SLEEP BREATHING PHYSIOLOGY AND DISORDERS • ORIGINAL ARTICLE

Effects of 12 weeks of regular aerobic exercises on autonomic nervous system in obstructive sleep apnea syndrome patients

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Abstract

Introduction Regular exercise is confirmed as a lifestyle treatment option for all obstructive sleep apnea (OSA) patients. It has beneficial effects other than weight loss, although the mechanisms remain unclear. Autonomic function imbalance plays an important role in OSA, so that it is meaningful to observe the effect of exercise on autonomic function.

Methods Seventy mild to moderate OSA patients were divided into two groups. The exercise group received a 12-week exercise program prescribed according to their first cardiopulmonary exercise tests, while the control group kept previous lifestyle. All patients underwent blood tests, cardiopulmonary exercise tests, and polysomnography studies at enrollment and at the 12-week's follow-up.

Results At the end of 12 weeks, three patients of the exercise group did not complete the program due to lack of adherence. The current study showed 12-week aerobic exercises could improve body mass index (27.6 ± 4.7 kg/m^2 vs. 24.5 ± 4.2 kg/m^2, $P < 0.05$), exercise capacities, apnea-hypopnea index (total AHI 20.2 ± 7.5 vs. 16.4 ± 5.2, $P < 0.05$; supine AHI 22.1 ± 6.3 vs. 18.3 ± 4.9, $P < 0.05$), average oxyhemoglobin saturation (AverSpO2), time/percentage SpO2 below 90%, and heart rate recovery (HRR) of OSA patients. Moreover, AverSpO2 change was significantly associated with HRR change in the exercise group.

Conclusions Our findings suggested regular aerobic exercise had beneficial effects on body mass index, functional capacity, intermittent hypoxia, and parasympathetic tone of OSA patients, and whether parasympathetic tone modification plays a role in improving intermittent hypoxia or not deserves further exploration.

Keywords Exercise · Obstructive sleep apnea · Heart rate recovery · Therapy

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Introduction

Obstructive sleep apnea (OSA) is a common clinical condition in which repetitive episodes of partial or complete occlusion of the upper airway occur during sleep. These episodes lead to intermittent hypoxia (IH) and arousals from sleep. OSA is diagnosed by polysomnography (PSG), which is a test that allows the calculation of the apnea-hypopnea index (AHI), the ratio of the total number of apneas, and hypopneas to total sleep time. The severity of OSA is determined by the AHI: 5.0–14.9 events/h, mild OSA; 15–30 events/h, moderate OSA; and ≥ 30 events/h, severe OSA [1]. Growing evidence has indicated that OSA is independently correlated with metabolic dysregulations such as diabetes, arterial hypertension, hypercholesterolemia, obesity, increased risk of cardiovascular events as well as atherosclerosis [2–4]. The underlying mechanisms are still unclear, but some fundamental features have been identified, such as increased sympathetic activation,

endothelial dysfunction, oxidative stress, and possibly systemic inflammation [5].

Recommended therapy, such as continuous positive airway pressure (CPAP) treatment, oral appliance therapy, and upper airway surgery can relieve symptoms; especially, CPAP still remains the first-line therapy for severe and moderate OSA patients. Data from small trials provide evidence that CPAP treatment improves not only patient-reported outcomes such as sleepiness, quality of life, and mood but also intermediate cardiovascular end points such as blood pressure, cardiac ejection fraction, vascular parameters, and arrhythmias. However, data from large-scale randomized controlled trials do not consistently support a role for reducing cardiovascular mortality [6]. The American Academy of Sleep Medicine recommends exercise as a lifestyle treatment option for all OSA patients [7]. Recent studies have focused on exercise programs because they constitute a low-cost easy-to-use treatment modality, and have been shown to be effective in mitigating several harmful consequences of OSA, including AHI severity [8], glucose intolerance [9], and inflammatory profiles [10].

