported: bedtime, wake time, how much time taken to fall asleep (SOL), how much time spent awake after sleep onset (WASO), and 7-point Likert ratings of sleep initiation difficulty, restfulness, sleep quality, level of physical tension at bed, level of mental activity at bed, and overall functioning on the previous day. In the current study, the sleep diary served as a subjective measure of sleep difficulties (i.e., sleep onset and sleep maintenance insomnia).

Pre-Sleep Arousal Scale

The Pre-Sleep Arousal Scale (PSAS; Nicassio, Mendelowitz, Fussell, & Petras, 1985) is a 16-item self-report questionnaire that assesses both somatic and cognitive manifestations of arousal prior to sleep. The PSAS has demonstrated good internal consistency (Cronbach alphas = .76 - .88), test-retest reliability (r’s = .72-.76), and concurrent validity (Nicassio et al., 1985). The PSAS also reliably distinguishes between good sleepers and those with insomnia, particularly on the basis of the cognitive subscale score; those with insomnia report higher cognitive arousal than somatic arousal and more cognitive arousal than good sleepers. Both subscales of the PSAS were used in this study to measure arousal prior to sleep and both scales demonstrated good internal consistency (Cronbach’s alphas; somatic subscale = .88 and cognitive subscale = .88).

Sleep Hygiene Diary

The Sleep Hygiene Diary (Lacks, 1987) is a 20-item checklist inquiring about behaviors/situations, which may have interfered with the previous night’s sleep. The sleep hygiene diary was adapted from the Sleep Hygiene Awareness and Practice Scale, which examines knowledge about environmental factors that can interfere with sleep and gathers information about inadequate sleep hygiene over the past week (Lacks, 1987). The Sleep
Hygiene Diary used in the current study required participants to rate sleep hygiene practices on a daily basis. Questions were responded to in a Yes/No format, and the inadequate sleep hygiene behaviors that were endorsed were assigned a score of 1. All items were summed to provide a total score; higher scores indicated poorer sleep hygiene.

**CPAP Compliance, Discomfort, and Interference**

For those participants who participated in the post-treatment phase of the study, a subjective measure of CPAP compliance was obtained by asking participants to rate, each morning, how many hours they wore the CPAP the previous night. Self-reported CPAP use is the most common method of collecting information about CPAP compliance (Hui et al., 2001; Monasterio et al., 2001; Montserrat et al., 2001). Two additional questions were asked regarding discomfort/stress and sleep interference from the CPAP. Participants responded on a 7-point Likert scale indicating the extent of any discomfort/stress or interference in sleep that was a function of the CPAP (i.e., uncomfortable, noisy, difficulty breathing, dry nasal passages). These questions were created to ensure that the insomnia and pre-sleep arousal reported during the post-treatment phase of the research were not a consequence of the CPAP treatment itself.
Results

Preliminary Analyses

Data Screening and Outlier Analysis

All measures were initially assessed for multivariate outliers, univariate outliers, and distribution skew. First, the data were assessed for multivariate outliers by entering all measures into a multiple regression and computing Mahalanobis distance. A chi-square cut-off of $p < .001$ was used as the criterion for multivariate outliers (Tabachnick & Fidell, 1996). No multivariate outliers were identified; therefore all cases were retained in the subsequent analyses. Univariate outliers were identified by taking 1.5 times the inter-quartile range, and declaring as outliers all data points either that distance above the upper quartile or that distance below the lower quartile (Hoaglin, Mosteller, & Tukey, 1983). Ten data points in total were identified as univariate outliers. Univariate outliers were Windsorized, which involved replacing the outlying data with non-outlying values while retaining the sequential order among the outliers (Hoaglin et al., 1983). Finally, univariate summary statistics were computed to identify non-normally distributed measures. Measures that were non-normally distributed (i.e., skew $> |0.7|$) were transformed according to the direction of the skew. The diagnostic A + HI, SOL, WASO, sleep hygiene, somatic arousal, and cognitive arousal average scores were positively skewed and transformed accordingly (i.e., square root or logarithmic transformations). Although there were broad ranges of responses across all variables, the majority of the participants fell at the lower (i.e., less severe) end of the distribution, thus creating the positive skew. These results suggest that this particular group of sleep disordered patients experienced symptoms that were mild to moderate in severity, albeit severe enough to warrant both OSAS and insomnia diagnoses.
Sample Characteristics

