Sleep Breath (2011) 15:49–56
DOI 10.1007/s11325-009-0311-1

ORIGINAL ARTICLE

The effect of exercise on obstructive sleep apnea: a randomized and controlled trial

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Received: 17 July 2009 / Accepted: 14 October 2009 / Published online: 7 November 2009

Abstract
Purpose The aim of the study was to assess the effect of breathing and physical exercise on pulmonary functions, apnea-hypopnea index (AHI), and quality of life in patients with obstructive sleep apnea syndrome (OSAS).
Methods Twenty patients with mild to moderate OSAS were included in the study either as exercise or control group. The control group did not receive any treatment, whereas the exercise group received exercise training. Exercise program consisting of breathing and aerobic exercises was applied for 1.5 h 3 days weekly for 12 weeks. Two groups were assessed through clinical and laboratory measurements after 12 weeks. In the evaluations, bicycle ergometer test was used for exercise capacity, pulmonary function test, maximal inspiratory-expiratory pressure for pulmonary functions, polysomnography for AHI, sleep parameters, Functional Outcomes of Sleep Questionnaire (FOSQ), Short Form-36 (SF-36) for quality of sleep and health-related quality of health, Epworth Sleepiness Scale for daytime sleepiness, and anthropometric measurements for anthropometric characteristics.
Results In the control group, the outcomes prior to and following 12-weeks follow-up period were found to be similar. In the exercise group, no change was found in the anthropometric and respiratory measurements ($P>0.05$), whereas significant improvements were found in exercise capacity, AHI, and FOSQ and SF-36 ($P<0.05$). After the follow-up period, it was shown that improvement in the experimental group did not lead to a statistically significant difference between the two groups ($P>0.05$).
Conclusions Exercise appears not to change anthropometric characteristics and respiratory functions while it improves AHI, health-related quality of life, quality of sleep, and exercise capacity in the patients with mild to moderate OSAS.

Keywords Mild to moderate obstructive sleep apnea syndrome · Breathing exercise · Physical exercise

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Introduction

Obstructive sleep apnea (OSA) can be described as a condition characterized by repetitive obstruction of the upper airway resulting in oxygen desaturation and awakening from sleep, loud snoring, and increased daytime sleepiness [1]. Many studies have shown that a link exists between OSA and cardiovascular disease, chronic heart failure ischemia, hypertension, obesity, and impaired glucose tolerance [2, 3]. A number of factors are likely to play role in development of clinical OSA syndrome (OSAS) ranging from upper airway anatomy to central respiratory control mechanisms. The pathophysiology of OSA is unclear and complex. Several previous studies have explored pulmonary function in the OSAS patients [4, 5]. Interestingly, OSAS has been found to be highly correlated

with lower airway obstruction, although it is originally defined as an upper airway disease [4]. In the recent studies, metabolic factors, especially inflammatory cytokines (IL-1, IL-6, and TNF-α) are implicated for leading to this condition because of its systemic interactions [6, 7]. Another important effect of pro-inflammatory cytokines is to cause collapse by leading inflammation in dilator muscles in the upper airway. Thus, the observed hypercytokinemia, hyperleptinemia, and hyperinsulinemia/visceral adiposity, through central and peripheral effects, may lead to a collapse of the upper airway during sleep [7, 8].

The most commonly accepted interventions in the treatment of OSA include administration of continuous positive airway pressure (CPAP) and oral appliances during sleep and surgery [9–11]. American Sleep Apnea Association considers exercise as a non-pharmacological treatment modality of sleep disorders [12]. Theoretical reviews and hypotheses on the effects of exercise in OSA have suggested thermoregulatory, metabolic, and biochemical mechanisms although clinical trials on the topic are inadequate [6, 7, 12, 13].

Hargens et al. [14] indicated that alteration in chemoreflex sensitivity and breathing efficiency exists in patients with OSA. Exercise studies showed that an improvement occurs in respiratory drive (and chemoreceptor sensitivity) after physical exercise in athletes. Netzer et al. [15] reported a significant improvement in apnea-hypopnea index (AHI) following a 6-month exercise program in the patients with moderate to severe OSAS. They suggested that improvement of OSA severity might be due to a possible rise in the respiratory drive or a stabilized muscle tonus in the upper airway after exercise. Nevertheless, Norman et al. [16] noted that regular exercise training provided improvement in sleep by decreasing body weight. Consequently, the studies have shown that regular physical activity reduces severity of OSA symptoms either by decreasing body weight or through positive effects on the respiratory muscles and sufficiency of breathing (ventilatory abilities) [13, 15–17].

