NEW RESEARCH

JCSM
Journal of Clinical Sleep Medicine

SCIENTIFIC INVESTIGATIONS

Outcome of CPAP Treatment on Intimate and Sexual Relationships in Men with Obstructive Sleep Apnea

Judith L. Reishtein, Ph.D.[1]; Greg Maislin, M.S., M.A.[2]; Terri E. Weaver, Ph.D.[2,3]; and the Multisite Study Group

[1]College of Nursing and Health Professions, Drexel University, Philadelphia, PA; [2]Division of Sleep Medicine, Department of Medicine and Center for Sleep and Respiratory Neurobiology, School of Medicine, University of Pennsylvania, Philadelphia, PA; [3]Biobehavioral and Health Sciences Division, School of Nursing, University of Pennsylvania, Philadelphia, PA

Study Objectives: To examine intimate and sexual relationships in patients with obstructive sleep apnea (OSA), the association with daytime sleepiness, and the change in these outcomes with continuous positive airway pressure treatment (CPAP).

Design: Pre-post test, quasi-experimental study

Setting: Seven sleep disorders centers in the US and Canada

Participants: 123 males with OSA (AHI ≥ 20), aged 21 to 60 years

Interventions: Nasal CPAP for ≥ 3 months

Measurements and Results: Compared to normal values, at baseline patients were significantly sleepier, as measured by the Multiple Sleep Latency Test and Epworth Sleepiness Scale. They were also more impaired in intimate and sexual relationships, as measured by the Intimate and Sexual Relationships subscale of the Functional Outcomes of Sleep Questionnaire. Neither race nor marital status was significantly associated with impaired intimate and sexual relationships. Following treatment, patients were significantly more alert and had reported improved intimate and sexual relationships, with the greatest change occurring in those with the most disease severity.

Conclusions: OSA has an adverse impact on intimate and sexual relationships that is related to subjective sleepiness and improved with CPAP treatment.

Keywords: CPAP, functional status, obstructive sleep apnea, relationships, sexual, sleepiness

Citation: Reishtein JL; Maislin G; Weaver TE. Outcome of CPAP treatment on intimate and sexual relationships in men with obstructive sleep apnea. *J Clin Sleep Med* 2010;6(3):221-226

Intimate and sexual relationships are important aspects of quality of life, as they reinforce human connections, aliveness, and caring.[1] However, for some individuals with obstructive sleep apnea (OSA), this essential behavior may be adversely affected. Indeed, 30% to 68% of men with OSA suffer from some level of sexual dysfunction,[2,3] and loss of interest in sex has been associated with the number of apneic events and level of oxygen desaturation.[3-6]

A commentary on this article appears in this issue on page 227.

The impairments in sexual functioning associated with OSA, including the impact on relationships and negative effect on desire, arousal, and orgasm, have not been well described. The limited studies that have examined alterations in intimate and sexual behavior in this population have utilized small samples and have only explored one aspect of intimacy and sexual functioning.[2,3,7,8] Moreover, the efficacy of CPAP in improving sexual dysfunction has been poorly documented.[3,7,9] For example, Zhuraviev and colleagues treated 5 men who had both OSA and erectile dysfunction with CPAP for 6 months. Even though sleep phases normalized, no changes occurred in the men's sexual function.[8]

The purposes of this study were to examine OSA patients' perceptions of their intimate and sexual relationships, the association with daytime sleepiness, and degree of impairment compared to normal values, and to document change in this behavior with CPAP treatment. We hypothesized that (1) compared to normal values on the Intimate and Sexual Relationships subscale of the Functional Outcomes of Sleep Questionnaire (FOSQ),[10] scores for individuals with OSA, especially for men with more severe disease, would indicate greater impairment; (2) limitations in sexual functioning would be associated with both objective and subjective sleepiness; and (3) following 3 months of CPAP treatment there would be improvement in scores on the intimate and sexual relationships subscale as well as on measures of objective and subjective sleepiness.

BRIEF SUMMARY

Current Knowledge/Study Rationale: Many studies of OSA and the effects of treatment on functional status of patients fail to include sexual function as a variable, and thus little is known about this important aspect of life. This secondary analysis using data from an investigation of the effect of CPAP treatment on the lives of people with OSA compared subjective self-reported intimate and sexual relationships before and after CPAP treatment.

Study Impact: In demonstrating that people who have OSA have impaired intimate and sexual relationships that generally normalize following CPAP treatment, this study has revealed another benefit of CPAP and a potential stimulus for improved adherence to treatment.

METHODS

Sample

This article is a secondary analysis of data gathered during a larger prospective study of CPAP effectiveness.[11] Participants were recruited from 7 sleep disorders centers in the US and

Canada. The institutional review boards at each agency approved the original study protocol.

