Effects of Exercise and Weight Loss in Older Adults with Obstructive Sleep Apnea

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ABSTRACT

DOBROSIELSKI, D. A., S. PATIL, A. R. SCHWARTZ, K. BANDEEN-ROCHE, and K. J. STEWART. Effects of Exercise and Weight Loss in Older Adults with Obstructive Sleep Apnea. *Med. Sci. Sports Exerc.*, Vol. 47, No. 1, pp. 20–26, 2015. **Purpose**: Obstructive sleep apnea (OSA) is prevalent among older individuals and is linked to increased cardiovascular disease morbidity. This study examined the change in OSA severity after exercise training and dietary-induced weight loss in older adults and the association of the changes in OSA severity, body composition, and aerobic capacity with arterial distensibility. **Methods**: Obese adults (n = 25) with OSA, age 60 yr or older, were instructed to participate in supervised exercise (3 d·wk^{-1}) and follow a calorie-restricted diet. Baseline assessments of OSA parameters, body weight and composition, aerobic capacity, and arterial distensibility were repeated at 12 wk. **Results**: Nineteen participants completed the intervention. At 12 wk, there were reductions in body weight (−9%) and percentage of total body fat (−5%) and trunk fat (−8%) whereas aerobic capacity improved by 20% (all $P < 0.01$). The apnea–hypopnea index decreased by 10 events per hour ($P < 0.01$) and nocturnal SaO_2 (mean SaO_2) improved from 94.9% at baseline to 95.2% after intervention ($P = 0.01$). Arterial distensibility for the group was not different from that at baseline ($P = 0.99$), yet individual changes in distensibility were associated with the change in nocturnal desaturations ($r = -0.49$, $P = 0.03$) but not with the change in body weight, apnea–hypopnea index, or aerobic capacity. **Conclusions**: The severity of OSA was reduced after an exercise and weight loss program among older adults, suggesting that this lifestyle approach may be an effective first-line nonsurgical and nonpharmacological treatment for older patients with OSA. **Key Words:** WEIGHT LOSS, MEAN SAO_2, APNEA–HYPOPNEA INDEX, ARTERIAL DISTENSIBILITY

Obstructive sleep apnea (OSA) is characterized by repeated episodes of upper airway obstruction that are associated with reductions in ventilation, arousals, and/or oxyhemoglobin desaturations during sleep (33). OSA can lead to excessive daytime sleepiness and fatigue, which, in turn, may cause vehicular and industrial accidents (39). In addition to neurocognitive sequelae (5), OSA is associated with increased cardiovascular disease (CVD) morbidity and mortality (35).

Although the pathology linking OSA with CVD is complex, it is well accepted that obesity plays an important role in OSA–CVD interaction. Obesity is the strongest predictor of OSA (34), and lifestyle interventions that promote weight loss are recommended in clinical guidelines as effective treatment strategies (30). The evidence in support of this recommendation has been recently reviewed by Araghi et al. (3), who report that lifestyle programs are associated with a decrease in apnea–hypopnea index (AHI) of between six and 12 events per hour of sleep. However, despite these promising findings, there are several gaps in the existing knowledge that warrant further attention.

First, the prevalence of OSA in adults >60 yr is considerably higher compared with that in middle-age adults (44), yet the abundance of data demonstrating the efficacy of lifestyle interventions for improving OSA severity has been shown in younger to middle-age cohorts (3). Increasing age is correlated with greater pharyngeal collapsibility, independent of body mass index (BMI), and some studies have demonstrated increased airway resistance during sleep in older adults versus younger adults (32). Moreover, compared with weight-matched younger adult control subjects, healthy older adults demonstrate wider oscillations in upper airway resistance during supine sleep that may contribute to greater tendency for periodic breathing (21). Interestingly, exercise, independent of changes in body weight, reduces the severity of OSA (23). These previously mentioned data suggest that factors beyond obesity contribute to the OSA phenotype in older adults. Given recent trends showing rising rates of obesity among the elderly (13) and evidence associating OSA with

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increased CVD morbidity in this demographic (42), it would be clinically relevant and of great public health importance to examine the effectiveness of a lifestyle program consisting of both structured exercise and weight loss diet on OSA in this vulnerable cohort.

