Adherence, Reports of Benefits, and Depression Among Patients Treated With Continuous Positive Airway Pressure

RACHEL D. WELLS, PHD, KENNETH E. FREEDLAND, PHD, ROBERT M. CARNEY, PHD, STEPHEN P. DUNTLEY, MD, AND EDWARD J. STEPANSKI, PHD

Objectives: To examine if reported obstructive sleep apnea (OSA) symptom improvement, baseline depressive symptoms, or polysomnographically measured sleep parameters are associated with adherence to continuous positive airway pressure (CPAP). CPAP is a highly effective treatment for OSA. Low adherence to CPAP therapy is common and poorly understood. Depression and lack of perceived benefits from CPAP are possible reasons for low adherence. Methods: Seventy-eight patients evaluated for OSA at a sleep medicine center agreed to participate in the study; 54 patients completed all study assessments. The Beck Depression Inventory (BDI) and the functional outcomes of sleep questionnaire (FOSQ) were administered before polysomnographic evaluation. A card embedded in the CPAP device electronically recorded adherence. The BDI and FOSQ were administered 1 to 2 months after the baseline measurements were obtained. Results: Baseline depressive symptoms were not correlated with mean duration of CPAP use per night. Reported improvements in OSA symptoms were correlated positively with CPAP adherence. There were significant positive correlations between improvement in depressive symptoms and OSA symptoms after initiation of CPAP therapy. The polysomnographic variables measured did not predict improvement in daytime OSA symptoms or CPAP adherence. Post hoc analyses suggested that those individuals with baseline Apnea Hypopnea Index (AHI) between 40 and 80 experienced more symptom improvement than those with AHI <40 or >80. Conclusions: Patients with the greatest level of CPAP adherence also reported the greatest improvement in OSA symptoms. Patients who continued to experience OSA symptoms after CPAP treatment also tended to have more depressive symptoms after CPAP treatment. Key words: sleep apnea, depression, adherence, continuous positive airway pressure, fatigue.

AHI = Apnea Hypopnea Index; **BDI** = Beck Depression Inventory; **CPAP** = continuous positive airway pressure; **FOSQ** = functional outcomes of sleep questionnaire; **OSA** = obstructive sleep apnea.

INTRODUCTION

Obstructive sleep apnea (OSA) is characterized by repetitive interruptions in respiration during sleep, causing excessive daytime sleepiness (1) and fatigue (2). Interruptions in respiration result from the collapse of the upper airway during sleep associated with hypotonia of the upper airway muscles during sleep. Central sleep apnea also involves interruptions in breathing, but the cause is absent respiratory effort that may result from various cardiopulmonary or neurologic causes (2). OSA is also associated with increased risk of automobile accidents (3), cardiovascular disease (4), hypertension (5), stroke, (4,6) and insulin resistance (7). Individuals with OSA typically show deficits in memory, attention, and vigilance (8). The prevalence of undiagnosed OSA is estimated to be 5% among adults in the United States (6).

Nasal continuous positive airway pressure (CPAP) is a highly effective treatment for OSA (9). It significantly reduces sleepiness (10,11) and seems to reduce hypertension (12) and

Received for publication July 8, 2006; revision received February 4, 2007. DOI: 10.1097/psy.0b013e318068b2f7

improve sensitivity to insulin (13). The most common reason for lack of patient benefit from CPAP is low adherence (14). In the first study to use covert, electronic measurements of adherence, Kribbs, Pack, Kline, Smith, and colleagues found that only 46% of their participants used CPAP "regularly," defined as >4 hours per night on >70% of nights (15). Subsequent studies have shown that low rates of CPAP adherence are typical (16–18).

The determinants of nonadherence are poorly understood. Psychological variables have been examined as possible predictors of CPAP adherence, and some significant relationships have been identified. Internal locus of control, less belief that powerful others controlled health, and stronger belief in the value of health were important predictors of increased adherence in one study (19). Stepnowsky et al. found that self-efficacy, positive outcome expectations for CPAP, social support, knowledge about CPAP, and decisional balance measured 1 week after participants began using CPAP predicted CPAP adherence at 1 month (20). Decisional balance refers to an individual's judgment of the relative benefits and disadvantages of CPAP. Readiness to change, self-efficacy about using CPAP despite minor problems, and the Decisional Balance Index measured after 1 week of CPAP use have also been found to predict CPAP adherence at 6-month follow-up (21).