Potential participants were recruited at a sleep disorders center if they were 21 to 60 years old, if they had an apnea hypopnea index (AHI) ≥ 20 events per hour/sleep, and were candidates for CPAP treatment. Exclusion criteria were: coexisting sleep disorder, history of sedative-hypnotic use, cerebrovascular accident, chronic obstructive pulmonary disease, other pulmonary disease, or congestive heart failure.

Procedure

The procedure for this study has been reported previously.[11,12] Briefly, patients who had been referred to a participating sleep disorders center for evaluation of sleep were approached by the physician supervising the sleep study. The research project was explained and written informed consent was obtained. The physician managing the patient's care and soliciting participation in the study did not participate in data collection.

As part of their routine medical care, participants suspected of having OSA underwent a full night PSG followed by a night of CPAP titration (74.3% of sample) or a split night study (24.8% of sample). Before beginning CPAP treatment, participants underwent a day of testing that included completion of the instruments described above, as well as a neurobehavioral assessment battery, the Psychomotor Vigilance Task, and the Multiple Sleep Latency Test (MSLT). Only the results from the MSLT, ESS, and FOSQ are reported here. Upon completion of testing, subjects received instruction in use of the CPAP machine and commenced treatment. After they had used CPAP ≥ 3 months, participants returned to the sleep disorders center and underwent the same testing as they did pre-treatment. The subjects slept at home prior to the day of testing using their CPAP machine.

Measures

Polysomnography

All subjects underwent nocturnal polysomnography (PSG) in the sleep laboratory as part of their routine clinical care. The number of apneas and hypopneas per hour was determined by a full PSG that included electroencephalogram, electrooculogram, electromyogram, and electrocardiogram. A finger pulse oximeter measured oxygen saturation, nasal thermistors measured airflow, and respiratory inductance plethysmography measured thoracic and abdominal respiratory movement.[13] Sleep stages were scored using the protocol of Rechtschaffen and Kales.[14] For analysis purposes, participants with sleep apnea were classified by AHI into 3 levels of severity: AHI of 20 to ≤ 40, AHI 40 to ≤ 60, and AHI > 60. No desaturation cutoff point was used to determine either diagnosis or disease severity classification.

Intimate and Sexual Relationships

Intimate and sexual relationships were assessed using the Intimate and Sexual Relationships subscale of the Functional Outcomes of Sleep Questionnaire (FOSQ).[10] This disease-specific, self-administered instrument contains 30 questions and 5 stand-alone subscales: General Productivity, Vigilance, Social Outcome, Activity Level, and Intimate and Sexual Relationships. Respondents rate each activity for degree of difficulty on a 5-point Likert scale, with higher scores indicating better function. There is also an option to indicate nonperformance of an activity for reasons unrelated to sleepiness. The FOSQ was designed to make each subscale psychometrically strong and capable of being used independently. This paper reports on values for the Intimate and Sexual Relationships subscale: a general evaluation of how intimate behaviors and sexual relationships have been affected by sleepiness or tiredness, consisting of questions related to desire for intimacy or sex, ability to become sexually aroused, and ability to have an orgasm. The FOSQ has been used in numerous studies of sleep outcomes and has demonstrated reliability and validity.[15-19] Cronbach α for the Intimate and Sexual Relationships subscale was originally reported as 0.89[10] and was 0.90 in this study.

Sleepiness

Objective daytime sleepiness was measured using the MSLT.[20-22] The MSLT is measured by polysomnogram, the time required for the subject to fall asleep (latency time) in a 20-min period and is administered every 2 h starting at 10:00. The MSLT is a sensitive and valid measure of physiologic sleepiness and response to treatment.[23,24] Sleep was terminated with the first epoch of stage 1 sleep to avoid sleep recovery effects on subsequent naps and neurobehavioral testing (results not reported here), as recommended by Carskadon and colleagues when the MSLT is used in research.[21] The MSLT value was the average time of 4 latencies to sleep.

Subjective sleepiness was measured using the Epworth Sleepiness Scale (ESS).[25,26] A score ≥ 11 indicates manifest sleepiness.[26] The ESS has a single factor structure,[27] and has demonstrated both test-retest reliability (r = 0.82) and internal consistency (Cronbach α = 0.88) in patients with OSA.[28,29] It is a validated clinical and research tool in the assessment of daytime sleepiness.[26]

Adherence

Subjects received the CPAP device and mask prescribed for them by the physician managing their sleep apnea. Adherence to CPAP treatment was documented using an overt monitor attached to the CPAP machine.[30] This external monitor employs a microprocessor that utilizes an algorithm for the detection of mask-on pressure. The electric cord of the CPAP machine was plugged into the adherence monitor. Subjects were instructed to turn their CPAP device on and off via the monitor activation switch. The monitor is also attached to the CPAP apparatus via high-pressure tubing connected to a "T" tube inserted in its outflow port. A pressure transducer housed in the monitor senses the circuit pressure level and records mask-off events when the therapeutic pressure drops > 5 cm for ≥ 10 sec. Thus, the duration of therapeutic pressure delivery per day and number of missed days of use can be determined. Subjects were informed that their CPAP use was being monitored.

