Obesity Surgery (2021) 31:1082–1091
https://doi.org/10.1007/s11695-020-05080-4

ORIGINAL CONTRIBUTIONS

Weight Loss as the First-Line Therapy in Patients with Severe Obesity and Obstructive Sleep Apnea Syndrome: the Role of Laparoscopic Sleeve Gastrectomy

Bilge Yılmaz Kara[1] · Süleyman Kalcan[2] · Songül Özyurt[1] · Aziz Gümüş[1] · Neslihan Özçelik[1] · Dilek Karadoğan[1] · Ünal Şahin[1]

Received: 25 August 2020 / Revised: 14 October 2020 / Accepted: 21 October 2020 / Published online: 27 October 2020

Abstract

Purpose The objective of this study is to investigate the effects of laparoscopic sleeve gastrectomy (LSG) on the polysomnographic parameters related to OSAS.

Materials and Methods We conducted this 3-year prospective cohort study in a tertiary care center between December 2016 and December 2019. In total, we enrolled 31 patients with severe obesity who underwent full-night polysomnography (PSG) before LSG. Later, the patients were re-evaluated by full-night PSG 12 months after the surgery.

Results The mean age of the patients was 44.1 ± 9.6 years. The mean body mass index (BMI) decreased significantly from a mean value of 49.8 ± 8.5 kg/m^2 at baseline to 33.2 ± 8.2 kg/m^2 and a percent BMI (%BMI) reduction of 33.8 ± 10.4% and a percent total weight loss (%TWL) of 35.4 ± 10.8% was achieved on the same day of the postsurgical PSG ($p < 0.001$). There was a remarkable improvement in the AHI (baseline: 36.1 ± 27.1, 12 months after the surgery: 10.3 ± 11.8; difference: 25.8 ± 22.8 events per hour) ($p < 0.001$). Importantly, there was a decrease in the percentage of non-rapid eye movement (NREM) 2 ($p < 0.001$), whereas NREM 3 and REM stages witnessed a significant increase ($p = 0.001$ and $p < 0.001$, respectively) after the surgery.

Conclusion The results of this study showed that weight loss after LSG yields improvement not only in AHI but also in many polysomnographic parameters such as sleep quality and desaturation indices.

Keywords Bariatric surgery · Obesity · Obstructive sleep apnea · Weight loss · Laparoscopic sleeve gastrectomy

Introduction

Obesity is a well-known risk factor for obstructive sleep apnea syndrome (OSAS), with strong evidence suggesting that it is also a complication of OSAS [1–3]. The consecutive obstruction of the upper airways of patients with OSAS during sleep causes hypoxemia, which leads to an increased respiratory effort and, subsequently, recurrent arousals (near awakenings), further causing the disruption of sleep architecture. Research evidence in the literature suggests that being overweight directly affects the pathophysiology of the narrowing of the upper airways and involvement of the oropharyngeal muscles [4]. Muscles are not solely responsible for causing respiratory obstruction. Moreover, a layer of excess fat along the soft palate and the tonsillar region that practically involves all the face and neck muscles are also responsible for the same. This phenomenon causes the narrowing of the pharynx, thus giving it a more rounded shape; the thickness of the fat layer is directly related to the increase in body mass index (BMI) [5]. There are several studies that present an association between excess weight and OSAS with approximately 70% of the patients with OSAS also having obesity [6]. This vicious cycle of disease coexistence must be broken to prevent the undesired outcomes of OSAS such as congestive heart failure, atrial fibrillation, refractory hypertension, type 2 diabetes, metabolic syndrome, nocturnal dysrhythmias, stroke, erectile dysfunction, and pulmonary hypertension.

✉ Bilge Yılmaz Kara
drbilgeyilmaz@hotmail.com; drbilgeyilmazkara@gmail.com

[1] Department of Pulmonology, Faculty of Medicine, Recep Tayyip Erdoğan University, Rize, Turkey

[2] Department of General Surgery, Faculty of Medicine, Recep Tayyip Erdoğan University, Rize, Turkey

Only a few studies have explored the effects of bariatric surgery on OSAS until the 1990s [7]. Subsequently, similar studies were published after 2000. In many cases, bariatric surgery has resulted in a remarkable reduction in weight and was associated with a significant improvement in the indicators of sleep-related disorders [1]. This surgery is indicated for patients with a BMI above 40 kg/m^2 and aged 18–65 years. Patients who have a BMI above 35 kg/m^2 and comorbidities such as resistant hypertension, proven heart disease, or respiratory failure are also the candidates for bariatric surgery. The mechanisms for the development of OSAS in these patients are not fully elucidated; however, these mechanisms are believed to be related to the tendency of upper airways to be narrowed and closed with fatty tissues [8]. Recent years have witnessed an increase in the importance and accessibility of treatment by surgical methods with the increased frequency of obesity. The objective of this study is to investigate the effects of the decrease in body weight (BW) on the polysomnographic parameters in the patients with severe obesity undergoing LSG. It is also known that OSAS is a neglected condition by clinicians in most of the patients with obesity. Therefore, the novel approach in many centers should include a routine investigation for the presence of OSAS in the candidates of bariatric surgery, regardless of their sleepiness scores or sleep-related complaints [9].