Our study was planned to assess the effects of physical and breathing exercise training on pulmonary function, AHI, and quality of life in order to investigate the place of exercise in management of OSAS.

Methods

Subjects

Study protocols and written informed consents were approved by the institutional review committee on clinical research of the Dokuz Eylul University School of Medicine. The inclusion criteria for this study included men 40 to 65 years of age, in good general health (stability of clinic state), with OSAS symptoms (snoring, breathing cessations, and daytime sleepiness), and polysomnographic evidence (AHI, sleep efficiency percentage, minimum saturation percentage, and total sleep time) consistent with mild ($5<$ AHI<15) to moderate ($16<$AHI<30) OSAS. Medical conditions that would make exercise dangerous such as angina pectoris, congestive heart failure, cardiomyopathy, emphysema, lung cancer, recent upper respiratory surgery, chronic obstructive pulmonary disease, or other serious medical problems such as neurological, psychological, and cooperation problems that would prevent successful participation in and completion of the protocol by the subject served as exclusion criteria.

The participants were divided into control and study groups according to the table of random numbers. The control group did not receive any treatment, the study group received exercise training. Both groups underwent similar clinical and physiotherapeutic assessment. In addition, the control group was not advised any information and/or exercise apart from routine clinical treatment and proposals. In contrast to the control group, the study group received breathing exercise (approximately; 15–30 min) and aerobic exercises (approximately; 45–60 min) lasting progressively 1–1.5 hour three times weekly for 12 weeks.

Exercises were given by a single physiotherapist at the same time and place for 12 weeks. Exercises were taught to the patients repetitively until they said "I understood". The patients were enabled to get used to the exercises by giving them exercise booklets and requiring them to participate with sports outfits. Several data such as the patients' respiration rates, heart rates, and blood pressures were recorded before and after the exercises. Participants' dyspnea severity and leg tiredness were evaluated with Modified Borg Scale [18]. The exercises were maintained under the control of a physiotherapist.

Breathing exercises Exercises were started with the pursed lips breathing training in which the patient is taught to inhale the air through the nose and exhale slowly by slightly opening his/her lips. During entire breathing exercises, relaxation training and its importance were explained to the patients in order to minimize contribution on the shoulder girdle and neck muscles to breathing. The patients were placed in appropriate position during diaphragmatic and thoracic expansion exercises. For kinesthetic stimulation, the patient was asked to place his/her hand on the relevant pulmonary areas to increase the amount of expiration through resistance in the inspiration period and through pressure in the expiration period. All exercises were combined with postural exercises. Exercise program was progressed according to fatigue severity of patients. Breathing exercises, which had been done in

sitting position, were done resistively in flat position by the help of gravity and 250-g weights [19, 20].

Aerobic exercises The height of the bicycle was adjusted specifically for each patient. After the breathing exercises, the patients did warm up exercises consisted of slow jogging, calisthenics, and stretching. Then they did aerobic exercises, resistance, and duration of which were increased according to the patients' tolerance on bisergo and treadmill. The training program began at a low to moderate intensity over the first 1–2 weeks and progressed to a moderate intensity program. During the treadmill and bicycle exercises, which were applied at submaximal intensity at 60–70% of maximal oxygen consumption, it was ensured that the intensity of fatigue that the patients perceived is at the interval of 4–5 according to the Modified Borg scale. During the exercises, the Palco Laboratories Model 400 pulse oxymeter has been used in order to observe the heart rate and peripheral oxygen saturation. After the bicycle and treadmill exercises, the exercise program was finished with the cooling down period which was consisted of low-tempo walking, posture, and stretching exercises. It was ensured in all exercises to maintain respiration control [21, 22].

Patients suitable for inclusion criteria were asked if they had smoking and exercise habits. Anthropometric measurements, pulmonary functions, exercise capacity, quality of sleep, and health-related quality of health were repeatedly measured at the end of week 12 in order to describe the effects of exercise treatments in patients with OSA.

Anthropometric data

Anthropometric measurements including height, weight, and circumference measurements were obtained before and after the study. From the height (m) and weight (kg) measurements, the body mass index (BMI) was calculated (kg/m^2). Circumference measurements were made with a flexible tape and included neck, upper chest, waist, and hip circumferences. The waist and hip girths were used to calculate the waist-to-hip ratio for each individual. A single investigator performed all measurements.

