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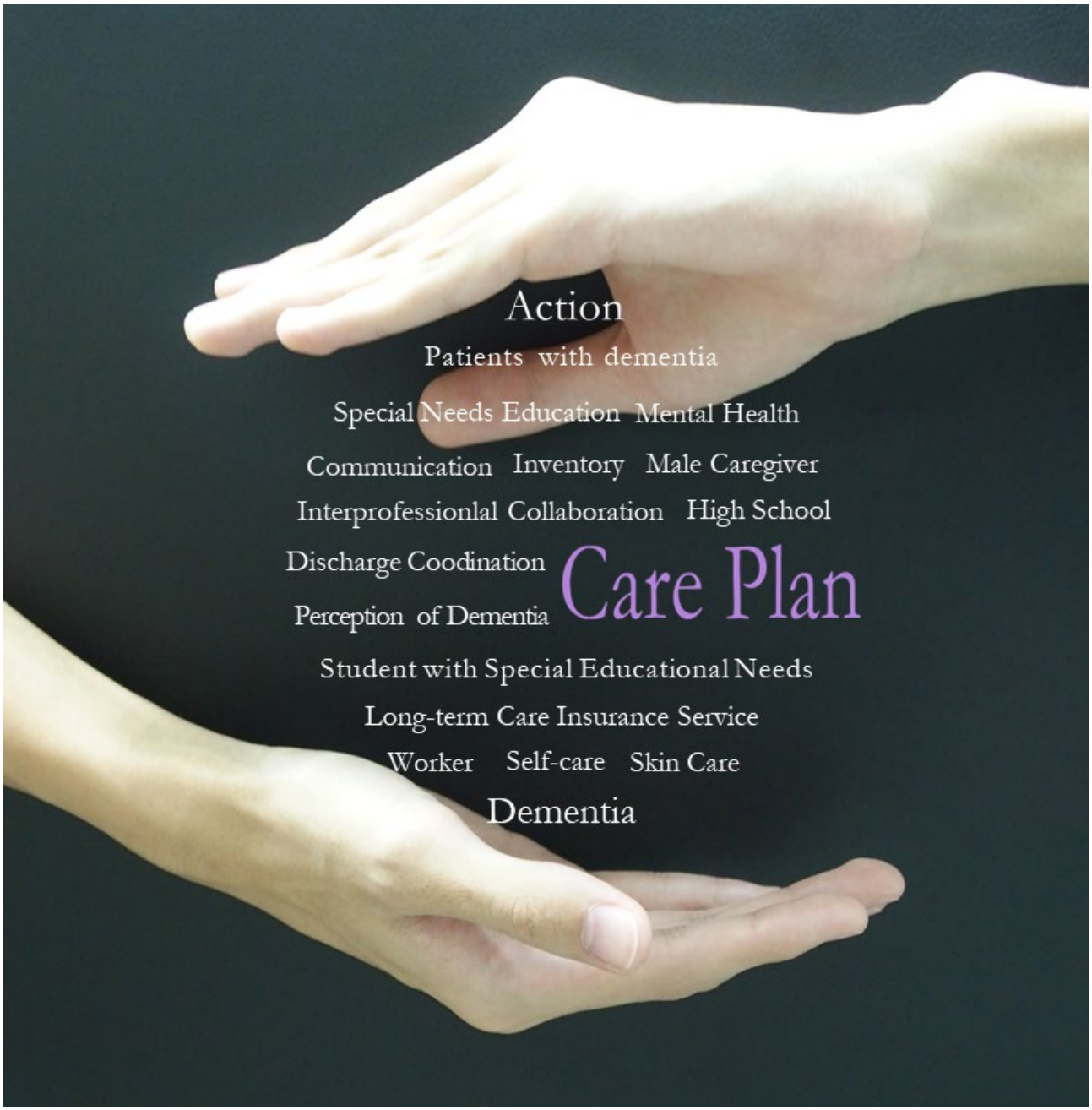

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REVIEW ARTICLE

Effect of Weight Loss and Exercise Therapy on Obesity-related Respiratory Disorders

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ABSTRACT

Obesity is an established risk factor for various diseases and weight loss is important to improve these comorbidities. Although exercise is effective to suppress obesity, obesity-related respiratory disorders are likely deterrents of physical activity. Therefore, it is important to clarify the role of exercise therapy in the treatment of obesity, especially the extent to which it is valid for obesity-related respiratory disorders.