Second, although it is likely that lifestyle changes will translate to improved cardiovascular profiles among OSA patients, there have been few studies that have examined intermediary mechanisms of CVD (e.g., endothelial function, inflammation, increased sympathetic activity) in the context of an intervention that also reduces obesity and improves aerobic capacity. Because obesity, especially central adiposity (9), and aerobic capacity (28) also predict CVD risk and mortality, it is unclear whether improvements in these outcomes or reductions in OSA severity are primarily responsible for mitigating CVD risk.

Accordingly, the principal aim of this study was to examine the change in OSA severity after a combined exercise- and dietary-induced weight loss intervention in a group of older men and women. A secondary aim was to examine the association among changes in OSA severity, body weight, body composition, and aerobic capacity with arterial distensibility, a marker of vascular wall damage (43) that is highly correlated with cardiovascular outcomes in older adults (40).

MATERIALS AND METHODS

Participant recruitment and screening. Obese adults (BMI, between 30 and 43 $kg \cdot m^{-2}$) age 60 yr and older were eligible for the study. Individuals were excluded if they 1) were currently being treated for OSA, 2) were engaged in moderate-intensity exercise for 90 $min \cdot wk^{-1}$ (19), 3) followed a hypocaloric diet for weight loss (in the last 3 months), 4) had history of CVD as defined by medical history, 5) had untreated hypothyroidism or hyperthyroidism, 6) had moderate-to-severe hypertension diastolic blood pressure ≥100 mm Hg or systolic blood pressure (SBP) ≥160 mm Hg, 7) were diagnosed with central sleep apnea of more than five events per hour, 8) had AHI >60 events per hour and nocturnal oxygen saturation <85% for >15% of the record, 9) had diabetes mellitus requiring insulin, 10) had an exercise stress test that was positive for ischemia, complex arrhythmias, or symptoms indicative of ischemia or (11) underlying osteoarthritis that would limit the ability to exercise.

Recruitment occurred through media advertisements. Phone screenings were conducted to determine initial eligibility and to administer the Berlin questionnaire (31) to identify patients at risk for OSA. A score ≥2 was used to suggest a positive screen for OSA. Those who were previously diagnosed with OSA or who were classified as high risk for OSA were invited to undergo baseline screening. The baseline screening consisted of an orientation to the study and obtaining a written informed consent that was approved by the Johns Hopkins University institutional review board. A physician performed a medical history and physical examination that was followed by a graded exercise test to screen for CVD as outlined in item number 10 mentioned previously.

Individuals who qualified were scheduled to undergo overnight polysomnography monitoring that served as the baseline measure of OSA severity. Upon waking up the next morning, participants underwent vascular testing, which was immediately followed by body composition assessment. After eating a light breakfast, participants underwent cardiopulmonary exercise testing. Once baseline assessments were completed, participants were enrolled in an exercise and dietary weight loss intervention for 12 wk. All assessments were repeated in the same order within a week after the end of the intervention.

Polysomnographic assessment. Patients were admitted to the unit at 5:00 p.m. for the sleep study. Thereafter, a standardized meal was provided for dinner at 6:00 p.m. Sleep study recording sensors were applied. Standardized recording methods using Remlogic 1.3 (Natus Medical Inc., Broomfield, CO) were used (22) and included continuous monitoring of left and right electrooculogram, submental EMG, F3-M2, C_3-A_2, and $O_{1\text{-}M2}$ electroencephalogram, anterior tibialis EMG, oronasal airflow as assessed by both a pressure-sensitive nasal cannula and a thermistor, pulse oximetry and thoracic and abdominal movements with respiratory inductive plethysmography, and a modified V5 ECG lead for cardiac rhythm monitoring. The sleep study commenced at 10:00 p.m. (lights out) and ended at 6:00 a.m. the following morning. Apnea was defined as a significant decrease (>90%) in oronasal flow ≥10 s, and hypopnea, as an evident decrease in airflow >30% but <90% and associated with either oxygen desaturation of ≥3% and/or arousal. The AHI was defined as the sum of apnea and hypopnea events per hour of sleep. Mean oxyhemoglobin saturation (mean SaO_2) and average nadir SaO_2 associated with each disordered breathing event (average Low SaO_2) during sleep were retained as markers of OSA severity. Patients were classified as having OSA if the AHI was equal to or exceeded five events per hour of sleep.