Skinfold thickness were measured on the right side of the body with a caliper (Holtain Tanner/Whitehouse skinfold caliper; Holtain Ltd, UK) to the nearest millimeter. To assess subcutaneous fat, the skinfold thickness of the following sites was measured: subscapular, triceps, and chest in accordance with procedures described by The American College of Sports Medicine. From the skinfold thickness, body density was calculated using the predictive equations proposed by Jackson and Pollock. Relative body fat was estimated from the equations proposed by Brozek and colleagues [21].

Pulmonary function testing

Subjects underwent pulmonary function testing to evaluate the lung functions. Spirometry was performed by an expert using a Sensormedics Vmax 22 machine (SensorMedics Inc., Anaheim, CA, USA) conforming to the American Thoracic Society criteria [23]. Forced vital capacity, first second forced expiratory volume (FEV_1), and FEV_1/FVC values were recorded.

Respiratory muscle strength Mouth pressures were measured by Sensormedics Vmax 22 (SensorMedics Inc., Anaheim, CA, USA) using a previously reported technique [24]. Inspiratory muscle strength ($P_i$max) was measured from functional residual capacity, whereas expiratory muscle strength ($P_E$max) was measured near total lung capacity. Tests were run three times (at the beginning of the study period, after 3 months, and at the end of the study period), and three measurements were made in each testing session.

Exercise testing

The subjects exercised on an electronically braked cycle ergometer (ercometrics 800, ergoline, Germany) by "Maximal incremental cycle ergometry protocols". This protocol consists of 3 min of rest, followed by 3 min of unloaded pedaling and by incremental load (3 W per 10 s) until reaching maximal load [25]. Criteria for terminating exercise testing include the patient reaching volitional exhaustion or the test has been terminated by the medical monitor. Pretest and posttest dyspnea severity and leg tiredness were evaluated with Modified Borg Scale [18].

Quality of life and sleep

Quality of life was assessed by two questionnaires as OSA specific measures and a general health-related quality of life questionnaire. Functional Outcomes of Sleep Questionnaire (FOSQ), which has been developed specifically for patients with sleep disorders leading to excessive sleepiness. FOSQ-tr was applied in a decreased 26-item Turkish version without the sexual functioning subscale [26].

The general health-related quality of life was assessed using the Short Form-36 (SF-36) questionnaire. As well as a transition question, the SF-36 consists of eight multi-item dimensions, which are physical functioning, role physical (role limitations due to physical problems), vitality, social functioning, role emotional (role limitation due to emotional problems), bodily pain, general health, and mental health. Each of the dimensions is scored from 0 to 100, with higher scores indicating better health-related quality of life. The

SF-36 was administered during a face-to-face interview by the physiotherapist [27].

Daytime sleepiness evaluation

The subjective sleepiness was assessed using Turkish Epworth Sleepiness Scale (ESS). The ESS is a questionnaire containing eight items that shows the likelihood of dozing during typical daytime activities. The dozing probability ranges from 0 (never) to 3 (high probability). Normal values range from 2 to 10, with scores >10 indicating daytime sleepiness [28].

Polysomnography

Polysomnography (PSG) evaluation of at least 8 h was performed on Embla A 10 (Flaga, Reyjavick, Iceland) and Schwarzer Comlab 32 polysomnographic device (Comlab 32; Schwarzer Medical Diagnostic Equipment, Baermannstr, Germany) sleep systems. The following variables were monitored: four channel EEG (C_3/A_2-C_4/A_1-O_1/A_2-O_2/A_1 according to the 10–20 international electrode placement system), right and left electrooculogram, chin electromyogram, and electrocardiogram. Airflow was monitored by nasal pressure cannula. Respiratory movements were assessed by thoracic and abdominal strain gauges. Snoring was evaluated with neck microphone. The oxygen saturation during sleep was measured continuously using a pulse oxymetry. Leg movements were recorded by left and right tibial electromyograms. PSG recordings were scored according to the standard criteria of Rechtschaffen and Kales in 30-s epochs [29].

Data analysis

Statistical analyses of data obtained before and after the treatment were performed with "SPSS for Windows Ver 11.0" software. Wilcoxon signed rank test was used to compare the pre- and post-treatment data of the study groups and chi-square test to compare determined changes and Mann–Whitney *U* test to compare the groups. Statistical significance level was chosen as p values less than 0.05.

