ORIGINAL ARTICLE

Changes in Depressive Symptoms after Continuous Positive Airway Pressure Treatment for Obstructive Sleep Apnea

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ABSTRACT

It is generally believed that obstructive sleep apnea (OSA) causes depression in some patients, yet it is unknown whether this depression is an actual clinical phenomenon or purely a result of overlapping somatic/physical symptoms shared by both disorders. The present study investigated changes in both somatic and affective/cognitive symptoms of depression associated with the introduction of continuous positive airway pressure (CPAP) treatment for OSA. Participants were 39 outpatients (35 males, 4 females) with no current or past mental health problems, diagnosed with OSA in a hospital sleep disorders clinic. The Beck Depression Inventory (BDI) was administered prior to treatment and again 3 months after CPAP. Total BDI scores improved after CPAP, independent of objectively monitored CPAP compliance rates. Both somatic and affective/cognitive symptoms of depression improved in a similar manner after treatment. Our findings suggest that depressive symptoms experienced by OSA patients are not solely the result of physical OSA symptoms but include a mood component as well. We introduce a hypothetical model to conceptualize the relationship between OSA and depression.

KEYWORDS: Sleep apnea, depression, CPAP

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Obstructive sleep apnea (OSA) is a common and debilitating sleep disorder, affecting 2 to 4% of adults.[1,2] It is characterized by repeated breathing pauses (apneas) during sleep, which cause sleep fragmentation and hypoxemia. Daytime symptoms of OSA include excessive sleepiness, fatigue, cognitive dysfunction, and impaired quality of life.[1,3,4]

One of the predominant psychological correlates of OSA is depression, although the nature of the relationship between depression and OSA is poorly understood. Many studies have reported elevated rates of depression ranging from 20 to 63% in untreated OSA patients,[5–14] yet some researchers have failed to find pathologic levels of depression.[15–18] This discrepancy may be due in part to the types of questionnaires used to assess depression. When structured clinical interviews are employed using DSM-III-R or DSM-IV criteria, current depressive episodes have been reported in 23%[19] and 34%[20] of OSA patient samples.

A more complete understanding of the relationship between OSA and depression can be appreciated by examining changes in depressive symptoms once OSA is successfully treated. Numerous outcome studies have documented reductions in depression after continuous positive airway pressure (CPAP) treatment, the standard treatment for OSA.[7,11,14,21–27] Mood improvements have been detected as early as 1 month post-treatment[22,28,29] and are maintained for the long term.[11,14,27,30] Despite these findings, other researchers have not found CPAP to be associated with significant improvements in depression.[31–33]

Depressive symptoms in OSA patients may be an emotional reaction to having a chronic medical illness or perhaps the result of the excessive sleepiness and hypoxemia characteristic of OSA.[20,34] Of note, many symptoms of clinical depression (e.g., sleep problems, fatigue, concentration difficulties, irritability, social withdrawal) overlap with symptoms of OSA.[35] Some experts argue that depression measures in OSA patients are inflated by these predominantly physical symptoms common to depression and OSA, such that post-treatment changes in depression reflect improvement in OSA and not mood per se.[36,37] Indeed, this possibility is supported by studies with medically ill inpatients, showing that because of the high frequency and severity of somatic depressive symptoms (e.g., sleep disturbance, weight change, fatigue) caused by medical illnesses, affective/cognitive symptoms of depression (e.g., sadness, guilt, low self-esteem, indecisiveness, suicidal thoughts) are better discriminators of true depression.[38,39] Studies that have reported type of depressive symptoms in OSA patients have found that many patients endorse affective/cognitive symptoms in addition to somatic symptoms.[7,12,40–42] Millman et al[7] noted that both somatic and affective/cognitive complaints improved after CPAP treatment. However, no studies have specifically investigated type of depression symptom change (i.e., affective/cognitive vs. somatic) associated with treatment for OSA. Such knowledge is essential for understanding and treating OSA patients with depressive symptoms.