Weight loss, consequent to body fat reduction, is crucial for the improvement in obesity-related respiratory disorders. In addition, exercise therapy has been suggested to be effective in reducing dyspnea on exertion and sleep apnea or hypopnea, independent of the effects of weight loss. Furthermore, exercise therapy is expected to improve peak oxygen uptake (peak VO_2) and reduce the risk of cardiovascular disease and mortality. Irrespective of diet therapy or surgical procedures towards weight reduction, a comprehensive program combined with exercise therapy may be effective for obese patients with respiratory disorders.

＜Key-words＞
obesity-related respiratory disorders, weight loss, exercise, sleep apnea, comprehensive program

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I. Obesity and weight loss therapy

Obesity is a condition wherein the excess energy of an individual accumulates over a certain level as fat, and is defined to be BMI≧30 kg/m^2 as per the WHO (World Health Organization) standards. The prevalence of obesity has increased worldwide over the decade, which is now not only a personal health complication but also a serious socio-economic problem.

Obesity is known to be a risk factor for various diseases such as hypertension, type 2 diabetes mellitus, dyslipidemia, cardiovascular disease, orthopedic disease, and respiratory disease.

Weight loss is important to improve obesity-related comorbidities. Various methods that are employed to lose weight includes diet, exercise therapy, drug therapy, behavioral therapy, and surgical treatment. A comprehensive care involving a designated medical team is of utmost importance in mitigating the perils associated with obesity.

Exercise therapy not only increases energy consumption but also has several consequences such as improved exercise tolerance, better coronary circulation, augmented skeletal muscle function, enhanced insulin sensitivity, and controlled inflammation. (Shuler, Hambrecht, Schlierf, 1992. Stefanyk and Dyck, 2010. Kasapis and Thompson, 2005).

It has been reported earlier, that an increase in physical activity suppresses obesity (Koh-Banerjee, Chu, Spiegelman et al., 2003). We note, however, that obesity-related respiratory disorders such as dyspnea on exertion, hypoxemia due to obesity hypoventilation syndrome (OHS), and daytime sleepiness due to sleep apnea syndrome (SAS) are potential barriers towards the improvement in physical activity. Therefore, it is important to clarify the role of exercise therapy in the treatment of obesity, especially the extent to which it is valid for obesity-related respiratory disorders.

II. Obesity related respiratory disorders

Various respiratory disorders occur with the progression of obesity. Specifically, the effects of obesity on respiratory function have been reported to decrease lung volume, reduce respiratory compliance, increase respiratory rate, and increase airway resistance (Littleton, 2013). In particular, it is known that a decrease in expiratory reserve volume (ERV) and functional residual capacity (FRC) among lung volumes is related to the degree of obesity (Jones & Nzekwu, 2006) (Figure 1). In obese individuals, fat deposits around the ribs, around the diaphragm and in the abdominal cavity reduce respiratory thorax compliance. In severe obesity, tidal volume (TV) decreases due to decreased chest wall compliance and decreased lung volume, thereby increasing the respiratory rate which tends to be shallow with fast breathing. In addition, the airway diameter decreases due

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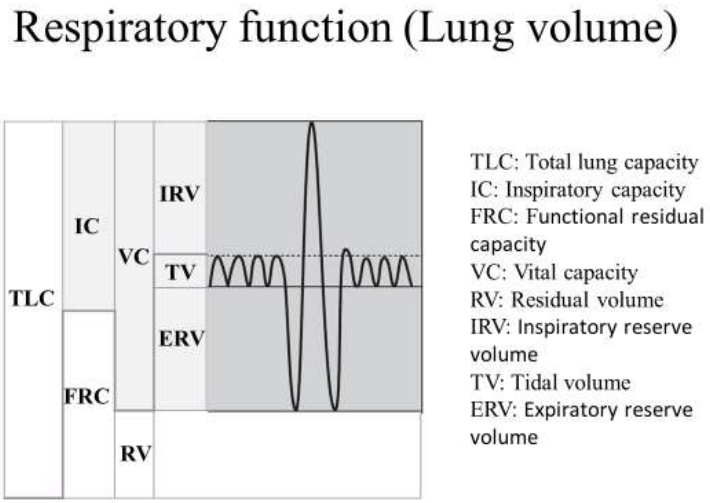
to a decrease in lung volume, indicating an increase in airway resistance (Brazzale, Pretto, Schachter, 2015).