Arterial distensibility. Beat-by-beat pulse wave amplitude was captured using fingertip peripheral arterial tonometry (ENDOPAT 2000; Itamar Medical, Caesarea, Israel), as previously described (7), and used in the determination of arterial distensibility (26). Plethysmographic finger cuffs were placed on the index fingers of both hands while the subjects lay in a supine position. The arterial pressure waveform is a composite of the forward pressure (P1) wave created by ventricular contraction and a wave reflected from the periphery (P2) mainly at branch points or sites of impedance mismatch. From the second derivative of the pulse pressure waveform obtained by the finger plethysmograph, the amplitudes of the second (P2) and first (P1) inflections were obtained (41). The augmentation index (AI) was calculated from the ratio of the difference between P1 and P2 systolic peaks of the waveforms relative to P1 expressed as a percentage $[(P2 - P1)/P1 \times 100)]$ (20). An elevated AI derived from peripheral arterial tonometry is associated with increased severity of atherosclerosis (41) and abnormal ventricular–vascular coupling (20). This method for calculating AI correlates well ($R = 0.68$, $P < 0.001$) with radial artery tonometry (18).

Body composition. Dual-energy x-ray absorptiometry was used to measure total and regional (trunk) fat mass in the frontal plane. These data are presented as percentages. Trunk fat was defined from the body of the mandible to the neck of the femur and laterally to the glenohumeral joint. We used a GE Lunar Prodigy (software version 13; Milwaukee, WI), which uses advanced fan-beam array mode technology.

Aerobic capacity. Aerobic capacity testing was done on a treadmill integrated with a Viasys Metabolic/ECG system using a modified Balke protocol, beginning at 3 mph, 0% grade, and increasing by 2.5% grade every 3 min until volitional fatigue was reached. A 12-lead ECG was recorded at each stage. Blood pressure was measured during the last 30 s, and the RPE, using the Borg 6–20 scale, (8) was obtained during each stage. Subjects were urged to push themselves to volitional fatigue. An RPE of 18–20 and an RER >1.1 was used to confirm whether maximal effort was achieved by every participant. The highest observed 30-s average value of $\dot{V}O_2$ was considered maximal.

Exercise training and dietary weight loss intervention. Participants were prescribed cardiorespiratory training and resistance exercise using American College of Sports Medicine guidelines (15). In accordance with these recommendations, cardiorespiratory conditioning consisted of a combination of moderate- and vigorous-intensity exercise on a treadmill, stationary cycle, or stair stepper to achieve a total energy expenditure of >500–1000 MET·min·wk^{-1}. Participants attended three supervised exercise sessions per week. A gradual progression of exercise duration was used until each participant could accumulate 45 total minutes of cardiorespiratory conditioning per session, after which intensity was raised incrementally and ultimately varied between 60% and 85% HR_{max}. Participants wore HR monitors (Polar Inc., Lake Success, NY) continuously and were monitored by study staff. American College of Sports Medicine equations were used to estimate MET minutes per week. On the same day, participants performed two sets of 10–15 repetitions of the following exercises: latissimus dorsi pulldown, leg extension, leg curl, bench press, leg press, shoulder press, and seated midrowing. An increase in weight was used once participants could successfully complete two sets of a given resistance for 15 repetitions. Resistance training volume per week was reported as the sum of the total amount of weight lifted (weight × repetitions × sets) per session.

In addition to the exercise intervention, each participant was given dietary advice by a study dietician that was consistent with American Heart Association Diet and Lifestyle Recommendations (29). The overarching goal of the diet intervention was to help participants achieve weight loss between 8% and 10% baseline body weight in 12 wk. Participants met with a study dietician once per week for the first 4 wk and biweekly thereafter to review food diaries and measure body weight because this is the most practical outcome to monitor and is the outcome most likely to be of interest to participants. Upon reviewing food records and body weight, the dieticians offered counseling and dietary education to help each participant reach his/her goal weight. Three-day food records were completed at baseline and after the intervention and were subsequently analyzed using the Food Processor software (ESHA Research, Salem, OR).