Materials and Methods

We conducted this 3-year prospective cohort study in a tertiary care center between December 2016 and December 2019. We followed a stepwise approach for the inclusion of the patients: first, we selected surgery candidates with a BMI of > 40 kg/m^2 or > 35 kg/m^2 with comorbidities associated with obesity, patients who agreed to undergo a full-night PSG before and after the surgery regardless of their symptoms and sleepiness or quality of life scores. Next, we enrolled patients with an AHI of ≥ 5 events per hour in the preoperative PSG. Importantly, we excluded the patients under the age of 18 years or over 65 years; patients with an untreated endocrine disease causing obesity such as Cushing's syndrome, hypothyroidism, or insulinoma; patients with a psychiatric disorder such as major depression, psychosis, drug or alcohol abuse or eating disorders; patients with severe coagulopathies, malignant diseases, portal hypertension, or severe gastroesophageal reflux; pregnant patients or patients planning for pregnancy in a 12–18-month period after bariatric surgery; patients with cardiac or general contraindications that prevent anesthesia administration; and patients who would fail to comply with dietary recommendations such as lifelong vitamin replacement or calorie restrictive diet from the study. In total, we evaluated 58 patients with severe obesity, who were assigned as the candidates for LSG with preoperative and postoperative full-night PSG recordings. According to the results, 7 patients did not have OSAS in PSG evaluations (AHI < 5), and the surgery was canceled in 11 patients because of various reasons (eight patients refused to undergo postoperative PSG because they did not think it was necessary, two were out of the city, and one was unreachable by phone). In total, 40 patients underwent laparoscopic sleeve gastrectomy (LSG) without any major complications. Importantly, 34 postoperative PSG evaluations were conducted, and three of these recordings were not eligible due to inadequate sleep efficiency. There were 17 patients with severe OSAS before surgery and they were informed that the surgery may decrease or eliminate the need for OSAS treatment; none of the patients with severe OSAS chose to have continuous positive airway pressure (CPAP) instead of surgery. Eventually, we enrolled 31 patients in our study. The BMIs of all the patients were above 40, except there was one patient with additional glucose intolerance and systemic hypertension.

Epworth sleepiness scores (ESS), anthropometric measurements such as BMI, %BMI reduction (the %BMI reduction was defined as operative BMI minus the follow-up BMI, divided by the operative BMI, and multiplied by 100), weight loss, percent total weight loss (%TWL) (the %TWL was defined as operative weight minus the follow-up weight, divided by the operative weight, and multiplied by 100), neck circumferences (NCs) and waist circumferences (WCs), PSG parameters such as apnea–hypopnea index (AHI), mean obstructive and central apneas, hypopneas, mean percentages of the sleep stages, durations and percentages of desaturations, and mean numbers of arousals and snores were recorded 12 months after the surgery. We used the term "index" to designate the number of events per hour of sleep. Thereafter, we compared the preoperative and postoperative recordings of the patients. The primary outcome was the change in AHI, whereas the secondary outcomes were the changes in desaturations and percentages of sleep stages after loss of weight. We also evaluated the factors associated with the improvements in the polysomnographic parameters.

Statistical Analysis

We used the SPSS 20.0 software (SPSS Inc., Chicago, IL, USA) for statistical analyses in this study. Descriptive statistics were presented as mean ± standard deviation (minimum–maximum), frequency distribution, and percentage. We used visual (histogram and probability plots) and analytical methods (Shapiro–Wilk test) to examine the conformity of the data with the normal distribution. For the comparisons between the two dependent groups, we used the Wilcoxon signed-rank test for the non-normally distributed variables and paired sample *T* test as the statistical method for normally distributed variables. We evaluated the relationship between the variables with the Spearman correlation test. The

correlation coefficient was interpreted as "weak" for the scores of 0–0.25, "moderate" for 0.26–0.50, "strong" for 0.51–0.75, and "very strong" for 0.76–1.00. Statistical significance level was accepted as $p < 0.05$ for this study.

