ORIGINAL ARTICLE

Effect of Continuous Positive Airway Pressure Treatment on Health-Related Quality of Life and Sleepiness in High Cardiovascular Risk Individuals With Sleep Apnea: Best Apnea Interventions for Research (BestAIR) Trial

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Study Objectives: The long-term effect of continuous positive airway pressure (CPAP) on health-related quality of life (HRQOL) in patients with high cardiovascular disease risk and obstructive sleep apnea (OSA) without severe sleepiness is uncertain. We aimed to determine the effect of CPAP treatment on HRQOL in individuals with moderate or severe OSA and cardiovascular disease (CVD) or multiple CVD risk factors without severe sleepiness.

Methods: In this randomized, controlled, parallel group study, 169 participants were assigned to treatment with CPAP or the control group (conservative medical therapy [CMT] or CMT with sham CPAP). Analyses were based on an intention-to-treat approach. Linear mixed effect models were fitted to compare the changes in the Medical Outcomes Study Short Form-36 (SF-36) and in subjective sleepiness (Epworth Sleepiness Scale [ESS]) between groups from baseline to the average of 6- and 12-month measurements.

Results: CPAP improved several domains of HRQOL including bodily pain (treatment effect 9.7 [95% confidence interval, CI 3.9 to 15.4]; p = .001), vitality (5.7 [95% CI 1.5 to 9.9]; p = .008), general health (8.2 [95% CI 3.7 to 12.7]; p < .001), physical functioning (5.5 [95% CI 1.1 to 10.0]; p = .016), and the physical health summary score (3.3 [95% CI 1.4 to 5.3]; p = .001). CPAP also resulted in less daytime sleepiness (mean change in ESS −1.0 point [95% CI −2.0 to −0.0]; p = .040).

Conclusions: In patients with moderate–severe OSA at high risk of cardiovascular events and without severe sleepiness, CPAP improved daytime sleepiness and multiple domains of HRQOL over 6 to 12 months of follow-up, with the largest improvement observed for bodily pain.

Keywords: sleep apnea, CPAP, quality of life, clinical trial, sleepiness.

Statement of Significance

Obstructive sleep apnea (OSA) and cardiovascular disease (CVD) each impairs health-related quality of life (HRQOL). However, the effectiveness of CPAP in improving outcomes in patients without severe sleepiness is debated. We evaluated the effect of CPAP on long-term changes in HRQOL in high-risk patients with moderate to severe OSA and without severe sleepiness in one of the largest clinical trials to date. Despite only modest CPAP compliance and exclusion of participants with severe daytime sleepiness, CPAP improved several domains of HRQOL, including bodily pain and daytime sleepiness, compared to the control arm. Our findings may motivate clinicians and patients when considering treatment for OSA and may also provide data for policy makers to reexamine the current insurance criteria for CPAP use.

INTRODUCTION

Cardiovascular disease (CVD) remains the leading cause of death in the United States and is a significant cause of morbidity and health-care expenditures.[1] Obstructive sleep apnea (OSA) affects an estimated 26% of the general population, and its prevalence among individuals with CVD is even higher.[2–4] Increasing evidence suggests that OSA is an important modifiable risk factor for CVD, including stroke, coronary artery disease, heart failure, and atrial fibrillation.[5–8] OSA is also associated with excessive daytime sleepiness, an increased risk of injuries from motor vehicle crashes and industrial injuries, and impaired health-related quality of life (HRQOL).[9–14]