Among respiratory diseases, OHS and obstructive sleep apnea (OSA) are closely related to obesity. As obesity progresses, fat accumulates around the airway and can directly affect the anatomy of the upper airway, increasing the likelihood of airway collapse (Jordan, McSharry, Malhotra, 2014). Hypoxia and reoxygenation by OSA (intermittent hypoxemia) and disruption of sleep are physiological stresses, which might play an important role in the development of insulin resistance and hypertensive cardiovascular disorders (Arnardottir, Mackiewicz, Gislason et al., 2009).



<Figure 1> Lung volume parameters

III. Effect of weight loss on obesity-related respiratory dysfunction

There are several studies on the effects of weight loss on respiratory function (Table 1). Surgical treatment enables massive weight loss in morbid obese patients and improves respiratory function significantly (Santana, Souza, Martins et al., 2006; Hewitt, Humerfelt, Sovik et al., 2014; Campos, Peixoto-Souza, Alves et al., 2017). Conversely, in diet therapy (calorie restriction), although the weight loss range is mild, respiratory function parameters such as ERV and FEV1 (forced expiratory volume in one second) have improved with weight loss (Lorenzo, Luca, Sasso et al.,1999; Aaron, Fergusson, Dent et al.,2004).

The correlation between the improvement in respiratory function and changes in body weight have been reported in several studies (Held, Mittnacht, Kolb et al., 2014; Takahashi, Ebihara, Kohzuki, 2017; Santana, Souza, Martins et al., 2006), suggesting that the improvement in respiratory function is influenced by the improvement of

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mechanical load due to weight and adiposity loss. The reduction in chest wall fat and abdominal fat might have enhanced chest wall compliance and thoracic mobility with a concomitant downward displacement of the diaphragm, possibly leading to improved respiratory function.