The concept of decisional balance is similar to the Health Belief Model, which posits that patients tend to discontinue treatments that do not produce subjective benefits that outweigh the negative aspects of treatment (22). This suggests that CPAP adherence is likely to decrease if a patient tries it but does not perceive any treatment-related improvement in his or her daytime sleepiness or fatigue. Patients expect CPAP to help them feel more alert during the day (23), but some people do not experience this benefit (24,25). Bédard and colleagues found that objectively measured somnolence levels did not return to normal after CPAP treatment (26). In a small study sample, some patients treated with CPAP became sleep-

0033-3174/07/6905-0449

Copyright © 2007 by the American Psychosomatic Society Copyright © Lippincott Williams & Wilkins. Unauthorized reproduction of this article is prohibited.

From the Department of Psychology (R.D.W.), Washington University, St. Louis, Missouri; Department of Psychiatry (K.E.F., R.M.C.), Washington University School of Medicine, St. Louis, Missouri; Washington University School of Medicine Multidisciplinary Sleep Medicine Center (S.P.D.), St. Louis, Missouri; and Rush University Medical Center Sleep Disorders Service and Research Center (R.D.W., E.J.S.), Chicago, Illinois.

Because co-author Kenneth E. Freedland is an associate editor of this journal, the review of this article was overseen by a guest editor. He was not involved in the decision-making process and, like all authors, he was blinded to the identity of the peer reviewers.

Address correspondence and reprint requests to Rachel D. Wells, Behavioral Medicine Institute, 1129 Macklind Avenue, St. Louis, MO 63110. E-mail: rdmwells@gmail.com

This study was supported, in part, by Grant RO1HL65356 from the National Heart, Lung, and Blood Institute (R.M.C., Principal Investigator).

ier (27). Although a meta-analysis (10) supports the finding that CPAP improves daytime symptoms, some patients using CPAP fail to experience subjective benefit. The variability in the effect of CPAP on sleepiness is poorly understood. As Black argued, more research is needed on this lack of improvement in daytime symptoms among patients using CPAP (28).

Depression may diminish the subjective benefits of CPAP, thereby depriving the patient of one of the most important reasons to maintain this intrusive treatment. Across medical illnesses, depressed individuals report more symptoms, independent of the physiological severity of the medical condition (29–31). This seems to be true for individuals with OSA as well. A recent study found higher levels of depressive symptoms were associated with higher levels of self-reported day-time sleepiness (32). The severity of depressive symptoms also independently predicts self-reported sleep quality (33). These findings suggest that depressed individuals with OSA may have more complaints about sleepiness and fatigue than nondepressed individuals with OSA. The improvements in daytime sleepiness caused by CPAP may not be as salient if depression-related fatigue is present.

Depression may also directly impede the use of CPAP, because it is associated with reduced adherence to medical treatment regimens in many different patient populations (34). Previous research has provided contradictory findings about whether or not depression is associated with reduced adherence to CPAP. Lewis and colleagues found no correlation between the Hospital Anxiety and Depression Scale and subsequent adherence to CPAP therapy among patients with OSA (35). Stepnowsky et al. also found that baseline measures of depressive symptoms did not predict CPAP adherence, although this was an intervention study, and the intervention may have confounded the relationship between depression and adherence. That is, the sleep center staff's phone calls to patients may have influenced the CPAP adherence behavior in this study (36). Three other studies found that adherence was predicted by baseline levels of depression: a) Edinger et al. used self-report measures of adherence (37), which are likely to be less accurate; b) Sandberg et al. included patients who recently had suffered cerebrovascular accidents (38); and c) Ayalon et al. included patients with Alzheimer's disease. Consequently, the results may not generalize to other patient populations (39).

In summary, low adherence presents a significant obstacle to the successful treatment of OSA, and the perception of symptom improvement may influence adherence. Depression is associated with a) low adherence to a variety of medical treatments, b) increased sensitivity to symptoms, and c) daytime fatigue similar to that caused by OSA. The relationships among perception of symptom improvement, depression, and CPAP adherence are not well understood.