Continuous positive airway pressure (CPAP) is currently the treatment of choice for OSA. CPAP has been shown to improve both subjective and objective measures of sleepiness.[9] A number of cohort studies and randomized controlled trials have also evaluated the impact of CPAP on HRQOL in OSA patients although not all studies have shown an improvement in HRQOL with treatment.[15–17] Most of these studies had relatively short study durations (3 months or less), and only a few studies have evaluated the impact of CPAP on HRQOL in patients with OSA and CVD.[18–21] Mansfield et al. found that 3 months of CPAP therapy improved several domains of HRQOL in patients with heart failure.[18] In comparison, a similar effect was not observed in a 6-week randomized controlled crossover study comparing autotitrating CPAP to sham CPAP in patients with stable symptomatic heart failure and OSA.[19] More recently, McEvoy et al. showed improvements in HRQOL with CPAP use in patients with coronary or cerebrovascular disease.[21] Given the limited and inconsistent data in patients with CVD and the short duration of follow-up in many of these studies, we aimed to investigate the impact of 6 to 12 months of CPAP therapy compared to control on key measures of HRQOL, using data from a randomized control trial.

The Best Apnea Interventions for Research (BestAIR) trial was designed as a planning study to assess key feasibility and optimal study design features in the context of a cardiovascular intervention trial in OSA.[22,23] A secondary, prespecified objective was to compare the changes in HRQOL as assessed by the Medical Outcomes Study Short Form-36 questionnaire

(SF-36) and subjective daytime sleepiness as measured by the Epworth Sleepiness Scale (ESS) between participants with cardiovascular comorbidity randomized to CPAP versus control. We hypothesized that in this group of high-risk patients without severe sleepiness CPAP treatment would significantly improve HRQOL and reduce sleepiness compared to the control group.

METHODS

The rationale and protocol for the BestAIR study has been previously reported.[22,23] Detailed methods are reported in the Online Supplement.

Study Design

BestAIR was a randomized, parallel-group clinical trial with blinded assessment of outcomes. Participants were recruited from outpatient clinics from three medical centers in Boston, Massachusetts. The four treatment arms were: conservative medical therapy (CMT), CMT + sham CPAP, CMT + CPAP, and CMT + CPAP + motivational enhancement (ME). CMT consisted of education on sleep hygiene, healthy lifestyle, and nasal dilator strips for use during sleep. ME consisted of a behavioral intervention to improve CPAP adherence, as described previously.[24] Owing to the longer-than-expected time to complete enrollment, patients randomized after January 2013 were only followed for 6 months.[22,23] The study was approved by the Institutional Review Board at each participating center. All participants provided written informed consent.

Eligibility criteria were used to identify those at high risk of cardiovascular events as may be recruited in future large-scale trials. Eligible subjects had an apnea–hypopnea index (AHI) 4% ≥10 events/hour or AHI 3% ≥15 events/hour and were either aged 45 to 75 years with established CVD (coronary artery disease, ischemic stroke, or diabetes) or aged 55 to 75 years with 3 or more CVD risk factors (male, body mass index [BMI] ≥30 kg/m^2, hypertension, dyslipidemia, or ≥10 pack-years of smoking). Major exclusion criteria were a cardiovascular event <4 months prior to enrollment, prior CPAP use, and excessive sleepiness (ESS score >14 or report falling asleep while driving within the past 2 years). Sleep studies to determine eligibility were completed either as part of routine care (in-laboratory polysomnography) or administered by the study investigators (Embletta Gold or X100, Embla, Ontario, Canada). Participants who had not undergone an in-lab CPAP titration used an auto-adjusting device for a minimum of 5 days to identify the pressure for ongoing fixed CPAP pressure.

Participants were asked to use a nasal CPAP mask open to atmosphere without a CPAP device and complete a diary during a 2-week run-in period. Those who reported wearing the mask for a majority of nights and were willing to continue in the study were randomized in a 1:1:1:1 ratio to one of the 4 study arms described earlier using sequence generated off-site with a block size of 4, based on 3 stratification factors: diagnostic type (full- or split-night titration), site, and CVD status (established or risk factors).