Statistical Analysis

SAS software was used to analyze the data. As data was not normally distributed, Spearman correlations were used to determine the relationship between sleepiness measures and the Intimate and Sexual Relationships subscale. Paired *t*-tests were used to assess the significance of the change from pre to post treatment on all items. Analyses of covariance (ANCOVA)

were conducted, with post hoc tests, to determine differences among the severity groups. Key variables (age and BMI) were used as covariates after significant differences were demonstrated among OSA severity groups. Categorical variables were compared using χ^2 statistics. Effect sizes were calculated by dividing the mean change score by the standard deviation of the change.[31] Statistical significance was set at $p < 0.05$.

RESULTS

Sample

One hundred seventy-six people met inclusion criteria for this study. Ten subjects failed to return for follow-up testing, and CPAP adherence data for 17 subjects (10% of total) was lost because of technical problems (broken monitor transformer, failed monitor battery, corrupted electronic data). Because of the relatively small number of female participants (10% of total) in the study, and the subjective differences in sexual expression between men and women, this analysis is based on data from 123 male participants only (77% of the men in the original sample).

As shown in **Table 1**, the sample analyzed for this report primarily consisted of middle-aged, obese white men with severe sleep apnea (mean AHI = 66.90 ± 28.70, range 20–155.9). By design, there were significant differences in the mean AHI between the 3 disease severity groups. As expected, there were also robust differences in age between the least and most severe groups as well as significantly higher BMI in the > 60 group compared to the AHI 40 to < 60 group.

Table 1—Demographic characteristics of participants

	All males with OSA n = 123	AHI 20 to < 40 n = 29	AHI 40 to < 60 n = 23	AHI > 60 n = 71
Age years				
mean	46.35	48.96*	47.92	44.79*
(SD)	8.47	9.50	7.52	8.06
BMI kg/m²				
mean	37.59	35.71	34.93*	39.22*
(SD)	8.42	6.42	6.33	9.38
Race[a]				
White	111	26	19	66
Non-white	11	2	4	5
Marital Status[b]				
Married	106	23	22	61
Single	10	3	1	6
Sep/Div	7	3	0	4
AHI				
Mean	66.90	30.94**	50.06**	87.04**
(SD)	28.70	6.37	5.16	19.26

[a]Not all subjects indicated race; [b]No widowers were in sample
*p < 0.05; **p < 0.001

Baseline

At baseline, the mean score for all participants on the Intimate and Sexual Relationships subscale (3.17 ± 0.79) was significantly lower than the normal value for this subscale (3.93 ± 0.17, $p < 0.001$).[10] This was a clinically meaningful difference with a large effect size of 0.96. Prior to treatment, there were no significant differences between disease severity groups (see **Table 2**). As displayed in **Table 2**, at baseline, 70% of the participants had an MSLT value < 10 min (mean = 7.26 ± 5.29) and an ESS ≥ 11 (mean = 14.48 ± 4.90). There were no significant differences in mean MSLT scores between disease severity groups, but scores on the ESS were significantly different between AHI 40 to < 60 and AHI > 60 groups.

The impact of perceived daytime sleepiness on the ability to be intimate and engage in sexual relationships was indicated by a moderate correlation between the Intimate and Sexual Relationships scale and subjective sleepiness ($r_s = -0.35$, $p < 0.0001$). However, there was no significant association between intimate and sexual relationships and objectively measured sleepiness ($r_s = 0.04$). More than half of the OSA participants reported that sleepiness had affected at least one behavior on the Intimacy and Sexual relationships scale (desire, arousal, or orgasm). Indeed, 63% reported problems with relationships, 69% experienced reduced desire, 46% decreased arousal, and 29% difficulty achieving orgasm (see **Table 3**).

Post Treatment

Participants used CPAP a mean of 4.75 ± 2.12 h/ night. There were no significant differences in CPAP use between disease severity groups. Mean Intimate and Sexual Relationships score for the OSA sample pre-treatment was weakly correlated with the number of days on which CPAP was subsequently used ($r_s = 0.17$, $p = 0.05$).