In order to assess differential changes in affective/cognitive and somatic symptoms, a measure that segregates these symptom clusters is needed. The Beck Depression Inventory (BDI)[43] may be particularly suited for such a purpose. Factor analyses have yielded two main factors (negative attitudes and performance difficulty) that correspond with the distinction between affective/cognitive and somatic symptoms.[44] The negative attitudes factor includes items relating to sadness, guilt, hopelessness, self-dislike, crying, and suicidal thoughts, whereas the performance difficulty factor includes items relating to indecisiveness, psychomotor retardation, sleep problems, fatigue, somatic concerns, and loss of sexual interest. These factors have shown differential improvement rates in a sample of psychiatric inpatients receiving treatment.[45]

On the BDI, untreated OSA patients score significantly higher than control groups.[33,46–48] Watson and associates[49] reported that 50% of their sleep apnea patient sample had elevated BDI scores, predominantly in the mild to moderately depressed

range. Yang and colleagues[48] reported clinically significant BDI scores in 25% of their sample of sleep-related breathing disorder patients, whereas McCullogh[50] found 20% of a large sample of apnea patients had BDI scores over 15. Some researchers have found significant decreases in BDI scores after OSA treatment,[21,50,51] whereas others have not.[31,33] None of these investigations considered type (i.e., somatic vs. affective) of symptom change on the BDI.

In the present study, patients undergoing routine clinical care in a hospital sleep disorders center for the diagnosis and treatment of OSA completed the BDI prior to and 3 months following treatment. We hypothesized that OSA patients would exhibit elevated depression levels at baseline (pre-treatment) compared to 3-month follow-up. Because research has demonstrated that patients with greater CPAP usage experience greater improvements in mood,[24,26] we also hypothesized that patients with high compliance to CPAP treatment would show greater improvements in BDI scores compared with those with low compliance. However, the main goal of our study was to explore differences in depressive symptoms in OSA patients treated with CPAP to determine whether there were improvements in affective/cognitive symptoms or purely improvements in somatic symptoms.

METHODS

Participants and Setting

Participants were 39 outpatients undergoing routine clinical evaluation for OSA at a sleep disorders center. Patients were eligible to participate if they received a diagnosis of OSA and were subsequently treated with CPAP therapy. Additional eligibility criteria included: (1) age 18 or older; (2) educational level 8th grade or above; (3) no psychiatric history (including no current or past diagnosis of depression); (4) no psychotropic medications; (5) not undergoing psychotherapy; and (6) no additional concurrent sleep disorders.

This study was conducted at three sites: Methodist Healthcare of Memphis, Memphis, TN (MHM; n = 7); Veterans Administration Medical Center, Durham, NC (VAMC; n = 31); and Duke University Medical Center, Durham, NC (DUMC; n = 1). Polysomnographic (PSG) monitoring at all sites minimally included the following channels: two electroencephalography channels, two electrooculography channels, chin electromyography, two anterior tibialis electromyography channels, electrocardiogram, nasal/oral thermistor, thoracic pneumography, abdominal pneumography, and pulse oximetry. PSG records were scored by trained sleep technologists using accepted scoring criteria.[52] At one site (VAMC), five participants underwent in-house unattended ambulatory PSG, which was scored according to a computer algorithm.

Outcome Measures

Beck Depression Inventory (BDI). The BDI is a 21-item self-report questionnaire measuring severity of depressive symptoms over the past week.[43] For each item, the respondent chooses one or more response options rated from 0 (absence of symptom) to 3 (most severe level). Total scores range from 0 to 63 and represent the sum of the highest level endorsed on each item. The BDI has demonstrated adequate reliability and validity.[53–55]

Sleep and respiratory variables. Because 12 patients underwent a full night diagnostic PSG and 27 patients underwent a "split night" PSG wherein they were treated with CPAP during the second portion of the night, it was not possible to equate all PSG sleep parameters for the entire patient sample. Sleep and respiratory measures for patients undergoing a split night protocol were calculated from the pre-CPAP sleep period. PSG variables that were equivalent between the two diagnostic protocols (full night and split night) were latency to sleep onset

(SOL), latency to rapid eye movement sleep (REML), and sleep efficiency (SE, a percentage score derived from total time slept divided by total time in bed). Respiratory measures of apnea severity included: apnea/hypopnea index (the number of apneic episodes greater than 10 seconds long per hour sleep), oxygen desaturation nadir, and apnea arousal index (the number of arousals per hour caused by apneic events).