This observational study investigated whether high baseline levels of depressive symptoms predict nonadherence to CPAP and tested the hypothesis that greater perceived improvement in OSA symptoms is associated with higher adherence. Because it is possible that objective improvements in sleep predict future adherence, polysomnographic measures of sleep quality were examined to determine if they predicted higher CPAP adherence. To identify which patients perceived the greatest benefits from CPAP, we examined the relationships among baseline OSA severity, depressive symptoms, polysomnographic measures of sleep quality, and subsequent reports of benefits from CPAP.

METHODS Participants

Participants admitted to the Multidisciplinary Sleep Medicine Center at Washington University School of Medicine in St. Louis, Missouri, between October 2003 and March 2004 were screened for this study. Individuals presenting for clinical polysomnographic evaluation were recruited, and those who were subsequently diagnosed with OSA and prescribed CPAP were included in the study. Eligibility criteria for this study included the ability to speak English and to complete the study questionnaires. Those who were <18 years old, who had previously used CPAP, who did not receive CPAP within 6 weeks of enrollment, or who had a comorbid sleep disorder, were excluded. All participants completed an informed consent form approved by the Human Studies Committee at Washington University Medical Center.

Adherence data were obtained from 78 participants, and 54 of those participants also completed the follow-up assessment (Figure 1). The 26 participants who did not complete the follow-up assessment mailed the Smart Card containing the adherence data, but could not be reached by telephone to complete the follow-up assessment.

Measures

Depressive Symptoms

The Beck Depression Inventory (BDI), a 21-item questionnaire, was used to measure self-reported depressive symptoms (40).



Figure 1. Recruitment flowchart. CPAP = continuous positive airway pressure; BDI = Beck Depression Inventory; FOSQ = functional outcomes of sleep questionnaire.

Psychosomatic Medicine 69:449-454 (2007)

OSA-Related Impairment

The functional outcomes of sleep questionnaire (FOSQ), a 30-item questionnaire, was used to measure the severity and impact of daytime sleepiness and fatigue caused by sleep disorders (41). The FOSQ assesses the impact of daytime sleepiness and fatigue on activity level, the ability to remain awake in low-stimulation environments, intimacy, and sexual relations, productivity, and the ability to converse with family and friends. On the FOSQ, respondents rate the amount of difficulty caused by "being sleepy or tired" during several everyday activities such as driving, conversing with friends, exercising, and managing finances.

Polysomnographic Measures

Standard polysomnographic techniques were used. Data from the electrooculogram, electrocephalogram, electrocardiogram, electromyogram, airflow measurement, and pulse oximeter were recorded with Sandman digital recording equipment (Tyco, Ottawa, Canada). The Apnea Hypopnea Index (AHI) was calculated from the polysomnographic data, using widely accepted criteria (42).

CPAP Adherence

Adherence to CPAP therapy was measured by Respironics Smart Cards (Respironics, Inc., Murrysville, PA). The cards are embedded in the CPAP device and unobtrusively record the clock times during which the CPAP apparatus delivers positive airway pressure through the mask.

Procedure

All participants signed the informed consent document, and then completed the BDI and FOSQ. Participants then underwent split-night or all-night polysomnography (PSG). The AHI was obtained either from an all-night diagnostic polysomnogram, or from the diagnostic portion of a split-night polysomnogram. Other variables, including arousal frequency, sleep onset latency, and sleep efficiency were obtained from polysomnographic recordings during CPAP titration. During split-night studies, CPAP was initiated if 2 hours of diagnostic data were obtained and the patient's AHI was >25. The diagnostic portion of the PSG was truncated if oxygen saturation decreased <70% during lateral sleep or if the attending physician had ordered a lower AHI threshold for clinical reasons.

In accordance with the clinic's standard practice, nurses or respiratory therapists instructed participants on CPAP use and informed them that their adherence would be monitored with the Smart Cards. Participants were phoned to verify when they received the CPAP apparatus. Approximately 30 days after receiving the CPAP apparatus, participants received letters, asking them to mail in the Smart Card. Participants were phoned to remind them to mail the Smart Card. They were also asked to complete the follow-up FOSQ and BDI over the phone.

Statistical Analysis

Pearson correlation coefficients were used to describe univariate relationships among CPAP adherence, depressive symptoms, change in daytime OSA symptoms, and polysomnographic variables. Significance thresholds were corrected to control for multiple tests. Changes in daytime OSA and depressive symptoms were calculated as the follow-up scores minus the baseline scores for the FOSQ and BDI, respectively. All analyses were performed using SPSS version 12.0 (SPSS Inc., Chicago, IL).