Post-treatment scores for Intimacy and Sexual Relationships subscale, MSLT, and ESS are shown in **Table 2**. As hypothesized, following 3 months of CPAP treatment, the Intimate and Sexual Relationships subscale mean score for the total sample (3.62 ± 0.59, $p < 0.0001$) demonstrated clinically important improvement as indicated by a moderate effect size (mean change 0.44 ± 0.73, effect size, 0.61). The magnitude of the change (effect size) in intimacy and sexual function following treatment increased across disease severity groups, with the least severe OSA participants achieving the smallest change, and the greatest improvement being realized by the most severe group (effect sizes for three groups = 0.34, 0.53, and 0.77, respectively). However, only in the group with the highest AHI (AHI ≥ 60) was this change statistically significant.

There were large changes in subjective sleepiness following treatment (ESS scores −6.15 ± 5.97, effect size 1.03, $p < 0.001$). Similar to the changes in intimacy and sexual function, the smallest change in subjective sleepiness occurred in the less severe group and the largest change in the group with the highest level of disease severity, all changes within severity groups were statistically reliable. Although not as robust, the mean change in objective sleepiness (MSLT scores) was also statistically significant (2.03 ± 4.85 min, effect size 0.42, $p = 0.003$). Moderate effect sizes in the MSLT following treatment were observed for persons with the highest levels of disease severity (effect size 0.61, $p < 0.01$).

The degree of change post-treatment for intimate behaviors and sexual function was significantly related to both objective and subjective sleepiness (r_s = 0.31 and −0.31, respectively, $p < 0.009$). The change in the Intimate and Sexual Relationships

Table 2—Comparisons of variables among OSA-severity groups

Outcome	Instrument	Group	Baseline (Mean ± SD)	Post treatment (Mean ± SD)	Change Score (Mean ± SD)	p	Effect Size*
Intimate/Sexual Relationships	FOSQ	All OSA subjects	3.17 ± 0.79	3.62 ± 0.59	0.44 ± 0.73	< 0.001	0.61
		AHI 20 to < 40	3.30 ± 0.79	3.55 ± 0.68	0.25 ± 0.73	ns	0.34
		AHI 40 to ≤ 60	3.22 ± 0.72	3.59 ± 0.70	0.37 ± 0.70	ns	0.53
		AHI > 60	3.10 ± 0.81)	3.65 ± 0.52	0.55 ± 0.72	< 0.001	0.76
Sleepiness	MSLT	All OSA subjects	7.26 ± 5.29	9.24 ± 6.01	2.04 ± 4.85	< 0.001	0.42
		AHI 20 to < 40	8.55 ± 5.41	9.52 ± 5.75	0.93 ± 4.41	ns	0.21
		AHI 40 to ≤ 60	7.78 ± 4.50	8.74 ± 6.05	0.66 ± 5.03	ns	0.13
		AHI > 60	6.56 ± 5.42	9.29 ± 6.19	2.97 ± 4.84	< 0.01	0.61
	ESS	All OSA subjects	14.48 ± 4.90	8.39 ± 5.03	-6.14 ± 0.97	< 0.001	6.33
		AHI 20 to < 40	13.40 ± 4.97	10.33 ± 5.80[b]	-2.80 ± 4.92	< 0.05	0.57
		AHI 40 to ≤ 60	13.05 ± 4.63[a]	8.30 ± 5.13	-4.95 ± 5.52	< 0.01	0.90
		AHI > 60	15.32 ± 4.84[a]	7.64 ± 4.50[b]	-7.82 ± 5.90	< 0.01	1.33

*Effect Size: mean of change scores/SD change; [a]significant difference at baseline between these two groups, p < 0.05; [b]significant difference post treatment between these two groups, p < 0.05

Table 3—Proportion of participants reporting difficulty with intimacy and sexual relationships

	Desire	Intimacy	Arousal	Orgasm
Baseline	69%	63%	46%	29%
Post CPAP	40%**	34%**	2%**	18%*

Change in proportion baseline to post treatment; *p < 0.05; **p < 0.001

score was not significantly correlated with age, BMI, AHI, or CPAP use (h /day, proportion of days with any CPAP use, or proportion of days when CPAP use was more than either 4 or 6 h).

With CPAP treatment, scores for the total sample on the Intimacy and Sexual Relationships subscale, although much improved, remained significantly different from normal values (p < 0.01). However as shown in **Table 3**, following treatment the proportion of participants indicating problems with relationships, desire, arousal, and orgasm significantly decreased (40%, 34%, 2%, and 18%, respectively).

DISCUSSION

This is the first large study that has examined how people who have OSA perceive their intimate and sexual relationships. Compared to people without OSA, men with OSA have poorer intimate and sexual relationships which are significantly related to daytime sleepiness. Following treatment with CPAP, intimate and sexual functioning improves substantially, with the most severely affected showing the largest improvement. This improvement is robustly related to changes in daytime sleepiness.