Outcomes

The key patient reported outcomes in the current analysis were HRQOL as assessed by the Medical Outcomes Study Short Form-36 questionnaire (SF-36) and subjective daytime sleepiness as measured by the ESS. Outcomes were measured at baseline, 6, and 12 months. The SF-36 is a generic HRQOL instrument that has been shown to have excellent reliability and validity in patients with OSA.[25] The questionnaire measures 8 domains of health: physical functioning, role limitation due to physical problems, bodily pain, general health, vitality/energy, social functioning, role limitation due to emotional problems, and mental health. Scores from the 8 domains and the 2 derived summary scores (physical health and mental health summary scores) are standardized such that a mean score of 50 with an SD of 10 would reflect the mean score in the U.S. general population. The ESS is a self-administered 8-item questionnaire that is the most widely used index to measure subjective sleepiness in OSA. Scores range from 0 to 24, with a cutoff of >10 representing clinically significant sleepiness.[26,27]

Statistical Analysis

Analyses reported in this article compare data from the combined control arms (CMT and CMT + sham CPAP) with the combined CPAP arms (CMT + CPAP and CMT + CPAP + ME) as specified in the protocol, to provide greater power to detect any CPAP effect than pairwise comparisons among individual subarms. Between-group cross-sectional comparisons were made using Fisher's exact tests for categorical data and 2-sample *t*-tests or Wilcoxon rank-sum tests for continuous data depending on whether or not substantial departure from normality in data distributions was observed. The primary analysis was based on an intent-to-treat approach. Mixed effects linear regression models were used to compare the longitudinal profiles between the combined CPAP and control groups. Observational time (0, 6, and 12 months) was modeled as a categorical variable. Stratification factors were included as covariates. To further explore the influence of adherence on study findings, we performed a sensitivity analysis comparing the differences in changes in outcomes from baseline to 6 months by adherence status among CPAP users (adherence defined using Medicare definition of ≥ 4 hours per night for 70% of days over 6 months).

Mixed effects linear regression analyses were performed using R version 3 or higher, with functions from the "lme4" package.[28] All other analyses were performed using SAS version 9.4 (SAS Institute, Inc., Cary, NC). Two-sided p values <.05 were considered statistically significant.

RESULTS

Study Population

Screening began in February 2011, and randomization took place from April 2011 to August 2013, with the final follow-up visit completed in March 2014.[22] A total of 475 patients completed screening sleep studies (in-laboratory polysomnography n = 36, home sleep apnea test n = 439), with 227 patients identified with moderate or severe OSA and completing the run-in phase (Figure 1). Of the 169 participants randomized, 108 were recruited before January 2013 and were followed for 12 months, and the remainder who were recruited later was followed for 6 months.

Baseline characteristics of the participants were comparable for the CPAP and control groups (Table 1). The majority