The mechanisms whereby physical exercise attenuates OSA have not yet to be well defined. Several hypotheses have been proposed: increasing upper airway dilator muscle tone, increasing slow-wave sleep, reducing fluid accumulation in the neck, reducing body weight, and reducing the systemic inflammatory response [11]. As we know, autonomic dysfunction is closely associated with cardiovascular disease development. Previous research also revealed autonomic dysfunction in OSA patients who belong to a specific population with a high risk of developing cardiovascular diseases. Different indexes were used in those studies, such as sympathetic skin responses from the neck [12] and multi-unit muscle sympathetic nerve activity [13] to represent sympathetic tone, heart rate variability to represent sympathovagal modulation [14], and heart rate recovery (HRR) at 1 min after exercise termination to represent vagal tone [15]. However, there are few data to indicate whether there is improvement in autonomic dysfunction in OSA patients after regular aerobic exercise training. Moreover, personalized patient management is encouraged in OSA patients. In this respect, we prescribed a 12-week exercise program based on the patient's cardiopulmonary exercise test (CPET) performance to observe if there was a beneficial effect on autonomic dysfunction. We adopted serum norepinephrine (NE) levels and HRR to represent sympathetic nervous tone and parasympathetic nervous tone, respectively. We also tried to investigate if there is any association with any other improvement in OSA patients, which would be very meaningful to explore underlying pathways.

Patients and methods

Study population

Seventy patients were consecutively enrolled as they were referred to the sleep laboratory in Tongji Hospital to be examined for suspected sleep apnea. The inclusion criteria comprised newly diagnosed mild to moderate OSA (AHI 5 to 30/h with clinical symptoms) and rejection of CPAP therapy, none of whom received surgical or mechanical ventilation treatment prior to inclusion. The exclusion criteria were patients with diabetes mellitus, hypertension, coronary artery disease (angina and/or electrocardiogram signs of ischemia on treadmill-exercise test), peripheral arterial disease, thyroid disorder, severe ventricular arrhythmia, severe reduction of LVEF (left ventricular ejection fraction, LVEF ≤ 45%), valvular disease requiring surgery, severe renal dysfunction (i.e., creatinine > 2.5 mg/dl), severe orthopedic problems that would prohibit exercise, history of psychiatric or neurodegenerative disorders, acute systemic illness, or circadian desynchrony (e.g., shift workers). The enrolled OSA patients were assigned by alternate randomization in a 1:1 ratio into two groups, stratified by age and sex. One group received 12 weeks of supervised exercise training (exercise group), and the other was the control group who maintained their previous lifestyle. Our study met the ethical standards, and it was also approved by the Ethics Committee of Tongji Hospital. All patients gave their written informed consent.

Laboratory measurement

A venous blood sample was collected from each participant under fasting condition. Fasting blood glucose, total cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides, and creatinine were measured using standard laboratory methods. Serum NE level was determined by sandwich immunoassay with an ELISA-kit (IBL, Germany), according to the manufacturer's methods.

Polysomnographic data collection

Polysomnographic recording was performed using Alice 6 (Philips, Netherlands). All participants underwent 7-h nighttime monitoring with PSG in bed. No alcohol or caffeine consumption was permitted during the study. Apnea was defined as being without respiration for more than 10 s. Hypopnea was defined as the deduction of at least 50% ventilation causing a drop in arterial saturation of at least 4%. OSA could be viewed as apnea or hypopnea occurring at least five times per hour, persisting for more than 10 s. The AHI documents the number of apnea-plus-hypopnea incidents every hour during sleep and was summarized by body position.

Total AHI and supine AHI were recorded. The oxygen desaturation index (ODI) assesses the average number of oxygen desaturation incidents every hour during sleep. Oxyhemoglobin saturation during wakefulness ($SpO2_{wakefulness}$), average oxyhemoglobin saturation (AverSpO2), minimum oxyhemoglobin saturation (MinSpO2), arousal index, and time/percentage SpO2 below 90% were collected.