<Table 1> Effect of weight loss on respiratory function in obese patients

	Authors (year)	Characteristics of patients	Weight loss Intervention	Weight outcomes	Lung function Outcomes	Correlation
Diet	Lorenzo et al. (1999)	16 obese patients (8 males, 8 females) Age 36±11 years mean BMI 32.1 kg/m²	Mediterranean diet 3 months Restricted hypocaloric diet ,calculated individually based on resting energy expenditure	Weight (kg) 89.70 to 82.1 Fat mass(kg) 35.92 to 29.01 Lean body mass(kg) 50.84 to 50.15 Waist/hip ratio 0.92 to 0.88	VC(L) 4.19 to 4.47 IC(ml) 3.08 to 2.99 ERV(ml) 1.16 to 1.53 FEV1(ml) 3.36 to 3.51 MVV(l/min) 123.53 to 141.21	Before the diet, a significant correlation was observed between lean body mass and VC,FEV1,PEF,ERV,MVV.
	Aaron et al. (2004)	58 obese women Age 44±13 years mean BMI 43.1 kg/m²	Calorie restriction (900kcal/day) 6 months	Weight (kg) 115.3 to 20kg reduction (17.4% reduced of their pretreatment weight)	FVC 92mL, FEV1 73mL improved for every 10% relative loss of pretreatment weight	Change FEV1 and relative weight loss (r=0.29,P=0.04) Change FVC and relative weight loss (r=0.27,P=0.057)
Exercise + Diet	Babb et al. (2011)	9 males Age 37±5 years mean BMI 35 kg/m²	Supervised 12-week diet and exercise(aerobic and resistive)weight-loss program	Weight (kg) 112 to 104 Body fat (%) 38 to 35 Waist/hip ratio 0.98 to 0.96	FVC(L) 5.03 to 5.21 FVC(%) 97 to 102 FEV1 (%) 92 to 95 EELV↑, gastric pressure↓ at Ventilatory threshold(VTh)	Change in peak expiratory gastric pressure at VTh and the change in the sum of chest,waist,and hip circumference (r=0.91,P<.01)
	Held et al. (2014)	74 asymptomatic obese subjects (32 males, 42 females) Age 42±12 years mean BMI 43.4 kg/m²	Structured weight reduction Program 1year A follow up examination was performed 4 month after start of the program.	Weight (kg) 125.6 to 99.6 Body fat (%) 45.1 to 37.8 Waist/hip ratio 0.93 to 0.90	VC(%) 98.5 to 104 FEV1 (%) 97.7 to 107.5 TLC (%) 100.7 to 101.1	Change FEV1 and change weight(r=0.31, p=0.03) Change corrected body fat and change VC (r=0.35,P=0.03),FEV1(r=0.44, p<0.01)
	Takahashi et al. (2017)	29 obese patients (14 males, 15 females) Age 42±12 years mean BMI 43.8 kg/m²	Comprehensive Obesity rehabilitation 1-3 months	Weight (kg) 118.7 to 106.7 Body fat (%) 47.8 to 44.9 Musculoskeletal mass(kg) 34.1 to 33.4	VC(L) 3.37 to 3.65 ERV(L) 0.62 to 0.93 FRC(L) 2.14 to 2.40 FEV1(L) 2.66 to 2.93 TLC(L) 4,91 to 5.15	Change in ERV and Change in BW (r=0.74, p<0.01) Change in TLC and Change in BW (r=0.68, p<0.01)
Surgical treatment	Mafort et al. (2014)	52 participants Age 18 to 65 years 40 patients completed the study mean BMI 39.1 kg/m²	Intragastoric balloon 6 months	Weight (kg) 111 to 93.8 Body fat (%) 48.7 to 44.6 Waist circumference (cm) 113 to 105	ERV(L) 0.39 to 0.74 FRC(L) 1.56 to 2.08 FEV1(L) 2.75 to 2.88 TLC(L) 4,42 to 4.68	delta TLC and delta waist circumference (ρ= -0.34,p= 0.03) delta FRC and delta IMC (ρ=-0.39,P=0.01) ・Significant correlation between delta ERV and delta truncal,android,gynoid,total fat.
	Santana et al. (2006)	39 morbid obese BMI 40-59.9 (n=28) BMI≧60 (n=11) Age 38±8 years mean BMI 52.5 kg/m²	Bariatric surgery Spirometry was performed at baseline and 12±4 months Postoperatively	Weight (kg) 137.5 to 35.8 reduction	FVC(%) 93.1 to 105.4 FEV1(%) 92.5 to 104.4	Weight loss and changes in FVC(r=0.526,p=0.001),FEV1(r=0.568,P<0.001)
	Hewitt et al. (2014)	113 morbid obese (33 males, 80 females) Age 40±9 years mean BMI 47.4 kg/m²	Bariatric surgery Spirometry was performed at baseline and 5 years Postoperatively	Weight (kg) male 158 to 38kg reduction female 133 to 42kg reduction	FVC(L): 4.59 to 4.94 (male) 3.63 to 3.84(female) FEV1(L): 3.57 to 3.81 (male) 2.91 to 3.02(female)	FEV1 improved significantly by percent weight reduction. FVC improved by percent weight reduction, male gender and high baseline BMI.
	Campos et al. (2017)	24 females with morbid obesity Age 40±7 years mean BMI 47.4 kg/m²	Bariatric surgery Spirometry was performed at baseline and 6 month Postoperatively	Weight (kg) 124.1 to 92.3 Body fat (%) 44.1 to 36.1 Lean mass(kg) 68.2 to 58.1	SVC(%) 92.2 to 99.8 ERV (L) 0.41 to 0.96 FVC (L) 3.16 to 3.45 FEV1(%) 92.8 to 98.7	delta IRV and fat mass (r=0.73) delta IRV and neck circumference (r=0.76) Delta ERV and neck circumference (r= -0.74)

VC, vital capacity; IC, inspiratory capacity; ERV, expiratory reserve volume; IRV, inspiratory reserve volume; FRC, functional residual capacity; TLC, total lung capacity; FEV1, forced expiratory volume in one second; FVC, forced vital capacity;

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IV. Effect of exercise on obesity-related respiratory dysfunction

When compared with diet therapy alone (Lorenzo, Luca, Sasso et al.,1999; Aaron, Fergusson, Dent et al., 2004), exercise with diet therapy (Babb, Wyrick, Chase et al., 2011; Held, Mittnacht, Kolb et al., 2014; Takahashi, Ebihara, Kohzuki, 2017) display improvement in the parameters of respiratory function as ERV, FEV1 seems to be slightly higher in the exercise combination group.

