Obstructive Sleep Apnea and Self-Reported Functional Impairment in Revascularized Patients with Coronary Artery Disease in the RICCADSA Trial

Lynn M. Baniak, PhD, RN[1,2]; Eileen R. Chasens, PhD, RN, FAAN[1]; Faith S. Luyster, PhD[1]; Patrick J. Strollo, Jr., MD[3,4], Erik Thunström, MD, PhD[5,6], Yüksel Peker, MD, PhD[3,5,7,8]

[1] School of Nursing, University of Pittsburgh, Pittsburgh, PA
[2]Department of Psychiatry, University of Pittsburgh School of Medicine, Pittsburgh, PA
[3] Division of Pulmonary, Allergy, and Critical Care Medicine, University of Pittsburgh School of Medicine, Pittsburgh, PA
[4] VA Pittsburgh Healthcare System, Pittsburgh, PA
[5]Department of Molecular and Clinical Medicine/Cardiology, Sahlgrenska Academy, University of Gothenburg, Sweden
[6] Department of Cardiology, Sahlgrenska University Hospital/Ostra, Gothenburg, Sweden
[7] Department of Clinical Sciences, Respiratory Medicine and Allergology, Faculty of Medicine, Lund University, Lund, Sweden
[8] Department of Pulmonary Medicine, School of Medicine, Koc University, Istanbul, Turkey

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Corresponding Author:
Lynn Baniak
3500 Victoria Street
Victoria Building 415
Pittsburgh, PA 15261 USA
E-mail: baniakl@pitt.edu
Fax: 412-624-1201
Phone: 412-648-3582

ABSTRACT

Purpose: Daytime sleepiness, a frequent symptom of obstructive sleep apnea (OSA), can impact functional status. In patients with coronary artery disease (CAD) and concomitant OSA, the distinction between sleep-related functional impairment from underlying CAD versus OSA is unclear. This study evaluated the impact of OSA on sleep-related functional impairment in patients with CAD and compared the effect of 1-year continuous positive airway pressure (CPAP) use on change in impairment between those with and without excessive daytime sleepiness (EDS) and OSA. We hypothesized that sleep-related functional impairment is impacted by EDS independent of OSA in patients with CAD.

Methods: 105 CAD patients without OSA and 105 with moderate-to-severe OSA from the RICCADSA trial were matched on disease severity and included in the current substudy. Of those with OSA, 80 were allocated to CPAP. Functional Outcomes of Sleep Questionnaire (FOSQ) score <17.9 corresponded to sleep-related functional impairment.

Results: Following revascularization, CAD patients with and without OSA frequently report sleep-related functional impairment (35% and 27.3%, respectively; $p = .29$). Moderate-to-severe OSA was not related to baseline FOSQ scores <17.9 in regression analyses; EDS was (OR 4.82, 95% CI 2.12-11.0; $p < .001$). CPAP use significantly improved FOSQ scores from baseline to 1-year follow-up in OSA patients with EDS (17.2±2.0 to 18.15±1.7, $p = .002$) despite suboptimal adherance.

Conclusions: Sleep-related functional impairment may be reflective of persistent EDS, independent of OSA. Diagnosing OSA and initiating treatment is worthwhile in individuals with CAD and EDS, as both are important to guide appropriate therapy in patients with CAD.

Keywords: coronary artery disease, obstructive sleep apnea, functional status, and continuous positive airway pressure

Obstructive Sleep Apnea and Sleep-Related Functional Impairment in Revascularized Patients with Coronary Artery Disease

INTRODUCTION

Functional status, the ability to manage daily activities and meet basic needs, is an important measure of health.[1] Optimal functional status is vital to adequate disease self-management and survival in persons with chronic disease. Functional impairment can negatively impact adherence to therapies and lead to worse health outcomes.[2] In persons with coronary artery disease (CAD), decrements in functional status are only partially explained by the disease process and can occur even when the desired outcomes from therapies are obtained (e.g. absence of angina). Percutaneous coronary intervention (PCI) and coronary artery bypass graft surgery (CABG) improve survival after an initial event,[3] however, patients still have an increased risk for recurrence[4] and may not regain their pre-cardiac disease functional status.