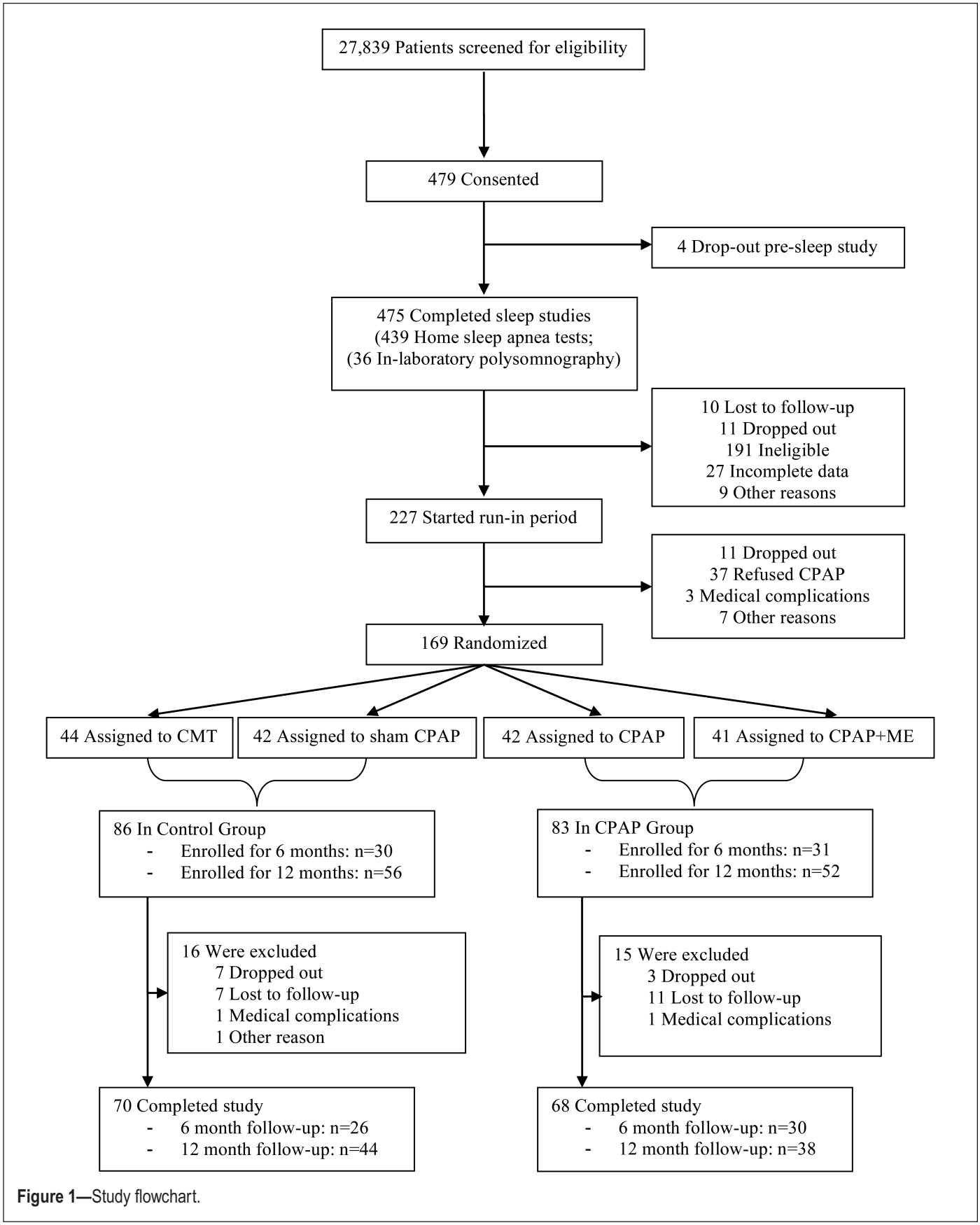

Figure 1—Study flowchart.

(85.2%) of the participants had hypertension, 34.3% had coronary artery disease, and 37.3% had diabetes. Mean (SD) age was 63.8 (7.3) years, and 65.1% were men. Mean BMI and AHI were 31.7 (5.9) kg/m^2 and 29.2 (16.6) events/hr, respectively. At 6- and 12 months, the mean (SD) CPAP use was 3.82 (2.86) and 3.44 (2.99) hours per night, respectively.

Table 1—Baseline characteristics of the study population[a].