Participants in this study ranged in age from 30-78 years old with a mean age of 54.0 years (SD = 10.74). Of the 41 participants, 56% (n = 23) were male and 44% (n = 18) were female. Racial composition of the sample consisted of 74% (n = 28) Caucasian, 23% (n = 9) African American, and 3% (n = 1) other. Multivariate analyses of variance (MANOVA) were computed to test for gender and race differences across the variables used in this study. Neither the gender MANOVA, $F(7, 32) = 1.46, \ p > .05$ nor the race MANOVA, $F(7, 30) = 3.55, \ p > .05$, were significant, indicating that there were no differential effects of gender or race on the dependent variables in this study. The majority of participants, 78%, were married, with 12% divorced, and 10% single.

Analyses of Baseline Data

Baseline data was collected on 41 participants, which included measures of OSAS, insomnia, and sleep-related behaviors over a 1-week period prior to receiving CPAP treatment for OSAS. Table 2 presents the means and standard deviations of the study variables. In an effort to determine if OSAS was associated with insomnia, daily self-monitoring scores were averaged over the entire study week and entered into a correlational analysis.

Table 2.

<table>
<thead>
<tr>
<th></th>
<th>A+HI diagnostic</th>
<th>Daily Fx</th>
<th>Sleep Hygiene</th>
<th>Somatic Arousal</th>
<th>Cognitive Arousal</th>
<th>SOL</th>
<th>WASO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>29.84</td>
<td>2.72</td>
<td>2.08</td>
<td>11.57</td>
<td>16.63</td>
<td>32.11</td>
<td>44.34</td>
</tr>
<tr>
<td>Standard Deviation</td>
<td>20.15</td>
<td>.76</td>
<td>1.29</td>
<td>3.68</td>
<td>5.41</td>
<td>20.81</td>
<td>30.15</td>
</tr>
</tbody>
</table>

Note. A + HI = apnea plus hypopnea index; Daily Fx = daily functioning; SOL = sleep onset latency; WASO = wake time after sleep onset
The first hypothesis of this study proposed that diagnostic A + HI, sleep hygiene and pre-sleep arousal would be significantly correlated with measures of insomnia and daily functioning. To test this hypothesis, correlations were computed among diagnostic A + HI, sleep hygiene, pre-sleep arousal, daily functioning, and self-reported measures of insomnia. The results of the correlational analyses are presented in Table 3.

Table 3.

Correlations between sleep apnea, insomnia, and sleep-related behaviors

<table>
<thead>
<tr>
<th></th>
<th>A+HI dx</th>
<th>ISI</th>
<th>SOL</th>
<th>WASO</th>
<th>Fx</th>
<th>SH</th>
<th>SA</th>
<th>CA</th>
</tr>
</thead>
<tbody>
<tr>
<td>A+HI dx</td>
<td>.01</td>
<td>.06</td>
<td>.09</td>
<td>.25</td>
<td>.44**</td>
<td>.31*</td>
<td>.10</td>
<td></td>
</tr>
<tr>
<td>ISI</td>
<td>.01</td>
<td>.09</td>
<td>.25</td>
<td>.44**</td>
<td>.31*</td>
<td>.10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SOL</td>
<td>.06</td>
<td>.25</td>
<td>.44**</td>
<td>.31*</td>
<td>.10</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WASO</td>
<td>.09</td>
<td>.25</td>
<td>.44**</td>
<td>.31*</td>
<td>.10</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fx</td>
<td>-.02</td>
<td>.14</td>
<td>.07</td>
<td>.07</td>
<td>.21</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SH</td>
<td>.13</td>
<td>.10</td>
<td>.07</td>
<td>.07</td>
<td>.21</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>SA</td>
<td>.20</td>
<td>.01</td>
<td>.45**</td>
<td>.27</td>
<td>.45**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CA</td>
<td>-.11</td>
<td>.23</td>
<td>.38*</td>
<td>.29</td>
<td>.50**</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. * = p < .05; ** = p < .01; A + HI dx = diagnostic apnea plus hypopnea index; ISI = insomnia severity index; SOL = sleep onset latency; WASO = wake time after sleep onset; Fx = daily functioning; SH = sleep hygiene; SA = somatic arousal; CA = cognitive arousal.