OSA, characterized by repetitive apneas and hypopneas from upper airway collapse during sleep, is highly prevalent (up to 73%) in patients with CAD.[5,6] Growing evidence indicates that severe OSA is a risk factor for CAD and is associated with an increased risk for recurrence and poor functional recovery in revascularized patients with CAD.[7,8] The resultant sympathetic activation, sleep fragmentation, and intermittent hypoxia can lead to reduced daytime function and health-related quality of life (HRQoL), fatigue, and sleepiness.[5,9] Treatment of OSA using continuous positive airway pressure (CPAP) therapy may help to improve daytime symptoms. Large clinical trials[10,11] evaluated the effect of CPAP in patients with OSA and cardiac disease. While the results did not support benefit on cardiovascular outcomes,[10,11] CPAP therapy did result in significant improvements to daytime sleepiness[11] and HRQoL.[10,11]

Excessive daytime sleepiness (EDS) can negatively impact functional status (i.e. sleep-related functional impairment).[12] In patients with CAD and OSA, factors beyond OSA may contribute to EDS including cardiac function, surgery, depression, or medications.[13,14] The distinction between sleep-related functional impairment due to CAD versus impairment due to OSA in a CAD population with concomitant OSA has not been fully examined. The majority of studies that have explored the effect of OSA therapy on change in sleep-related functional impairment have been in non-cardiac populations.[15–18] A CAD population with comorbid OSA may yield discrete findings given that complaints of EDS as a result of OSA, are reported less frequently, suggesting factors other than OSA contribute to sleep-related functional impairment.[6] This study evaluated the impact of OSA on sleep-related functional impairment in patients from a CAD cohort and compared the effect of 1-year continuous positive airway pressure (CPAP) use on change in impairment between those with and those without excessive daytime sleepiness (EDS) and OSA. We hypothesized that sleep-related functional impairment is impacted by EDS independent of comorbid OSA in patients with CAD.

METHODS

The current study was a secondary analysis of baseline data from the *R*andomized *I*ntervention with *C*PAP in *C*oronary *A*rtery *D*isease and *S*leep *A*pnea (RICCADSA) trial.[19] The RICCADSA trial was a randomized controlled trial designed to examine whether CPAP therapy in newly revascularized CAD patients with OSA reduced cardiovascular mortality or the need for an additional revascularization over three years. The trial complied with the Declaration of Helsinki and was approved by the local ethics committee. All persons gave their informed consent prior to their inclusion in the trial. Men and women with angiography-verified CAD who

recently (within six months) underwent either PCI or CABG were recruited between 2005 and 2010 from Sweden and follow-up completed in May 2013.

The current baseline sample comprised of patients with either no OSA (apnea-hypopnea index [AHI] <5 per hour) or moderate to severe OSA (AHI ≥15 per hour), **Figure 1**. Case control matching was performed to control for age and CAD severity. For each OSA patient, one patient without OSA was randomly matched by age ± 2 years, left ventricular ejection fraction (LVEF) ± 5%, and baseline status of treated hypertension (HTN; no/yes). The final baseline sample (N = 210) consisted of 105 non-OSA patients matched to 105 OSA patients. Of those with OSA, 80 went on to use CPAP therapy for 1 year and were included in follow-up analyses.

Procedures

Patients were screened for EDS using the Epworth Sleepiness Scale (ESS), EDS defined as an ESS score ≥ 10. Sleep apnea was evaluated with in-home sleep testing using the EmblettaR PDS (Portable Digital System) device (Embla, Broomfield, CO, USA). Apneas were defined as the (≥ 90%) cessation of airflow. Hypopneas were defined as either a ≥50% reduction in chest-abdominal movement, a ≥50% decrease in the nasal airflow for ≥10 seconds, or a ≥30% reduction in chest-abdominal movement or nasal airflow accompanied by oxygen desaturation (≥4%). Patients were excluded if they had mild OSA (AHI 5-14) or predominantly central sleep apneas with Cheyne-Stokes respiration. Baseline data were collected in 511 patients meeting inclusion criteria. Patients with an OSA diagnosis during home sleep testing underwent an unattended overnight polysomnography in hospital at baseline. Those with nonsleepy OSA (ESS <10) were randomized to CPAP or no-treatment, and patients with sleepy OSA (ESS ≥10) received CPAP. Patients returned at 1 year for a post-evaluation.

Outcome Measures

Sleep-related functional impairment was assessed using the Functional Outcomes of Sleep Questionnaire (FOSQ).[20] The FOSQ is a validated measure[20] and includes 30-items categorized into five subscales (i.e., Activity Level, Vigilance, Intimacy and Sexual Relationships, General Productivity, and Social Outcomes). Item responses range from no difficulty (4) to extreme difficulty (1). The total score is the sum of the subscale scores. Total FOSQ scores range from 5 to 20 with lower scores indicate greater functional impairment. Impairment is indicated by a total FOSQ score of <17.9.[16] A change of 2.0 or more points in the FOSQ score is considered to indicate a clinically meaningful improvement in daily functioning.[16]

Daytime sleepiness was assessed by the ESS.[21] The ESS is a validated [22] subjective measure that assesses patients' tendency to fall asleep in eight different situations. Responses are on a 0 to 3 Likert scale where 0 signifies "no chance of dozing" and 3 signifies a "high chance of dozing"; total scores range from 0 to 24.[21] Excessive daytime sleepiness (EDS) was indicated by a total score of ≥10.