Results

We enrolled 31 patients with severe obesity and OSAS (mean age: 44.1 ± 9.6 years, male/female: 14/17) who underwent LSG. The average time between the first and the second PSG performed after the surgery was 12 months. The anthropometrical measurements were recorded both before and 12 months after the surgery: The mean BW decreased from 139.4 ± 26.7 to 92.7 ± 24.6 kg (kg), and the BMI decreased significantly from a mean of 49.8 ± 8.5 kg/m^2 at baseline to 33.2 ± 8.2 kg/m^2 after the surgery ($p < 0.001$). We recorded a reduction in the mean BW by 48.8 ± 15.7 kg, BMI by 16.6 ± 5.2 kg/m^2, WC by 32.7 ± 18.0 cm, NC by 7.1 ± 4.0 cm, and the ESS by 3.5 ± 3.4 points. The %TWL achieved was 35.4 ± 10.8% and %BMI reduction was 33.8 ± 10.4% 12 months after the surgery. (Table 1).

Table 2 shows the change in the recorded apneas and hypopneas before and after the surgery. The mean baseline AHI was 36.1 ± 27.1 (min–max: 5–93.8) events per hour. After the surgery, the mean AHI was 10.3 ± 11.8 (min–max: 1.3–53.1) events per hour with an improvement of 25.8 ± 22.8 events per hour, which was highly significant ($p < 0.001$). While a significant decrease in all the apneas and hypopneas was recorded, the change in central apneas did not show a statistical significance ($p > 0.05$).

Table 3 shows the changes in the sleep stages (non-rapid eye movement [NREM] 1, 2, and 3) and some sleep quality-related indices before and after the surgery. The percentage of NREM2 decreased ($p < 0.001$), whereas the percentages of NREM3 and REM (R) stages were significantly increased ($p = 0.001$ and $p < 0.001$, respectively). The percentage of NREM1 did not change significantly. There was a statistically significant improvement in the number of arousals, periodic snores, and total snores ($p < 0.001$).

Table 4 shows the parameters associated with oxygen saturation. Desaturation episodes are generally described as a decrease in the mean oxygen saturation of ≥ 4% (over the last 120 s) that lasts for at least 10 s. Total O_2 desaturation numbers and desaturations per hour of sleep along with the time and percentage spent below the saturation of 88% (minute) and 90% (percentage) were significantly lower in the postoperative PSG ($p < 0.001$).

Table 5 summarizes the correlation analysis between the differences in the anthropometric measurements and sleep parameters before and after the surgery. The change in BW was positively correlated with a decrease in arousals ($r = 0.444$, $p < 0.05$), all hypopneas ($r = 0.370$, $p < 0.05$), mean hypopneas in REM sleep ($p = 0.358$, $p < 0.05$), and the percentage of time during which oxygen saturation is measured between 80 and 89% during the entire sleep period ($r = 0.458$, $p < 0.05$). However, the change in BW was negatively correlated with the change in the central apneas ($r = -0.385$, $p < 0.05$) and average O_2 saturation ($r = -0.382$, $p < 0.05$).

The change in BMI was positively correlated with the change in arousals ($r = 0.445$, $p < 0.05$), mean hypopneas in REM sleep ($r = 0.375$, $p < 0.05$), oxygen desaturations ($r = 0.405$, $p < 0.05$), and the percentage of time during which oxygen saturation is measured between 80 and 89% during the entire sleep period ($r = 0.452$, $p < 0.05$). However, the change in BMI was negatively correlated with the change in mean apneas in the REM stage ($r = -0.402$, $p < 0.05$).

Table 1 The anthropometric measurements and the Epworth sleepiness scores before and after the LSG

$n = 31$	Before surgery Mean ± SD (min–max)	After surgery Mean ± SD (min–max)	Difference Mean ± SD (min–max)	p
Body weight (kg)	139.4 ± 26.7 (96–228)	92.7 ± 24.6 (62–182)	48.8 ± 15.7 (20–86)	< 0.001[a]**
%TWL	35.4 ± 10.8 (13.7–60)			
BMI (kg/m^2)	49.8 ± 8.5 (37.4–68.8)	33.2 ± 8.2 (21.4–55.0)	16.6 ± 5.2 (6.2–26.0)	< 0.001[a]**
%BMI	33.8 ± 10.4 (13.0–53.1)			
Waist circumference (cm)	138.2 ± 15.6 (109–170)	105.4 ± 16.5 (75–150)	32.7 ± 18.0 (5–71)	< 0.001[b]**
Neck circumference (cm)	44.4 ± 4.2 (36–54)	37.4 ± 4.0 (27–45)	7.1 ± 4.0 (2–23)	< 0.001[b]**
Epworth sleepiness score	5.1 ± 3.7 (0–16)	1.6 ± 1.6 (0–5)	3.5 ± 3.4 (− 4–13)	< 0.001[a]**

n number of patients, *SD* standard deviation, *min–max* minimum–maximum, *BMI* body mass index, *%BMI* percent of BMI difference, *%TWL* percent total weight loss