Our findings of diminished intimate and sexual functioning in men with OSA are consistent with findings in other smaller studies.[5,16,32-34] For example, in a study of mild OSA, Barnes found that compared to the normal value for the Intimate and Sexual Relationships subscale score, participants (N = 42) had reduced intimate and sexual activity scores of clinical importance.[32] Bradshaw and colleagues also reported limitations in intimate and sexual behavior of OSA patients to be of similar magnitude (2.88 ± 0.88) to that found in our study.[16] In 59 men who snored or had other symptoms of OSA, 59.3% of those who had OSA confirmed by PSG also reported erectile dysfunction, as indicated by low scores on the Korean version of the International Index of Erectile Function, while 29.6% of the men who did not have OSA reported erectile dysfunction.[34] In a study of 15 men treated with CPAP for 12 weeks, scores on the International Index of Erectile Function, which assesses intercourse satisfaction and overall satisfaction, increased significantly.[33] In a study of 50 patients with erectile dysfunction and presumed OSA based on clinical history, Teloken and colleagues demonstrated significant differences between those manifesting sleepiness (ESS scores > 10) and those who did not (ESS ≤ 10) on the total and erectile function domains of the International Index of Erectile Function.[5] However, unlike our findings, they did not report differences between the two groups on the domains of intercourse satisfaction, orgasm, libido, and overall satisfaction. Without having polysomnographic confirmation of OSA for the participants, it is difficult to know whether differences in findings between the Teloken study and our investigation are related to disease severity, measures employed, or other factors related to OSA.

Most of the investigations of the relationship between OSA and intimate relationships and sexual functioning have focused on the ability to have an erection. In 1981, Schmidt and Wise noted the association between OSA and erectile dysfunction, with 46% of the 15 participants having abnormal nocturnal penile tumescence, with measurements that were significantly lower than those with psychogenic impotence or normal men.[35] Examining the prevalence of OSA in a sample of 1,025 patients with ED, Hirshkowitz and associates found that 91% had an AHI > 5, indicative of OSA.[36] We did not find differences in scores on the Intimacy and Sexual Relationship score between levels of disease severity. However, using cut points for respiratory disturbance similar to our study, but including patients with mild OSA, Margel and coworkers did find a significant dose-response association between severity of ED as measured by the International Index of Erectile Function and the respiratory disturbance in-

dex.[37] These inconsistencies could be due to the wider spectrum of disease severity evaluated, measures used to evaluate intimate and sexual behavioral, and greater number of participants at each category of respiratory disturbance employed.

Several possible physiologic mechanisms have been explored for decreased intimate and sexual relationships in OSA. Those who are sleepier, as we found, may be less interested and more impaired in sexual activity. Indeed, Barnes and colleagues also found among a group of 98 men with OSA, those with ED were significantly sleepier in the daytime than those without ED.[32] Margel and colleagues reported that morning tiredness was predictive of sexual satisfaction and being able to have a morning erection.[37] In addition to excessive sleepiness, other potential mechanisms have been considered, including hormone levels as well as hypoxic-induced neural dysfunction affecting the erectile process. Comparing 10 men who had OSA with 5 healthy controls, Luboshitzky and others found that the OSA patients had lower mean luteinizing hormone (LH) and testosterone levels, secreting less LH and testosterone during sleep.[38] In a study of testosterone levels in 1312 men selected from a study of osteoporosis in elderly men, Barrett-Connor and colleagues found that men in the lowest testosterone quartile had higher AHI ($p = 0.02$) and spent more of their sleep time with oxygen saturation less than 90% ($p = 0.0001$).[39]

In a rodent model of intermittent hypoxemia during daylight hours analogous to that experienced by patients with OSA, Soukhova-O'Hare evaluated the impact of chronic intermittent hypoxemia on sexual function and erectile activity, particularly changes in endothelial nitric oxide, which mediates penile erection, and phosphodiesterase enzyme type 5, which metabolizes cGMP into 5´GMP inhibiting the erectile process.[40] In addition to finding ED in mice exposed to chronic hypoxemia, this team also documented that chronic intermittent hypoxemia produced a 55% decrease in the number of daily spontaneous erections that were reversed after 6 weeks of recovery in normoxia. Associated with this decrease in erectile functioning was reduced endothelial nitric oxide expression. They also found reduced sexual drive and mating activity. These effects were not testosterone-dependent, and no disruptions of testicular anatomy, including the number and appearance of Leidig and Sertoli cells, were found. Thus, the findings from these studies suggest that the effect of OSA on intimate and sexual behaviors may be multifactorial.