The objective measure of sleep apnea severity (diagnostic A + HI) was not correlated with any subjective measure of insomnia or behaviors associated with insomnia. However, apnea severity was significantly correlated ($r (39) = .37, p < .05$) with the objective measure of WASO, as measured by the PSG. That is, the more severe the sleep apnea, the higher percentage of time the patient spent awake after sleep onset. This finding would be expected given the tendency for apneic events to produce transient arousals, thus producing light and discontinuous sleep. Interestingly, A + HI was not correlated with self-reported WASO or perceived daily functioning. Perhaps some participants are unaware of the severity of their
OSAS because they do not fully awaken from the apneas during the night and therefore, their perception of WASO and daily functioning is based on other information (e.g., SOL, fatigue, anxiety). Finally, the lack of association between the subjective measures of insomnia (i.e., ISI and SOL, ISI and WASO) and the significant correlation between ISI and daily functioning \( (r(39) = .31, p < .05) \) suggests that the ISI may be measuring both the symptoms and sequelae of insomnia.

Consistent with the research literature, both somatic arousal and cognitive arousal were significantly correlated with onset insomnia (i.e., SOL). However, in the present study of patients diagnosed with OSAS, somatic arousal was more strongly associated with insomnia compared to cognitive arousal; a finding that is contrary to previous research, which suggests that cognitive arousal, plays a more important role in the development and maintenance of insomnia (Lichstein & Fanning, 1990; Nicassio et al., 1985). Neither somatic nor cognitive arousal were correlated with the ISI, providing further support for the hypothesis that the ISI may be a more general measure of the consequences of insomnia (i.e., interference with quality of life, daily functioning, noticeability to others) rather than a measure of insomnia symptoms.

Both somatic arousal and cognitive arousal were also significantly correlated with the participant’s reported level of daily functioning. Partial correlations were computed in an effort to determine if any of the relations between somatic arousal, cognitive arousal, insomnia, and functioning could be accounted for by apnea severity. None of the partial correlations were significant (\( p > .05 \)), that is, the partial correlations did not change in magnitude compared to the original bivariate correlations, when controlling for A + HI. Therefore, it appears that apnea severity does not mediate any of the relationships between
pre-sleep arousal, insomnia, and perceived daily functioning. These results provide further evidence suggesting a lack of association between OSAS and insomnia.

Two stepwise multiple regression analyses were conducted to examine how well apnea and insomnia-related behaviors predicted sleep onset and sleep maintenance insomnia. These analyses tested the second hypothesis of the study, which proposed that A + HI and pre-sleep arousal would predict significant unique variance in insomnia. The daily self-monitoring scores were averaged over the first 4 days and the last 3 days of the measured baseline week. The data were analyzed in this manner in order to maximize the number of data points while maintaining the temporal precedence among the variables. That is, the predictor variables (diagnostic A + HI, sleep hygiene, somatic arousal, and cognitive arousal) were averaged over days 1-4, and examined in relation to the criterion variables (sleep onset latency (SOL) and wake time after sleep onset (WASO)), which were averaged over days 5-7. With respect to SOL, the linear combination of the predictor variables was significantly related to onset insomnia, \( F(1, 38) = 16.67, p < .001 \). The sample multiple correlation coefficient was .55, indicating that approximately 30% of the variance of sleep onset insomnia in the sample could be accounted for by the linear combination of the independent variables. Of all of the independent variables included in the analysis, only somatic arousal was identified as a unique predictor of onset insomnia, \( t = 4.08, p < .001 \). Similar results were found with respect to WASO, in that the regression was significant, \( F(1, 38) = 8.00, p < .01 \), with only somatic arousal emerging as a significant predictor variable, \( t = 2.82, p < .01 \).

Consistent with the pattern of correlations, the severity of sleep apnea (A + HI) did not account for any significant variance in the reported sleep onset or sleep maintenance insomnia in patients with OSAS. It appears that somatic arousal, or physiological symptoms reported
prior to sleep, may play an important role in sleep onset and maintenance insomnia in patients diagnosed with OSAS.

**Analyses of Post CPAP Treatment Data**

The results of the analyses involving the baseline data set the context for the primary analyses of this study, by suggesting that there may be no relation between OSAS and insomnia. A more rigorous test of this hypothesis would involve following patients over the course of OSAS treatment and examining any changes in reported insomnia. In order to test these primary hypotheses regarding the potential association between OSAS and insomnia, individuals who completed the baseline assessment, were followed over the course of treatment for OSAS ($n = 15$). If patients received an effective treatment for OSAS (i.e., CPAP), experienced improvement in sleep apnea symptoms, but did not report any changes in insomnia, further evidence would be provided supporting the independence of these two sleep disorders. However, the relatively small number of participants who completed this phase of the research limits the statistical power and therefore precludes anything other than tentative conclusions.

