*Obesity Surgery, **17**, 95-99*

Review Article

Obesity and Obstructive Sleep Apnea-Hypopnea Syndrome: the Impact of Bariatric Surgery

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Obstructive sleep apnea-hypopnea syndrome (OSAHS) is characterized by successive episodes of cessation or decrease in respiratory airflow, in which obesity is an important risk factor. The prevalence of the disease in morbidly obese patients is ~70%. Treatment is based on the use of *continuous positive airway pressure* (CPAP) and weight loss in obese patients. Weight loss by dieting often produces unsatisfactory results, and the use of CPAP does not show good adherence because of being long-term and uncomfortable. Bariatric surgery has emerged as the treatment for morbid obesity and various associated co-morbidities. This article reviews the principal studies that evaluate the modifications in obstructive sleep apnea after bariatric surgery, showing that surgery is an effective treatment for the management of OSAHS in morbidly obese patients.

Key words: Morbid obesity, bariatric surgery, obstructive sleep apnea, apnea-hypopnea index, sleep disorder

Introduction

The prevalence of obesity has been increasing in recent decades in developed countries.[1-3] Slight and moderate obesity is associated with various co-morbidities. However, the more severe form of obesity, known as *grade III* or *morbid obesity*, is consistently associated with the development of various health problems[4,5] and with a reduction in life expectancy of ~12 years.[6,7] Diseases commonly associated with this condition are dyslipidemia, hypertension, diabetes, arthritis and obstructive sleep apnea.[8-10]

Obstructive sleep apnea-hypopnea syndrome (OSAHS) is a disease with multifactorial etiology, marked by the occurrence of frequent episodes of nocturnal apnea and hypopnea caused by repeated collapse in the upper airways. The patients also have diurnal symptoms, usually excessive somnolence. This clinical situation is involved in the genesis of systemic diseases, particularly cardiovascular.[8-10]

The diagnosis of OSAHS is based on *polysomnography*, which consists of monitoring nocturnal sleep, performed in special laboratories, which includes electroencephalographic and respiratory parameters. The main objective of polysomnography is to verify and quantify the occurrence of episodes of respiratory airflow cessation, accompanied by loud intermittent snoring and associated or not with nocturnal awakening and episodes of desaturation, measured by the *apnea-hypopnea index* (AHI).[8-10]

Physiopathology

One of the factors frequently related to this syndrome is obesity. Although the mechanisms are still poorly understood, OSAHS is associated with deposition of fat about the upper airways, causing their collapse. Alterations in the central regulatory mechanisms of respiratory control and in airway muscular tone are also active in the physiopathology of the disease.[8]

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As obesity increases the risk of OSAHS, the latter can predispose patients to developing further obesity, because patients with somnolence exercise less. Evidence also suggests that there is interference in endocrinological regulation of leptin, the hormone that suppresses appetite and alters energy expenditure.[11,12]

Patients with morbid obesity have higher levels of leptin, indicating that they are resistant to the effects of this hormone. This could be the cause of obesity, ie., they cannot get satiated. Individuals with OSAHS have 50% higher leptin levels than healthy controls and also do not show satiety. Thus, there appears to be potentiation of resistence to leptin in OSAHS, predisposing the patient to a cycle of weight gain and worsening apnea.[11,12]

Epidemiology

The prevalence of apnea in the general population has been demonstrated in epidemiological studies. In the most extensive study, Young and colleagues[13] using polysomnography, evaluated a random sample of 602 workers in Wisconsin, USA, and found a prevalence of 24% and 9% with an AHI >5 in males and females, respectively. Of these, 4% of the men and 2% of the women, despite this elevated index, showed diurnal somnolence, which confirmed the diagnosis of OSAHS. The main risk factors of the disease were obesity and male sex.

These patients were followed-up for 4 years with the aim of determining eventual alterations in sleep disturbance after changes in weight. The authors determined that an increase of 10% in weight was associated with an increase of ~32% in AHI, and that with a decrease of 10% in weight, there was a 26% reduction in AHI.[14]

The prevalence of OSAHS in patients with morbid obesity has been studied especially in those who are planning bariatric surgery. It is known that its detection implicates greater surgical risk, and OSAHS is therefore considered as an important preoperative evaluation.[15]

In a cohort of 99 patients with a BMI >35 kg/m^2 and who were candidates for bariatric surgery, the prevalence of apnea was 70%.[16] However, the intensity of symptoms due to apnea did not show a good correlation with intensity of disease measured by AHI. In another study of 170 candidates for bariatric surgery, only 15% had a prior diagnosis of OSAHS. However, this percentage climbed to 77% with routine polysomnography, independent of having had symptoms or not.[17] Similar results were found in another study with the same characteristics, verifying a prevalence of OSAHS in 71%.[18]

Treatment of OSAHS

The treatment of choice for OSAHS is through the use of a device that supplies *continuous positive airway pressure* (CPAP) during sleep, by way of a nasal or oronasal mask, for the purpose of preventing the collapse of airways and thereby preventing apnea or hypopnea.[19,20] CPAP should be combined with weight loss in those with an elevated BMI. However, losing weight is not an easy task.