RESULTS

The mean age of participants was 47.9 ± 10.9 (standard deviation) years. Forty-five percent of the participants were women. Caucasians comprised 56% of the sample, African-Americans were 40%, and the remaining 4% were Latino. Medical records indicated that 22% had been previously diagnosed with depression, and 24% were currently prescribed antidepressants. The mean body mass index was 40.5 ± 10.9 , and the mean baseline AHI was 36.3 ± 33.1 . Ninety-seven

 TABLE 1. Summary of Participant Characteristics at Baseline and Follow-Up

	Mean \pm SD
Age, years	47.6 ± 10.7
BMI	40.7 ± 11.3
Baseline AHI	36.6 ± 33.3
CPAP pressure	11.2 ± 4.6
CPAP use per night, minutes	281.5 ± 145.8
Baseline BDI score	12.2 ± 9.3^{a}
Follow-up BDI score	6.2 ± 8.7
Baseline FOSQ score	15.2 ± 2.6^{a}
Follow-up FOSQ score	17.6 ± 3.1

SD = standard deviation; BMI = body mass index; AHI = Apnea Hypopnea Index; CPAP = continuous positive airway pressure; BDI = Beck Depression Inventory; FOSQ = functional outcome of sleep questionnaire.

^a The *t* test difference between baseline and follow-up means is significant; p = .0001.



Figure 2. Mean number of minutes of CPAP use per night. SD = standard deviation; N = number; CPAP = continuous positive airway pressure.

percent of the participants underwent split-night PSG. Participants used CPAP 281.5 \pm 145.8 minutes per night (Table 1 and Figure 2). The mean number of nights recorded on the Smart Card was 40.4 \pm 22.4; on average, participants used CPAP 77% of the nights during the follow-up period. Depressive symptoms improved significantly during the follow-up (t = 4.9; p = .0001). OSA symptoms, as measured by the FOSQ, also improved (t = 6.9; p = .0001) (Table 1).

On average, participants received 4.1 ± 2.6 phone calls during the study, reminding them to mail in the Smart Card and requesting they complete the FOSQ. The FOSQ and BDI were completed 46 ± 19 days after the PSG and baseline FOSQ and BDI. The 24 participants who did not complete the FOSQ at follow-up did not differ from those who completed the follow-up FOSQ with regard to age, gender, or racial distributions, presence of bed partners, antidepressant prescriptions, past history of depression, baseline FOSQ scores, baseline BDI scores, baseline AHI, CPAP adherence, obesity, or number of phone contacts with research staff. The first analyses examined predictors of CPAP adherence. Baseline depressive symptoms were not correlated with mean number of minutes of CPAP use per night. Larger reported improvements in OSA symptoms, as measured by the FOSQ, were associated with higher CPAP adherence (r = .50; p = .0001). Improvements in depressive symptoms were not associated with CPAP use. CPAP adherence was not associated with PSG measures of sleep quality, including total sleep time, sleep onset latency, sleep efficiency, arousals due to leg movements during CPAP titration, or frequency of idiopathic arousals (arousals not attributed to a particular cause). Prescribed CPAP pressure did not predict adherence.

The next group of analyses examined which variables predicted greater reports of benefits from CPAP, as measured by the FOSQ. More severe baseline depression scores did not predict change in daytime OSA symptoms (r = .19; p = .17). However, improvement in depressive symptoms during the follow-up period was associated with more improvements in daytime OSA symptoms (r = -.54, p = .0001).

Neither idiopathic arousals during CPAP titration, sleep onset latency, sleep efficiency, arousals due to periodic leg movements, baseline AHI, CPAP pressure nor time spent at optimal CPAP pressure during CPAP titration predicted daytime symptom improvement after CPAP use.

