Benefits of the physical exercise in the obstructive apnea syndrome of the sleep (sahos) and its comorbilities, recommendations in cardiopulmonary rehabilitation

Diego Sosa Rojas[1], Javier Bejarano[2], Emmanuel Jiménez Castro[3*]

Diego RS, Bejarano J, Castro EJ. Benefits of the physical exercise in the obstructive apnea syndrome of the sleep (Sahos) and its comorbilities, recommendations in cardiopulmonary rehabilitation. J Pulmon 2017; 1(1):6-8.

ABSTRACT

In obstructive sleep apnea syndrome (OSAHS) airway obstruction is present in the upper airway, associated with desaturation, multisystemic involvement and deterioration of the quality of life. Usually the treatment is based only on continuous positive airway pressure (CPAP), the pathology is usually associated with other concomitant diseases that worsen the clinical picture. In specific cases of OSAHS, physical training in a complementary way to conventional treatments could benefit and improve the quality of life, physical condition and pulmonary and systemic affections of the disease and diseases coexisting with OSAHS. In view of the growing number of cardiopulmonary rehabilitation centers and fitness centers, to gather recommendations for physical exercise and to demonstrate the potential benefits, it will serve as an important theoretical guide for the adequate prescription of physical exercise in this population with an association of other concomitant pathological conditions

OBJECTIVES

- To show that the interaction between CPAP and physical exercise improves the systemic response to OSAHS pathological condition against CPAP as a single therapy.
- Confirm how combined CPAP/exercise therapy may cause regulation in the vasoconstriction of small arteries
- To show in real percentages how much obesity affects the appearance of OSAHS
- To show the present relationship between OSAHS and the prevalence metabolic syndrome

BACKGROUND

Obstructive Sleep Apnea Syndrome (OSAHS) is a multifactorial pathological condition with excessive sleepiness, cognitive-behavioral, respiratory, cardiac, metabolic and inflammatory disorders secondary to repeated episodes of upper airway obstruction during sleep. These events produce inflammatory, cardiovascular, neurocognitive and metabolic responses, which increase patients' morbidity and mortality (1-7). The American Academy of Sleep Medicine (AASM) mentions that prevalence of OSAHS increases each year and is between 2 and 4% in the adult population (4).

The pathophysiology of OSAHS is still not entirely clear, but there is consensus among researchers that sleep during upper airway permeability is compromised by a decrease in neuromuscular activation, which tends to collapse during inspiration, airway permeability is dependent on the stabilizing capacity of the dilator muscles of the pharynx and other factors that can be improved through physical exercise (8,9).

This activation and tone of the pharyngeal musculature is highly related to the ability of the central respiratory center to maintain airway permeability, in OSAHS disorders have been found in the central control of ventilation and alterations in central chemoreceptors by the increase chronic carbon dioxide (CO_2) levels, altering the ventilatory response during sleep (10).

It is estimated that 40% to 60% of patients with OSAHS are obese and a 10% increase in body weight represents an increase of up to 30% in the apnea and hypoapnea index during sleep (11-14). When obesity is present, fat deposits exist between muscle fibers, reducing muscle contractility, exacerbating upper airway occlusion, and limiting the expansion of the thoracic cage by the weight of adipose tissue on the thorax causes the muscles to generate greater inspiratory force, increasing negative pressures and favoring collapse during inspiration (15-22).

Studies have found high levels of cytokines such as IL-1 and IL-6 that cause inflammation in the upper airway dilator muscles by altering their response during sleep (23).

Scientific evidence has associated heart disease and cardiovascular events with OSAHS, the appearance of vasoconstricting alterations of the sympathetic system, the hormonal renin - angiotensin - aldosterone system, and hypoxemia and chronic hypercapnia favor the appearance of arterial hypertension (24-28). Presence of cardiac arrhythmias, coronary diseases and hypertrophy of the cardiac chambers has been associated with an aggravation of chronic heart failure (11).

Studies have correlated hypoapnea apnea index (AHI) greater than 30, with a higher incidence of coronary disease and with a higher score on the Framingham scale for a cardiovascular event (18).