Results

Twenty-five patients were randomized as exercise and control group and were screened. Twenty patients completed the trial. Data were presented for the control group (CG; $n=10$ patients) and exercise group (EG; $n=10$ patients). All of the ten patients in the exercise group participated the training. No problems occurred during the exercises.

Characteristics of the subjects

Baseline characteristics of the subjects for each group are presented in Table 1. There were no significant differences in height and weight ($P>0.05$). Patients in CG (48.0± 7.49 years) were slightly younger than those in the EG (54.40±6.57 years; $P<0.05$).

In regard to exercise habits of the subjects, seven subjects in the exercise group (70%) and nine subjects in the control group (90%) were found not to physically exercise at all. No statistically significant difference was found between the control and exercise groups in exercise and smoking habits ($\chi^2=1.25$, $P=0.26$; $\chi^2=2.80$, $P=0.25$; Table 1).

Physical changes

BMI, body fat percentage (BF percentage), skinfold thickness, neck and waist girth measures, and waist-to-hip ratios were similar in both groups at the baseline and the end of the 12-week program ($P>0.05$). The patients' anthropometric parameters did not change significantly during the exercise period ($P>0.05$; Table 2).

Respiratory parameter results

At the baseline, there were no significant differences in all of the pulmonary function tests, except FEV_1/FVC ratio, $P_i$max,

Table 1 Baseline subject characteristics of patients with OSAS

	Exercise group $n=10$	Control group $n=10$	P value
Age (year)	54.40 (6.57)	48.0 (7.49)	0.04[a]
Height (cm)	170.40(6.17)	176.60(5.40)	0.40[a]
Weight (kg)	86.40(8.04)	88.47(16.24)	0.70[a]
Percentage of current smoker	30.0	30.0	0.25[b]
Percentage of Never	60.0	30.0	
Percentage of former Smoker	10.0	40.0	
Regular exercise	30	10	0.26[b]
Not regularly exercise	70	90	

Data are presented as mean± standard deviation

BMI body mass index

[a] P values were derived by Mann–Whitney *U* test

[b] P values were derived by chi-square test

Table 2 Anthropometric changes

	Exercise group			Control group			*P***
	Pre	Post	*P**	Pre	Post	*P**	
BMI (kg/m2)	29.79 (2.66)	29.20 (3.07)	0.17	28.42 (5.42)	28.28 (5.52)	0.89	0.41
Neck circumference (cm)	41.15 (1.53)	42.15 (2.79)	0.31	41.30 (3.47)	41.60 (3.10)	0.47	0.54
Waist circumference (cm)	104.25 (5.51)	104.45 (6.85)	0.88	103.5 (14.83)	101.25 (11.57)	0.21	0.16
Waist-hip ratio	0.97 (3.33)	0.97 (3.24)	0.79	0.96 (6.30)	0.96 (5.59)	0.96	0.33
Body density	1.03 (9.38)	1.04 (7.7)	0.17	1.04 (7.23)	1.04 (5.65)	0.29	0.50
Body fat percentage (%)	28.41 (4.01)	26.87 (3.27)	0.17	24.84 (3.06)	25.81 (2.40)	0.29	0.53

Data are presented as mean±standard deviation

BMI body mass index

**P*<0.05, Wilcoxon signed rank test

***P*<0.05, Mann–Whitney *U* test

and $P_e$max between the EG and CG. There were increases in FVC percentage and FEV_1/FVC ratio, but unchanged in $P_i$max-$P_e$max and respiratory parameters after exercise. All pulmonary parameters were the same in both groups ($P<0.05$), while the FEV_1/FVC ratio was significantly higher in the CG on follow-up ($P>0.05$; Table 3).

Responses to the exercise testing

Dyspnea severity after the test was higher in the exercise group than in the control group at first exercise test ($P<0.05$). Significant improvements in $MaxVO_2$, metabolic equivalent of task (MET), and maximal work load were noted on follow-up exercise testing in the exercise group ($P<0.05$). The dyspnea and leg fatigue severity was the same before and after the exercise program. However, after the exercise test, the dyspnea and leg fatigue severity were significantly decreased ($P<0.05$). In the control group, they did not change after exercise test at week 12 ($P>0.05$). In control evaluation of the subjects included and not included in the exercise program, maximal tolerated load was significantly higher and dyspnea severity perceived following the test was significantly lower in the exercise group ($P<0.01$; Table 3).