There was no overall association between baseline AHI and subsequent improvement in daytime OSA symptoms (r = .16; p = .25) (Figure 3). However, a post hoc examination of the scatterplot suggested that the 12 patients with baseline AHI between 40 and 80 showed more improvement in daytime OSA symptoms than those with AHI <40 or AHI >80. All participants with AHI between 40 and 80 reported improve-



Baseline AHI

Figure 3. Relationship between baseline Apnea Hypopnea Index (AHI) and subsequent change in functional outcomes of sleep questionnaire (FOSQ) scores (n = 54; r = .17; p = .24). Positive change in FOSQ indicates improvement in obstructive sleep apnea (OSA) symptoms.

ments, with a mean FOSQ improvement of 4.2 ± 1.8 (range = 0.35–7.1). The 35 patients with baseline AHI ranging from 0 to 40 had a variable response to CPAP. Their mean change in FOSQ scores at follow-up was $\pm 1.9 \pm 2.8$ and ranged from -3.3 to ± 8.5 . Seven of the 35 patients had a negative change in FOSQ score, indicating their daytime symptoms worsened with CPAP use. The seven patients with baseline AHI >80 also had a variable response to CPAP (2.3 ± 1.9 ; range = -0.65 to ± 5.3). A second-order polynomial regression was run, and this model fit these data significantly better than the first-order regression (difference in fit between full versus reduced model, $F_{1,51} = 9.7$; p = .003). This suggests a curvilinear relationship between baseline AHI and OSA symptom change.

DISCUSSION

Baseline levels of depression did not predict CPAP adherence in this sample of patients. CPAP use was associated with improvement in self-reported daytime OSA symptoms. Selfreported depressive symptoms also improved during the follow-up period, but this was not associated with the amount of CPAP use. The improvement in depressive symptoms is both statistically significant and clinically meaningful because the initial mean BDI score was 12.5, consistent with mild depression, and the mean improved to 6.2 at follow-up, which is within the normal range (43). Depressive symptoms and daytime OSA symptoms seem to improve concurrently during OSA treatment. Those who remain depressed after using CPAP continue to report that daytime sleepiness and fatigue interfere with their daytime functioning. Sleep onset, sleep efficiency, frequency of limb movements, time spent at optimal CPAP pressure, and idiopathic arousals did not predict improvement in daytime OSA symptoms among CPAP users.

The association between daytime OSA symptoms and CPAP adherence may reflect a "dose-response" relationship, with higher CPAP adherence causing greater improvement. The association may also be attributed to greater willingness to continue CPAP use among those patients who noticed improvement in daytime symptoms. The direction of causality cannot be determined from this correlational study. It is possible, however, that there is a bidirectional relationship, with adherence improving symptoms, and symptom improvement promoting sustained adherence to CPAP.

The feedback loop between symptom improvement and CPAP adherence may be attenuated by the presence of depression. Individuals with depression tend to report more symptoms, regardless of the severity of their illness (29–31). The strong association between self-reports of depressive symptoms and OSA symptoms may reflect the tendency of depressed individuals to be unusually sensitive to symptoms and to report more impairments of all types on self-report measures. However, fatigue, low energy, insomnia, and altered sleep architecture are also symptoms of depression (44) and may mimic daytime OSA symptoms. Individuals with depressive symptoms that continue during CPAP treatment are more likely to report continued fatigue and/or sleepiness,

Psychosomatic Medicine 69:449-454 (2007)

ADHERENCE, BENEFIT, AND DEPRESSION AFTER CPAP

even with adequate CPAP treatment. Individuals who experience an improvement in depressive symptoms also report an improvement in OSA symptoms. Kawahara and colleagues reported that depressive symptoms and self-reported measures of daytime sleepiness both improved post CPAP treatment (45). The factors that determine if depressive symptoms will improve during CPAP treatment are unknown. Baseline depression was not related to the level of CPAP adherence in the present study. Regardless of the causal mechanism, this study suggests that it is important to inquire about depressive symptoms if an individual does not report improvement in fatigue or sleepiness after using CPAP regularly.

It is also possible that depressive symptoms may respond differently during the course of CPAP treatment, depending on whether they precede the onset of OSA, develop before but are exacerbated by OSA, or are secondary to OSA. Perhaps depressive symptoms that begin during the course of OSA respond more readily to CPAP, although long-standing symptoms of recurrent major depressive disorder tend to persist even after successful CPAP treatment.