Babb et al. observed an increase in end-expiratory lung volume during submaximal exercise in obese men after a 12-week diet and exercise program (Babb, Wyrick, Chase et al., 2011). We showed improvement of lung volume parameters after 1-3 month of comprehensive obesity rehabilitation program (Takahashi, Ebihara, Kohzuki, 2017). The exercise therapy in these studies used a combination of aerobic exercise and resistance exercises, which changed body composition through a reduction in body fat, even during short-term interventions. The effect of upper body fat distribution for lung volumes has been described previously (Collins, Hoberty, Walker et al., 1995). Given that exercise intervention significantly reduces abdominal visceral fat (Giannopoulou, Ploutz-Snyder, Carhart et al., 2005), exercise therapy is expected to be effective in improving respiratory function through changes in fat distribution.

In addition, Bernhardt et al. have shown that an aerobic exercise intervention, without weight loss, reduced dyspnea on exertion in obese women (Bernhardt et al, 2016). Since dyspnea on exertion is also a forestalling factor of physical activity in obese patients, reduction of dyspnea is considered important to the improvement of the motivation to continue the exercise therapy.

For the improvement of respiratory function, bariatric surgery that can significantly results in weight loss is considered to be the most effective. However, at the time of significant weight loss, there is a concern about the decrease in muscle mass and exercise capacity (Dereppe, Forton, Pauwen et al., 2019). Combining exercise therapy is expected to improve VO_2peak and reduce the risk of cardiovascular disease and mortality.

V. Effect of weight loss in obese patients with SAS

Table 2 lists the major randomized controlled trial (RCT)s for the effectiveness of medical weight loss for obese patients with OSA. Regardless of whether the diet was based on very low-calorie diet (VLCD) or lifestyle intervention, apnea-hypopnea index (AHI) improved with weight loss (Johansson, Neovius, Lagerros et al., 2009; Foster, Borradaile, Sanders et al, 2009).

The sleep AHEAD study (Foster, Borradaile, Sanders et al, 2009) compared intensive lifestyle intervention group to diabetes support and education group in obese patients with type 2 diabetes. The physical activity in the intensive lifestyle intervention was 175

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minutes a week of moderate-intensity activity. At 1 year, body weight reduced significantly in the intervention group (-10.8kg) associated with AHI reduction (-5.4).

Tuomilehto et al. compared VLCD program with supervised lifestyle modification to routine lifestyle counseling. In the intervention group, the subjects were recommended to increase their daily physical activity and aerobic exercise. In the lifestyle intervention group, body weight was markedly reduced (-10.7kg) associated with AHI reduction (-4.0). Also, OSA was cured in 22 of 35 patients (63%) in the intervention group compared with 13 of 37 patients (35%) in the control group. (Tuomilehto, Seppä, Partinen et al., 2009)