	All patients, *N* = 169	Control group, *N* = 86	CPAP group, *N* = 83
Age (years)	63.8 (7.3)	63.7 (6.9)	63.8 (7.8)
Male sex, n (%)	110 (65.1)	55 (64.0)	55 (66.3)
Race/ethnicity, n (%)[b]			
White	151 (89.3)	77 (89.5)	74 (89.2)
Black	11 (6.5)	6 (7.0)	5 (6.0)
Hispanic	6 (3.6)	2 (2.3)	4 (4.8)
Other	7 (4.1)	3 (3.5)	4 (4.8)
Education, n (%)[b]			
<High school	2 (1.2)	1 (1.2)	1 (1.2)
High school graduate	57 (33.7)	24 (27.9)	33 (39.8)
Bachelors or higher	110 (65.1)	61 (70.9)	49 (59.0)
Body mass index (kg/m^2)[c]	31.7 (5.9)	32.3 (6.5)	31.1 (5.2)
Neck circumference, cm	41.5 (3.9)	41.9 (3.9)	41.1 (4.0)
Smoking history, n (%)			
Current	13 (7.7)	5 (5.8)	8 (9.6)
Former	85 (50.3)	40 (46.5)	45 (54.2)
Never	71 (42.0)	41 (47.7)	30 (36.2)
History of hypertension, n (%)	144 (85.2)	73 (84.9)	71 (85.5)
History of CVD or diabetes, n (%)			
Coronary artery disease	58 (34.3)	28 (32.6)	30 (36.1)
Diabetes	63 (37.3)	35 (40.7)	28 (33.7)
Stroke	4 (2.4)	2 (2.3)	2 (2.4)
AHI, events/hr			
Mean (SD)	29.2 (16.6)	32.0 (19.1)	26.2 (12.9)
Median (IQR)	23.9 (17.4–33.4)	26.1 (18.2–37.4)	22.7 (16.6–31.4)
Sleep time with SpO_2 <90%, %			
Mean (SD)	9.2 (14.3)	9.9 (15.2)	8.5 (13.3)
Median (IQR)	3.5 (0.9–11.0)	3.7 (1.2–12.0)	3.2 (0.6–10.8)

Abbreviations: ACE, angiotensin-converting enzyme; AHI, apnea–hypopnea index; CVD, cardiovascular disease; IQR, interquartile range; CPAP, continuous positive airway pressure; SD, standard deviation; SpO_2, oxygen saturation measured by pulse oximetry.
[a]Data presented as mean (SD) unless otherwise specified.
[b]Education, race and ethnic group were self-reported.
[c]The body-mass index is the weight in kilograms divided by the square of the height in meters.

Outcomes

Baseline scores in all 8 domains of the SF-36 and the 2 component summary scores were similar between the CPAP and the control groups (Table 2). Overall, participants had above population average scores, with some scores higher than those reported in prior studies of OSA patients.[18,29,30] No significant differences in SF-36 scores or ESS were observed within the 2 CPAP or within the 2 control subarms at baseline or at follow-up (results not shown). At the end of the study, CPAP improved several domains of the SF-36 compared to the control group. The greatest effect was observed for bodily pain, with an estimated treatment effect of 9.7 points (95% confidence interval [CI] 3.9 to 15.4; p = .001) comparing the CPAP to control group. Significant improvements were also seen for vitality (estimated treatment effect 5.7 points [95% CI 1.5 to 9.9]; p = .008), general health (8.2 points [95% CI 3.7 to 12.7]; p < .001), and physical functioning 5.5 points [95% CI 1.1 to 10.0]; p = .016). The physical health summary score improved by 3.3 points (95% CI 1.4 to 5.3; p = .001), but no significant improvement in the emotional or social role functioning, mental health subscale, or summary score was noted (all p > .05).

The baseline ESS was similar between the CPAP and the control groups (Table 2). Despite the exclusion of participants with severe daytime sleepiness (mean ESS 8.3 [SD 4.5]), CPAP led to greater

Table 2—Patient-reported outcomes (Epworth Sleepiness Scale and SF-36).