Cardiopulmonary Exercise Test

All OSA patients underwent an incremental Cardiopulmonary Exercise Test (CPET) on a bicycle ergometer at the beginning of the study and 12 weeks later. To stabilize respiratory exchange, patients were asked to remain still on the ergometer for at least 3 min before starting the exercise. CPET was performed according to a symptom-limited Bruce's protocol with continuous electrocardiographic monitoring, and cuff blood pressure was manually recorded every 2 min. Blood pressure was recorded at rest, at the end of each stress stage, at peak stress, and at recovery. The test was stopped for any of the following reasons: a rating of perceived exertion > 17 (Borg scale), achievement of > 90% of age-predicted maximum heart rate (HRmax = 220 − age), if the subject was too fatigued to continue the test safely, systolic blood pressure > 200 mmHg, typical chest discomfort, severe arrhythmias, or more than 1 mm horizontal or down-sloping ST segment depression. Respiratory gas exchange measurements were obtained breath-by-breath with the use of a computerized metabolic monitor (Innocor 5.0, Innovision Cor., Denmark). VO2peak was recorded as the mean value of VO_2 during the last 20 s of the test and expressed in ml × kg^{-1} × min^{-1}. Power$_{max}$ was the maximal workload during exercise. The ventilatory anaerobic threshold (AT) was assessed mathematically, and VO2 at AT (VO2$_{AT}$) was recorded. After achievement of peak exercise, the test was almost immediately terminated while the subjects were in a seated position. The HRR was obtained by subtracting the HR at the first minute of recovery from the peak HR obtained during the exercise. Therefore, the subjects in our CPET did not go through a "cool-down" phase. It was reported that the value of HRR might be affected by the small workload during the "cool-down" phase, decreasing its diagnostic sensitivity [16, 17]. The whole process was supervised by a cardiologist and a nurse who were unaware of the subject's clinical history.

Training protocol

Exercise group patients attended exercise training three times per week. Training sessions, performed under continuous electrocardiogram monitoring, were supervised by a cardiologist and a nurse. Each session was preceded by a 15-min warm-up and followed by a 15-min cool-down. Exercise

was performed for 30 min on a bicycle ergometer at VO2AT (using target HR as a scale), which was determined at the initial symptom-limited CPET.

Statistics

Descriptive statistics were reported as mean values ± SD. Comparison between groups for continuous variables were made using a *t* test or one-way analysis of variance where appropriate. Correlations were determined with the Pearson's or Spearman's correlation tests as appropriate. Differences between the two groups and changes over time within each group were assessed by two-way repeated measures ANOVA. A *P* value below 0.05 was considered statistically significant. All statistical analyses were performed using the software package SPSS, version 16.0 (SPSS Inc., Chicago, USA).

Results

During the course of the study, three patients did not complete the exercise program due to lack of adherence to the training regimen. No statistically significant differences were found at baseline between the exercise group and the control group in clinical, demographic, PSG characteristics as well as CPET indexes (Table 1).

At the end of 12 weeks, we found the patients' exercise capacities (VO2peak, Powermax, AT, and VO2$_{AT}$) elevated and body mass index decreased in the exercise group (*P* < 0.05, Table 2), and there was no change in the control group. Moreover, HRR in the exercise group increased significantly (*P* < 0.01, Table 2), while there was no significant change in NE level in either group.

As for those PSG parameters, it was found that AHI (total AHI and supine AHI) and time percentage SpO2 below 90% decreased significantly, while AverSpO2 increased significantly in the exercise group. But there was no obvious change in ODI, MinSpO2, and arousal index in either group (Table 2). In the exercise group, there was a significant positive correlation between the change of AverSpO2 and the change of HRR (*r* = 0.217, *P* < 0.01, Fig. 1).