Treatment

Titration to therapeutic CPAP pressure levels was performed by an experienced technologist either during the patient's initial PSG (split night) or during a second night-time PSG (full night). In five cases at VAMC, the patients did not receive CPAP titration in the lab, but instead received an autotitrating CPAP unit at home. The protocols for conducting a full night diagnostic PSG versus a split night PSG differed between sites; all participants at MHM completed a full night PSG before returning for a CPAP trial study. All participants at DUMC and VAMC (except for the five patients mentioned above) underwent a split night protocol.

CPAP units were delivered to the patient's home by a home care company representative typically within 7 to 10 days after the PSG evaluation. During this visit, patients were individually instructed on the use and care of the CPAP unit. The CPAP model type received by patients depended on insurance coverage and patient preference, but all patients received either Healthdyne®, Respironics®, or ResMed® brand CPAP units. CPAP units were equipped with factory-made internal meters or microchips designed to measure usage. Objective compliance to CPAP at follow-up was determined by these usage measurements. Patients were considered high compliers if they used CPAP at least 4.5 hours per night on average over the 3-month follow-up period. This value was based on average CPAP use rates in published literature.[56] Low compliers used CPAP under 4.5 hours per night.

Procedure

Human subjects Internal Review Board approval was obtained at each institution and informed consent was obtained from all study participants. Patients consenting to participate completed the BDI during their diagnostic evaluation prior to receiving home CPAP. Approximately 3 months after initiation of CPAP therapy, participants again completed the BDI, either by mail, during a home visit by a research assistant, or a during a follow-up appointment with the first author. Objective CPAP compliance data were collected at this time.

RESULTS

Sample Characteristics

A total of 63 sleep apnea patients were enrolled in this study. Thirty-nine patients (62%) completed baseline and follow-up. Nine patients (14%) did not tolerate or declined CPAP treatment. Three patients (5%) did not return baseline data. Four patients (6%) were diagnosed and treated for depression during the course of the study and were subsequently excluded from analyses. Eight patients (13%) patients were lost to follow-up.

All data analyses were conducted using SPSS-PC version 10.0 software (SPSS Inc., Chicago, IL). Demographic variables are presented in Table 1. Because of the low number of participants recruited at the private medical center settings (MHM and DUMC), these groups were combined when testing for differences between sites. Gender distribution between sites was significantly different (Fisher's Exact Test, $p = 0.001$), with no female patients recruited at VAMC and 4 female patients recruited at the private hospital settings. There were no significant differences between settings for ethnicity (Fisher's Exact Test, $p = 0.58$) or education (χ^2 [3, N = 39] = 1.39, $p = 0.71$). Independent t-tests for differences between settings in age, self-

Table 1 Demographic Characteristics of Apnea Patients by Site (N = 39)

	VA Med. Center (N = 31)		Private Med. Center (N = 8)		Combined (N = 39)	
	M	**SD**	**M**	**SD**	**M**	**SD**
Age	58.7	11.2	54.5	11.0	57.8	11.2
Body Mass Index	33.3	6.4	35.1	8.2	33.7	6.8
	n (%)		**n (%)**		**n (%)**	
Gender						
Male	31 (100%)		4 (50%)		35 (90%)	
Female	0 (0%)		4 (50%)		4 (10%)	
Ethnicity						
African-American	4 (13%)		2 (25%)		6 (15%)	
Caucasian	27 (87%)		6 (75%)		33 (85%)	
Education						
Grades 8–11	3 (10%)		0 (0%)		3 (8%)	
High school	7 (23%)		3 (37.5%)		10 (26%)	
Some college	12 (39%)		3 (37.5%)		15 (39%)	
College degree or higher	9 (29%)		2 (25%)		11 (28%)	
Marital Status						
Married or living with partner	27 (87%)		8 (100%)		33 (89%)	
Divorced/Separated	2 (6.5%)		0 (0%)		2 (5%)	
Single	2 (6.5%)		0 (0%)		2 (5%)	

M, mean; SD, standard deviation.

reported duration of sleep problem, baseline BDI score, sleep variables (SOL, REML, SE), and respiratory measures of apnea severity (apnea/hypopnea index, apnea arousal index, oxygen desaturation nadir) were all nonsignificant, except that patients studied in the private medical center setting had a significantly longer latency to REM sleep (M = 173.5 min, SD = 82.6) than VAMC patients (M = 87.2 min, SD = 84.6; t [23] = 2.40, $p < 0.05$).