CPAP imposes a heavy burden on patients, because of the cumbersome and long-term use of the equipment, in addition to the cost, which makes its utilization difficult, especially for individuals with mild sleep respiratory diseases.[21]

In addition, results of nutritional diets with a very low calorie level (<800 kcal/day), even when supervised, have been disappointing. Of the patients who succeed in losing weight, only 5-10% keep the weight off over the course of some years.[6]

The treatment of OSAHS through only weight loss by intensive dieting, without the use of CPAP or other devices, was evaluated in a group of 205 obese patients. Successful weight loss (decrease of >10% of initial weight) was achieved in 104 patients (51%) after 1 year. Of these, 30 were cured of OSAHS (IAH <10 associated with resolution of symptoms of diurnal somnolence). After 8 years, 24 of these 30 patients were re-evaluated, showing that only 9 of them did not have OSAHS. That is, out of a total of 205 patients intially treated with dieting, 30 (15%) were considered cured of OSAHS in the short-term but only 9 (5%) remained cured in the long-term.[22]

Treatment of OSAHS by Bariatric Surgery

The surgical treatment for morbid obesity is increasing each year, especially since the introduction of video-laparoscopic techniques. Bariatric surgery affords extensive, rapid and sustained weight loss,

usually providing excellent long-term results in the reduction of commonly associated co-morbidities. The impact of weight loss after bariatric operations on OSAHS has been studied through specific questionnaires for the evaluation of symptoms and changes in pressure levels needed in CPAP and through alterations on polysomnography. Reports have found a significant cure rate of apnea in morbidly obese subjects after bariatric surgery.[23]

The first large study, conducted in the 1980s, evaluated patients with severe apnea and a mean excess body weight of 222%, and found that 6 months after surgery AHI was lowered from 88.8 to 11.8 events/hour.[24] The same group, some years later, published a similar study with a larger sample, reporting a 70% reduction in excess weight and a decrease in mean AHI from 58.8 to 36.1 events/hour after 6 months of follow-up.[25]

Few studies have been conducted to determine the effect of bariatric surgery after a longer follow-up. This could lead to a bias in the interpretation of results, because the final weight in many studies after bariatric surgery is usually established 1 to 2 years after the operation, without reports of later response of obstructive sleep apnea to the operation.

Another study showed a reduction in AHI from 64 to 26 events/hour, while the BMI decreased from 58 to 39 kg/m^2 in a group with a follow-up of 1 year. After 2 years, two-thirds of the patients were considered asymptomatic.[26] Similar results were recorded more recently with the mean AHI falling from 56 to 23 events/hour.[27]

Valencia-Flores and colleagues[28] in a study using methods similar to those above after bariatric surgery, reported a decrease in BMI from 56.5 to 39.2 kg/m^2 and disappearance of sleep apnea in 46% of the patients, in addition to improved oxygen saturation. Another report indicated a reduction of 75% in AHI 28 months after surgery, where 5 of the 8 patients no longer needed CPAP during the course of this period.[29]

Dixon and colleagues,[30] in a study with more complete follow-up, observed after 17 months a reduction in excess weight of 50 ± 15% and a decrease in AHI from 61.6 ± 34 to 13.4 ± 13 events/hour, in addition to a large improvement in quality of sleep, in diurnal somnolence and in quality of life.

Frigg and colleagues[31] followed-up patients who had a gastric band for 4 years with moderate weight loss. This study using polysomnography or indirect measurements found that 75% of the patients no longer needed the use of CPAP and the other 25% had at least improvement in symptoms.

Only one study has been reported with a postoperative follow-up period >5 years. In this work, there was great initial improvement in AHI (reduction from 45 to 11 events/hour over 4 months). However, over 7 years this index returned to an elevated level (AHI 24), although at a lower level than originally, without concomitant weight gain.[32]

Interesting results were also shown in a group of patients with very severe apnea. In this study of 15 patients who did not adapt to CPAP, including 8 patients with a tracheostomy, there was a significant improvement in symptoms, in AHI and in basal O_2 saturation after 1 year of follow-up after bariatric surgery. Nine patients were considered cured, and all the patients with a tracheostomy were able to have it removed.[33]

Studies that evaluated OSAHS using a questionnaire of symptoms or of modifications in pressure necessary in the use of CPAP, without objective polysomnographic data, have also found favorable results after obesity surgery.[34,35] Table 1 summarizes the main results described in the text.