Statistical Analyses

The sample distribution of demographic and clinical characteristics was examined using descriptive statistics. Continuous variables were reported as mean ± standard deviation; categorical variables were described as numbers and percentages. Comparisons between patients with and without OSA and between OSA patients with and without EDS who used CPAP therapy were done using Student's independent t-test or Mann-Whitney U tests for continuous variables and Chi-square tests for categorical variables. Wilcoxon signed rank test was used to examine differences in FOSQ and ESS scores between baseline and 1-year follow-up within

groups. Unadjusted linear regression modeling examined binary relationships between baseline FOSQ scores, demographics, and clinical characteristics selected a priori.

Binary logistic regression analysis evaluated the impact of OSA on the likelihood of patients reporting sleep-related functional impairment. The model was adjusted for age, sex, BMI, and additional covariates identified from the unadjusted linear regression analyses and between group comparisons with a *P* value <0.10. EDS was categorized as no (ESS <10) and yes (ESS ≥10) and OSA was categorized as no OSA (AHI <5) or moderate-to-severe OSA (AHI ≥ 15). Only cases with complete data were used. Results are reported as odds ratio (OR) and 95% confidence interval (CI). A *P* value of <.05 was considered statistically significant. Analyses were performed using SPSS 24 Windows (IBM Corp., Armonk, NY). The normality of the dependent variable (sleep-related functional impairment) was violated, showing both skewness (-1.45) and kurtosis (1.56). The Kolmorgorov-Smirnov test was significant at $p < .001$. However, in reasonably large sample sizes (N ≥ 200), the risk from bias or ineffectiveness due violation of the normality assumption is adequately reduced [23].

RESULTS

Table 1 details baseline characteristics of the 105 patients with moderate-to-severe OSA and 105 patients without OSA. The sample was mostly men (87.6 %, n = 184); none of the women (n = 26) had OSA. Those with OSA were significantly more likely to be overweight, male, have EDS, higher oxygen desaturation index (ODI), higher C-reactive protein (CRP) values, and were less likely a smoker or have a baseline acute myocardial infarction (AMI) compared to those without OSA (all *p*-values <.05). The OSA group had significantly lower mean FOSQ total scores (18.11 ± 1.9 versus 18.50 ± 1.9; $p = .023$) and lower mean scores on the "general productivity", "activity level", and "vigilance" subscales compared to the no OSA

group (all *p*-values <.05) (**Table 2**). There was no difference in the percentage of patients with sleep-related functional impairment between groups, 35% with OSA versus 27.3% without OSA (p = .288).

Baseline Associations between Sleep-Related Functional Impairment and Covariates in Matched Sample (N=210)

Univariate analysis found two variables significantly associated with sleep-related functional impairment: higher CRP values (rho = .165) and daytime sleepiness (rho = .352). Comorbidities (atrial fibrillation, diabetes, stroke, AMI, previous PCI/CABG), ODI and LVEF were not associated with impairment (all *p*-values >0.05). The final regression model (**Table 3**) contained eight independent variables: sex, age, BMI, CRP, smoking status, and baseline AMI, OSA presence, and EDS. The full model was statistically significant, χ^2 (9, N = 200) = 27.0, p = .001 and explained 17.8% (Nagelkerke R square) of the variance in sleep-related functional impairment. The model identified EDS and female sex as significant contributors. Those with EDS were 4.8 (95% CI 2.12-11.0, p < .001) times more likely to report sleep-related functional impairment, after controlling for all other factors in the model. Women were 3.8 (95% CI 1.37-10.6, p = .01) times more likely than men to report impairment.