It is also estimated that 7% to 11% of cases of moderate to severe OSAHS suffer from the metabolic syndrome or diabetes mellitus (25) associated with a cognitive impairment which increases the risk of accidents and serious injuries (14).

DISCUSSION

Traditionally, airway positive pressure ventilation (CPAP) has been considered the best and sometimes only treatment, but there are reviews that analyze greater benefits with multidimensional treatments, associating CPAP therapies with physical exercise and better eating habits, showing greater health benefits and decreasing the severity of OSAHS in a more significant way than treatments with only CPAP (16).

Different studies with low-calorie diets and exercise programs showed a decrease of up to 7 points in body mass index, weight reduction has been shown to be an important factor allowing one to reduce the pressures of CPAP systems, minor disorders respiratory disorders such as snoring and a decrease in the apnea-hypoapnea index (AHI) of up to 50%, with improvements in sleep quality and quality of life (3,15,20,21,24,27).

Different respiratory care societies recommend that patients with OSAHS participate in cardiopulmonary rehabilitation programs, they mention that after a supervised physical exercise program for at least 8 weeks and nutritional control, it is possible to reduce AHI, improve sleep quality, improve the affections on other physiological and pathophysiological aspects, increases life expectancy, improves cardiorespiratory health, regulates metabolic

[1]Terapeuta Respiratorio Registrado- Universidad Santa Paula, [2]Médico General- Hospital de la Mujer, [3]Fisiologo del ejercicio- Hospital de la Mujer- Universidad Santa Paula

Correspondence: Dr. Emmanuel Jiménez Castro, Fisiologo del ejercicio-Hospital de la Mujer- Universidad Santa Paula, Telephone: 506) 88601236, e-mail melterapia@gmail.com

Received: October 10, 2017, Accepted: October 13, 2017, Published: October 20, 2017

processes, generates weight loss, improves muscle mass and skeletal muscle health, improves cardiac function, improves depression and improves quality of life (1,2,4,5,6,7,9).

It has also been shown that after an aerobic exercise period, the sensitivities of the respiratory chemoreceptors responded better to changes in carbon dioxide and oxygen, optimizing the respiratory pattern in patients with OSAHS, this improves the functioning of muscles stabilizers of the pharynx, which leads to a significant improvement in the levels of hypoapnea apnea index, without generating an excessive increase in inspiratory pressures (10). Patients who are overweight or have significant obesity, the factor limiting the thoracic expansion of the abdominal contents against the diaphragm when the patient is in the supine position, together with the increase of the adipose tissue on the rib cage, is reduced proportionally to the reduction of weight, also observed significant improvements in the patients' perception regarding quality of life and daytime sleepiness (20,23).

With respect to OSAHS and cardiovascular comorbidities, OSAHS has been associated with the presence of heart failure and generalized inflammation; it has been documented that after physical exercise, improvements can be obtained in the levels of some pro-inflammatory cytokines in patients with insufficiency cardiaca, which means less systemic inflammation post physical exercise. Also, physical exercise per se produces an increase in the ejection fraction of the left ventricle by an improvement in the effectiveness of the heart; this in patients with congestive heart failure leads to a reduction of edema in the upper airway and thus to a decrease in upper airway occlusion at night. Some patients in the research also had a history of pre-hypertension or hypertension associated with OSAHS, where they have found that after exercise the levels of hypertension decrease, this effect is explained by the vasodilator response of the central nervous system, of angiotensin II levels and an increase in the production of endothelial nitric oxide, normal vasodilator factors during physical activity, evidenced that in these patients this antihypertensive effect is maintained over time, provided that physical exercise is maintained as a style of lifetime (26,28).