Discussion

A growing amount of published data has confirmed that regular physical exercise could have valid benefits to OSA patients, including improving both sleeping quality and some cardiovascular risk factors. To our knowledge, there is a lack of studies evaluating the effects of regular aerobic training on OSA patients in China. The current study showed that 12 weeks of aerobic exercise could improve the exercise capacities, body mass index, AHI, AverSpO2, time percentage

Table 1 Baseline clinical characteristics of the exercise group and the control group

	Control group ($n=35$)	Exercise group ($n=32$)	P value
Age (years)	48.6 ± 7.2	46.3 ± 6.4	0.419
Male/female	24/11	22/10	0.360
BMI (kg/m^2)	27.1 ± 3.5	27.6 ± 4.7	0.227
Neck circumference (cm)	38.2 ± 4.1	39.3 ± 5.6	0.504
Waist circumference (cm)	115.4 ± 16.7	117.3 ± 18.4	0.268
Waist-to-hip ratio	0.93 ± 0.08	0.92 ± 0.07	0.577
Smokers (n %)	18(51.4%)	17(53.1%)	0.475
FBG (mmol/L)	5.4 ± 0.7	4.9 ± 0.8	0.143
TG (mmol/L)	2.4 ± 0.5	2.5 ± 0.6	0.612
TC (mmol/L)	5.9 ± 1.0	5.6 ± 1.2	0.149
HDL-C (mmol/L)	1.1 ± 0.2	1.2 ± 0.3	0.305
LDL-C (mmol/L)	3.4 ± 0.7	3.6 ± 0.9	0.241
Cr (umol/L)	76.4 ± 10.7	68.3 ± 12.9	0.542
NE (ng/L)	293.6 ± 24.8	284.7 ± 26.1	0.332
HRR (b/min)	17.7 ± 5.6	18.1 ± 6.4	0.267
SpO$_{2wakefulness}$ (%)	97.0 ± 2.2	97.2 ± 2.4	0.605
Total AHI (events/h)	19.5 ± 6.1	20.2 ± 7.5	0.164
Supine AHI (events/h)	21.7 ± 5.8	22.1 ± 6.3	0.238
ODI (events/h)	12.4 ± 7.9	13.0 ± 8.2	0.415
AverSpO$_2$ (%)	87.1 ± 8.2	88.6 ± 7.4	0.461
MinSpO$_2$ (%)	81.5 ± 12.4	79.1 ± 13.6	0.378
Arousal index (events/h)	27.6 ± 9.3	28.1 ± 10.5	0.607
Time/percentage below 90% SpO$_2$ (%)	7.8 ± 1.4	7.6 ± 1.7	0.429
VO$_{2peak}$ (ml $\times$ kg $\times$ min^{-1})	29.5 ± 6.7	28.4 ± 7.1	0.328
Power$_{max}$ (W)	68.9 ± 8.4	67.3 ± 9.1	0.810
AT (W)	59.1 ± 7.2	58.9 ± 8.3	0.141
VO$_{2AT}$ (ml $\times$ kg $\times$ min^{-1})	26.1 ± 5.1	25.3 ± 5.2	0.203

BMI, body mass index; *FBG*, fasting blood glucose; *TG*, triglyceride; *TC*, total cholesterol; *HDL-C*, high-density lipoprotein cholesterol; *LDL-C*, low-density lipoprotein cholesterol; *Cr*, creatinine; *NE*, norepinephrine; *SpO$_{2wakefulness}$*, oxygen saturation during wakefulness; *HRR*, heart rate recovery; *AHI*, apnea-hypopnea index; *ODI*, oxygen desaturation index; *AverSpO$_2$*, average of oxygen saturation; *MinSpO$_2$*, minimum oxygen saturation; *AT*, anaerobic threshold; *VO$_{2AT}$*, VO$_2$ at anaerobic threshold

SpO2 below 90%, and HRR of OSA patients. Moreover, the change in AverSpO2 was significantly associated with the change in HRR in the exercise group. Those findings suggested that the beneficial effects of exercise on parasympathetic tone might be partly responsible for improvement of IH in OSA patients, which is a vital pathway deserving further exploration.