<Table 2> Effect of medical weight loss on obese patients with OSA

Authors (year)	Characteristics of patients	Weight loss Intervention	Weight outcomes	Sleep apnea Outcomes
Foster et al. (2009)	264 participants with type 2 diabetes mild/moderate/severe OSA: 38.7%/35.2%/26.1%	RCT Intervention: Diet + Intensive lifestyle intervention(n=139) Control: Diabetes support education(n=125) 12 months	Intervention vs. Control Weight(kg) -10.8 vs. -0.6. BMI(kg/m^2) -3.8 vs. -0.2	Intervention vs. Control AHI(events/h): -5.4 vs. 4.2 Oxygen desaturation index,≧4%: -5.5 vs. 1.2
Tuomilehto et al.(2009)	72 overweight patients (BMI 28-40) with mild OSA	RCT Intervention: Very low calorie diet(VLCD) with supervised lifestyle counseling(n=35) Control: Single session of counseling with a physician and nurse(n=37) 1year	Intervention vs. Control Weight(kg) -10.7 vs. -2.4 Waist Circumference(cm) -11.6 vs. -3.0	Intervention vs. Control AHI(events/h): -4.0 vs. 0.3 All common symptoms related to OSA, and some features of 15D-QOL improved after the lifestyle intervention
Johansson et al.(2009)	63 obese men(BMI 30-40, age 30-65 years) with moderate to severe OSA (AHI≧15)	RCT Intervention: Very low energy diet(n=30) Control: usual diet(n=33) 9 weeks	Intervention vs. Control Weight(kg) -18.7 vs. 1.1 BMI(kg/m^2) -5.7 vs. 0.3	Intervention vs. Control AHI(events/h): -25 vs. -2 Oxygen desaturation episodes: -19 vs. -1 Epworth sleepiness scale: -3 vs. 1

VI. Effect of exercise on obese patients with SAS

Table 3 lists the RCTs for the effectiveness of exercise for OSA. Most studies showed a reduction in AHI in the exercise group. Moreover, peak VO_2 was increased in the exercise group in the report evaluating exercise tolerance.

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<Table 3> Effect of exercise on obese patients with OSA