*$p < 0.05$, **$p < 0.01$

[a] Wilcoxon signed-rank test

[b] Paired sample *T* test

Table 2 The change in apneas and hypopneas before and after the surgery

$n = 31$	Before surgery Mean ± SD (min–max)	After surgery Mean ± SD (min–max)	Difference Mean ± SD (min–max)	p^a
Obstructive apnea	122.2 ± 167.1 (0–537)	38.9 ± 60.0 (0–245)	83.3 ± 144.9 (− 49; 536)	0.001**
Central apnea	4.6 ± 14.4 (0–77)	1.6 ± 2.9 (0–12)	3.0 ± 14.7 (− 12; 77)	0.615
All apneas	116.7 ± 169.1 (0–537)	40.4 ± 60.8 (0–245)	76.3 ± 159.2 (− 245; 535)	0.006**
All hypopneas	78.2 ± 79.4 (0–260)	30.1 ± 29.6 (0–127)	48.0 ± 91.9 (− 113; 254)	0.016*
Apnea + hypopnea	204.9 ± 150.9 (2–538)	70.6 ± 75.1 (8–280)	134.3 ± 129.3 (− 78; 487)	< 0.001**
Apnea index	22.6 ± 30.3 (0–92.8)	6.1 ± 10.0 (0–46.4)	16.5 ± 26.1 (− 6.7; 82.9)	< 0.001**
Hypopnea index	13.2 ± 13.8 (0–46.6)	4.1 ± 3.5 (0–16.1)	9.1 ± 15.1 (− 9.3; 45.4)	0.006**
A/H index (AHI)	36.1 ± 27.1 (5.0–93.8)	10.3 ± 11.8 (1.3–53.1)	25.8 ± 22.8 (− 6.0;73.5)	< 0.001**

n number of patients, *SD* standard deviation, *min–max* minimum–maximum

*$p < 0.05$, **$p < 0.01$

[a] Wilcoxon signed-rank test

The change in WC was positively correlated with the change in arousals ($r = 0.483$, $p < 0.01$), arousal index ($r = 0.404$, $p < 0.05$), apneas + hypopneas ($r = 0.458$, $p < 0.05$), AHI ($r = 0.397$, $p < 0.05$), oxygen desaturations ($r = 0.447$, $p < 0.05$), and the percentage of time when the oxygen saturation is recorded between 80 and 89% during the entire sleep period ($r = 0.521$, $p < 0.01$), ≤ 88% (time ≤ 88% [min]) ($r = 0.365$, $p < 0.05$), and $\%SaO_2 \leq 90\%$ ($r = 0.439$, $p < 0.05$). However, the change in WC was negatively correlated with the average O_2 saturation ($r = -0.452$, $p < 0.05$), the time when the oxygen saturation is recorded between 90 and 100% ($r = -0.432$, $p < 0.05$), and the change in the non-REM 3 stage ($r = -0.365$, $p < 0.05$).

The change in NC was positively correlated with the change in arousals ($r = 0.393$, $p < 0.05$), arousal index ($r = 0.415$, $p < 0.05$), apneas + hypopneas ($r = 0.400$, $p < 0.05$), AHI ($r = 0.419$, $p < 0.05$), oxygen desaturations ($r = 0.564$, $p < 0.01$), and oxygen desaturation index ($r = 0.412$, $p < 0.05$). The improvement in ESS was not correlated with the changes in the sleep parameters.

Discussion

Weight loss has been proven effective in the resolution of OSAS in the patients with obesity. There are three modalities of weight loss therapies: (1) lifestyle modifications, which include changes in dietary intake and physical exercises along with behavioral interventions; (2) pharmacological agents; and (3) bariatric surgery [10]. Weight reduction

Table 3 The change in sleep stages and some sleep quality related indices before and after the surgery