We found, as have others, that intimate and sexual activity recovers following CPAP treatment. A clinically meaningful change was found in our sample following 3 months of treatment and increased across disease severity groups. Similarly, Bradshaw and associates demonstrated significant increased in the Intimate and Sexual Relationships score in participants who received 2 weeks of CPAP treatment.[16] Akashiba and colleagues reported that in patients with severe OSA, following 6 weeks CPAP treatment, Intimacy and Sexual Relationships scores improved 2.59 points.[41] Using other outcome measures, Margel and colleagues[3] documented improvement on the International Index of Erectile Function-5 in 20% of patients with OSA after being treated with CPAP for more than a year, and Goncalves and colleagues[7] reported ED resolved in 13 of 17 OSA patients treated for one month with CPAP.

Not all studies show improvement in intimate and sexual functioning associated with CPAP therapy. Margel and coworkers found that 62% of their participants showed no change in the International Index of Erectile Function-5 after CPAP treatment, with 18% having a worse score.[3] Massie and Hart found that 3 weeks of CPAP treatment did not lead to significant change in Intimacy and Sexual Relationships among 39 people with severe OSA.[18] A trial of 8 weeks of CPAP in 80 people with mild OSA also did not demonstrate significant improvement.[32] Zhuraviev and colleagues found no improvement in sexual function of five men with both OSA and ED following 6 months of CPAP treatment, even though their sleep normalized,[8] and Hoekema and colleagues found non-significant improvements in self-reported erectile dysfunction and sexual dissatisfaction in 27 men with OSA following 2 months CPAP treatment.[42] It may be that compared to other outcomes, such as daytime sleepiness, changes in intimacy and sexual relationships may take longer to be realized or noticed, given its social nature. Differences in level of disease severity, degree of treatment experience, and presence of a type II error, may be other reasons for the inconsistent results. Larger controlled trials with optimal levels of adherence are needed to more adequately understand the contribution of CPAP treatment to the recovery of intimacy and sexual activity in this population.

Despite significant improvement following 3 months of CPAP therapy, with decreased proportions of the sample reporting difficulty in desire, intimacy, arousal, and orgasm, we also found that the mean Intimacy and Sexual Relationships subscale score for the sample was not at normal levels. We have previously reported residual impairment in optimally adherent patients.[11] Indeed, 43% of OSA patients using CPAP for 3 months still experienced reduced daily functioning even with nightly use > 6 hours.[11] In a sample of optimally adherent CPAP-treated patients with residual daytime sleepiness and impaired intimate and sexual functioning, significant improvement occurred with modafinil treatment.[19]

CONCLUSION

The present study is the only large multicenter investigation to have explored OSA patients' subjective assessments of their intimate and sexual relationships both before and after treatment. The results of this study demonstrate again that OSA affects patients' waking function, and emphasizes the benefits of CPAP treatment in an area of function not often investigated within the context of sleepiness. It points out the necessity of evaluating all areas of function when assessing a person's complaints of excessive daytime sleepiness and including this important area of functioning in the assessment of treatment effectiveness.

REFERENCES

1. Wilmoth MC, ed. Sexuality in chronic illness: impact and interventions. Boston: Jones and Bartlett; 2002.
2. Guilleminault C, Eldridge FL, Tilkian A. sleep apnea syndrome due to upper airway obstruction: a review of 25 cases. *Arch Intern Med.* 1977;137:4.
3. Margel D, Tal R, Livne PM, Pillar G. Predictors of erectile function improvement in obstructive sleep apnea patients with long-term CPAP treatment. *Int J Impot Res* 2005;17:186-90.
4. Arruda-Olson AM, Olson LJ, Nehra A, Somers VK. Sleep apnea and cardiovascular disease. Implications for understanding erectile dysfunction. *Herz* 2003;28:298-303.