Comparison of FOSQ and ESS scores - baseline to 1 year follow-up

Among those patients with baseline OSA who went on to use CPAP for 1 year, 35 did not have EDS (nonsleepy OSA) and 43 had EDS (sleepy OSA). There was no significant difference in AHI between groups (p = .74). At baseline (**Table 4**), the sleepy OSA group had a mean FOSQ total score corresponding to impairment (17.2 ± 2.0) and was significantly lower (p <.05) versus the mean FOSQ total score of the nonsleepy OSA group (18.52 ± 1.6). At 1 year, the sleepy OSA group demonstrated significant improvements in the FOSQ total score (p = .002)

from baseline which corresponded to a medium effect size [23] of 0.33. The median score went from being within the impaired range to being within the normal range (17.67 to 18.75) and at 1 year was comparable to the non-sleepy group. The sleepy OSA group also demonstrated significant improvements in the FOSQ subscales of "General productivity", "Activity Level", and "Vigilance". The OSA sleepy group had significant reductions in ESS median scores (12.6 ± 2.5 to 9.4 ± 2.8, p <.001) which corresponded with a large effect size [23] of 0.53 and a significant reduction in the proportion with EDS (58%; n= 45 versus n=19). Overall, only the sleepy OSA group showed significant improvements in both the FOSQ and ESS scores after 1 year (**Figure 2)** when compared to the nonsleepy OSA and non-OSA group. Relative change in FOSQ total score from baseline to 1-year **(Table 4)** was significantly greater in the sleepy OSA group compared to the non-sleepy OSA group after 1 year of CPAP use (6.1% versus 0.9%, p = .004). Mean CPAP use **(Table 4)** did not differ between groups at 1 year (2.93 ± 2.93 versus 3.0 ± 3.09, p = .57). To explore if change in ESS has a significant effect on change in FOSQ (total) when controlling for CPAP adherence and other covariates, a post-hoc multiple linear hierarchical regression was completed. However, the fully adjusted model was found to not be significant (p > .05).

DISCUSSION

In this study, we assessed the impact of OSA on sleep-related functional impairment in a CAD cohort matched for CAD severity. In addition, we investigated the effect of long-term CPAP use on change in functional impairment from baseline to 1 year follow-up. We found that following revascularization, CAD patients frequently report sleep-related functional impairment regardless of the presence of comorbid OSA. Secondly, moderate-to-severe OSA was not related

to baseline sleep-related functional impairment in this CAD cohort. Finally, treatment of OSA with CPAP therapy in patients with moderate-to-severe sleepy OSA significantly improved FOSQ scores despite suboptimal adherence (approximately 3 hours per night).

In support of our hypothesis, moderate-to-severe OSA was not associated with sleep-related functional impairment in the full CAD cohort. The lack of a cross-sectional association between the FOSQ and AHI is consistent with prior studies in young and middle-aged adults without cardiac disease.[24–27] The FOSQ assesses impairment in function due to daytime sleepiness as a result of impaired sleep; the cause of sleepiness cannot be determined from this measure. In cardiac populations, OSA may not be the dominant cause of sleepiness and impaired functioning may not be entirely sleep related but rather associated with recovery of cardiac functioning following intervention, recovery from the intervention itself, or medications. Even with an improved LVEF, difficulty in performing daily activities could be perceived by patients as due to post-surgical deconditioning, fatigue[14], or depression[13]. Beta-adrenergic blocking agents, 86% use in the current sample, can also exacerbate vulnerability to daytime fatigue, somnolence, falls, and functional decline.[28] These non-sleep related factors, unavailable for the current study, may have contributed to the low variance in FOSQ scores that was explained by the full regression model.

The FOSQ, specifically developed to measure sleep-related impairment to function, correlated with EDS. Prior work emphasizes the contribution of daytime sleepiness to daily functioning [12] especially in those persons with OSA [5]. Daytime sleepiness was significantly associated with AHI in our study. In the current study, only 35% of the OSA patients reported sleep-related functional impairment and of those, less than half reported EDS. FOSQ scores did improve in the sleepy OSA group with CPAP use, but this improvement was not considered

clinically meaningful. ESS significantly improved with CPAP use in this same group. These results suggest that the improvement in daytime sleepiness may have contributed to improvement in FOSQ scores, not OSA, which further corroborates our hypothesis.

Female sex was significantly associated with an increased risk for reporting impairment in functional outcomes. Evidence suggests a gender effect in the expression of impaired sleep including sleep-related functional impairment among women and men with suspected sleep disordered breathing.[29] However, the present study was underpowered to fully examine sex differences and highlights the need for a sufficiently larger sample of women.