Statistical methods. Primary outcomes of interest are presented as median (interquartile range) for continuous variables. Baseline associations between markers of OSA severity, body weight, body composition, aerobic capacity, and arterial distensibility were evaluated with the Spearman rank test. Dietary data (baseline and after intervention) and exercise programming data (months 1, 2, and 3) were evaluated for change over time using a dependent *t*-test and repeated-measures ANOVA, respectively. These data are represented as mean (SD). Baseline and postintervention values for the primary outcomes of interest were compared using the Wilcoxon signed-rank test. We calculated Spearman correlations (rho) between 12-wk changes in measures of body weight, total body and trunk fat percentage, aerobic capacity, and OSA parameters with arterial distensibility to examine the weight loss-induced changes in these variables with arterial distensibility. The level of statistical significance was set at $P < 0.05$ (two tailed). Analyses were performed using STATA version 12 (StataCorp LP, College Station, TX).

RESULTS

Baseline characteristics. One hundred and thirty-nine individuals responded to advertisements. Eighty-one did not meet the inclusion criteria, and 18 could not be contacted for subsequent follow-up. Of the remaining 40 individuals who underwent phone screening, one died before providing an informed consent and four declined future participation. Thirty-five individuals were invited to visit the laboratory on the basis of the Berlin score, provided an informed consent, and underwent diagnostic screening on a treadmill. Five individuals were excluded because of ECG abnormalities during the treadmill test, suggestive of underlying CVD, or BMI below 30 kg·m^{-2}. Of the remaining 30 individuals who underwent polysomnography, five were excluded because of AHI <5.

Fifteen women and 10 men enrolled in the study (mean age of the sample, 67 ± 4 yr). None of the participants had been previously diagnosed with OSA. A summary of the baseline associations between markers of OSA severity and body composition and arterial distensibility is provided in Table 1. Of note, lower mean SaO_2 was associated with higher body weight (rho = −0.41, $P = 0.04$) as well as total body fat percentage (rho = −0.45, $P = 0.02$) and trunk fat percentage (rho = −0.54, $P < -0.01$). The average low SaO_2 was inversely related to body weight (rho = −0.46, $P = 0.02$) and BMI (rho = −0.42, $P = 0.04$). Interestingly, more severe AHI was associated with lower total body fat percentage (rho = −0.42, $P = 0.04$). To explore the potential for confounding by sex, scatter plots and Spearman rho coefficients were produced separately by sex strata. The sex-specific analyses proved similar to each other and the overall analyses, contraindicating substantial sex-related confounding as a concern.

TABLE 1. Associations between OSA severity, body composition, and vascular parameters at baseline (n = 25).

	AHI	Mean $SaPO_2$	Average Low $SaPO_2$
Weight (kg)	0.13	−0.41	−0.46*
BMI ($kg \cdot m^{-2}$)	0.18	−0.24	−0.42*
Total body fat (%)	−0.42*	−0.45*	0.14
Trunk fat (%)	−0.37	−0.54**	0.16
SBP (mm Hg)	0.19	0.30	0.09
DBP (mm Hg)	0.15	0.10	−0.12
AI (%)	−0.01	−0.01	−0.03

Data are presented as Spearman rank correlations.
*$P < 0.05$.
**$P < 0.01$.
DBP, diastolic blood pressure; SBP, systolic blood pressure.

Effects of exercise and dietary weight loss. Of the 25 participants who completed baseline testing, two individuals suffered an injury unrelated to the study and were withdrawn from the intervention, one individual lost a family member to death and withdrew voluntarily, and three individuals dropped out of the program because of their inability to adhere to the exercise or diet appointments. The remaining 19 participants completed the exercise and diet intervention, which was followed by postintervention testing. Nutrition data acquired from food diaries are reported in Table 2. As expected, there was a marked reduction in total calories consumed ($P < 0.01$) and in percentage of total calories derived from fat ($P < 0.01$), whereas the amount of calories derived from dietary protein increased ($P < 0.01$). Exercisers who completed postintervention testing attended a mean (SD) of 34 (2.8) of their prescribed 36 sessions (95% compliance). Table 2 summarizes the exercise training data according to the month enrolled in the program. The average MET minutes per week and resistance training volume per week increased from month 1 (weeks 1–4) to month 3 (weeks 9–12).