Post hoc examination of the data demonstrated that those patients with baseline AHI between 40 and 80 had the largest and most consistent improvement in daytime symptoms after CPAP use. Those with baseline AHI <40 benefited less from CPAP, and in some cases reported more sleepiness and fatigue after CPAP use. This finding is similar to the report by Monasterio et al., showing that individuals with mild OSA (AHI 10-30) had no significant improvement in FOSQ scores after using CPAP (46), and a meta-analysis that found the largest improvements in self-reported daytime sleepiness when the analysis was restricted to samples with baseline AHI >30 and Epworth Sleepiness Scores ≥ 11 (10). Perhaps the perceived benefits of treating milder OSA do not outweigh the annoyances of wearing CPAP for some individuals. The individuals in this sample with AHI > 80 also showed smaller improvements in FOSQ scores than those individuals with AHI between 40 and 80. Perhaps other health problems caused individuals with AHI > 80 to continue to sleep poorly, despite CPAP use. The regression analyses indicate a curvilinear relationship exists between baseline severity of OSA and perceived improvements post CPAP treatment in this sample. This was a post hoc analysis, however, so replication is needed before any firm conclusions can be drawn.

This study has several limitations. Of the 78 individuals who provided adherence data, only 54 individuals agreed to complete the FOSQ at follow-up. Providing adherence data only required participants to mail in the Smart Card; completing the follow-up FOSQ required a 15- to 20-minute phone conversation, which likely accounts for some of the failure to complete the study. Those who completed the follow-up FOSQ did not differ from those who did not complete it in any of the characteristics tested, but it remains possible they differed in variables that were not tested.

A randomized control trial design with sham CPAP would provide stronger evidence than the present observation study. However, ethical considerations prohibited the use of this study design in the project's clinical setting. Performing follow-up PSG or pulse oximetry would provide a measure of how well OSA had been controlled, at least on the night of the follow-up measures.

Another limitation comes from the measurement of daytime OSA symptoms. The FOSQ is a self-report measure of daytime fatigue and sleepiness and, as such, is a subjective measure. It was chosen to gain information about the participant's perception of his/her daytime functioning. However, it would be useful to repeat this study with objective measures of wakefulness and daytime sleepiness. The BDI is also a self-report measure and cannot be used to diagnose major depressive disorder. Using diagnostic interviews in future studies might provide additional data.

Daytime fatigue and sleepiness are often the most troublesome aspects of OSA for patients. More research is needed to learn which patients experience improvement in these symptoms post CPAP therapy. When approaching this question, it is important to consider the strong relationship between depressive symptoms and reports of daytime fatigue and sleepiness.

We thank the volunteer participants who made this study possible, and the staff at the Washington University Multidisciplinary Sleep Medicine Center for their assistance.

REFERENCES

- Bassiri A, Guilleminault C. Clinical features and evaluation of obstructive sleep apnea-hypopnea syndrome. In: Kryger M, Roth T, Dement W, editors. Principles and Practice of Sleep Medicine. 3rd ed. Philadelphia: WB Saunders; 2000.
- Orr W. Obstructive sleep apnea: natural history and varieties of the clinical presentation. In: Pressman M, Orr W, editors. Understanding Sleep: the Evaluation and Treatment of Sleep Disorders. Washington, DC: American Psychological Association; 1997.
- Howard M, Desai A, Grunstein R, Hukins C, Armstrong J, Joffe D, Sqann P, Campbell D, Pierce R. Sleepiness, sleep-disordered breathing, and accident risk factors in commercial vehicle drivers. Am J Repir Crit Care Med 2004;170:1014–21.
- 4. Malhotra A, White D. Obstructive sleep apnea. Lancet 2002;360:237-45.
- Narkiewicz K, Phillips B, Somers V. Influence of sleep and sleep apnea on autonomic control of the cardiovascular system. In: Bardley T, Floras J, editors. Sleep Apnea: Implications in Cardiovascular and Cerebrovascular Disease. New York: Marcel Dekker; 2000.
- Young T, Peppard P, Gottlieb D. Epidemiology of obstructive sleep apnea. Am J Repir Crit Care Med 2002;165:1217–39.
- Punjabi N, Sorkin J, Katzel L, Goldberg A, Schwartz A, Smith P. Sleep-disordered breathing and insulin resistance in middle-aged and overweight men. Am J Repir Crit Care Med 2002;165:677–82.
- Aloia M, Arnedt J, Davis J, Riggs R, Byrd D. Neuropsychological sequelae of obstructive sleep apnea-hypopnea syndrome: a critical review. J Int Neuropsychol Soc 2004;10:772–85.
- American Thoracic Society. Indications and standards for use of nasal continuous positive airway pressure (CPAP) in sleep apnea syndromes. Am J Repir Crit Care Med 1994;150:1738–45.
- Patel R, White D, Malhotra A, Stanchina M, Ayas N. Continuous positive airway pressure therapy for treating sleepiness in a diverse population with obstructive sleep apnea. Arch Intern Med 2003;163:565–71.
- Giles T, Lasserson T, Smith B, White J, Wright J, Cates C. Continuous positive airways pressure for obstructive sleep apnoea in adults. Cochrane Database Syst Rev 2006;3:CD001106.
- Hla K, Skatrud J, Finn L, Young T. The effect of correction of sleepdisordered breathing on breathing on BP in untreated hypertension. Chest 2002;122:1125–32.
- 13. Babu A, Herdegen J, Fogelfeld L, Shott S, Mazzone T. Type 2 diabetes,