According to the American Academy of Sleep Medicine, CPAP should be the first-line treatment for moderate to severe OSA (AHI > 15 events/h). However, many patients struggle with this therapy, for which adherence remains unacceptably low [18, 19]. Changes in lifestyle habits, including weight loss, regular physical activity, discontinuation or replacement of drugs that directly interfere with upper airway muscle function, reducing alcohol consumption, and smoking cessation are always encouraged in all OSA patients [7]. Among those lifestyle modifications,

regular exercise has gradually gained great attention because clinical trials have illustrated its beneficial effects other than weight loss. Kline et al. enrolled 43 patients with moderate OSAS and carried out a 12-week supervised exercise program [20]. They found reduced AHI and ODI. Similarly, it was reported that AHI, functional outcomes of sleep questionnaire (FOSQ) scores, and short form-36 (SF-36) scores for quality of sleep and health-related quality of life were improved after 12 weeks of exercise training (1.5 h, 3 days weekly) in mild to moderate OSA patients [21]. In the current study, we found that 12 weeks of exercise training decreased AHI and increased AverSpO2. Our sample size, OSAS severity of enrolled subjects and exercise duration were similar to those previous investigations. Recently, a meta-analysis of nine studies revealed that exercise as the sole treatment for OSA could lead to reduction in the AHI and in daytime sleepiness [22].

Table 2 CPET and PSG parameters, clinical characteristics at baseline and after 12 weeks in two patient groups

	Control group			Exercise group			Differences in changes between groups
	Baseline	12 weeks' follow-up	P value	Baseline	12 weeks' follow-up	P value	P value
BMI (kg/m^2)	27.1 ± 3.5	26.8 ± 3.7	0.614	27.6 ± 4.7	24.5 ± 4.2	0.023	0.018
Neck circumference (cm)	38.2 ± 4.1	39.1 ± 4.4	0.305	39.3 ± 5.6	37.4 ± 5.9	0.716	0.829
Waist circumference (cm)	115.4 ± 16.7	111.6 ± 18.3	0.081	117.3 ± 18.4	115.2 ± 20.6	0.305	0.214
Waist-to-hip ratio	0.93 ± 0.08	0.92 ± 0.07	0.614	0.92 ± 0.07	0.91 ± 0.07	0.683	0.415
FBG (mmol/L)	5.4 ± 0.7	5.1 ± 0.9	0.127	4.9 ± 0.8	4.8 ± 1.0	0.216	0.343
TG (mmol/L)	2.4 ± 0.5	2.2 ± 0.5	0.754	2.5 ± 0.6	2.3 ± 0.8	0.429	0.582
TC (mmol/L)	5.9 ± 1.0	6.0 ± 1.2	0.516	5.6 ± 1.2	5.4 ± 0.9	0.147	0.651
HDL-C (mmol/L)	1.1 ± 0.2	1.2 ± 0.3	0.207	1.2 ± 0.3	1.3 ± 0.3	0.513	0.748
LDL-C (mmol/L)	3.4 ± 0.7	3.7 ± 0.9	0.115	3.6 ± 0.9	3.2 ± 1.1	0.821	0.537
Cr (umol/L)	76.4 ± 10.7	78.2 ± 11.6	0.328	68.3 ± 12.9	70.2 ± 12.4	0.248	0.516
NE (ng/L)	293.6 ± 24.8	289.5 ± 26.7	0.153	284.7 ± 26.1	280.1 ± 27.6	0.360	0.401
HRR (b/min)	17.7 ± 5.6	18.1 ± 6.2	0.215	18.1 ± 6.4	21.9 ± 8.1	0.008	0.009
SpO$_{2wakefulness}$ (%)	97.0 ± 2.2	96.8 ± 2.3	0.327	97.2 ± 2.4	97.5 ± 1.6	0.169	0.215
Total AHI (events/h)	19.5 ± 6.1	20.1 ± 7.0	0.429	20.2 ± 7.5	16.4 ± 5.2	0.032	0.020
Supine AHI (events/h)	21.7 ± 5.8	21.8 ± 6.1	0.505	22.1 ± 6.3	18.3 ± 4.9	0.024	0.011
ODI (events/h)	12.4 ± 7.9	12.6 ± 5.8	0.527	13.0 ± 8.2	11.4 ± 8.3	0.062	0.134
AverSpO2 (%)	87.1 ± 8.2	86.5 ± 7.4	0.297	88.6 ± 7.4	91.2 ± 9.3	0.044	0.032
MinSpO2 (%)	81.5 ± 12.4	81.8 ± 13.7	0.109	79.1 ± 13.6	79.9 ± 12.4	0.341	0.258
Arousal index (events/h)	27.6 ± 9.3	28.5 ± 8.7	0.263	28.1 ± 10.5	27.2 ± 11.6	0.183	0.092
Time/percentage SpO$_2$ below 90% (%)	7.8 ± 1.4	7.5 ± 1.2	0.384	7.6 ± 1.7	6.1 ± 1.5	0.031	0.027
VO$_{2peak}$ (ml × kg × min^{-1})	29.5 ± 6.7	29.4 ± 8.1	0.714	28.4 ± 7.1	32.1 ± 8.5	0.208	0.016
Power$_{max}$ (W)	68.9 ± 8.4	69.1 ± 9.0	0.526	67.3 ± 9.1	70.4 ± 10.2	0.041	0.039
AT (W)	59.1 ± 7.2	58.8 ± 6.9	0.367	58.9 ± 8.3	61.6 ± 9.6	0.015	0.024
VO$_{2AT}$ (ml × kg × min^{-1})	26.1 ± 5.1	26.8 ± 7.6	0.192	25.3 ± 5.2	28.1 ± 7.3	0.026	0.017