Further Discussion and Conclusions

Sleep apnea is a very prevalent disease, especially in the obese population. The pressure of the parapharyngeal fat pads contributes to the collapse of the retroglossal space during sleep.[38] Fatty deposition results in airway reduction and predisposes to airway collapse, contributed to by neurologic loss of the normal dilator muscle tone in the neck.[39] This leads to loud snoring, then apneic episodes, restless sleep agitation, hypoxemia, hypercapnia, pulmonary vasoconstriction, daytime somnolence, and pulmonary hypertension.[40] Polysomnographic sleep, recording is confirmatory, and demonstrates the severity of OSAHS.[39] The airway deformity improves after weight loss.[38]

The current therapeutic focus centers on the use of CPAP, where the importance of weight loss should be emphasized to all patients. However, improvement requires attainment and maintenance of goal weight loss. Bariatric surgery is the best option for lowering and maintaining weight at much lower levels, as well

Table 1. Effect of surgery/procedure on the severity of sleep apnea

Source	n	Bariatric Operation	Time months	BMI before	BMI after	AHI before	AHI after	*P*-value
Busetto[36]	17	IB	6	55.8	48.6	52.1	14	<0.01
Charuzi[24]	13	GBP	6	70.8†	55†	88.8	8	<0.05
Charuzi[25]	46	GBP and VBG	6	47.5†	32.1†	58.8	7.8	<0.001
Dixon[30]	25	LAGB	17	52.7	37.2	61.6	13.4	<0.01
Valencia-Flores[28]	29	GBP	13.7	56.5	39.2	53.7	8.6	<0.01
Guardiano[29]	8	GBP	28	49	34	55	14	0.01
Lankford[35]	15	GBP	12	48	32	11*	9*	–
Pillar[32]	14	GBP	90	45	35	40	24	<0.05
Rasheid[27]	11	GBP	3-21	62	40	56	23	<0.05
Scheuller[33]	15	GBP and VBG	12-144	160‡	105‡	97	11.3	<0.001
Sugerman[26]	40	GBP, VBG and HG	69.6	56	40	64	26	0.001
Summers[23]	1	VBG	6	54	37	40	<5	–

GBP = gastric bypass; VBG = vertical gastroplasty with band or ring; HG = gastroplasty; IB = intragastric baloon; LAGB = laparoscopic adjustable gastric banding; BMI = body mass index; AHI = apnea-hypopnea index.
* = Pressure used in CPAP; ‡ = Weight in kg; † = Calculated by the equation from Lemmens et al.[37] *P*=Calculated by Student's *t*-test.

as for the resolution of co-morbidities associated with morbid obesity, among them OSAHS. The morbi-mortality of this surgery is relatively low and has been decreasing with greater experience among surgeons in video-laparoscopic techniques.

OSAHS is a multi-factorial disease and the correction of one of the factors, such as obesity, is valid for a large portion of the patients. Improvement in OSAHS symptoms with reduction in AHI in the majority of patients studied after bariatric surgery has been well-documented. These effects occur early, as well as over a longer period of 1 to 2 years. It is believed, however, that apnea can return with the passing of years but at a clearly lesser severity.

The reports should be interpreted with caution, because the majority are from series with loss of follow-up of many patients after surgery. This no doubt could produce biases, because the patients who are available for repeat polysomnographic study can be the ones most satisfied with their results and with the greatest improvement.

In addition, the results cannot be generalized for the whole population of patients with sleep apnea and morbid obesity, because in the studies described the majority of the patients' main complaint was morbid obesity and not sleep apnea. Therefore, they cannot represent the entire population whose main complaint is sleep apnea symptoms.

Sleep apnea is considered a chronic disease, and its treatment with CPAP and caloric restriction is long-term and is often required indefinitely. Finding an alternative curative treatment for a large portion of this population, is of great importance. Bariatric surgery represents this therapeutic alternative in patients with sleep apnea and morbid obesity, aimed at alleviating the respiratory disease. More prolonged studies of cohorts, with 2 to 5 years of follow-up and with clinical tests as well, should consolidate these conclusions.

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(Received July 15, 2006; accepted August 31, 2006)