Baseline and postintervention data for those who completed the program (12 women, seven men) are displayed in Table 3. Of note, the intervention resulted in 9% loss of body weight ($P < 0.01$) and reduced AHI by 10 events per hour of sleep ($P = 0.03$). Similarly, we observed marked reductions in total body fat percentage (−5%), trunk fat percentage (−8%), and waist circumference (−6%) (all P <0.01), whereas aerobic capacity improved by 20% ($P < 0.01$). In addition, total sleep time increased by 27 min (<0.01) and nocturnal SaO_2 also improved (mean SaO_2) from 94.9% at baseline to 95.2% after the intervention ($P = 0.02$). The average low SaO_2 at baseline was 89.9% compared with 91.0% after the intervention ($P = 0.02$). No significant change in AI was observed.

Comparisons of changes in arterial distensibility with changes in OSA severity, body composition, and aerobic capacity. The relation between the change in arterial distensibility and markers of OSA severity, body composition, and aerobic capacity was examined and is expressed in Table 4. A reduction in AI after the intervention was associated with improved nocturnal mean SaO_2 (rho = −0.47, $P = 0.04$) and average low SaO_2 (rho = −0.57, $P = 0.01$), whereas the relation with the reduction in total body fat percentage fell short of statistical significance (rho = 0.45, $P = 0.05$). By contrast, no association was observed for the change in AI and the change in weight, AHI, or aerobic capacity. Moreover, the change in body weight was not related to any marker of OSA severity.

DISCUSSION

It is well established that continuous positive airway pressure (CPAP) therapy is an effective first-line nonsurgical and nonpharmacological intervention for management of OSA (25), yet the use of CPAP is limited by poor patient adherence. We found marked reductions in disordered breathing events and improvements in nocturnal oxyhemoglobin desaturation among older adults with OSA after a 12-wk exercise and diet intervention. These positive changes in OSA severity were accompanied by weight loss and reduced total body fat, as well as an increase in aerobic capacity, thus supporting the view that a comprehensive lifestyle approach may be a desirable and effective treatment for patients with OSA, particularly in those with poor adherence to CPAP. In contrast, no significant reduction in mean arterial distensibility was observed after the intervention. However, correlational analysis revealed that positive changes in arterial distensibility were related to improvements in nocturnal desaturations but not to reductions in weight, disordered breathing events, or improvements in aerobic capacity.

TABLE 2. Dietary and exercise programming data for participants who completed the intervention (n = 19).

Dietary Data	Baseline		Postintervention	P Value
Total calories (kcal)	2062 (683)	—	1439 (431)	<0.01
Calories from fat (%)	37 (8)	—	31 (8)	<0.01
Calories from CHO (%)	43 (7)	—	46 (7)	0.07
Calories from protein (%)	17 (4)	—	20 (4)	<0.01
Calories from alcohol (%)	3 (4)	—	3 (3)	0.25
	Month Enrolled			
Exercise Data	1	2	3	P Value
MET·min·wk^{-1}[a]	668 (295)	889 (414)	1046 (544)*	0.04[b]
Resistance volume[c]	21,809 (8866)	25,442 (8267)	29,138 (8592)*	0.04[b]

Data are presented as mean (SD). Significance levels are set at $P < 0.05$.
[a]Represents the mean of the sum of the metabolic cost during each week for each participant.
[b]ANOVA main effect.
[c]Represents the mean of the sum of the total weight lifted during each week for each participant.
*$P < 0.05$ vs month 1.

TABLE 3. Baseline and postintervention values for primary outcomes among those who completed the intervention.

Variable	Baseline	Postintervention	*P* Value
Body composition			
Weight (kg)	97.5 (82.0–114.2)	88.4 (78.4–105.3)	<0.01
BMI ($kg \cdot m^{-2}$)	33.8 (32.6–36.9)	31.6 (30.3–34.7)	<0.01
Waist circumference (cm)	108 (101–114)	102 (97–113)	<0.01
Total body fat (%)	42.2 (38.3–47.6)	39.9 (34.4–46.9)	<0.01
Trunk fat (%)	46.6 (41.9–49.4)	43.0 (37.9–48.2)	<0.01
Aerobic capacity			
$\dot{V}O_{2max}$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	22.3 (18.8–25.4)	26.8 (23.1–28.6)	<0.01
$\dot{V}O_{2max}$ ($L \cdot min^{-1}$)*	1.91 (1.79–2.80)	2.17 (2.05–2.97)	<0.01
Vascular parameters			
SBP (mm Hg)	129 (114–147)	123 (112–131)	0.20
DBP (mm Hg)	69 (65–76)	67 (65–72)	0.50
AI (AU)	36 (22–44)	26 (10–49)	0.84
Sleep parameters			
Total sleep time (min)	389 (323–401)	416 (395–444)	<0.01
Mean SaO_2 (%)	94.9 (94–95.4)	95.2 (94.4–95.7)	0.02
Average low SaO_2 (%)	89.9 (88.5–91.5)	91.0 (89.4–92)	0.02
AHI (events per hour)	22 (14–44)	12 (5–26)	0.03