Based on recently proposed criteria for sleep-related breathing disorders,[57] the level of nocturnal breathing disruption was rated as severe in 58% of the sample, moderate in 29%, and mild in 13%. Patients had an average of 46.9 (SD = 33.8) respiratory events per hour, with 30.4 (SD = 30.1) arousals per hour related to apnea. Average oxygen desaturation nadir was 77.8% (SD = 12.3). Sleep efficiency was 76.5% (SD = 19.3). Patients reported sleep difficulties for an average of 7.22 years (SD = 7.26). Average BMI was in the "obese" weight range (i.e., greater than or equal to 30 kg/m^2).[58]

CPAP Compliance

Objective compliance measures downloaded from CPAP units at 3-month follow-up were available on 27 patients. For the remaining 12 patients, compliance was estimated from subjective reports, as this variable was most highly correlated with objective compliance (Pearson $r = 0.88$, $p < 0.001$). Simple regression analysis performed with objective compliance entered as the dependent variable and subjective compliance entered as the predictor was significant ($\beta = 0.88$, t[26] = 9.08, $p < 0.001$). The subsequent regression equation was used to estimate objective compliance for the 12 patients missing objective compliance data. On average, patients overestimated CPAP compliance by about 30 minutes per night; objective CPAP usage was 4.31 (SD = 2.31) hours per night, with patients reporting usage at 4.82 (SD = 2.49) hours. Percent compliance (derived from dividing objective compliance by reported numbers of hours slept per night) was 65%. Using

the previously mentioned usage rate of 4.5 hours per night as the cut-off score to determine level of compliance, 20 patients (51%) were considered high compliers, while 19 patients (49%) had low compliance.

CPAP meter values were also obtained after the first week of CPAP for 25 patients. Average CPAP use after 1 week was highly related to use at 3 months (Pearson $r = 0.88$, $p < 0.001$), consistent with compliance literature indicating that long-term compliance to CPAP is determined relatively early on in treatment.[59,60] Side effects related to CPAP were experienced by 46% of patients and included sinus congestion, nose soreness, dry mouth, and mask leaks.

Treatment Outcome

As expected, patients demonstrated statistically significant reductions in BDI scores after CPAP treatment (baseline: M = 9.59, SD = 7.63; follow-up: M = 7.26, SD = 5.85; F [1, 38] = 8.52, $p < 0.01$). Higher BDI scores at baseline were associated with lower oxygen desaturation nadir (Pearson $r = -0.32$, $p < 0.05$), but were not significantly related to any other sleep or respiratory variables.

Table 2 presents the distribution of BDI scores by severity of depression before and after CPAP treatment. Although most patients (64%) were in the nondepressed range prior to CPAP, there were fewer moderately and severely depressed patients after CPAP. Chi-square analysis for differences between pre- and post-CPAP was significant (χ^2 [6, N = 39] = 34.93, $p < 0.001$).

Table 2 Distribution (%) of BDI Scores in Patients at Baseline and Follow-up

	Nondepressed	Mild	Moderate	Severe
Pre-CPAP	64.1	25.6	7.7	2.6
Post-CPAP	69.2	25.6	5.1	0.0

BDI, Beck Depression Inventory.
Note: BDI scores as follows: Nondepressed < 10; Mild = 10–18; Moderate = 19–29; Severe > 29. Beck et al.[53]

To test the hypothesis that patients with high compliance to CPAP would show greater BDI improvements compared with patients with low compliance, a linear regression analysis was conducted with compliance entered as the independent variable and pre- to post-CPAP change scores on the BDI entered as the dependent variable. CPAP compliance (both actual use and percent compliance) did not significantly predict BDI change, although the analysis approached significance. Partial correlation coefficient between compliance and post-treatment BDI score controlling for baseline score was also nonsignificant.