BMI, body mass index; *FBG*, fasting blood glucose; *TG*, triglyceride; *TC*, total cholesterol; *HDL-C*, high-density lipoprotein cholesterol; *LDL-C*, low-density lipoprotein cholesterol; *Cr*, creatinine; *NE*, norepinephrine; *HRR*, heart rate recovery; *SpO$_{2wakefulness}$*, oxygen saturation during wakefulness; *AHI*, apnea-hypopnea index; *ODI*, oxygen desaturation index; *AverSpO$_2$*, average of oxygen saturation; *MinSpO$_2$*, minimum oxygen saturation; *AT*, anaerobic threshold; *VO$_{2AT}$*, VO$_2$ at anaerobic threshold

As an optimal exercise program for OSA treatment should identify the type, frequency, and intensity of exercise; length of program; duration of individual session; and number of sessions per week, we prescribed exercise based on each OSAS patient's CPET performance in an effort to approach personalized therapy. As we expected, the exercise capacity of the exercise group was improved at the 12-week follow-up. VO2peak is considered to be one of the best measurements of cardiovascular fitness. It was reported that supervised exercise enhanced the VO2peak/exercise capacity not only in mild to moderate OSAS patients [23] but also in severe OSA patients [24], even in OSA patients with heart failure [25]. After 12 weeks of training, we observed enhanced VO2peak, Powermax, AT, and VO2AT, which suggested that both exercise capacity and aerobic metabolism were improved, because AT represented the point at which anaerobic metabolism started with production of lactic acid. The

reasons for the increases in exercise parameters were out of our study scope and we will try to explore it in subsequent studies.