Data are presented as median (interquartile range).
*Absolute $\dot{V}O_{2max}$ at baseline for women (mean, 1.78 $L \cdot min^{-1}$) and men (mean, 2.89 $L \cdot min^{-1}$).
DBP, diastolic blood pressure; SBP, systolic blood pressure.

The present finding of a reduction of 10 apneic events per hour of sleep compares favorably with previous studies that have used similar therapeutic approaches. Barnes et al. (4) found that 4 months of supervised exercise, consisting of both aerobic and resistance training, combined with very low energy diet led to a reduction of AHI from 24 to 18 events (−25%) and a 12-kg weight loss among 12 middle-age OSA patients. In the Sleep AHEAD trial (14), older (mean age, 61 yr) obese adults with type 2 diabetes underwent an intensive lifestyle intervention for 1 yr and lost 8.6% of body weight, which was associated with a decrease in AHI of 10 events per hour of sleep. However, while Sleep AHEAD encouraged physical activity, this was not closely evaluated. Thus, the implication of the current study is that older adults seem to accrue benefits on OSA severity similar to that of younger cohorts when prescribed a regimen of structured exercise and a weight loss diet.

Parceling out the independent contributions of exercise or weight loss to reducing OSA severity is beyond the scope of the present study, although we suspect that exercise plays a significant role partly because of lack of association between the change in body weight and the change in apneic events. Others (16,24,37) have reported improvements in AHI of between four and 10 events per hour of sleep, independent of weight loss, in patients who have been prescribed structured exercise for between 4 and 6 months. It has been speculated that exercise may improve pharyngeal muscle tone and strength or result in shifts in peripheral fluid accumulation (23). Furthermore, we suspect that improvements in OSA severity are more closely linked to the patterns of regional weight loss rather than to the total amount of weight lost because abdominal visceral fat elevates mechanical loads on the upper airway and is an abundant source of proinflammatory cytokines, which may lead to depression of CNS activity and upper airway neuromuscular control when released (36). That said, we (38) have shown that exercise training alone, independent of weight loss, leads to a significant reduction in visceral fat among older adults. Thus, exercise may mitigate OSA severity by reducing visceral adipose tissue. While changes in OSA severity were not associated with improvement in trunk fat in our sample, our dual-energy x-ray absorptiometry methods lack the sensitivity to detect specific changes in the abdominal visceral compartment.

Contrary to other exercise (11) and weight loss (17) studies in non-OSA groups, we did not observe a change in arterial distensibility among this group of patients with OSA after the intervention. The reason(s) for the lack of change is unclear, and we cannot discount the possibility that we made a type 2 error because of our small sample. Furthermore, apart from having OSA, our participants were relatively healthy, as demonstrated by the fact that 1) they had no history of CVD and 2) aerobic capacity at baseline for the men and women exceeded predicted normal values by 10% and 70%, respectively (See Table 14 in reference (2)). Therefore, there may have been limited room for vascular improvement, although we did not have an appropriate control group to make

TABLE 4. Associations between changes in OSA severity, body composition, aerobic capacity, and arterial distensibility.

	ΔMean $SaPO_2$	ΔMean Low $SaPO_2$	ΔAHI	ΔWeight	ΔBMI	ΔTotal Body Fat	ΔTrunk Fat	Δ$\dot{V}O_{2max}$
ΔAverage low $SaPO_2$ (%)	0.66*	—						
ΔAHI (events per hour)	−0.15	−0.53*	—					
ΔWeight (kg)	−0.40	−0.49*	0.25	—				
ΔBMI ($kg \cdot m^{-2}$)	−0.26	−0.48*	0.38	0.92*	—			
ΔTotal body fat (%)	−0.36	−0.32	0.11	0.64*	0.41	—		
ΔTrunk fat (%)	−0.26	−0.28	0.08	0.59*	0.38	0.96*	—	
ΔVO_{2max} ($mL \cdot kg^{-1} \cdot min^{-1}$)	−0.07	0.25	−0.11	−0.24	0.14	−0.27	−0.25	—
ΔAI (%)	−0.47*	−0.57*	0.40	0.20	0.12	0.45	0.34	−0.38