Quality of life and sleep outcomes

Baseline SF-36 and ESS scores (Table 4) were same in both groups ($P>0.05$). However, activity level scores of the

Table 3 Results of pulmonary function tests and the exercise test

	Exercise group			Control group			*P***
	Pre	Post	*P**	Pre	Post	*P**	
FEV_1% predictive	104.3 (10.65)	96.60 (17.57)	0.13	112.50 (22.03)	103.40 (25.00)	0.11	0.62
FVC% predictive	108.50 (12.06)	98.60 (18.90)	0.05	105.80 (16.85)	99.40 (21.92)	0.11	0.94
FEV_1/FVC%	77.0 (4.69)	78.80 (3.80)	0.04	85.50 (4.93)	83.70 (4.74)	0.19	0.05
P_E (cmH_2O)	122.40 (16.60)	117.40 (23.37)	0.67	113.00 (33.40)	113.00 (33.40)	0.58	0.68
P_I (cmH_2O)	91.90 (31.46)	90.20 (31.51)	0.88	89.20 (36.22)	96.90 (44.88)	0.14	0.97
$MaxVO_2$ (mL/min)	15.38 (3.58)	17.48 (5.63)	0.02	16.6 (4.87)	18.37 (3.52)	0.31	0.47
MET	4.40 (1.00)	5.37 (1.04)	0.02	4.76 (1.40)	5.27 (1.01)	0.26	0.47
Maximum work load (W)	112.50 (35.54)	155.00 (18.11)	0.02	141.50 (35.90)	147.50 (25.50)	0.31	0.40

Data are presented as mean±standard deviation

FEV1 forced expiratory volume in 1 s as a percent of the predicted, *FVC* forced vital capacity as a percent of the predicted, *FEV1/FVC* forced expiratory volume in the first second as a percent of the predicted value/forced vital capacity, $P_i max$ maximal inspiratory pressure, $P_e max$ maximal expiratory pressure, *Max VO2* maximal oxygen consumption, *MET* metabolic equivalents

**P*<0.05, Wilcoxon signed rank test

***P*<0.05, Mann–Whitney *U* test

Table 4 Results of the polysomnographic and intra- and inter-group differences for the control group and exercise group following the intervention period

	Exercise group			Control group			*P***
	Pre	Post	*P**	Pre	Post	*P**	
AHI	15.19 (5.43)	11.01 (5.28)	0.02	17.92 (6.45)	17.36 (11.18)	0.58	0.11
Sleep efficiency%	80.58 (6.34)	86.35 (13.97)	0.14	89.05 (5.96)	88.30 (9.53)	0.80	0.91
Saturation%	83.90 (4.53)	83.60 (4.48)	0.94	82.50 (5.44)	84.03 (4.50)	0.51	0.94
Total sleep time (min)	361.30 (55.27)	361.16 (79.99)	0.80	388.15 (57.27)	351.82 (89.64)	0.29	0.76
ESS	8.20 (6.14)	7.00 (6.65)	0.40	3.42 (507)	5.30 (4.191)	0.43	0.65

Data are presented as mean±standard deviation

ESS Epworth Sleepiness Scale, *AHI* apnea hypopnea index

*$P<0.05$, Wilcoxon signed rank test

**$P<0.05$, Mann–Whitney *U* test

FOSQ-tr were low when the exercise group was compared to the control group ($P<0.05$).

The exercise intervention significantly improved SF-36 (vitality and mental health domains) and FOSQ-tr (activity level subscale; $P<0.05$). In the control group, they unchanged after quality of life and sleep at week 12 ($P>0.05$). FOSQ-tr, SF-36, and ESS scores (Table 4) were not significantly different between control and exercise groups after 12 weeks ($P>0.05$).

Polysomnography

Means and standard deviation of selected sleep parameters for both groups are shown in Table 4. There was not a significant difference between groups. Total sleep efficiency was higher the control group than in the exercise group ($P<0.05$). Significant improvements in AHI were noted on follow-up polysomnograms in the exercise group ($P<0.05$). AHI was unchanged in the control group while it was similar in both groups at the end of 12 weeks ($P>0.05$; Table 4).

Discussion

This randomized, controlled trial has shown that supervised exercise training reduced the severity of OSA. Although AHI decreased the body weight, BMI and other anthropometric characteristics remained unchanged. Increase in exercise capacity was less in the control group that was younger with better quality of life at baseline. For the patients with OSA, with reduced physical activity, we found increase in exercise capacity, decrease in leg tiredness and severity of dyspnea perceived following exercise, and improvements in quality of life and sleep parameters of mental health, activity level, and vitality.