Somatic and Affective/Cognitive Symptom Change on the BDI

Factor scores on the BDI for affective/cognitive symptoms (Factor 1) and somatic symptoms (Factor 2) were computed (see Tanaka and Huba[44] for factor loadings). Symptom change was analyzed using a 2 (conditions: high compliance, low compliance) × 2 (time: repeated measures at baseline and follow-up) multivariate analysis of variance (MANOVA), with total BDI and factor scores entered as dependent variables. This MANOVA yielded a significant effect for time (Wilks' Λ [3, 36] = .80, F [3, 36] = 2.98, $p < 0.05$). Univariate F tests were significant for all three dependent variables: total BDI (F [1, 38] = 8.52, $p < 0.01$), Factor 1 (F [1, 38] = 5.39, $p < 0.05$), and Factor 2 (F [1, 38] = 4.70, $p < 0.05$). Regression analyses with BDI and factor change scores entered as dependent variables and CPAP compliance entered as the independent variable were nonsignificant. Compliance was not significantly related to BDI factor scores.

To compare change in depressive symptoms, z-scores were calculated using the overall mean and standard deviation, following the procedures of Neimeyer and associates.[45] The results are plotted in Figure 1. These analyses suggest that although BDI scores improve after treatment, there is no indication that somatic symptoms improve to any greater extent than affective/cognitive symptoms.

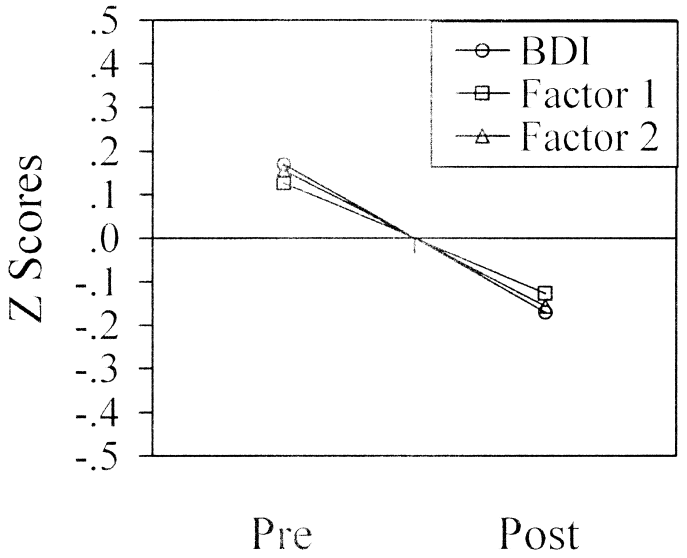

Figure 1 Improvement in BDI and factor scores after CPAP treatment in apnea patients.

Post Hoc Analysis of Daytime Sleepiness

A post hoc analysis of subjective daytime sleepiness was performed in response to recent literature indicating that CPAP does not improve quality of life functioning in patients with severe sleep-related breathing disruption but no subjective sleepiness.[61] Two authors (M.K.M. and J.D.E.) retrospectively reviewed available medical charts on 31 participants and independently rated the initial clinical sleep evaluation for level of reported sleepiness (i.e., none, mild, moderate, severe) according to accepted criteria.[57] When disagreement in sleepiness ratings arose, the raters discussed each case to reach a consensus. Most participants reported severe daytime sleepiness (52% severe; 10% moderate; 35% mild; 3% none). The severity of daytime sleepiness prior to treatment was not significantly related to any measures of apnea severity or CPAP compliance. Sleepiness was correlated with pre- to post-treatment change in BDI score (Spearman $r_s = .40$, $p < 0.05$), such that individuals who reported greater sleepiness before CPAP showed greater improvement on their BDI scores.

DISCUSSION

The main findings from this study were: (1) patients demonstrated significant, albeit small, improvements in BDI scores following treatment; (2) both somatic and affective/cognitive symptoms on the BDI improved equally after 3 months' CPAP treatment for OSA; and (3) patients who were more compliant with CPAP did not show better outcomes on the BDI.