There were a number of participants who completed the baseline assessment but were not included in the post-treatment analyses for the following reasons: they were never prescribed CPAP treatment for OSAS, they were prescribed, but did not accept CPAP treatment, or they did not complete the self-monitoring forms and were considered dropouts from the post-treatment phase. In an effort to ensure that there were no systematic differences between those individuals who only completed the baseline assessment and those who were involved in both phases of the research, analyses were conducted that compared the two groups of participants on demographic variables and the sleep variables of interest in this
study. A multivariate analysis of variance (MANOVA) was conducted, where the independent variable was whether or not the participant was included in both sets of analyses, and the dependent variables were those measures that were assessed during the baseline assessment such as, age, diagnostic A + HI, insomnia severity index, sleep hygiene, somatic arousal, and cognitive arousal. The overall MANOVA was not significant ($p > .05$) indicating that there were no significant differences between the groups on the continuous measures used in this study. Chi-square tests of independence were performed on the two nominal variables (gender and race) to determine if the proportion of participants in each demographic category differentially changed from baseline to post-treatment. Neither the gender chi-square $\chi^2 (1) = .53$, $p > .05$, nor the race chi-square $\chi^2 (1) = .13$, $p > .05$ was significant indicating that the baseline and post-treatment groups did not differ in gender or racial distribution. Taken together, these analyses suggest that there were no significant differences, based on demographics or symptoms measured, between those participants involved in the two phases of the research.

To examine the post-treatment research hypotheses, the same set of analyses were performed with the post-treatment data as were conducted with the baseline data, as well as other analyses intended to specifically address the hypotheses regarding change in insomnia as a function of OSAS treatment with CPAP. All of the results based on the analyses of the post-treatment data should be interpreted with caution due to the small number of participants included in this portion of the research.

Self-monitoring data was collected for a 2-week period after the initiation of treatment of OSAS with CPAP. Only data from the last week was utilized in the analyses in an attempt to allow the participants time to acclimate to the CPAP stimulus. As with the baseline data, all
scores were averaged over the entire week and correlations were computed among the sleep variables. Table 4 presents the means and standard deviations of the post-treatment sleep apnea and sleep variables.

Table 4.

Means and standard deviations for all variables after treatment with CPAP.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>A+HI titration</td>
<td>11.56</td>
<td>7.33</td>
</tr>
<tr>
<td>Daily Fx</td>
<td>2.21</td>
<td>0.59</td>
</tr>
<tr>
<td>Sleep Hygiene</td>
<td>1.55</td>
<td>0.70</td>
</tr>
<tr>
<td>Somatic Arousal</td>
<td>9.65</td>
<td>1.52</td>
</tr>
<tr>
<td>Cognitive Arousal</td>
<td>14.22</td>
<td>5.45</td>
</tr>
<tr>
<td>SOL</td>
<td>29.38</td>
<td>26.17</td>
</tr>
<tr>
<td>WASO</td>
<td>31.47</td>
<td>22.75</td>
</tr>
</tbody>
</table>

Note. A + HI = apnea plus hypopnea index; Daily Fx = daily functioning; SOL = sleep onset latency; WASO = wake time after sleep onset

Table 5 presents the correlations that were computed among the sleep variables measured after CPAP treatment was initiated. Similar to the baseline data, there continued to be an association between somatic arousal, cognitive arousal and sleep onset latency, while there was no relationship between post-treatment A + HI and any of the sleep variables.

Two stepwise multiple regression analyses were conducted to determine if the post-treatment results replicate the findings from the baseline data, which identified somatic arousal as a unique predictor of onset and maintenance insomnia. Once again, the daily self-monitoring scores were averaged over the first 4 days and the last 3 days of the measured week. The predictor variables were, post-treatment A + HI, post-treatment sleep hygiene, post-treatment somatic arousal, post-treatment cognitive arousal and the criterion variables were post-SOL and post-WASO, respectively. Neither of the regressions were significant; however, a power analysis with 4 independent variables and power set at .80, revealed that a minimum of 23 individuals would be needed to detect a large effect size. Given that the
magnitude of the correlations between somatic arousal and SOL at baseline and post-treatment are similar, it is likely that the lack of statistical power greatly impacted these regression analyses.

Table 5.