	Control group			CPAP Group			Treatment Effect (95% CI)	p value
	Baseline	6-month follow-up	12-month follow-up	Baseline	6-month follow-up	12-month follow-up		
ESS	8.5 (4.5)	7.6 (4.2)	7.7 (4.0)	8.0 (4.5)	6.2 (3.8)	6.0 (4.0)	−1.0 (−2.0 to −0.0)	.040
ESS >10, n (%)	24 (27.9)	16 (23.5)	12 (27.9)	20 (24.1)	10 (14.7)	5 (15.2)	–	–
SF-36 Scales								
Vitality	56.0 (19.8)	58.8 (21.6)	60.9 (18.1)	56.0 (20.0)	63.5 (21.2)	63.5 (18.8)	5.7 (1.5 to 9.9)	.008
General Health	57.9 (20.1)	57.5 (21.9)	57.7 (21.2)	56.5 (21.8)	61.8 (21.8)	61.5 (17.8)	8.2 (3.7 to 12.7)	<.001
Physical Functioning	69.4 (25.5)	67.6 (26.4)	69.6 (23.4)	73.2 (24.9)	75.5 (23.1)	76.4 (21.7)	5.5 (1.1 to 10.0)	.016
Bodily Pain	64.7 (23.9)	61.8 (25.7)	56.3 (22.3)	62.3 (25.0)	65.7 (23.0)	68.1 (22.8)	9.7 (3.9 to 15.4)	.001
Emotional Role Functioning	79.8 (24.7)	84.4 (21.6)	84.8 (24.6)	81.3 (24.6)	84.8 (21.4)	83.1 (23.8)	−0.4 (−6.5to 5.8)	.904
Physical Role Functioning	72.5 (26.1)	70.2 (25.8)	76.2 (23.5)	73.8 (26.0)	73.5 (27.2)	73.1 (30.1)	1.1 (−5.0 to 7.2)	.718
Social Role Functioning	80.9 (22.5)	80.9 (22.7)	86.3 (20.6)	79.7 (27.0)	81.5 (27.0)	83.0 (25.6)	1.0 (−3.9 to 5.9)	.682
Mental Health	75.7 (16.6)	76.9 (16.2)	80.9 (17.9)	74.8 (19.8)	77.7 (18.0)	76.2 (14.8)	1.2 (−2.6 to 5.0)	.541
Physical Health Summary Score	44.3 (9.6)	42.7 (10.1)	42.9 (8.7)	45.2 (9.4)	45.6 (9.7)	46.2 (9.6)	3.4 (1.4 to 5.3)	<.001
Mental Health Summary Score	50.3 (9.6)	52.2 (9.7)	54.0 (11.5)	49.7 (12.0)	52.2 (11.3)	51.3 (9.7)	0.2 (−2.1 to 2.4)	.869

Abbreviations: CI, confidence interval; ESS, Epworth Sleepiness Scale; SF-36, Medical Outcomes Study Short Form 36 Health Survey.
[a]Data presented as mean (SD) unless otherwise specified. *N* = 86 subjects in the control group and *N* = 83 subjects in the CPAP group had one or more data points and were included in the analysis of ESS. *N* = 85 in the control group and *N* = 81 in the CPAP group had one or more data points and were included in the analysis of the various SF-36 scales.

reduction in the ESS score (estimated treatment effect −1.0 point [95% CI −2.0 to −0.0]; p = .040 compared to the control group.

Over the first 6 months, the mean (SD) CPAP use was 3.8 (2.9) hours. Only 51.8% of participants in the treatment group used CPAP for an average of ≥4 hr/night over 6 months, and only 43.4% of participants were adherent by Medicare definition (≥ 4 hours per night for 70% of days). The degree of improvement in HRQOL did not correlate with CPAP compliance, but higher average nightly CPAP use was associated with more improvement in ESS scores (Pearson correlation $r = -0.29$, p = .018; Supplemental Table 1). Changes in HRQOL indices over 6 months in those who met or did not meet Medicare adherence criteria are shown in Supplemental Table 2. The magnitude of changes in ESS and HRQOL were not statistically different between individuals characterized by low or higher CPAP adherence; however, the power to detect differences was low.

DISCUSSION

The present study is one of the largest long-term randomized controlled trials to examine the effect of CPAP on HRQOL in patients with moderate or severe OSA at high risk of CVD. In this group of participants without severe sleepiness and with generally above-average HRQOL at baseline, CPAP led to significant 6- to 12-month improvements in several domains of HRQOL, with the greatest improvement seen in bodily pain. CPAP also led to a modest but significant improvement in subjective daytime sleepiness. These improvements were seen despite an average CPAP use of less than 4 hours per night.