Especially anatomy of upper airway is affected by obesity while opinions exist that metabolic activity of the visceral (central) fat tissue plays role in development of sleep apnea [7]. Thus, we determined the sample of our study as males in whom visceral fat formation occurs frequently.

Giebelhaus et al. [30] found that no change occurred in BMI and body weight of the patients with OSA included in exercise program for 6 months consisting of power and aerobic exercises. Similarly, Peppard et al. [31] showed that exercise habits of different extent had no effect on body shape and composition.

Opinions in the literature are conflicting on that exercise in the patients with OSAS generates changes in anthropometric features and BMI and, thus, lead changes in AHI [16, 17, 30, 31]. Type, frequency, and duration of given exercise differed with no consensus being reached among a few studies on the effect of exercise on OSA [16, 30]. We found that supervised exercise of respiratory and aerobic type for 12 weeks had no effect on BMI, body fat percentage, neck, waist, and hip circumferences, and waist-to-hip ratio in our prospective, randomized, controlled study on the effect of mild to moderate OSAS.

Norman et al. [16] showed that improvements occurred in MET levels and resting blood pressures of the patients with mild to moderate OSAS receiving aerobic exercise and diet program for 6 months. Giebelhaus et al. [30] noted that exercise performance did not change with the program consisting of aerobic and power exercises in the patients with severe OSAS.

The fact that neither type of exercise nor patients' groups were standardized prevented the exercise outcomes to become clear. In the present study, respiratory and aerobic exercises we applied for 3 months increased $MaxVO_2$, MET, and maximal load value. We found decrease in leg tiredness and dyspnea severity as important determination

of exercise capacity. Changes occurred in sleep parameters with increases in determination of exercise capacity without changes in breathing reserve during exercise testing. We believe that these improvements in sleep parameters result from increase in functional capacity contributing to improvement in exercise capacity.

It has been reported that CPAP treatment improves ESS, mental vitality, and social function domains of SF-36, and especially "activity level" and "vigilance" subscales of FOSQ [32, 33]. Decrease in daytime sleepiness with increased level of physical activity has been shown to improve quality of sleep and life, especially vitality and physical role [16, 17]. Studies considered this result as a positive effect of decreased body weight on quality of life and sleep. The present study showed that applied exercise treatment did not change ESS score but significant improvements occurred in vitality and mental health subscales of SF-36. At the baseline, “Activity level” score and total score of FOSQ were better in the control group than in the exercise group. This remained unchanged in the control group while changed in the exercise group. We felt that as with CPAP treatment and improved physical activity, efficient sleep occurred; and this improved vital activities such as work productivity and vitality.

Although a number of the studies looking at the effects of chronic exercise on AHI are low, there are many hypotheses and theoretical reviews [6, 7, 12]. Basic hypotheses proposed by Driver and Taylor [12] are those of thermogenic, conversation of the energy, and body restoration and are that these mechanisms affect sleep-awake cycle and slow wave sleep [13]. Netzer and colleagues [15] studied the effects of exercise in the patients with sleep apnea based on the idea that chemoreceptor sensitivity enhanced following physical exercise in the athletes, and, thus, breathing improves. As a result of their study, they advocated that decrease in AHI resulted from improved muscle tonus of the upper airway or improved breathing although body weight did not change. Similarly, Giebelhaus et al. [30] suggested that possible cause of decrease in AHI, without change in body weight following 6-month exercise training they give to the patients with sleep apnea syndrome, was increased strength of pharyngeal and glossal muscles. Another hypothesis alternative to decreased severity of OSAS through improved tonus of tongue muscles is that strength of the respiratory muscles, i.e., inspiratory and expiratory pressures increase with exercise training [34]. Based on the study by O’Donnel et al., Norman et al. [16] noted that decrease could occur in apnea and hypopnea with increasing inspiratory and expiratory pressure although they had no enough data on the topic. In contrast to Norman et al., we measured strength of inspiratory and expiratory muscles and similar to the study by O’Donnell et al.. We found that exercise did not alter strength of ventilatory muscles although we give breathing exercises to our study group.