5. Teloken PE, Smith EB, Lodowsky C, Freedom T, Mulhall JP. Defining association between sleep apnea syndrome and erectile dysfunction. *Urology* 2006;67:1033-7.
6. Watson R, Greenberg G, Bakos L. Sleep apnea and depression. *Sleep Res* 1987;16:6.
7. Goncalves MA, Guilleminault C, Ramos E, Palha A, Paiva T. Erectile dysfunction, obstructive sleep apnea syndrome and nasal CPAP treatment. *Sleep Med* 2005;6:333-9.
8. Zhuravlev VN, Frank MA, Gomzhin AI. Sexual functions of men with obstructive sleep apnoea syndrome and hypogonadism may improve upon testosterone administration: a pilot study. *Andrologia* 2009;41:193-5.
9. Luboshitzky R, Lavie L, Shen-Orr Z, Lavie P. Pituitary-gonadal function in men with obstructive sleep apnea. The effect of continuous positive airways pressure treatment. *Neuro Endocrinol Lett* 2003;24:463-7.
10. Weaver TE, Laizner AM, Evans LK, et al. An instrument to measure functional status outcomes for disorders of excessive sleepiness. *Sleep* 1997;20:835-43.
11. Weaver TE, Maislin G, Dinges DF, et al. Relationship between hours of CPAP use and achieving normal levels of sleepiness and daily functioning. *Sleep* 2007;30:711-19.
12. Chasens ER, Pack AI, Maislin G, Dinges DF, Weaver TE. Claustrophobia and adherence to CPAP treatment. *West J Nurs Res* 2005;27:307-21.
13. American Academy of Sleep Medicine. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine task force. Sleep 1999;22:667-89.
14. Rechtschaffen A, Kales A. A manual for standardized terminology: techniques and scoring system for sleep stages of human subjects. Los Angeles: UCLA Brain Information Service/Brain Research Institute; 1968.
15. Blanco J, Zamarron C, Abeleira Pazos MT, Lamela C, Suarez Quintanilla D. Prospective evaluation of an oral appliance in the treatment of obstructive sleep apnea syndrome. *Sleep Breath* 2005;9:20-5.
16. Bradshaw DA, Ruff GA, Murphy DP. An oral hypnotic medication does not improve continuous positive airway pressure compliance in men with obstructive sleep apnea. *Chest* 2006;130:1369-76.
17. Faccenda JF, Mackay TW, Boon NA, Douglas NJ. Randomized placebo-controlled trial of continuous positive airway pressure on blood pressure in the sleep apnea-hypopnea syndrome. *Am J Respir Crit Care Med* 2001;163:344-8.
18. Massie CA, Hart RW. Clinical outcomes related to interface type in patients with obstructive sleep apnea/hypopnea syndrome who are using continuous positive airway pressure. *Chest* 2003;123:1112-8.
19. Schwartz JRL, Hirshkowitz M, Erman MK, Schmidt-Nowara W. Modafinil as adjunct therapy for daytime sleepiness in obstructive sleep apnea: a 12-week, open-label study. *Chest* 2003;124:2192-9.
20. Carskadon MA, Dement WC. The multiple sleep latency test: what does it measure? *Sleep* 1982;5:Suppl 2:S67-72.
21. Carskadon MA, Dement WC, Mitler MM, Roth T, Westbrook PR, Keenan S. Guidelines for the multiple sleep latency test (MSLT): a standard measure of sleepiness. *Sleep* 1986;9:519-24.
22. Mitler MM, Carskadon MA, Hirshkowitz M. Evaluating sleepiness. In: Kryger M, Roth T, Dement WC, eds. Principles and practice of sleep medicine, 4th ed. Philadelphia: Elsevier Saunders 2005: 1417-23.
23. Seneviratne U, Puvanendran K. Excessive daytime sleepiness in obstructive sleep apnea: prevalence, severity, and predictors. *Sleep Med* 2004;5:339-43.
24. Sforza E, Lugaresi E. Daytime sleepiness and nasal continuous positive airway pressure therapy in obstructive sleep apnea syndrome patients: effects of chronic treatment and 1-night therapy withdrawal. *Sleep* 1995;18:195-201.
25. Johns MW. Daytime sleepiness, snoring, and obstructive sleep apnea. the Epworth Sleepiness Scale. *Chest* 1993;103:30-36.
26. Johns MW. Sensitivity and specificity of the multiple sleep latency test (MSLT), the maintenance of wakefulness test and the Epworth Sleepiness Scale: failure of the MSLT as a gold standard. *J Sleep Res* 2000;9:5-11.
27. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep* 1991;14:540-545.
28. Hardinge FM, Pitson DJ, Stradling JR. Use of the Epworth Sleepiness Scale to demonstrate response to treatment with nasal continuous positive airways pressure in patients with obstructive sleep apnoea. *Respir Med* 1995;89:617-20.
29. Johns MW. Sleepiness in different situations measured by the Epworth Sleepiness Scale. *Sleep* 1994;17:703-10.
30. Kribbs NB, Pack AI, Kline LR, et al. Objective measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. *Am Rev Respir Dis* 1993;147:887-95.
31. Cohen J. Statistical power analysis for the behavioral sciences. 2nd ed. New York: Academic Press; 1977.
32. Barnes M, McEvoy RD, Banks S, et al. Efficacy of positive airway pressure and oral appliance in mild to moderate obstructive sleep apnea. *Am J Respir Crit Care Med* 2004;170:656-64.
33. Karkoulias K, Perimenis P, Charokopos N, et al. Does CPAP therapy improve erectile dysfunction in patients with obstructive sleep apnea syndrome? *Clinica Terapeutica* 2007;158:515-8.
34. Shin HW, Rha YC, Han DH, et al. Erectile dysfunction and disease-specific quality of life in patients with obstructive sleep apnea. *Int J Impot Res* 2008;20:549-53.
35. Schmidt HS, Wise HA 2nd. Significance of impaired penile tumescence and associated polysomnographic abnormalities in the impotent patient. *J Urol* 1981;126:348-52.
36. Hirshkowitz M, Karacan I, Arcasoy MO, Acik G, Narter EM, Williams RL. Prevalence of sleep apnea in men with erectile dysfunction. *Urology* 1990;36:232-4.
37. Margel D, Cohen M, Livne PM, Pillar G. Severe, but not mild, obstructive sleep apnea syndrome is associated with erectile dysfunction. *Urology* 2004;63:545-9.
38. Luboshitzky R, Aviv A, Hefetz A, et al. Decreased pituitary-gonadal secretion in men with obstructive sleep apnea. *J Clin Endocrinol Metab* 2002;87:3394-8.
39. Barrett-Connor E, Dam TT, Stone K, Harrison SL, Redline S, Orwoll E; Osteoporotic Fractures in Men Study Group. The association of testosterone levels with overall sleep quality, sleep architecture, and sleep-disordered breathing. *J Clin Endocrinol Metab* 2008;93:2602-9.
40. Soukhova-O'Hare GK, Shah ZA, Lei Z, Nozdrachev AD, Rao CV, Gozal D. Erectile dysfunction in a murine model of sleep apnea. *Am J Respir Crit Care Med* 2008;178:644-50.
41. Akashiba T, Kawahara S, Akahoshi T, et al. Relationship between quality of life and mood or depression in patients with severe obstructive sleep apnea syndrome. *Chest* 2002;122:861-5.
42. Hoekema A, Stel A, Stegenga B. Sexual function and obstructive sleep apnea-hypopnea: a randomized clinical trial evaluating the effects of oral-appliance and continuous positive airway pressure therapy. *J Sexual Med* 2007;4:1153-62.