$n = 31$	Before surgery Mean ± SD (min–max)	After surgery Mean ± SD (min–max)	Difference Mean ± SD (min–max)	p
NREM 1 (%)	3.8 ± 1.7 (1.3–9.5)	4.0 ± 2.0 (1.4–9.2)	− 0.20 ± 2.61 (− 6.3; 7.2)	0.440[a]
NREM 2 (%)	68.5 ± 14.4 (41.9–90.5)	53.4 ± 11.3 (14.3–75.4)	15.1 ± 18.0 (− 17.3; 76.0)	< 0.001[a]**
NREM 3 (%)	13.7 ± 11.0 (0–33.5)	23.7 ± 13.2 (3.1–79.2)	− 10.0 ± 17.3 (− 79.2; 16.2)	0.001[a]**
REM (%)	14.0 ± 6.3 (2.7–23.8)	18.9 ± 5.8 (4.9–30.3)	− 4.90 ± 7.0 (− 21.9; 6.6)	< 0.001[b]**
Arousal (AR)	204.3 ± 150.8 (25–537)	75.8 ± 77.3 (8–282)	127.5 ± 115.3 (− 80; 426)	< 0.001[a]**
AR index	35.7 ± 27.2 (5.0–93.6)	10.8 ± 12.1 (1.3–53.5)	25.1 ± 21.9 (− 7.1; 72.0)	< 0.001[a]**
Periodic snores (p.s.)	1003.0 ± 905.7 (0–3923)	258.3 ± 547.3 (0–2338)	745.5 ± 1021.7 (− 2063; 3834)	< 0.001**
Index (p.s.)	146.5 ± 153.5 (0–565.8)	40.6 ± 91.9 (0–410.2)	105.9 ± 171.9 (− 369.9; 553.7)	< 0.001**
Total snore (t.s.)	1104.0 ± 890.8 (9–3957)	305.2 ± 586.3 (0–2454)	798.8 ± 999.6 (− 1790; 3795)	< 0.001**
Index (t.s.)	162.0 ± 154.2 (0–570.7)	46.6 ± 97.3 (0–430.5)	115.4 ± 169.7 (− 333.2; 548.6)	< 0.001**

n number of patients; *SD* standard deviation; *min–max* minimum–maximum; *NREM1*, *NREM2*, *NREM3*, *REM* sleep stages; *NREM* non-REM; *Index* the number of events per hour of sleep

*$p < 0.05$, **$p < 0.01$

[a] Wilcoxon signed-ranked test

[b] Paired sample *T* test

Table 4 The change in oxygen saturations before and after the surgery

$n = 31$	Before surgery Mean ± SD (min–max)	After surgery Mean ± SD (min–max)	Difference Mean ± SD (min–max)	p^a
O_2 desaturation	207.0 ± 192.2 (5–695)	59.6 ± 76.6 (1–294)	147.4 ± 156.2 (− 17; 555)	< 0.001**
O_2 desaturation index	26.7 ± 29.5 (0–96.8)	7.5 ± 9.9 (0–34.8)	19.2 ± 24.2 (− 6.6; 77.2)	< 0.001**
Av. O_2 sat.	91.5 ± 3.7 (82.1–95.8)	94.0 ± 1.6 (90.9–97.2)	− 2.5 ± 3.4 (− 11.4; 3.9)	< 0.001**
Min. O_2 sat.	74.3 ± 12.1 (50–92)	79.2 ± 14.7 (8–94)	− 4.9 ± 19.3 (− 34; 78)	0.008**
Max. O_2 sat.	98.4 ± 1 (95–100)	98.6 ± 0.8 (97–100)	− 0.16 ± 1.10 (− 3; 1)	0.528
Time @ 90–100% (min)	70.8 ± 30.5 (9.6–100)	87.0 ± 19.0 (19.4–100)	− 16.2 ± 31.0 (− 70.2; 75.0)	0.001**
Time @ 80–89% (min)	22.9 ± 23.2 (0–80.3)	10.2 ± 14.3 (0–48.9)	12.6 ± 19.4 (− 30.6; 50.2)	< 0.001**
Time @ 70–79% (min)	5.0 ± 10.1 (0–40.7)	0.09 ± 0.25 (0–1.3)	5.0 ± 10.1 (− 0.5; 40.7)	0.003**
Time @ 60–69% (min)	1.0 ± 2.8 (0–10.9)	0.01 ± 0.04 (0–0.2)	1.0 ± 2.8 (0–10.9)	0.008**
Time @ 50–59% (min)	0.06 ± 0.24 (0–1.3)	0	0.06 ± 0.24 (0–1.3)	0.109
Time ≤ 88% (min)	24.0 ± 29.3 (0–84.8)	5.4 ± 7.0 (0–20.8)	18.6 ± 26.5 (− 15.3; 72.6)	< 0.001**
%SaO_2 ≤ 90%	29.0 ± 30.6 (0–90.4)	10.3 ± 14.3 (0–48.7)	18.6 ± 26.1 (− 30.9; 70.1)	< 0.001**

n number of patients, *SD* standard deviation, *min–max* minimum–maximum, *Index* the number per hour of sleep, *Av. $_{O2}$ sat.* average oxygen saturation, *Time @ 90–100% (min)* time when the oxygen saturation is recorded between 90 and 100%, *min* minutes