453

Psychosomatic Medicine 69:449-454 (2007)

glycemic control, and continuous positive airway pressure in obstructive sleep apnea. Arch Intern Med 2005;165:447–52.

- Kryger M. Management of obstructive sleep apnea-hypopnea syndrome: overview. In: Kryger M, Roth T, Dement W, editors. Principles and Practice of Sleep Medicine. 3rd ed. Philadelphia: WB Saunders; 2000.
- Kribbs N, Pack A, Kline L, Smith P, Smith P, Schwartz A, Schubert N, Redline S, Henry J, Getsy J, Dinges D. Objective measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. Am Rev Respir Dis 1993;147:887–95.
- Engleman H, Kingshott R, Wraith P, Mackay T, Deary I, Douglas, N. Randomized placebo-controlled crossover trial of continuous positive airway pressure for mild sleep apnea/hypopnea syndrome. Am J Repir Crit Care Med 1999;159:461–7.
- Kribbs, N. (1997). Methods and problems of treatment compliance in obstructive sleep apnea. In: Pressman M, Orr W, editors. Understanding Sleep: the Evaluation and Treatment of Sleep Disorders. Washington, DC: American Psychological Association; 1997.
- Noseda A, Jann E, Hoffman G, Linkowski P, Kerkhofs M. Compliance with nasal continuous positive airway pressure assessed with a pressure monitor: pattern of use and influence of sleep habits. Respir Med 2000; 94:76–81.
- Wild M, Engleman H, Douglas N, Espie C. Can psychologigcal factors help us to determine adherence to CPAP? A prospective study. Eur Repir J 2004;24:461–5.
- Stepnowsky C Jr, Marler MR, Ancoli-Israel S. Determinants of nasal CPAP compliance. Sleep Med 2002;3:239–47.
- Aloia M, Arnedt T, Stepnowsky C, Hecht J, Borrelli B. Predicting treatment adherence in obstructive sleep apnea using principles of behavior change. J Clin Sleep Med 2005;1:346–53.
- Janz N, Becker M. The health belief model: a decade later. Health Educ Q 1984;11:1–47.
- 23. Weaver T, Maislin G, Dinges D, Younger J, Cantor C, McCloskey S, Pack A. Self-efficacy in sleep medicine: instrument development and patient perceptions of obstructive sleep apnea risk, treatment benefit, and volition to use continuous positive airway pressure. Sleep 2003;26: 727–32.
- Morrison F, Décary A, Petit D, Lavigne G, Malo J, Monplaisir J. Daytime sleepiness and EEG spectral analysis in apneic patients before and after treatment with continuous positive airway pressure. Chest 2001;119: 45–52.
- Rosenthal L, Gerhardstein R, Lumley A, Guido P, Day R, Syron ML, Roth T. CPAP therapy in patients with mild OSAQ: implementation and treatment outcome. Sleep Med 2000;1:215–20.
- Bédard M, Montplaisir J, Malo J, Richer F, Rouleau I. Persistent neuropsychological deficits and vigilance impairment in sleep apnea syndrome after treatment with continuous positive airways pressure (CPAP). J Clin Exp Neuropsychol 1993;15:330–41.
- Meurice J, Paquereau J, Neau J, Caron F, Dore P, Ingrand P, Patte, F. Long-term evolution of daytime somnolence in patients with sleep apnea hypopnea syndrome treatment by continuous positive airway pressure. Sleep 1997;20:1162–6.
- 28. Black, J. Sleepiness and residual sleepiness in adults with obstructive sleep apnea. Respir Physiol Neurobiol 2003;136:211–20.
- 29. Dwight M, Kowdley K, Russo J, Ciechanowski P, Larson A, Katon W.