In conclusion, similar to the studies by Netzer [15] and Giebelhaus [30], the present study found decreased AHI without change in body weight following exercise in the patients with OSAS. We, however, do not believe that decreased AHI might be due to changes in the muscle groups of upper airway or improved breathing control as Netzer et al. [15] advocated because aerobic and breathing exercises we applied in our study did not affect $P_i$max, $P_e$max and other respiratory parameters.

It has been shown that the patients with OSA with regular physical activity have lower AHI [17, 31]. Several studies exists reporting that exercise does not change total sleep time [14, 30] despite others that found improvements in AHI, total sleep time, and sleep efficiency with exercise in the patients with OSA [16]. Thus, polysomnographic results of a few studies on effects of supervised exercise training are controversial. Decrease in AHI has been found in all studies while improvement in total sleep time has been found in only one study. Similar to all other studies, we found in our study that no change occurred in the control group although significant decreases were found in the group receiving regular exercise. Total sleep time and other polysomnographic results did not change after exercise. Although we determined in the first follow-up that the control group had more efficient sleep percentage than the exercise group, no difference was found after the follow-up between the two groups because it increased in the exercise group.

In recent years, "Metabolic hypothesis" has been proposed in explaining the effects of exercise on quality of sleep. In addition, inflammation of the upper airway related to the visceral fat tissue appears as a systemic condition [7, 34]. It has been advocated that plasma concentrations of the cytokines leading to airway collapse and inflammation alters, and their receptor antagonists increase through exercise [6, 34]. In conclusion, exercise training we applied to the patients with OSAS surprisingly found decreased AHI despite no changes in breathing function and BMI. The resultant decrease was supported by improved exercise capacity. Thus, an effect of exercise in OSAS that has been frequently discussed as a metabolic condition in the literature is to make possible metabolic changes by increasing which lowered functional activity level. It will be possible to resolve the discussion through investigations on plasma levels and further studies with more patients.

Our study is limited by the small sample size and by inclusion criteria. Due to the known effects of age and sex on OSA, interval of age was one of the inclusion criteria and only males were included in the study and this caused the study to have a small sample size. The situation has

been the same in similar studies which used elaborated protocols and advanced examinations.

Conflict of interest statement There is no related to the article or the research described and personal or financial support or author involvement with organizations with a financial interest in the subject matter.