*$p < 0.05$, **$p < 0.01$

[a] Wilcoxon signed-rank test

programs based on low-calorie diets with or without physical exercise directed by health professionals were proven effective in the resolution of OSAS [11, 12]. Perhaps, because of the challenges of implementing weight loss as a therapy, it has been somewhat unfairly neglected, especially in the setting of sleep clinics where specialized obesity management is usually not offered. This is particularly true when we consider the huge impact of medical weight loss on the severity of sleep apnea [13]. Surgical methods were shown to be more effective than intensive medical therapy in treating the complications of obesity such as insulin resistance and hyperlipidemia [14]. It is hypothesized that there was an improvement in the mortality of cardiovascular diseases after bariatric surgery as compared with lifestyle intervention, which is a function of the substantially greater weight loss that follows after surgery [15]. A reduction of 10% or more is better for improving OSAS severity indices as compared to the other groups of patients who lost 5–10% or less than 5% of their excess weight [16]. In our study, patients have lost approximately 35% of their preoperative excess BWs. The remarkable improvement in the polysomnographic parameters is mostly attributed to the decrease in NCs and WCs that are significantly correlated with AHI, which is the major index showing the severity of OSAS.

Conventional Treatments for OSAS

Oral appliance therapy should be considered as a viable alternative treatment to CPAP in patients with mild to moderate OSAS. In patients with severe OSAS, CPAP remains the preferred choice of treatment. Positive airway pressure (PAP) is the frontline therapy for all the patients with moderate to severe OSAS [17]. Patients who are unable to use PAP therapy may be the candidates for oral mandibular advancement splints, hypoglossal nerve stimulation, or other surgical procedures [18]. However, the conventional treatment modality for OSAS is PAP treatment, and its success or failure primarily depends on patient's compliance. Adherence to PAP therapies is low [19, 20], and their effects decrease over time [21]. Good compliance is defined as more than 4 hours of night use of the device. Factors such as upper airway symptoms (blocked or dry nose, mouth, or throat), pressure intolerance, cold sensation of airflow, and machine noise may cause poor compliance [22]. Strategies such as heated humidification during PAP treatment were studied to improve compliance but they did not appear to be effective [23]. Research comparing CPAP with surgery could not substantiate the superiority of PAP treatment on weight loss, predominantly because of patients' poor compliance to PAP therapy. In a randomized controlled study that compared weight loss as an add-on therapy for OSAS, AHI decreased more in the intervention group at 3 months (− 23.72 versus − 9 events per hour), but the difference was not significant at 12 months, even though 28% of patients from the intervention group had an AHI of less than 30 events per hour as compared to none in the controls ($p = 0.046$). There is an urgent need for more research that focuses on effective interventions to aggressively treat the association between obesity and OSAS.

Table 5 The relationship between the change in the anthropometric measurements and the change in the sleep parameters

Difference (before–after surgery)	Difference (before–after surgery)			
	BW (kg)	BMI (kg/m^2)	Waist circumference (cm)	Neck circumference (cm)
NREM 1 (%)	0.178	− 0.030	− 0.033	0.148
NREM 2 (%)	0.122	0.194	0.220	0.332
NREM 3 (%)	− 0.181	− 0.177	− 0.183	− 0.365*
REM (%)	− 0.066	− 0.184	− 0.272	− 0.206
Arousal (AR)	0.444*	0.445*	0.483**	0.393*
AR index	0.342	0.340	0.404*	0.415*
Obstructive apneas	− 0.015	− 0.073	0.175	0.185
Central apneas	− 0.385*	− 0.314	− 0.164	0.023
All apneas	− 0.020	− 0.117	0.111	0.147
All hypopneas	0.370*	0.339	0.337	0.255
Apneas + hypopneas	0.235	0.252	0.458*	0.400*
A/H index (AHI)	0.135	0.173	0.397*	0.419*
Apneas (mean)	− 0.263	− 0.092	0.088	0.150
Hypopneas (mean)	− 0.211	− 0.010	− 0.017	0.061
Apneas (REM mean)	− 0.177	− 0.402*	− 0.213	− 0.209
Hypopneas (REM mean)	0.358*	0.324	0.281	0.029
O_2 desaturations	0.340	0.405*	0.447*	0.564**
O_2 desaturation index	0.117	0.172	0.192	0.412*
Av. O_2 sat.	− 0.382*	− 0.333	− 0.452*	− 0.205
Min. O_2 sat.	− 0.036	0.072	− 0.211	− 0.130
Max. O_2 sat.	− 0.202	− 0.274	− 0.214	0.015
90–100%	− 0.337	− 0.294	− 0.432*	− 0.161
80–89%	0.458*	0.452*	0.521**	0.278
70–79%	0.085	0.031	0.209	0.092
60–69%	− 0.061	− 0.222	− 0.041	− 0.093
50–59%	0.041	− 0.238	− 0.010	− 0.105
Time ≤ 88% (min)	0.260	0.225	0.365*	0.195
%SaO_2 ≤ 90%	0.349	0.307	0.439*	0.201