Authors (year)	Characteristics of patients	Study design and duration	Exercise Protocol	Weight outcomes	Lung outcomes	Sleep apnea Outcomes	Physical outcomes
Kline et al. (2011)	43 sedentary overweight/obese patients with at least moderate-severity OSA	RCT Intervention: Aerobic and resistance exercise (n=27) Control: stretching (n=16) 12weeks	Aerobic exercise Frequency:4 times/wk Intensity:60% HRR Time:0-4week: exercise dose increase gradually 5-12week:150min/wk Resistance exercise 2 days/wk. 2sets of 10-12 repetitions for 8 different exercise Supervised by trained staff.	Weight (kg) IG:105.6 to 104.7 CG: 99.3 to 98.7 Total body fat(%) IG:42.1 to 41.0 CG:40.6 to 40.8	FVC(L) IG:3.7 to 3.6 CG:3.5 to 3.6 FEV1(L) IG:3.2 to 3.1 CG:3.1 to 3.1	AHI(events/h) IG:32.2 to 24.6 CG:24.4 to 28.9 ODI(events/h) IG:24.5 to 21.5 CG:16.8 to 23.2 Stage N3 sleep(%) IG:12.8 to 13.2	
Sengul et al. (2011)	20 patients with mild to moderate OSA	RCT Intervention: Aerobic and breathing exercise (n=10) Control: (n=10) 12weeks	Aerobic exercise Frequency:3 times/wk Intensity:60-70% of maxVO_2 Time:45-60min Type:treadmill or bicycle Breathing exercise combined with postual exercises.	BMI(kg/m^2) IG:29.79 to 29.2 CG:28.42 to 28.28 Total body fat(%) IG:28.41 to 26.87 CG:24.84 to 25.81	FEV1(%pred) IG:104.3 to 96.6 CG:112.5 to 103.4 Pe max (cmH^{2}O) IG:122.4 to 117.4 CG:113.0 to 113.0 Pi max (cmH^{2}O) IG:91.9 to 90.2 CG:89.2 to 96.9	AHI(events/h) IG:15.19 to 11.01 CG:17.92 to 17.36	PeakVO_2 (mL/kg/min) IG:15.4 to 17.5 CG:16.6 to 18.4 MET (kcal/kg/h) IG:4.4 to 5.4 CG:4.8 to 5.3
Desplan et al. (2014)	22 sedentary patients with moderate to severe OSA	RCT Intervention: Inpatient rehabilitation program (n= 11) Control: Education activity session (n=11) 4weeks	Aerobic exercise Frequency:6 sessions/wk Intensity:ventiratory threshold heart rate. Time:45min/1session Type:cycle ergometer. Resistance exercise 30min/1sessiom Postual and balance exercise 15min	BMI(kg/m^2) IG:29.9 to 29.1 CG:31.3 to 31.3 Fat mass (kg) IG:30.0 to 27.8 CG:28.9 to 30.2 Waist circum ference (cm) IG:99 to 96 CG:104 to 104		AHI(events/h) IG:40.6 to 28.0 CG:39.8 to 45.4 ODI(events/h) IG:23.1 to 17.6 CG:24.9 to 30.1 Epworth sleepiness scale IG:13.6 to 8.0 CG: 8.0 to 9.4	PeakVO_2 (mL/kg/min) IG:21.3 to 22.9 CG:23.2 to 19.8 MET (kcal/kg/h) IG:3.6 to 4.6 CG:3.3 to 2.91 Endurance test IG:770 to 1771 CG:448 to 668
Mendelson et al. (2016)	34 patients with coronary artery disease and OSA or CSA	RCT Intervention: Aerobic exercise (n=17) Control:(n=17) 4weeks	Aerobic exercise Frequency:5days/wk Intensity:60% peakVO_2 Time:30min/day Type:walking(track or treadmill)	Weight(kg) IG:81.1 to 80.6 CG: 85.9 to 85.8		AHI(events/h) IG:24.8 to 17.2 CG:21.6 to 21.8 Minimum SpO_2 IG:86.4 to 85.0 CG:83.2 to 83.2	
Karlsen et al. (2017)	30 patients with moderate to severe OSA	RCT Intervention: High Intensity interval training(HITT) (n=13) Control:(n=15) 12weeks	HITT exercise Frequency:2 times/wk 4×4min of treadmill walking or running at 90-95% of maximal heart rate, Supervised by exercise physiologist	BMI:no significant between-group changes in BMI. Total body fat was reduced (～2%) in HITT group.		AHI IG:24% improve. CG: no change. Epworth sleepiness scale was improved significantly in the HITT group.	PeakVO_2 (mL/kg/min) IG: 28.2 to 30.2 CG:27.0 to 27.0 Breathing reserve (L/min) IG: 22.8 to 10.1 CG:36.7 to 30.9
Yang et al. (2018)	70 patients with mild to moderate OSA	RCT Intervention: Aerobic exercise (n=32) Control:(n=35) 12weeks	Aerobic exercise Frequency:3times/wk Intensity:anaerobic threshold HR Time:30min/day Type:bicycle ergometer	BMI(kg/m^2) IG:27.6 to 24.5 CG:27.1 to 26.8 Waist circum ference (cm) IG:117.3 to 115.2 CG:115.4 to 111.6		AHI(events/h) IG:20.2 to 16.4 CG:19.5 to 20.1 ODI(events/h) IG:13.0 to 11.4 CG:12.4 to 12.6 Minimum SpO_2 IG:79.1 to 79.9 CG:81.5 to 81.8	PeakVO_2 (mL/kg/min) IG:28.4 to 30.2 CG:29.5 to 29.4 VO_2 at AT (mL/kg/min) IG:25.3 to 28.1 CG:26.1 to 26.8
Servantes et al. (2018)	65 patients with heart failure and OSA	RCT Intervention: <1>exercise (n=17) <2>CPAP (n=15) <3>exercise+CPAP (n=15) Control: (n=18) 3months	Aerobic exercise Frequency:3sessions/wk Intensity: anaerobic threshold HR. Time:0-1month 30 min 1-3minth 45min Type:treadmill and cycle ergometer. Resistance exercise 50% to 60% of 1RM for elbow and knee extension/flexion	Weight (kg) IG: <1> 81 to 79 <2> 86 to 87 <3> 86 to 87 CG: 78 to 78		AHI(events/h) IG: <1> 28 to 18 <2> 32 to 8 <3> 25 to 10 CG: 29 to 31 N3 sleep (%Total sleep time) <1> 19 to 20 <2> 14 to 23 <3> 19 to 26 CG: 15 to 16	PeakVO_2 (mL/kg/min) <1> 15 to 20 <2> 14 to 15 <3> 16 to 19 CG: 15 to 14 Max HR (beats/min) <1> 108 to 121 <2> 116 to 114 <3> 111 to 132 CG: 115 to 113