Pretreatment BDI scores were similar to those found by other investigators,[47,50,51] and as predicted, scores improved after CPAP treatment. However, the clinical significance of this finding is debatable, as the mean BDI score at baseline was in the nondepressed range. Because patients with known depression were excluded from participating, this finding likely reflects a selection bias. Nonetheless, the prevalence of undetected depression at baseline (BDI score greater than or equal to 10) was 42% in our sample. The correlation between baseline depression scores and oxygen desaturation also provides evidence of a link between OSA and depression. Furthermore, all 4 participants who were excluded

from analyses because they received treatment for depression during the course of the study endorsed suicidal ideation on the BDI. Thus, in at least some apnea patients with no prior mental health history, depression is a true clinical phenomenon requiring treatment. Our findings replicate other reports on the association between depression and untreated OSA and imply that routine screening for depression in apnea patients is warranted.

In analyzing the type of depressive symptoms that showed improvement after CPAP treatment, we found apnea patients were not only endorsing somatic symptoms of depression but affective/cognitive symptoms as well. Our findings support the position that depression in OSA patients is not entirely attributable to symptoms of apnea such as tiredness and poor concentration being mistaken as depression, as some have argued.[36,37] The pattern of depression involves affective/cognitive symptoms as opposed to strictly a "medical" depression characterized predominantly by increased somatic symptoms.

A hypothetical model depicting the relationship between OSA and depression is presented in Figure 2. In this model, hypoxemia and sleep fragmentation cause the *primary effects* seen in OSA, such as daytime sleepiness, fatigue, and irritability. These symptoms are analogous to depressive-somatic symptoms, but would not in and of themselves cause depression. Left untreated, the primary effects of OSA can lead to *secondary effects*, characterized by stress related to having a chronic medical illness. Secondary effects may include decreased coping skills, reduced activity levels, and fewer social interactions. These effects may contribute to the development of both affective/cognitive and somatic depressive symptoms. Increased susceptibility toward the development of depressive symptoms may be seen in some patients, for example, those with a predisposition towards depression and/or those experiencing a precipitating event or stressor. Furthermore, it is well known that weight gain is a clinical feature of depression. In our model, we have presented a path wherein weight gain could further exacerbate OSA symptoms.

The primary effects of OSA are reduced rather expediently with CPAP treatment. It seems reasonable to speculate that the secondary effects of OSA and both the affective/cognitive and somatic symptoms of depression would be alleviated within the first few weeks or months of CPAP treatment. Although this could explain our findings, the model we have presented herein was not explicitly tested in our study and thus remains hypothetical in nature. Future studies that include OSA patients with depression are needed to provide more knowledge of the complex relationship between OSA and depression.

Our final hypothesis, that patients who were more compliant with CPAP would show greater improvement on the BDI compared to low compli-

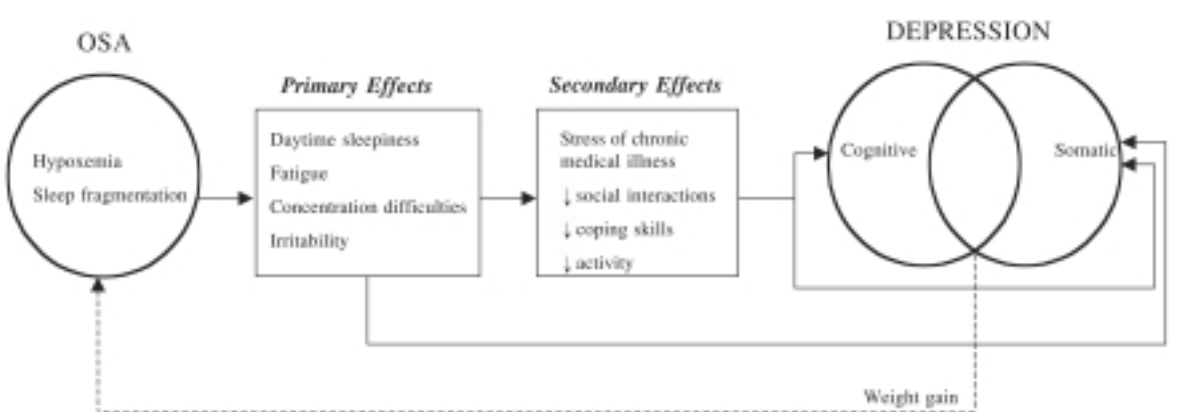

Figure 2 Hypothetical model of the relationship between obstructive sleep apnea and depression.