Variables are presented as Spearman correlation coefficient

BW body weight; *Circum* circumference; *ESS* Epworth sleepiness score; *NREM1*, *NREM2*, *NREM3*, *REM* sleep stages; *NREM* non-REM; *Index* is number per hour of sleep; *Av. O_2 sat.* average oxygen saturation; *Time ≤ 88% (min)* time when the oxygen saturation is recorded ≤ 88%, *min* minutes

*$p < 0.05$, **$p < 0.01$

Effects of Bariatric Surgical Procedures

The fundamental basis for bariatric surgery is the purpose of accomplishing weight loss in severe obesity, which is a disease associated with multiple adverse effects on health that can be reversed or improved by successful weight loss surgery in the patients who are unable to achieve weight loss by non-surgical means [15]. A growing body of evidence further suggests that bariatric surgical procedures (i.e., sleeve gastrectomy [SG], Roux-en-Y gastric bypass (RYGB), or one-anastomosis gastric bypass [OAGB]) can improve the outcomes of patients suffering from a number of cardiovascular diseases, such as heart failure [24].

A study comparing all the three bariatric surgical methods analyzed 1219 patients in which 74% underwent RYGB, 16% underwent OAGB, and 10% underwent SG after banding failure. The mean age of these patients was 38 years (± 10 years), and 82% of them were women. The mean follow-up time was 33 months. Percentage excess BMI loss of less than 50% was achieved by 75% of patients who underwent RYGB, 85% of patients who underwent OAGB, and 67% of patients who underwent SG. Postoperative complications occurred in 13% of patients after RYGB, 5% after OAGB, and 15% after SG. It was concluded that it is possible to achieve or maintain significant weight loss with an acceptable complication rate with all the three surgical options [25].

LSG is a relatively safe procedure with low mortality and complication rates [5]. In our study, all the patients underwent SG because our center specializes in SG. Of the 42 patients operated, two had major complications and three had minor complications after the surgery. There were no fatalities. SG is also an effective option for weight loss and the resolution of comorbidities with low complication rates in the elderly patients with obesity [26].

We have observed an improvement of 93% in the AHI (29 out of 31 patients had AHI of less than 30 events per hour) of the patients after LSG, which was consistent with the results reported by Del Genio et al. in a cohort of patients followed up for 5 years (80.6%, 29 out of 36 patients achieved the same result). The patients with no improvement in AHI had a high resistance in the upper airways due to nasal obstruction [27]. Two strategies might be beneficial for the patients whose AHI remained over 30 events per hour after the surgery. The first one is to wait and monitor the BW of the patient because the AHI might continue to decrease in correlation with the change in BMI. The second is the conventional approach with the re-evaluation of upper airways and CPAP treatment so that the exposition to complications related to apneas and desaturations might be prevented. We have started CPAP treatment in our patients who had severe OSAS documented by the postsurgical PSG. In our opinion, waiting for more than a year would not be appropriate because the vast majority of the patients undergoing surgery lose the maximum percentage of their excess BW in the first 12-month period following surgery [28]. There is a marked variability in the weight loss response during the first two postoperative years following RYGB and SG in the literature. The maximal percentage of weight loss occurs at the 12-month follow-up appointment in approximately one third of patients in both patient groups, which underwent RYGB or SG. Furthermore, the early postoperative weight loss is a major predictor of the ultimate weight loss caused by surgery, with a greater effect on outcomes rather than several well-established baseline clinical factors such as preoperative BMI, age, sex, and diabetes [29]. The results of a study searching for the long-term consequences of LSG show that the resolution of comorbid conditions was achieved in 91% of patients with OSAS, 68% of patients with type 2 diabetes, 53% of patients with hypertension, and 25% of patients with dyslipidemia [30].

No standard time for OSAS resolution has been notified after bariatric surgery. In our study, the average time that passed between the first and the second PSG performed after the operation was 12 months. In a study by Neagoe, 31 (70.4%) patients with preoperative OSAS reported resolution/improvement within a year after the surgery [31].

Anthropometric Measurements

The prevalence of OSAS in patients undergoing preoperative evaluation for bariatric surgery is approximately 75% [32]. In our study, 51 out of 58 (87%) patients with severe obesity had OSAS. The peak incidence of OSAS is in the age group of 50–60 years, but in individuals with a high BMI, it tends to peak in the age group of 40–50 years. The mean age of our patients was 44.1 ± 9.6 years (min–max: 25–60; median: 44 years). It is a fact, however, that OSAS can affect individuals in any age group. The presence of only one population sample profile, such as obesity, limits the study with respect to the comparison of anthropometric characteristics with the risk of developing OSAS. Nevertheless, the minimal variations in BMI, NC, WC, and waist-to-hip ratio may alter the risk of developing OSAS; the higher these values, the greater the risk of developing OSAS [33].