A series of studies and studies compared the benefits of aerobic exercise and resistance against metabolic variables, they found that both types of exercises had improvements on disorders such as diabetes mellitus type II and metabolic syndrome, they found a statistically significant decrease significant in glycosylated hemoglobin levels, which improves glucose tolerance and insulin sensitivity, an increase in the use of glucose available by muscle during physical activity, a reduction in the daily requirement of insulin or hypoglycemic agents, an improvement in hypercoagulability states and alterations in fibrinolysis, there is also an increase in energy expenditure and loss of body fat, an improvement in blood pressure, improvement in cardiac function, increase in levels of High-density lipoprotein helps reduce cholesterol levels l total, low density lipoproteins and triglyceride levels. All these improvements in exercise in relation to the metabolic pathologies improve, ultimately, the effects of OSAHS (12,13).

CONCLUSION

There is evidence of cases where OSAHS has been treated with CPAP and physical exercise simultaneously and a greater improvement in symptoms and signs of the disease has been demonstrated than in cases treated with CPAP alone.

It should be noted that in almost all the studies analyzed and following the recommendations of prestigious international organizations, it is summarized that the moderate aerobic type physical exercise, for periods greater than 8 weeks and with frequencies of 3 days per week or more, are the more recommended for working patients with OSAHS.

Among the benefits found in predominantly aerobic physical exercise programs for OSAHS, we find:

- The chemoreceptor sensitivity of central ventilation control improves, regulating the respiratory pattern and improving apneas.
- It improves the ventilatory response of the central respiratory control, which improves the tone of the muscles of the upper airway, preventing collapse.
- Sleep architecture improves in patients, associated with an increase in cerebral and muscular irrigation.
- A decrease in the severity of OSAHS symptoms has been observed, associated with decreased body weight and reduction of fat deposits in the upper airway.
- Because overweight and obesity are a major and determinant risk factor, decreases in body weight translate into benefits in all aspects of OSAHS.
- There are improvements in blood pressure due to vascular and nervous improvement, decrease in sympathetic activation and decrease in adrenaline release, decreasing generalized vasoconstriction.
- There are also improvements in the renin - angiotensin - aldosterone system, reducing fluid retention and its vaso-constricting properties.
- In patients with OSAHS and heart failure, the symptoms improve with a decrease in post-exercise generalized inflammation.
- Improved myocardium through cardiovascular and pulmonary rehabilitation programs have been shown to increase ejection fraction, reduce peripheral edema in cases of cardiac congestion, and decrease edema in the upper airway.
- Within the metabolic affections associated with OSAHS, improvements in glycemic control, insulin sensitivity, high density lipoproteins and triglycerides help to improve the symptoms associated with the disease and the generalized inflammatory factor due to increases in the circulating pro-inflammatory cytokines such as IL-1 and IL-6.

REFERENCES

1. American Association of Cardiovascular and Pulmonary Rehabilitation. AACVPR. Guidelines for Cardiac Rehabilitation and Secondary Prevention Programs. (5aedn). Champaign: Human Kinetics 2013.
2. American Association of Cardiovascular and Pulmonary Rehabilitation. AACVPR. Guidelines for Pulmonary Rehabilitation Programs. (4th edn). Champaign: Human Kinetics 2010.
3. American Academy of Sleep Medicine. AASM. Clinical practice guideline for the treatment of obstructive sleep apnea and snoring with oral appliance therapy: An update for 2015. Journal of clinical sleep medicine 2015;11(7):773–827.
4. American Academy of Sleep Medicine. AASM. Clinical Guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. Journal of clinical sleep medicine 2009;5(3):263–276.
5. American College of Sport Medicine. ACSM (2014). ACSM`s Guidelines for exercise testing and prescription. (9th edn). Philadelphia: Wolters Kluwer.
6. American Thoracic Society ATS. An Official American Thoracic Society/European Respiratory Society Statement: Key Concepts and Advances in Pulmonary Rehabilitation. American Journal Respiratory Critical Care, 2013;188 (8), e13-e64.
7. British Thoracic Society. BTS. BTS Guideline on Pulmonary Rehabilitation in Adults. Thorax,2013; 68 (2), i1-i30.
8. Goldman LY, Schafer A. Tratado de medicina interna. (24th edn). Barcelona: ELSEVIER 2013.
9. Güell R, Díaz S, Rodríguez G, et al. Normativa sobre rehabilitación respiratoria 64. Barcelona: Respira-Fundación Española del Pulmón SEPAR 2014.
10. Hargens, T, Guill S, Aron A, et al. Altered ventilatory responses to exercise testing in young adult men with obstructive sleep apnea. Respiratory Medicine Journal, 2009;103 (7), 1063–69.
11. Hedner J, Ejnell H. Caidahl K. Left ventricular hypertrophy independent of hypertension in patients with obstructive sleep apnoea. Hypertension Journal,1990;8(10), 941–6.
12. Hernández JY, Licea M. Papel del ejercicio físico en las personas con diabetes mellitus. Revista Cubana de Endocrinología, 2010;21(2), 182–201.
13. Jorquera C, Cancino J. Ejercicio, obesidad, y síndrome metabólico. Revista Condes, 2012;23 (3), 227–235.
14. Kasper D, Hauser S, Jameson J, et al. Harrison`s principles of internal medicine. (19aed.). Nueva York: McGraw-Hill, 2015.
15. Kline C, Crowley E, Ewing G, et al. The effect of exercise training on obstructive sleep apnea and sleep quality: A randomized controlled trial. Sleep, 2011;34 (12), 1631-40.