Patient-reported health status, a construct that includes functional status,[1] is becoming increasingly important to evaluate the benefits of CPAP therapy. Similar to our longitudinal results, the FOSQ has been shown to be responsive to CPAP therapy in sleepy OSA patients.[15,18] We found that while 3 hours of CPAP use per night significantly improved FOSQ scores after 1 year in patients with sleepy OSA, it was not adequate to change the proportion with impairment or to exceed the 2.0-point increase that is considered to be a clinically meaningful improvement. Weaver et al. found a linear dose-response relationship between increased CPAP use and normalized functioning with 7.5 hours of CPAP use needed to achieve normalization.[16] Antic et al.[18] failed to identify a linear dose-response relationship with many patients not achieving normal functioning after 3 months of CPAP therapy despite CPAP use of ≥4 hours. Baseline FOSQ scores in the Antic[18] and Weaver[16] studies were considerably lower than the baseline FOSQ scores in our study, which could account for the nonsignificant change in proportion with impairment and the lack of a clinically meaningful improvement.

Study Limitations

Strengths of our study include the ability to evaluate the unique association of OSA with sleep-related functional impairment in a modest sample of adults with CAD and OSA. Limitations include that, it is unknown whether patients had co-existing sleep disorders and/or depression that contributed to their self-reported sleep-related functional impairment. Secondly, although a validated instrument, the FOSQ may have lower levels of discrimination in persons with cardiac disease who may also have competing causes of sleepiness.

Conclusions

These findings suggest that following revascularization in patients with CAD, (a) perceived sleep-related functional impairment may be more reflective of persistent EDS independent of OSA, (b) exploring for OSA and pursuing treatment is worthwhile in persons with CAD and comorbid sleepy OSA, and (c) although the low CPAP use as seen in the current study may not be adequate to reduce cardiovascular risk, this level of adherence was helpful to improve patient-centered outcomes. Because OSA and EDS appear to be independently associated with patient outcomes, both are important to guide appropriate therapy in patients with CAD.

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Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent: For this type of study, formal consent is not required.

FIGURE LEGENDS

Figure 1. Flow diagram for the study sample. In bold, individuals who were included for baseline and follow-up analyses.

Figure 2. The figures depict group mean functional outcomes of sleep questionnaire (FOSQ) total scores and Epworth Sleepiness Scale (ESS) scores at baseline and 1 year for those who used CPAP (nonsleepy OSA and sleepy OSA) and those who did not use CPAP (no OSA). Only the sleepy OSA group demonstrated significant improvements in both FOSQ and ESS scores from baseline to 1-year follow-up.

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Table 1. Comparison of characteristics of the baseline sample between those with and without obstructive sleep apnea.

	With OSA (n = 105)	**Without OSA (n = 105)**	***P* Value**
Age, year	63.03 ± 8.0	62.96 ± 8.1	.946
Sex, male (%)	100% (105)	75.2% (79)	<.001
BMI (kg/m^2)	28.4 ± 3.8	25.5 ± 3.0	<.001
% >= 25	84.8% (89)	56.2% (59)	<.001
LVEF	60 (55-65)	60 (55-65)	.854
CRP (mg/dL)	3.5 ± 6.8	2.3 ± 3.3	.013
AHI	29.8 ± 12.9	3.1 ± 1.3	<.001
Moderate (AHI 15-29)	57% (60)		
Severe (AHI ≥ 30)	42.9% (45)		
ODI	17.8 ± 12.3	1.7 ± 1.3	<.001
Previous PCI/CABG	21.9% (23)	16.2% (17)	.292
Treated HTN	49.5% (52)	49.5% (52)	1.00
Hx A-Fib	14.3% (15)	8.6% (9)	.193
Hx Diabetes	18.1% (19)	13.3 (14)	.343
AMI_baseline	43.8% (46)	58.1% (61)	.038
Hx Stroke	7.7 % (8)	3.8% (4)	.234
Current Smoker	14.3% (15)	25.7% (27)	.038

Abbreviations: A-fib, atrial fibrillation; AHI, apnea/hypopnea index; AMI, acute myocardial infarction; BMI, body mass index; CABG, coronary artery bypass grafting; CRP, C-reactive protein; HTN, hypertension; IQR, interquartile range; LVEF, left ventricular ejection fraction; MI, myocardial infarction; ODI, oxygen desaturation index; OSA, obstructive sleep apnea; PCI, percutaneous coronary intervention.

Data are presented as % (n) or mean ± SD.

Table 2. Comparison of baseline FOSQ and ESS scores between patients with and without OSA.

	With OSA (n = 105)	**Without OSA (n = 105)**	***P* Value**
FOSQ (total)	18.11 ± 1.9	18.50 ± 1.9	**.023***
FOSQ Subscales			
General Productivity	3.77 ± .29	3.82 ± .03	**.011***
Social Outcome	3.87 ± .35	3.82 ± .41	.411
Activity Level	3.50 ± .49	3.59 ± .50	**.007***
Vigilance	3.57 ± .46	3.72 ± .38	**.009***
Intimacy	3.43 ± .78	3.51 ± .08	.450
FOSQ Scores < 17.9	35.0% (36)	27.3% (27)	.288
ESS	8.44 ± 4.4	5.63 ± 3.0	**<.001***
EDS (ESS ≥ 10)	42.9% (45)	6.7% (7)	**<.001***

Abbreviations: EDS, excessive daytime sleepiness; ESS, Epworth Sleepiness Scale; FOSQ, functional outcomes of sleep questionnaire.