Patient-reported outcomes such as symptoms and HRQOL are increasingly recognized as important components of disease management to improve patient well-being and are recommended to be included as secondary end points in clinical trials.[30] CVD is known to impair HRQOL.[31] Sleep apnea, even if mild, has also been associated with impaired HRQOL.[10,11,29,32] Thus, patients with both OSA and CVD may be at particular risk of poorer HRQOL. CPAP pneumatically stents open the airway, thereby abolishing apneas and hypopneas and reduces sleep apnea-related arousals and sleep fragmentation. Several previous studies have demonstrated that CPAP improves HRQOL in symptomatic patients with OSA.[29,33] However, the impact of CPAP on these outcomes in nonsleepy patients has been debated. In particular, Barbé et al. found that 6 weeks of treatment with CPAP compared to sham CPAP in nonsleepy patients with severe OSA did not improve HRQOL as measured by the SF-36 and the Functional Outcomes of Sleep Quality questionnaires.[34] In contrast, in our study of patients without severe sleepiness (mean ESS 8.3), significant improvements in several domains of SF-36 were seen. The difference between our study and Barbé et al. may be explained by the longer duration of treatment in our study (6- to 12 months vs. 6 weeks) and

the inclusion of patients with significant cardiovascular comorbidity in our study.

Prior studies in patients with heart disease have not consistently demonstrated a benefit of CPAP on HRQOL.[17–21] In a study of 55 patients with chronic heart failure, Mansfield and colleagues showed that 3 months of CPAP therapy significantly improved the physical role, vitality, social functioning, and mental health domains of the SF-36, compared to control.[18] In a separate study, Smith et al did not show an improvement in HRQOL with 6 weeks of autotitrating CPAP compared to sham CPAP in patients with stable symptomatic heart failure and OSA.[19] Similar to the Mansfield study, we showed that CPAP improved vitality. However, no significant improvement in physical role, social functioning, and mental health were noted in our study. One explanation may be that our participants did not have heart failure and tended to have high scores in the physical role, social functioning, and mental health domains at baseline (>70). More recently, improvements in HRQOL with CPAP use were also reported in an international trial of patients with moderate-to-severe OSA and coronary or cerebrovascular disease.[21] However, a direct comparison of our study results to that of the SAVE trial is not possible, given the latter did not report a detailed analysis of the HRQOL measures.

Unlike previous studies, which did not show significant improvements in pain domains of HRQOL with CPAP use,[18,35-37] we observed the greatest improvement in the bodily pain scale of the SF-36 (mean treatment effect 9.7, 95% CI 3.9 to 15.4, p = .001). Chronic pain has become the most common reason for outpatient medical visits, and its treatment has led to a dramatic rise in habitual narcotic use.[38] Studies show that patients with chronic pain have diminished HRQOL and are more likely to develop psychological disorders, cognitive impairment, and sexual dysfunction.[39] Chronic pain syndromes also have significant cost implications, both in terms of increased health-care costs and lost productivity with increased absenteeism from work and reduced performance while at work.[40] There is emerging data indicating the close association between sleep quality, duration, and pain, with sleep and pain sharing common neurological pathways.[41] New data suggest that sleep apnea may be associated with chronic pain syndromes.[42] Our results point to the possibility that improvement in sleep apnea may improve pain and suggest that sleep apnea may potentially be an important therapeutic target in the management of chronic pain syndromes.

Subjective sleepiness is absent in many individuals with significant sleep disordered breathing.[43] In particular, many patients with CVD and sleep apnea do not report excessive daytime sleepiness.[20,44] Therefore, our findings of improved HRQOL with CPAP use in a group of participants with ESS scores in the normal to mildly elevated range have important clinical implications. These findings suggest that treatment benefits occur even in the absence of a high ESS score. Furthermore, an expectation of improved HRQOL may motivate patients to undergo testing and treatment for sleep apnea. Given that symptom relief with CPAP treatment has been consistently shown among the strongest predictors of CPAP compliance,[45,46] improved HRQOL may promote better CPAP adherence. The larger changes in HRQOL measures compared to change in the ESS also suggest the potential utility in tracking change in HRQOL with treatment.