Correlations between post treatment apnea, insomnia, and sleep-related behaviors

<table>
<thead>
<tr>
<th></th>
<th>A+HI titration</th>
<th>ISI</th>
<th>SOL</th>
<th>WASO</th>
<th>Fx</th>
<th>SH</th>
<th>SA</th>
<th>CA</th>
</tr>
</thead>
<tbody>
<tr>
<td>A+HI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ISI</td>
<td>.50</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SOL</td>
<td>.25</td>
<td>.09</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WASO</td>
<td>-.14</td>
<td>.37</td>
<td>.62*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fx</td>
<td>-.46</td>
<td>.37</td>
<td>-.26</td>
<td>.21</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SH</td>
<td>.29</td>
<td>-.09</td>
<td>.12</td>
<td>-.32</td>
<td>-.21</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SA</td>
<td>.29</td>
<td>.08</td>
<td>.54*</td>
<td>.06</td>
<td>-.09</td>
<td>.47</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CA</td>
<td>.25</td>
<td>.13</td>
<td>.57*</td>
<td>.11</td>
<td>-.11</td>
<td>.45</td>
<td>.67**</td>
<td></td>
</tr>
</tbody>
</table>

Note.  * = p < .05; ** = p < .01; A + HI titration = apnea plus hypopnea index with CPAP at optimal pressure; ISI = insomnia severity index; SOL = sleep onset latency; WASO = wake time after sleep onset; Fx = daily functioning; SH = sleep hygiene; SA = somatic arousal; CA = cognitive arousal.

A series of dependent t-tests were conducted to test the third set of hypotheses involving the change in sleep apnea, insomnia, and sleep related variables from baseline to post-CPAP treatment. Table 6 presents the results and effect sizes for all variables of interest in this study. The significant decrease in the A + HI suggests that CPAP was an effective treatment for OSAS, which was accompanied by a significant improvement (lower score indicates less impairment) in daily functioning. Sleep hygiene did not significantly improve from baseline to post-treatment when sleep hygiene education was provided prior to initiating CPAP; however, mean sleep hygiene scores did decrease (lower score indicates better sleep hygiene) from baseline to post-treatment and produced a moderate effect size, which suggests that inadequate power (i.e., small sample size) may have been a factor in the failure of the
Finally, without any specific intervention targeting pre-sleep arousal, both somatic arousal and cognitive arousal decreased significantly after the initiation of CPAP; however, there was no concurrent improvement in any of the measures of reported insomnia (i.e., ISI, SOL, WASO).

Table 6.

Baseline and post-treatment results for sleep apnea and sleep-related variables.

<table>
<thead>
<tr>
<th></th>
<th>Baseline Mean</th>
<th>Post-CPAP mean</th>
<th>t</th>
<th>p</th>
<th>d</th>
</tr>
</thead>
<tbody>
<tr>
<td>A + HI</td>
<td>30.36</td>
<td>8.25</td>
<td>3.74</td>
<td>.002*</td>
<td>.99</td>
</tr>
<tr>
<td>ISI</td>
<td>15.47</td>
<td>14.13</td>
<td>1.76</td>
<td>.106</td>
<td>.44</td>
</tr>
<tr>
<td>SOL</td>
<td>31.09</td>
<td>29.39</td>
<td>.39</td>
<td>.702</td>
<td>.10</td>
</tr>
<tr>
<td>WASO</td>
<td>37.51</td>
<td>31.48</td>
<td>1.10</td>
<td>.286</td>
<td>.28</td>
</tr>
<tr>
<td>Daily Functioning</td>
<td>2.74</td>
<td>2.21</td>
<td>3.36</td>
<td>.005*</td>
<td>.87</td>
</tr>
<tr>
<td>Sleep Hygiene</td>
<td>2.24</td>
<td>1.55</td>
<td>2.04</td>
<td>.060</td>
<td>.53</td>
</tr>
<tr>
<td>Somatic Arousal</td>
<td>11.43</td>
<td>9.65</td>
<td>3.37</td>
<td>.005*</td>
<td>.87</td>
</tr>
<tr>
<td>Cognitive Arousal</td>
<td>17.29</td>
<td>14.22</td>
<td>2.13</td>
<td>.049*</td>
<td>.56</td>
</tr>
</tbody>
</table>

Note. A + HI = apnea plus hypopnea index; ISI = insomnia severity index; SOL = sleep onset latency; WASO = wake time after sleep onset