References

1. McNamara SG, Grunstein RR, Sullivan CE (1993) Obstructive sleep apnea. Thorax 48:754–764
2. Parish JM, Somers VK (2004) Obstructive sleep apnea and cardiovascular disease. Mayo Clinic Proc 79:1036–1046
3. Levinson PD, McGarvey ST, Carlisle CC, Eveloff SE, Herbert PN, Millman RP (1993) Adiposity and cardiovascular risk factors in men with obstructive sleep apnea. Chest 103:1336–1342
4. Zerah-Lancner F, Lofaso F, Coste A, Ricolfi F, Goldenberg F, Harf A (1997) Pulmonary function in obese snorers with or without sleep apnea syndrome. Am J Respir Crit Care Med 156:522–527
5. Öztürk L, Metin G, Cuhadaroğlu C, Utkusavaş A, Tutluoğlu B (2005) FEF (25–75)/FVC measurements and extrathoracic airway obstruction in obstructive sleep apnea patients. Sleep Breath 9 (1):33–38
6. Santos RV, Tufik S, De Mello MT (2007) Exercise, sleep and cytokines: is there a relation? Sleep Med Rev 11:231–239
7. Vgontzas AN, Bixler EO, Chrousos GP (2005) Sleep apnea is a manifestation of the metabolic syndrome. Sleep Med Rev 9 (3):211–224
8. Boyd JH, Petrof BJ, Hamid Q, Fraser R, Kimoff RJ (2004) Upper airway muscle inflammation and denervation changes in obstructive sleep apnea. Am J Respir Crit Care Med 170:541–546
9. Lim J, Lasserson TJ, Fleetham J, Wright J (2006) Oral appliances for obstructive sleep apnoea. Cochrane Database Syst Rev 25(1): CD004435
10. Sundaram S, Bridgman SA, Lim J, Lasserson TJ (2005) Surgery for obstructive sleep apnoea. Cochrane Database Syst Rev 19(4): CD001004
11. Giles TL, Lasserson TJ, Smith BH, White J, Wright J, Cates CJ (2006) Continuous positive airways pressure for obstructive sleep apnoea in adults. Cochrane Database Syst Rev 19(3):CD001106
12. Driver HS, Taylor SR (2000) Exercise and sleep. Sleep Med Rev 4(4):387–402
13. Youngstedt SD, O'Connor PJ, Dishman RK (1997) The effects of acute exercise on sleep: a quantitative synthesis. Sleep 20(3):203–214
14. Hargens TA, Guill SG, Aron A, Zedalis D, Gregg JM, Nickols-Richardson SM, Herbert WG (2009) Altered ventilatory responses to exercise testing in young adult men with obstructive sleep apnea. Respir Med 103(7):1063–1069
15. Netzer N, Lormes W, Giebelhaus V, Halle M, Keul J, Matthys H, Lehmann M (1997) Physical training of patients with sleep apnea. Pneumologie 51:779–782
16. Norman JF, Von Essen SG, Fuchs RH, McElligott M (2000) Exercise training effect on obstructive sleep apnea syndrome. Sleep Res Online 3(3):121–129
17. Hong S, Dimsdale JE (2003) Physical activity and perception of energy and fatigue in obstructive sleep apnea. Med Sci Sports Exerc 35(7):1088–1092
18. Weiser PC, Mahler DA, Ryan KP, Hill KL, Greenspon LW (1993) Dyspnea: symptom assessment and management. In: Hodgkin JE, Conors GL, Bell CW (eds) Pulmonary rehabilitation: guidelines to success, 2nd edn. JB Lippincott Company, Philadelphia, pp 478–511
19. Nici L, Donner C, Wouters E et al (2006) American Thoracic Society/European Respiratory Society statement on pulmonary rehabilitation. Am J Respir Crit Care Med 173(12):1390–1413
20. Bianchi R, Gigliotti F, Romagnoli I, Lanini B, Castellani C, Binazzi B, Stendardi L, Grazzini M, Scano G (2004) Chest wall kinematics and breathlessness during pursedlip breathing in patients with COPD. Chest 125(2):459–465
21. Dwyer GB, Davis SE (2005) ACSM's health-related physical fitness assessment manual. Lippincott Williams &Wilkins, Philadelphia
22. American College of Sports Medicine (2003) The ACSM Fitness Program. ACSM fitness book. Human Kinetics, Hong Kong
23. American Thoracic Society (1995) Standardization of spirometry, 1994 update. American Thoracic Society. Am J Respir Crit Care Med 152:1107–1136
24. Black LF, Hyatt RE (1969) Maximal respiratory pressures: normal values and relationship to age and sex. Am Rev Respir Dis 99:696–702
25. American Thoracic Society (2003) ATS/ACCP statement on cardiopulmonary exercise testing. Am J Respir Crit Care Med 167:211–277
26. Izci B, Firat H, Ardıc S, Kokturk O, Gelir E, Altınors M (2004) Adaptation of Functional Outcomes of Sleep Questionnaire (FOSQ) to Turkish population. Tuberk Toraks 52:224–230
27. Brazier J, Harper R, Jones N, O'Cathain A, Thomas KJ, Usherwood T, Westlake L (1992) Validating the SF-36 health survey questionnaire: new outcome measure for primary care. BMJ 305(6846):160–164
28. Johns MW (1991) A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. Sleep 14(6):540–545
29. Rechtschaffen A, Kales A (eds) (1968) A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects. Brain Research Institute, UCLA, Los Angeles
30. Giebelhaus V, Strohl KP, Lormes W, Lehmann M, Netzer N (2000) Physical Exercise as an adjunct therapy in sleep apnea—an open trial. Sleep Breath 4(4):173–176
31. Peppard PE, Young T (2004) Exercise and sleep-disordered breathing: an association independent of body habitus. Sleep 27 (3):480–484
32. Moore P, Bardwell WA, Ancoli-Israel S, Dimsdale JE (2001) Association between polysomnographic sleep measures and health-related quality of life in obstructive sleep apnea. J Sleep Res 10(4):303–308
33. Marshall NS, Barnes M, Travier N, Campbell AJ, Pierce RJ, McEvoy RD, Neill AM, Gander PH (2006) Continuous positive airway pressure reduces daytime sleepiness in mild to moderate obstructive sleep apnoea: a meta-analysis. Thorax 61(5):430–434
34. O'Donnell DE, McGuire M, Samis L, Webb KA (1998) General exercise training improves ventilatory and peripheral muscle strength and endurance in chronic airflow limitation. Am J Respir Crit Care Med 157:1489–1497