Data are presented as % (n) or mean ± SD.

Table 3.

Adjusted binary logistic regression analysis[a] examining predictors of impaired functional outcomes (FOSQ < 17.9) in 210 revascularized patients matched on CAD severity.

	OR	**95% CI**	***P* Value[b]**
Final Model			<.001*
BMI	1.67	.734-3.78	.222
Sex	4.42	1.6-12.24	.004*
Age	1.24	.611-2.50	.556
C-reactive protein	1.04	.976-1.11	.227
Current Smoker	1.23	.508-3.0	.642
AMI Baseline	0.91	.461-1.79	.782
OSA Severity			
None (AHI <5)			.881
Moderate (15-29)	0.96	.374-2.47	.933
Severe (≥ 30)	1.20	.446-3.22	.720
EDS	4.68	2.06-10.60	<.001*

Abbreviations: AMI, acute myocardial infarction; BMI, body mass index; EDS, Excessive Daytime Sleepiness; FOSQ, functional outcomes of sleep questionnaire; OSA, obstructive sleep apnea status.

[a] Logistic regression analysis adjusted for BMI, body mass index, dichotomized as 0 = normal (BMI < 25) and 1 = overweight (BMI ≥ 25); sex, 0 = male and 1 = female; age, dichotomized as 0 = < 65 and 1 = ≥ 65; CRP, C-reactive protein, continuous; Current smoker dichotomized as 0 = no and 1 = yes; AMI, acute myocardial infarction, dichotomized as 0 = no and 1 = yes. OSA severity categorized as 0=no OSA, 1=moderate, and 2=severe; and EDS, Excessive Daytime Sleepiness, dichotomized as 0= no (ESS < 10) and 1= yes (ESS ≥ 10) were entered into block 2.

[b] Final model after an enter stepwise approach, with entry and removal criteria of $P < 0.05$ and $p < 0.10$, respectively.

Table 4. Comparison of FOSQ and ESS scores at baseline to 1 year in patients with nonsleepy and sleepy OSA who used CPAP.

	Baseline	**1 year**	**Mean Difference (Baseline – 1 year)**	***P Value***
Non-Sleepy CPAP				
N	35	35		
FOSQ (total)	18.52 ± 1.6	18.58 ± 1.8	-.10 (-0.69, 0.49)[†]	
Median	19.0	19.1		.873
Interquartile Range	18.0-19.9	18.2-19.9		
FOSQ Subscales				
General Productivity	3.81 ± .21	3.82 ± .33		.169
Social Outcome	3.86 ± .45	3.82 ± .48		.785
Activity Level	3.58 ± .45	3.63 ± .41		.859
Vigilance	3.74 ± .35	3.79 ± .37		.361
Intimacy	3.65 ± .55	3.50 ± .75		.090
Sleepiness-Related Functional Impairment (FOSQ < 18)	22.9% (8)	20.6% (7)		1.00
ESS	5.4 ± 2.5	5.5 ± 3.2	-.03 (-1.1, 1.05)	
Median	6.0	5.0		.860
Interquartile Range	4.0-8.0	3.0-8.0		
CPAP Adherence[a]	N/A	2.93 ± 2.93		
Median		3.19		
Interquartile Range		(0-5.8)		
Sleepy CPAP				
N	45	45		
FOSQ (total)	17.2 ± 2.0	18.15 ± 1.7	-.89 (-1.5, -0.32)[†]	
Median	17.67	18.75		**.002***
Interquartile Range	16.2-18.86	17.11-19.46		
FOSQ Subscales				
General Productivity	3.66 ± .33	3.80 ± .21		**.016***
Social Outcome	3.81 ± .38	3.88 ± .30		.160
Activity Level	3.30 ± .54	3.53 ± .41		**.003***
Vigilance	3.31 ± .43	3.59 ± .30		**<.001***
Intimacy	3.12 ± .88	3.31 ± .92		.101
Sleepiness-Related Functional Impairment (FOSQ < 18)	55.8% (24)	40% (16)		.092
ESS	12.6 ± 2.5	9.4 ± 2.8	3.23 (2.2, 4.2)	
Median	12.0	9.0		**<.001***
Interquartile Range	11.0-14.0	7.0-12.0		
EDS (ESS ≥ 10)	100% (45)	42% (19)		
CPAP Adherence[a]	N/A	3.0 ± 3.09		
Median		3.09		
Interquartile Range		(0-5.11)		