Although there was no significant improvement in insomnia over the course of treatment of OSAS, it is possible that change in apnea symptoms was associated with change in sleep variables. To examine this hypothesis, residualized change scores were created for all sleep apnea and sleep variables, and correlations were computed among these change scores. The change in A + HI was unrelated (p > .05) to the change in all insomnia related variables (i.e., ISI, SOL, WASO, daily functioning, sleep hygiene, somatic arousal, cognitive arousal), thus reinforcing the findings from the baseline analyses, which suggested that there was no relation between OSAS and insomnia. There were however, significant correlations between the change in variables assumed to be related to insomnia. Change in sleep hygiene was significantly correlated with change in cognitive arousal (r (13) = .57, p < .05), and
although not significant, change in sleep hygiene was moderately correlated with change in somatic arousal ($r (13) = .46, p = .08$). Furthermore, change in cognitive arousal was significantly correlated with change in the length of time taken to fall asleep (SOL; $r (13) = .54, p < .05$). There was no significant correlation between change in sleep hygiene and change in daily functioning. These results, although correlational and very tentative given the small sample size, can be said to suggest that the sleep hygiene intervention provided in this study may have been useful in promoting change in behaviors found to be associated with the development and maintenance of insomnia, namely, somatic and cognitive arousal.

Although a number of important insomnia-related variables improved over the course of treatment for OSAS, the participant’s reported insomnia did not change. Two of the possible explanations for this finding include, noncompliance with CPAP treatment for OSAS and interference of the CPAP stimulus itself on sleep. It was hypothesized that greater CPAP compliance would be associated with greater improvement in insomnia, thereby providing evidence for the relations between OSAS and insomnia. Adequate compliance with CPAP was reported (4.89 hours per night), but there were no significant correlations between CPAP compliance and the measures of insomnia. There was however, a significant positive correlation between CPAP interference and SOL ($r (13) = .59, p < .05$), indicating that the more the participant perceived the CPAP to interfere with the ability to sleep, the longer it took to fall asleep (i.e., higher SOL). Although not significant, there was an indication that a similar relationship may exist between CPAP interference and WASO ($r (13) = .49, p = .06$). While the overall level of interference was not particularly high (a rating of 2 on a 0-6 scale), these results do suggest that the perceived interference from the CPAP itself may play a role in the persistence of insomnia in the face of significant decreases in other variables previously
demonstrated to maintain insomnia. That is, any potential improvements in insomnia that may have occurred as a result of changes in factors known to influence insomnia (i.e., pre-sleep arousal, sleep hygiene), may have been masked by the adverse effect CPAP had on the ability of the participants to fall asleep.
Discussion

The purpose of this study was to examine the association between obstructive sleep apnea syndrome (OSAS) and insomnia and determine if insomnia in OSAS patients is secondary to OSAS, or is a primary disorder that requires its own treatment. The baseline analyses of 41 individuals diagnosed with OSAS and insomnia provided preliminary evidence to suggest that OSAS and insomnia are independent conditions. The diagnostic Apnea plus Hypopnea Index (A + HI), a measure of OSAS severity, was not correlated with any self-reported insomnia or sleep related behaviors. Furthermore, partial correlations confirmed that A + HI was not mediating the associations among other sleep-related variables, namely: somatic arousal, cognitive arousal, daily functioning, and insomnia. Finally, multiple regression analyses failed to identify A + HI as a unique predictor of either sleep onset latency (SOL) or wake time after sleep onset (WASO).

Both somatic arousal and cognitive arousal were significantly correlated with sleep onset insomnia, which is consistent with the literature on psychophysiological insomnia (Lacks, 1992; Nicassio et al., 1985). However, in this study, somatic arousal was more strongly correlated with insomnia than cognitive arousal, and emerged as the only significant predictor of either SOL or WASO. Previous research has suggested that while both forms of pre-sleep arousal are important contributing factors to insomnia, cognitive arousal is more closely related to nightly SOL (Lichstein & Fanning, 1990; Nicassio et al., 1985; Wicklow & Espie, 2000). Perhaps somatic arousal plays a more prominent role in individuals diagnosed with both insomnia and OSAS. There are a number of well-documented acute physiologic changes that are associated with apneic events: Heart rate, systemic arterial blood pressures, and pulmonary arterial blood pressures increase markedly at the termination of apneas.
(Chervin & Guilleminault, 1996). It might be that because of these frequent physiologic changes during the night, patients with OSAS experience more somatic arousal in general compared to those individuals diagnosed with insomnia only. If the insomnia that is comorbid with OSAS is, in fact, conditioned psychophysiological insomnia, the somatic arousal experienced as a result of apneic events may become conditioned to the bedroom and attempts to sleep, thus producing a heightened state of somatic arousal prior to sleep onset. That is, the somatic arousal produced from the OSAS acts as a maintaining factor for the insomnia. This theory would be particularly relevant for those individuals who remember having OSA-related arousals at sleep onset and during the night.