ance participants, was not confirmed, even though CPAP compliance rates in the present study were comparable to those reported elsewhere.[62–64] The bulk of literature in this area shows that greater CPAP use improves outcomes (e.g., Douglas and Engleman[24]); however, similar to our findings, two studies have failed to find a relationship between CPAP compliance and mood changes.[30,65] Post hoc analyses suggest that our sample had a high degree of sleepiness prior to CPAP and argues against the possibility that CPAP was ineffective in improving BDI scores because our sample was not subjectively sleepy.[61]

A number of possibilities can explain the lack of a relationship between CPAP compliance and BDI improvements. First, low compliers may be receiving a sufficient "dose" of CPAP to promote similar improvements as high compliers. This explanation is supported by research suggesting that partial use of CPAP results in daytime benefits.[66] Alternatively, improvements may be caused by a placebo effect of CPAP and not the treatment dosage per se. Yu and associates[67] present some convincing evidence for this possibility. They tested the placebo effect of CPAP on mood by randomly assigning apnea patients to either CPAP treatment or sham CPAP. The sham treatment consisted of a CPAP unit delivering a low pressure with holes drilled into the mask to prevent effectiveness. After 1 week, both groups showed improvements on mood measures. It is likely that there is some placebo effect related to the patients' relief at finding a cause for their daytime sleepiness and receiving medical attention. However, whether a placebo effect would be maintained at the same level after 3 months of CPAP is questionable. Furthermore, randomized, placebo-controlled trials of CPAP therapy have been carried out and indicate that CPAP is associated with significant daytime mood benefits compared to an oral placebo pill.[5,32]

The design of our study must also be considered in interpreting our findings. In general, participants did not display significant depression scores prior to treatment. Patients with current or past mental health problems were screened out of the study, thereby restricting the range of scores on the BDI and increasing the probability of a Type II error. CPAP compliance may have influenced outcomes if our sample had demonstrated more severe impairment on the BDI at baseline. Additionally, the pre-post design of the current study does not permit a measure of statistical regression to the mean, which could account for improved post-treatment scores independent of compliance. However, the placebo-controlled studies mentioned above make this explanation less plausible. Finally, significant findings may have been achieved by a larger sample size, although significant results have been reported in other studies with similar sample sizes.[5,26]

The results of the present study should be interpreted in light of its limitations and considered preliminary until replication with larger sample sizes can be undertaken. Given the quasi-experimental study design, we cannot eliminate the possibility that our outcome reflects a placebo effect. Our results generalize to those OSA patients who accept CPAP treatment, as patients who did not get CPAP treatment or who rejected CPAP during the study period were not included. Our sample consisted primarily of Caucasian men and was representative of patients seen in both private and VA medical center sleep practices. Future studies are needed to determine whether our results generalize to other demographic groups such as African-Americans and women. Further research considering treatment effectiveness and outcomes in apnea patients with comorbid mental health problems such as depression is also warranted.

In summary, apnea patients treated with CPAP showed reductions in both affective/cognitive and somatic symptoms of depression, independent of CPAP compliance rates. Cumulative effects of undiagnosed sleep apnea, such as chronic sleep deprivation, affective/cognitive dysfunction, sleep disruption, and hypoxemia, likely contribute to psychological impairment. However, the exact nature of the relationship between sleep apnea and psychological factors such as depression remains to be

elucidated by future research. Larger controlled studies are needed to better appreciate the impact of sleep apnea on daytime functioning and the subsequent benefits bestowed by CPAP treatment.

ACKNOWLEDGMENTS

This project was part of the first author's doctoral dissertation, supported by The University of Memphis Department of Psychology. We appreciate the assistance and cooperation of the Sleep Disorders staff at Methodist Sleep Disorders Center, Durham VA Medical Center and at Duke University Medical Center. We are also grateful for the statistical assistance provided by William K. Wohlgemuth, Ph.D.

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