Data are presented as Spearman rank correlations.
* $P < 0.05$.

meaningful comparisons. However, our group-averaged data obscured some interesting observations, namely, that the change in AI was inversely related to the change in nocturnal desaturation but not to the changes in AHI, body weight, or aerobic capacity. Although this correlation was observed in a relatively small sample, it suggests that beneficial cardiovascular outcomes accrued through an exercise and diet program may be influenced to a greater extent by improvements in the severity of hypoxemia rather than the frequency of disordered breathing events or the change in body weight. It also implies that vascular impairment that exists in many patients with OSA may not be the cause or consequence of physical inactivity or cardiovascular deconditioning. Supporting this line of reasoning are data from Drager et al. (10) showing that treatment of OSA with CPAP improves arterial stiffness without affecting body weight. The reason why the change in AI was more strongly associated with hypoxemia rather than AHI is not immediately clear. Yet, chronic intermittent hypoxia with reoxygenation is thought to be similar to repeated ischemia–reperfusion damage with increased production of reactive oxygen species (27). This increased oxidative stress leads to increased expression of proinflammatory cytokines and adhesion molecules that are believed to activate monocytes, lymphocytes, and endothelial cells, resulting in vascular dysfunction. We believe that our data could have implications for how we design and implement therapies to reduce CVD risk in OSA especially with regard to lifestyle programs. While exercising and losing weight will undoubtedly have major implications for cardiovascular morbidity, concomitant use of CPAP to treat underlying OSA (1) may allow for exercise training and weight loss to result in more beneficial cardioprotective outcomes.

This study arose out of an initiative with the ultimate goal of ameliorating frailty and thereby prolonging independent living for older adults. Data from the Cardiovascular Health Study show an independent association between severe sleep disordered breathing and one or more indicators of frailty (12). On the surface, our data may seem to add little to this finding, as our cohort consisted of relatively healthy, but overweight, older adults with a history of snoring and daytime sleepiness (as determined by the Berlin questionnaire). However, while the frailty syndrome is typically viewed by lay persons as synonymous with underweight, given the rising prevalence of obesity among older adults (13) and that an obese frail phenotype has already been recognized (6), the most common phenotype of frailty in the future may be that of an obese disabled person. Whether underlying OSA accelerates the onset of frailty and disability remains to be determined, but we argue that the therapeutic approach with the largest clinical effect is one that targets those who are not yet frail but are at high risk for this syndrome.

Several limitations of our study deserve comment. First, we lacked a nonintervention control group. Second, the experimental design did not allow us to compare the effectiveness of diet or exercise alone versus a combined diet and exercise intervention on improvements in OSA. Because this is an understudied cohort, our ultimate decision to include a lifestyle group incorporating diet and exercise is based on our hypothesis that we would see the most change in OSA severity after the intervention and, therefore, we will be able to establish effect sizes that can, in turn, be used to power a larger study. In addition, from a clinical application perspective, dietary interventions are most effective for weight loss and maintenance when physical activity/exercise is added. Third, the issue of whether OSA attenuates the reduction in weight or improvements in vascular function is clinically important but one that we could not explore given the current design. To address whether underlying OSA moderates weight loss or improvements in cardiovascular function will require a non-OSA control. Fourth, we used very strict selection criteria. Accordingly, our results may not be easily extrapolated to older adults with comorbidities. Fifth, the study was relatively short and we cannot be certain of the physiological effects of a longer trial. Moreover, it is not clear whether individuals' willingness to persevere with this type of intervention extends beyond 12 wk, although we have reported high compliance rates among older adults who were participating in a similar exercise regimen for 6 months (38).

CONCLUSIONS

The role of lifestyle interventions on OSA severity in older adults has received little attention in the literature. This study has provided two unique findings. First, we observed that OSA severity is improved in a sample of older men and women after a weight loss intervention consisting of structured exercise and dietary change. Second, we found that a change in arterial distensibility is more closely associated with the change in nocturnal desaturations than with changes in apneic events, body weight, or aerobic capacity.

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The authors report no conflicts of interest.

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