ACKNOWLEDGMENTS

We would like to thank members of the Multisite Study Group, Site Principal Investigtors: Thomas J. Bloxham, M.D. Sleep Medicine Center of Kansas West, Wichita, KS, USA; Charles F. P. George, M.D. Univ. of Western Ontario, London, ON, Canada; Harly Greenberg, North Shore Long Island Jewish Medical Center, New Hyde Park, NY, USA; Gihan A. Kader, M.D., St. Luke's Hospital, Chesterfield, MO, USA; Mark W. Mahowald, M.D., Hennepin County Medical Center, Minneapolis, MN, USA; Joel B. Younger, M.D., Bon Secours Holy Family Regional Health System, Altoona, PA; and the Multisite Study Group Site Research Coordinators: Sandra Halko, University of Western Ontario; Bernadette Krug, Bons Secours Holy Family Regional Health System, Angie Randazzo, Ph.D., St. Lukes Medical Center; Deborah Sturgeon, Sleep Medicine Center of Kansas; Constance Ullevig, Hennepin County Medical Center; and research team at the University of Pennsylvania School of Nursing: David Dinges, Ph.D. and Sharon Hurley.

This research was supported by grants from the National Institutes of Health RR00040 (GCRC), National Institutes of Health National Heart Lung and Blood Institute P50-HL60287 (T. Weaver, A. Pack, and Greg Maislin) and HL53991 (T. Weaver), and by Philips Respironics, Nellcor Puritan Bennett Inc., DeVilbiss Health Care Inc., Healthdyne Technologies, Inc.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication July, 2009
Submitted in final revised form January, 2010
Accepted for publication February, 2010
Address correspondence to: Terri E. Weaver, Ph.D., R.N., FAAN, University of Pennsylvania School of Nursing, Claire Fagin Hall, 418 Curie Boulevard, Philadelphia, PA 19104-4217, Tel: (215) 898-2992; Fax: (215) 573-2249; E-mail: tew@nursing.upenn.edu

DISCLOSURE STATEMENT

This was not an industry supported study. Dr. Weaver has received research support from Respironics Respiratory and Sleep Research Foundation and Cephalon; has consulted for Apnex Medical and Cephalon; has FOSQ license agreements with Sanofi-Aventis, Merck, N.V. Organon, Apnex Medical, Ventus Medical, GlaxoSmith-Kline, and Philips Respironics; and has received the use of research equipment from Respironics and Protech. The other authors have indicated no financial conflicts of interest.