The 6- to 12-month treatment duration in our study is longer than most previous randomized controlled trials, which were generally 3 months or shorter in duration. Our results indicate that improvements in HRQOL seen with CPAP use are sustained in this patient population, even in a group of patients with an average duration of use of less than 4 hours per night. Long-term CPAP coverage is currently limited by the Center for Medicare and Medicaid Services to those who demonstrate adherence and subjective benefit during an initial 90-day trial period. Adherence is defined as CPAP use ≥4 hours per night for 70% of days within a consecutive 30-day period. Of note, although change in sleepiness was associated with level of CPAP adherence, we observed no significant associations with average hours of CPAP use and change in the HRQOL measures. Research by Weaver et al. suggest that there are different dose–response associations between CPAP use and different outcomes.[47] Additional research is needed to further investigate the nature of the dose–response relationship for CPAP and relevant outcomes. Given the importance of HRQOL to patients, these data suggest that more liberal CPAP coverage criteria should be considered.

Our study had several limitations. A potential limitation was the use of a generic HRQOL instrument. While the use of disease-specific instruments such as the Functional Outcomes of Sleep Questionnaire and the Sleep Apnea Quality of Life Index may detect more subtle effects of CPAP on HRQOL, the use of SF-36 allows for cross-study and cross-population comparisons.[10] The SF-36 is also one of the most frequently used HRQOL instrument in sleep research.[10] Although we did not have 12-month follow-up data for all of our study participants, the use of mixed effects linear regression models allowed use of all data points at 6- and 12-months. In addition, the participants who were followed to 12 months were those randomized before a certain date and not those who chose to remain in the study long term; thus, we do not believe that the 12-month data are biased toward those more motivated to use the therapy. Finally, despite intensive efforts to promote CPAP adherence, average use was only modest, and 56.6% of subjects did not meet the Medicare adherence criteria for coverage over 6 months. It is possible larger effects may have been observed with greater CPAP use, although we did not observe a correlation between improvements in HRQOL and CPAP compliance. Nonetheless, these findings suggest that even modest use may result in clinically significant improvements in HRQOL, and may motivate reexamination of current insurance criteria for CPAP use.

In conclusion, CPAP improved multiple domains of HRQOL in relatively asymptomatic patients with moderate to severe OSA at high risk of CVD. Our findings are likely important for patients and clinicians to help inform therapeutic choices and may also have important implications for policy makers and reimbursement decisions.

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FUNDING

This study was supported by the National Institutes of Health (NIH) and the National Heart, Lung, and Blood Institute (NHLBI) 1U34HL105277 and a supplemental grant from the ResMed Foundation. Equipment was donated by ResMed Inc. and Philips Respironics.

ACKNOWLEDGMENTS

We would like to thank all study participants as well as research staff including Hannah Buettner, Beatriz Oropeza, Erin Reese, Tricia Tiu, and Christina Zenobi. The study is also grateful to members of the Data Safety and Monitoring Board (Drs. Mark Espeland, Mike Sharma, Richard Bootzin, Mark Dyken, Ileana Piña), the external Medical Monitor, Dr. Sergio Waxman, the local Medical Monitor, Dr. Sanjay Patel, and Dr. Dennis Drotar for his expertise in adherence fidelity monitoring.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication November, 2016
Submitted in final revised form January, 2017
Accepted for publication February, 2017
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CLINICAL TRIAL REGISTRATION

URL: http://www.clinicaltrials.gov; Unique identifier: NCT 01261390.

DISCLOSURE STATEMENT

None disclosed.