Depression, fatigue, and functional disability in patients with chronic hepatitis C. J Psychosom Res 2000;49:311–7.

- Katon W, Ciechanowski P. Impact of major depression on chronic medical illness. J Psychosom Res 2002;53:859-63.
- 31. Katon W, Sullivan M, Walker E. Medical symptoms without identified pathology: relationship to psychiatric disorders, childhood and adult trauma, and personality traits. Ann Intern Med 2001;134:917–25.
- Kjelsberg F, Ruud E, Stavem K. Predictors of symptoms of anxiety and depression in obstructive sleep apnea. Sleep Med 2005;6:341–6.
- Wells R, Day R, Carney R, Freedland K, Duntley S. Depression predicts self-reported sleep quality in patients with obstructive sleep apnea. J Psychosom Med 2004;66:692–7.
- DiMatteo M, Giordani P, Lepper H, Croghan T. Patient adherence and medical treatment outcomes: a meta-analysis. Med Care 2002;40: 794-811.
- Lewis K, Seale L, Bartle I, Watkins A, Ebden P. Early predictors of CPAP use for the treatment of sleep apnea. Sleep 2004;27:134–8.
- Stepnowsky C, Bardwell W, Moore P, Ancoli-Israel S, Dimsdale J. Psychologic correlates of adherence with continuous positive airway pressure. Sleep 2002;25:36–40.
- Edinger J, Carwile S, Mikller P, Hope V, Mayti C. Psychological status, syndromatic measures, and compliance with nasal CPAP therapy for sleep apnea. Percept Mot Skills 1994;78:1116–8.
- Sandberg O, Franklin KA, Bucht G, Eriksson S, Gustafson, Y. Nasal continuous positive airway pressure in stroke patients with sleep apnoea: a randomized treatment study. Eur Respir J 2001;18:630–4.
- Ayalon L, Ancoli-Israel S, Stepnowsky C, Marler M, Palmer B, Liu L, Soredo J, Corey-Bloom J, Greenfield D, Cooke J. Adherence to continuous positive airway pressure treatment in patients with Alzheimer disease and obstructive sleep apnea. Am J Geriatr Psychiatry 2006;14: 176–80.
- Beck A, Ward C, Medelson M, Mock J, Erbaugh J. An inventory for measuring depression. Arch Gen Psychiatry 1961;4:561–71.
- Weaver T, Laizner A, Evans L, Maislin G, Chugh D, Lyon K, Smith, P, Schwartz, A, Redline, S, Pack, A, Dinges, D. An instrument to measure functional status outcomes for disorders of excessive sleepiness. Sleep 1997;20:835–843.
- 42. American Academy of Sleep Medicine Task Force. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. Sleep 1999;22:667–89.
- Beck A, Steer R, Garbin M. Psychometric properties of the Beck depression inventory: twenty-five years of evaluation. Clin Psychol Rev 1988; 8:77–100.
- Ware J, Morin, C. Sleep in depression and anxiety. In: Pressman, M, Orr W, editors. Understanding Sleep: the Evaluation and Treatment of Sleep Disorders. Washington, DC: American Psychological Association; 1997.
- 45. Kawahara S, Akashiba T, Akahoshi T, Horie T. Nasal CPAP improves the quality of life and lessens the depressive symptoms in patients with obstructive sleep apnea syndrome. Ann Innter Med 2005;44:422–7.
- 46. Monasterio C, Vidal S, Duran J, Ferrer M, Carmona C, Barbé F. Mayos M, Gonzalez-Mangado N, Juncadella M, Navarro A, Barreira R, Capote F, Mayoralas L, Peces-Barba G, Alonso J, Montserrat J. Effectiveness of continuous positive airway pressure in mild sleep apnea-hypopnea syndrome. Am J Respir Crit Care Med 2001;164:939–43.