A study examining changes in state and trait anxiety of OSAS patients treated with CPAP found that while trait anxiety decreased significantly after one month, state anxiety did not decrease until after three months of CPAP treatment (Sanchez, Buela-Casal, Bermudez, & Casas-Maldonado, 2001). The fact that, in this study, state anxiety took three times longer to decrease after treatment than trait anxiety, provides some evidence indicating that somatic arousal may be a conditioned response and therefore more difficult to extinguish (i.e., decrease) after the treatment of OSAS.

Since not all patients with OSAS also have insomnia, it would be interesting for future research to investigate if patients with both conditions are characteristically different than those individuals with either OSAS or insomnia alone. Vincent and Walker (2001) studied the construct of anxiety sensitivity, fear of anxiety and the consequences of anxiety, in patients with insomnia and found that anxiety sensitivity predicted sleep-related impairment. Perhaps there is an association between anxiety sensitivity and awareness of OSAS-related arousals, that is, higher anxiety sensitivity predisposes an individual to take more notice of
physiological changes in the body and thus perceive more somatic arousal. In order to fully test these hypotheses, it would be necessary to conduct another study, which had OSAS-only and insomnia-only control groups.

There were a number of post-CPAP treatment findings, which also suggested that OSAS and insomnia are independent sleep disorders. The significant decrease in A + HI suggested that CPAP was effective in reducing the number of apneic events throughout the night, and the significant improvement in functioning suggested that CPAP improved the participants’ functioning during the day. However, the significant decrease in A + HI had no relation to self-reported insomnia, as measured by the ISI, SOL, and WASO, but was associated with a decrease in somatic and cognitive arousal. Taken together and interpreted in the context of the results from the baseline analyses, it appears that insomnia in patients with OSAS is not caused by the OSAS itself; however, improvement in OSAS is related to other variables involved in the development and maintenance of insomnia (i.e., somatic arousal and cognitive arousal).

Despite there being no intervention implemented specifically for pre-sleep arousal in this study, both somatic arousal and cognitive arousal decreased significantly from baseline to post-CPAP treatment. Also, although not significant with 15 participants (p = .06), mean sleep hygiene scores decreased after the sleep hygiene education, suggesting that the intervention itself, or potentially the reactivity of the daily self-monitoring of sleep hygiene practices, had some impact on self-reported sleep hygiene practices. Alternatively, improved sleep due to CPAP treatment may have reduced the need for participants to engage in maladaptive attempts to obtain more sleep. Similarly, it is possible that somatic arousal and cognitive arousal improved as a function of receiving treatment for OSAS. That is, patients
may have less somatic arousal because they are having fewer apneas and associated arousals and less cognitive and somatic arousal because they are less worried and anxious about the cessation of breathing, disrupted sleep, and impairment of daily functioning.

Finally, it was found that the change in sleep hygiene (from baseline to post-treatment) was significantly correlated with change in cognitive arousal and that the change in cognitive arousal was significantly correlated with change in sleep-onset insomnia (i.e., SOL). Perhaps learning a variety of new sleep-promoting behaviors gave participants a sense of control over their sleep, which resulted in less cognitive arousal leading to a decrease in the length of time to fall asleep. Future studies utilizing structural equation modeling techniques would be necessary to test these causal hypotheses.

Given the significant decrease from baseline to post-treatment in three variables that are known to be associated with insomnia (i.e., somatic arousal, cognitive arousal, and sleep hygiene), it was expected that insomnia would have improved as well. Contrary to the hypotheses in this study, insomnia did not change after treatment of OSAS with CPAP. The moderate to strong correlation between CPAP interference and insomnia ($\tau = .59$) suggests that any improvement in insomnia that may have occurred as a result of change in other factors may have been negated by the interference of the CPAP stimulus itself. It would be interesting to determine if a point exists where the CPAP stimulus is no longer considered an interference and if changes in insomnia would occur at that time.