FEV1, forced expiratory volume in one second; FVC, forced vital capacity; Pemax, maximal expiratory pressure; Pimax, maximal inspiratory pressure; ODI, Oxygen desaturation index; peakVO_2, Peak oxygen consumption; VO_2 at AT, oxygen consumption at anaerobic threshold; MET, metabolic equivalents

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Kleine et al. randomized 43 moderate severity OSA patients in the exercise group and the stretch group and followed them up for 12 weeks. Compared to the stretch group, the exercise group showed significant AHI reduction (exercise; -7.6 vs. stretch; +4.5) and improved oxygen saturation (Kleine, Crowley, Ewing et al., 2011). These improvements are seen independently of weight loss, suggesting the effectiveness of exercise therapy for OSA improvement. In Sengul et al's RCT, exercise therapy combined with breathing exercise and aerobic exercises did not change breathing function and BMI, but significantly decreased AHI.

The detailed mechanism by which exercise reduces AHI in OSA patients has not been fully elucidated, but there are several possibilities, including changes in upper airway size and collapse, nasal resistance, lung volume alterations during sleep, and metabolic changes related to the visceral fat tissue. (Kleine, Crowley, Ewing et al., 2011)

Desplan et al. compared an inpatient rehabilitation program to a control group in OSA patients. In this rehabilitation program, a combination of aerobic exercise and resistance exercise was performed for a longer time compared to other reports. As a result, body fat mass and waist circumference were reduced significantly in the exercise group. Although the study was performed over only 4 weeks, it improved AHI to the same extent as that seen in longer duration studies. Changes in body composition and fat distribution due to intensive exercise, increased pharyngeal lumen size due to fat loss, may contribute to improving AHI.

Mendelson et al. compared 4 weeks of aerobic exercise training to a control group in OSA patients with coronary artery disease. AHI decreased significantly in the exercise group (31.1 to 20.5), in association with a greater reduction in the overnight change in leg fluid volume (Exercise: 579 to 466 vs. Control 453 to 434) and by a significantly greater increase in the overnight change in upper-airway cross sectional area in the exercise group (Mendelson, Lyons, Yadollahi et al., 2016). This study suggests that exercise therapy has further reduced nighttime fluid movement from the leg to the neck, and the accompanying dilation of the upper airway results in a decrease in AHI.

In an RCT comparing continuous positive airway pressure (CPAP) treatment with exercise training, the group using CPAP showed significant improvement in AHI, and the exercise group improved quality of life more than CPAP group (Servantes, Javaheri, Kravchychy et al., 2018). A previous study has shown improvements in CRP, insulin resistance, lipid metabolism, and blood pressure in the combination of weight loss and CPAP (Chirinos, Gurubhagavatula, Teff et al., 2014). In the treatment of OSA, it may be better to aim for weight loss comprehensively, considering the risk of complications and adherence to CPAP.

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VII. Conclusion

Weight loss and body fat loss are important for the improvement in conditions of respiratory disorders associated with obesity. In addition, exercise therapy has been suggested to be effective in reducing dyspnea on exertion and sleep apnea or hypopnea independent of the effects of weight loss. Furthermore, exercise therapy has the effect of improving exercise capacity (peak VO_2). Since peak VO_2 is an independent predictor of cardiovascular risks and mortality, exercise therapy is recommended to improve life prognosis. In surgical treatment for severe obesity, it is considered that a decrease in skeletal muscle mass and a decrease in exercise capacity can be prevented by combining exercise therapy before and after bariatric surgery.

In continuing exercise therapy, it is important to maintain patient motivation to exercise. Aerobic exercise below the anaerobic threshold level allows safe exercise and less breathlessness, leading to maintenance of compliance with exercise. In obese patients with OSA and OHS, use of CPAP is expected to improve daytime sleepiness and increase physical activity. Whether it is diet therapy or surgical weight loss, a comprehensive program combined with exercise therapy may be effective for obese patients with respiratory disorders.

Risk management for other obesity-related comorbidities is also important in promoting exercise therapy for obese patients. Evaluation of cardiovascular disease risk and attention to heart failure symptoms is necessary. In patients with musculoskeletal problems, it is important to consider the type of exercise and the amount of load. In order to continue exercise therapy for a longer period, it is important to set the program according to the lifestyle of each individual.

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