16. Leuza E, Alonso MJ, Febrero, C, et al. Manual SEPAR de procedimientos 30. CPAP y otros sistemas de tratamiento en el SAHS. Barcelona: Respira-Fundación Española del Pulmón SEPAR, 2014.

17. Lloberes P, Duran J, Martínez M, et al. Diagnóstico y tratamiento del síndrome de apneas – hipopneas del sueño. Arch Bronconeumol, 2011;47(3), 143–156.

18. Matthews K, Strollo P, Hall M, et al. Associations of Framingham risk score profile and coronary artery calcification with sleep characteristics in middle-aged men and women: Pittsburgh Sleep score study. Sleep,2011;34(6), 711–6.

19. Nogueira F, Nigro C, Cambursano, H, et al. Guías prácticas de diagnóstico y tratamiento del síndrome de apneas e hipopneas obstructivas del sueño. Recuperado el 2 de abril de 2015.

20. Norman J, Von S, Fuchs R. et al. Exercise training effect on obstructive sleep apnea syndrome. Sleep Research Online, 2000;3(3), 121–129.

21. Quan, S, O'Connor G, Quan J, et al. Association of physical activity with sleep-disordered breathing. Sleep Breath Journal. 2007;11,149–157.

22. Río V. Anías J. Mecanismos fisiopatológicos de las alteraciones cardiovasculares en el Síndrome de apnea obstructiva del sueño. Revista Cubana de Investigación Biomédica, 2009;28(1),1–7.

23. Salik Y, Ozalevli S, Oztura I, et al. The effect of exercise on obstructive sleep apnea: A randomized and controlled trial. Sleep Breath Journal, 2011;15,49-56.

24. Strobel R. Rosen R. Obesity and weight loss in obstructive sleep apnea: A critical review. Sleep, 1996;19(2),104–115.

25. Tumbeiro M, Acuña A, Alves M, et al. Síndrome metabólico y sus componentes en pacientes con síndrome de apnea-hipoapnea del sueño. Anales Sistema Sanitario Navarra, 2011;34(3), 363–2.

26. Ueno L, Drager L, Rodríguez A, et al. Effects of exercise training in patients with chronic heart failure and sleep apnea. Revista Sleep, 2009;32(5),637–7.

27. Veasey S, Guilleminault C, Strohl K, et al. Medical therapy for obstructive sleep apnea: A review by the medical therapy for obstructive sleep apnea Task Force of the Standards of Practice Committee of the American Academy of Sleep Medicine. Sleep, 2006;29(8),1036–44.

28. Weiss J, Remsburg S, Garpestad E, et al. Hemodynamic consequences of obstructive sleep apnea. Sleep, 1996;19(5),338–97.