This study represents the first attempt to understand the nature of the relations between obstructive sleep apnea syndrome and insomnia. Insomnia has a long history of being considered “secondary” to other medical or psychological problems. Harvey (2001) empirically examined the assumption that insomnia is better described as a symptom as
opposed to a stand-alone diagnosis. She reviewed numerous studies involving the presence of insomnia and another medical or psychological condition (primarily depression), and concluded that although insomnia is a symptom and often comorbid with a range of other disorders, the idea that insomnia is “secondary” was unfounded. Her conclusions were based on evidence which indicated that depression is predicted by the presence of prior insomnia, effective interventions for the primary disorder do not necessarily alleviate the insomnia (as was the case in the present study), and that insomnia has been identified as a risk factor for the development of other psychological disorders. As a result of these findings, she questioned the extent to which the primary/secondary distinction is valid.

There are limitations of the current study, which must temper the conclusions being drawn. The relatively small number of participants who were followed over the course of treatment for OSAS limited statistical power and the scope of analyses available. It was encouraging, however, that even with less than ideal power, a number of significant results emerged that corroborated the findings based on the baseline analyses with all participants. The nonequivalence between samples sizes at baseline and post-treatment also indicates that a number of participants dropped out from the study. Although analyses revealed no significant differences on baseline measures between those who completed the study compared to those who dropped out, numerous other variables exist, which were not tested that may have identified some systematic difference between the groups of participants (e.g., depression, anxiety, personality factors, other medical conditions).

The presence of two potential interventions for insomnia (i.e., CPAP treatment and sleep hygiene education) seriously limits any firm conclusions that can be drawn about the impetus for change in insomnia-related variables. In the present study, insomnia did not
significantly change over the course of treatment, which supports previous research that states that sleep hygiene interventions presented in isolation typically do not improve insomnia (Harvey, 2000), and also suggests that treatment for OSAS has little impact on insomnia. Despite the lack of change in insomnia, other factors that are correlated with insomnia, such as somatic arousal and cognitive arousal did significantly decrease over the course of treatment. Unfortunately, it is difficult to know whether the decrease in pre-sleep arousal is attributable to the OSAS treatment, the sleep hygiene education, or a combination of both interventions.

Future studies could address these issues by including an insomnia-only control group and ensuring that no other interventions for insomnia are presented during the CPAP treatment phase. Furthermore, it is recommended that a longer period be given for the participants to adjust to the CPAP stimulus. Collecting information about insomnia after a period of a month of wearing the CPAP should be sufficient time to rule out the possibility that the insomnia is being maintained or exacerbated by the CPAP stimulus itself.

A replication of the results of the present research from studies that incorporate the strategies suggested to address the current limitations, would have a number of implications for the treatment and management of patients with comorbid OSAS and insomnia. Patients who present to sleep clinics should be adequately assessed for both OSAS and insomnia. Since OSAS is often considered a more “serious” disorder, the majority of time and resource allocation is spent on its assessment and diagnosis. However, a thorough assessment of the insomnia will help identify the treatment strategies that will be of most benefit to the patient, since it appears unlikely that the insomnia would be helped by CPAP treatment. With respect to OSAS treatment, for those patients who present with comorbid psychophysiological
insomnia, it may be particularly important to spend time assisting the patient in acclimating to the CPAP stimulus before they begin using it at night. Previous research has shown that education and instruction can help patients become acclimated to the CPAP stimulus in a relatively short period of time (Edinger & Radtke, 1993; Rains, 1995). This may be an important part of treatment since the results from the present study suggest that interference from the CPAP stimulus is associated with continued sleep-onset insomnia. Although not tested in the current study, it is possible that for those patients with conditioned psychophysiological insomnia, the CPAP itself could be another stimulus to which hyperarousal and frustration about sleep difficulties may become conditioned.

Finally, it would be necessarily to provide treatment for both OSAS and insomnia. Future research would need to determine if there is an optimal order in which to present these treatments. Insomnia has been found to be among the most common psychological health problems (Canals, Domenech, Carbajo, & Blade, 1997) and is comorbid with a number of medical and psychological problems, including other sleep disorders. Poor sleepers consistently report impaired health, mood, work efficiency, relationships, and a sense of well-being (Lacks & Morin, 1992). Therefore, it is incumbent upon the health professionals within the field of sleep medicine to provide all the treatments necessary to allow the patient to experience improved sleep, daytime functioning, and quality of life.
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