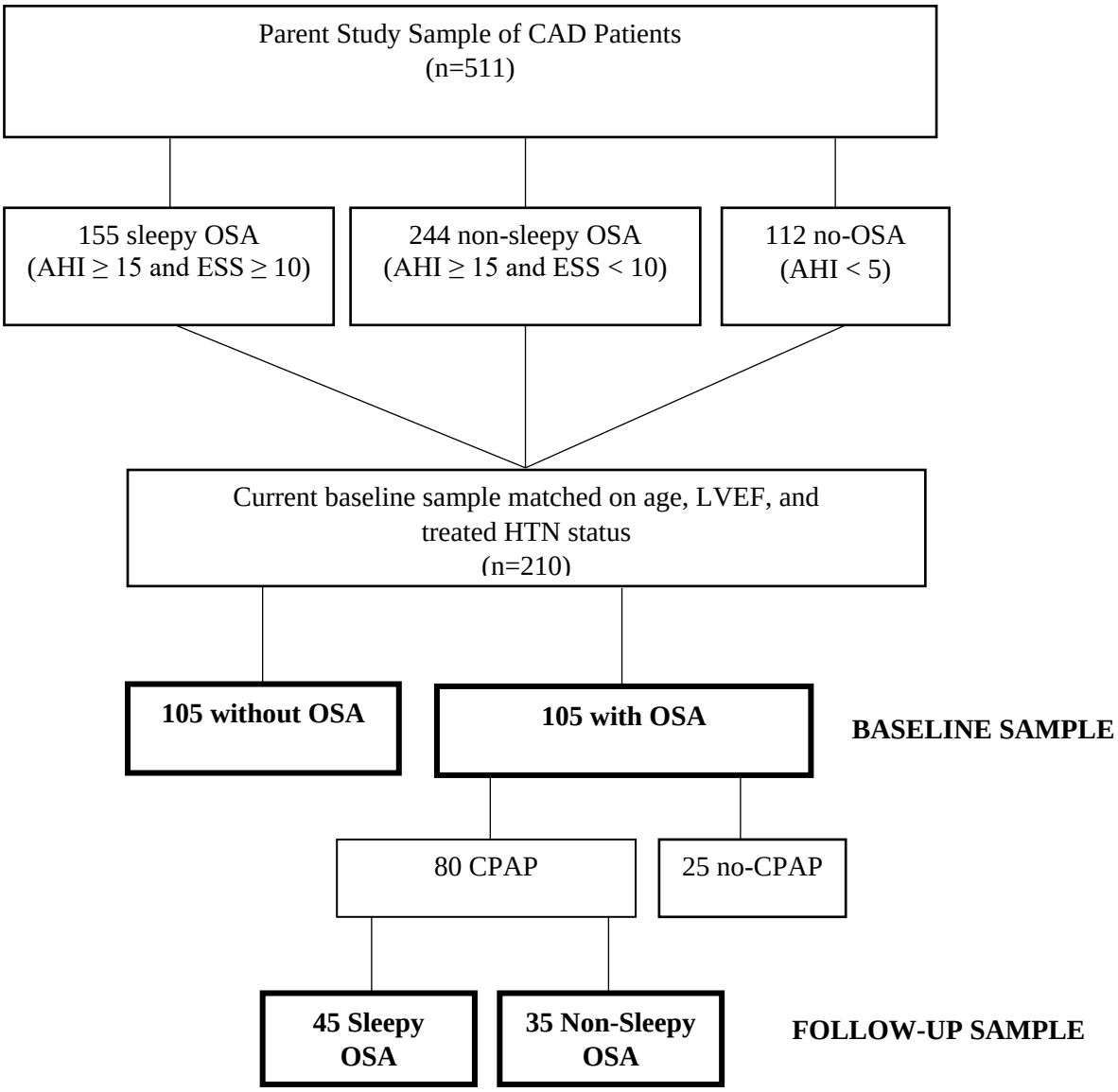
Abbreviations: CPAP, continuous positive airway pressure; EDS, excessive daytime sleepiness; ESS, Epworth Sleepiness Scale; FOSQ, functional outcomes of sleep questionnaire.

Data are presented as % (n), mean ± SD, or median (IQR).

[a] Data available for n = 35 nonsleepy and n = 44 sleepy patients.