Epidemiological research indicates that OSA is associated with the incidence and progression of cardiovascular diseases [6]. Although the underlying mechanisms are still not well defined, some common traits, including autonomic nervous system imbalance, endothelial dysfunction, and oxidative stress are confirmed as contributors to this increased risk [5]. As CPAP treatment is accepted as a classic therapeutic modality for moderate and severe OSA patients, earlier clinical observations have proved its beneficial effects on autonomic functions in this specific population [26–28]. Likewise, whether exercise training would bring about a favorable influence on autonomic nerve imbalance deserves our attention. Serum NE level, a typical sympathetic index, was used in our study. However, we did not find any change in NE levels in either of those two groups. It was reported that 1 month of
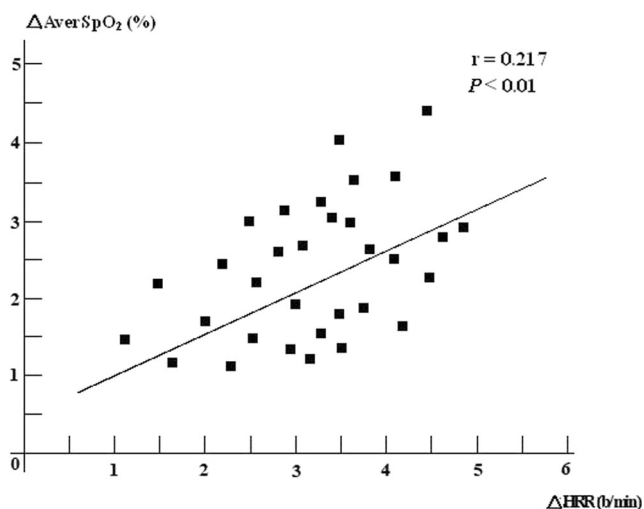
Fig. 1 Significant positive correlation between the change of AverSpO2 and the change of HRR ($r = 0.217$, $P < 0.01$)

CPAP treatment resulted in a significant reduction in 24-h urinary NE levels only in severe OSA patients, not in patients with mild-moderate OSA [29]. As for parasympathetic tone assessment, we used the index of HRR, which quantifies the decrease in heart rate following exercise. It is a useful straight-forward method and a highly reproducible tool. Slower HRR has been identified as a powerful prognostic parameter for mortality in cardiovascular patients [30, 31]. Maeder et al. reported that the severity of OSAS, expressed as higher AHI, was independently associated with lower HRR [15]. Meanwhile, a change in HRR after exercise training was found in patients with chronic obstructive pulmonary disease [32], type 2 diabetes mellitus [33], and chronic heart failure [34]. Kline et al. reported that blunted HRR was improved following 12 weeks' exercise training in overweight OSA patients and AHI change was associated with change in HRR at 5-min post-exercise [20]. Similarly, we found a significant increase in HRR in the exercise group at the end of the 12-week follow-up. Moreover, the enhancement of AverSpO2 was significantly associated with the increase in HRR, which suggested that the improvement of ANS induced by exercise might be an important pathway of ameliorating IH in OSA patients. As we know, IH is a key feature of OSA. Our findings were so encouraging that further studies enrolling more patients are needed to clarify the underlying mechanisms.

Certain limitations to the current study should be considered. First, this was an observational and a single-center study in which only a small number of OSA patients were available for analysis; second, we used the NE level as the measure of sympathetic activity. Although NE was previously shown to be elevated in OSA patients [35], it has limitations, in particular its nonspecificity. Therefore, we were cautious in our interpretation that sympathetic activity was unaffected after exercise training; third, we did not collect subjective sleep quality data, such as the Epworth Sleeping Scale (ESS) and

Pittsburgh Sleep Quality Index (PSQI). Those subjective parameters were included to evaluate the effects of regular exercises in earlier reports; fourth, we used the subjects' single night of laboratory PSG performances at baseline and at 12 weeks' follow-up, which likely introduced additional variability in measures of OSA severity [36]. Another limitation of our study was that we did not observe any correlation between HRR change and any other PSG index. The causative mechanisms of the only significant correlation between HRR change and AverSpO2 change were difficult to hypothesize.

Conclusions

In conclusion, 12 weeks of aerobic exercise induced a significant improvement in the body mass index, AHI, AverSpO2, time percentage SpO2 below 90%, and HRR in OSA patients. Furthermore, the increase in AverSpO2 was positively associated with HRR change. These findings suggested that regular personalized aerobic training could reverse OSA severity to a certain extent. Modification of the automatic nervous system might be a vital pathway for the exercise effect on IH of OSA patients, which will be explored in future research.

Compliance with ethical standards

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