[†] Relative difference (1 year – Baseline / Baseline x 100) in FOSQ (total) between groups was found to be significantly greater in the sleepy OSA group compared to the non-sleepy OSA group after 1 year of CPAP use (6.1% versus 0.9%, respectively).

Figure 1



Abbreviations: CAD, coronary artery disease; ESS, Epworth sleepiness scale; LVEF, left ventricular ejection fraction; HTN, hypertension; OSA, obstructive sleep apnea.

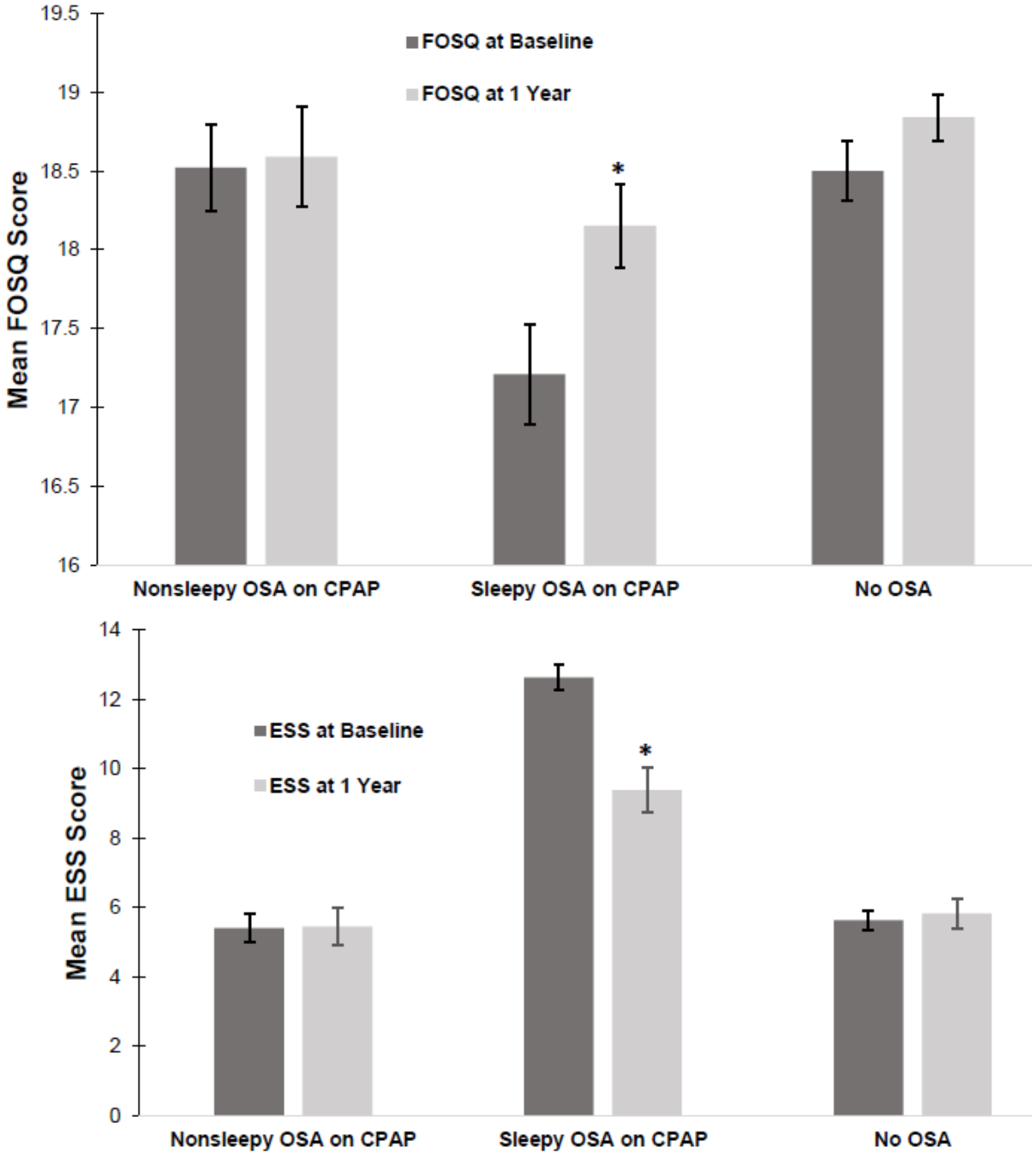



Figure 2. Comparison of Mean FOSQ and ESS scores at Baseline and 1 Year Follow-up in Nonsleepy OSA, Sleepy OSA, and no OSA Groups.