Ever since 1990, investigators have reported that the variation in NC measurement is the better clinical predictor for the development and severity of OSAS than other anthropometric measurements. We have observed a significant improvement in all the anthropometric measurements such as BW, BMI, NC, and WC ($p < 0.001$). A multivariate correlation analysis found a decrease in WC and NC to be positively and significantly correlated with the reduction in AHI ($r = 0.397$ and $r = 0.419$, respectively; $p < 0.005$). However, the change in all the four anthropometric measurements was correlated with the decrease in the number of oxygen desaturations, which is a more important parameter than AHI or any other parameter in assigning the sleep-disordered breathing [34].

The Change in Central Apneas

In our study, the effects of LSG on the PSG parameters came out as we anticipated, except the change in the central apneas. The mean number of central apneas decreased from 4.6 ± 14.4 to 1.6 ± 2.9, but the change was not significant ($p = 0.615$), especially in patients with severe OSAS (i.e., AHI> 30 events per hour). The number of the central apneas was increased even in the patients with no central apneas in the preoperative PSG. This phenomenon might be explained with the complex sleep apnea syndrome, which is characterized by emerging central apneas (> 5 events per hour) when the obstructive events are eliminated by PAP therapy. There is no clear cause of the central apneas such as narcotics or systolic heart failure, and the driving forces in its pathophysiology are believed to be associated with the instability in ventilator and oscillations in the arterial partial carbon dioxide pressure. An increased elimination of carbon dioxide and the activation of airway and pulmonary stretch receptors are some factors that are believed to trigger these central

apneas. In our study, the number of patients with postoperative central apneas (> 5 events per hour) was four, and there was a negative correlation between the change in BW and the central apneas ($r = -0.385$ and $p < 0.005$). The prevalence ranges from 0.56 to 18% with no clear predictive characteristics as compared to simple obstructive sleep apnea, with the similar prognosis. The central apnea component is resolved in most of the patients on follow-up who are using CPAP therapy. Other treatment options such as bilevel PAP, adaptive servo ventilation, permissive flow limitation, and/or drugs are recommended for individuals with continued central apneas on the simple CPAP therapy [35].

Models to Estimate the Effects of Bariatric Surgery

The reliability of the surveys to question the patients' sleep-related symptoms is low in patients with obesity and OSAS. ESS is not a good predictor of OSAS because it has a poor correlation with AHI and suggests that daytime sleepiness can also exist in snoring patient without OSAS [36]. In our study, the ESS after the surgery decreased significantly as compared to the baseline values but there was no correlation between the change in ESS and the improvement in polysomnographic parameters. Duarte et al. found out a six-item model to predict OSAS in the patients who are the candidates for bariatric surgery. In this NO-OSAS model (NC, obesity, observed apnea, snoring, age, and sex), a cut-off value of ≥ 3 was defined for identifying high-risk patients. For the diagnosis of moderate/severe OSAS, the model showed an accuracy of 70.8%, sensitivity of 82.8%, and specificity of 57.9%. This model might be helpful in determining the high-risk patients and its usage might be cost-effective in order not to refer PSG in all patients with obesity [32].

A different tool for the estimation of the potential outcomes of bariatric surgery has also been developed by researchers. It was designed to provide the estimates of absolute weight loss and resolution of four major comorbidities: type 2 diabetes, high blood pressure, hypercholesterolemia, and the obstructive syndrome of sleep apnea [37].

Sleep Quality

Slow wave activity (SWA) in NREM sleep, which is obtained by the spectral analysis via electroencephalogram, is a marker of the depth or intensity of NREM sleep. Higher levels of SWA are associated with lower arousability during NREM sleep and protect against sleep fragmentation [38]. It has been known for several decades that sleep is a major modulator of hormonal release, glucose regulation, and cardiovascular function. In particular, compared with the wake state, slow wave sleep (SWS), which is believed to be the most restorative sleep stage, is associated with decreased heart rate, blood pressure, sympathetic nervous activity, and cerebral glucose utilization. During SWS, the anabolic growth hormone is released, whereas the stress hormone cortisol is inhibited. In recent years, laboratory and epidemiologic evidence have converged to indicate that sleep loss may be a novel risk factor for obesity and type 2 diabetes. The increased risk of obesity is possibly linked to the effects of sleep deprivation on hormones that play a major role in the central control of appetite and energy expenditure, such as leptin and ghrelin. Reduced leptin levels and increased ghrelin levels correlate with the increase in subjective hunger when individuals are sleep deprived rather than well rested [39].

The results of our study showed a remarkable improvement in the sleep architecture related to sleep quality. The mean percentage of SWS (i.e., stage 3 of NREM sleep) has increased from 14.4 ± 11.1% (min–max: 0–33.5) to 23.7 ± 12.8% (min–max: 3.1–79.2) after surgery ($p = 0.002$). Arousals that are one of the very important indicators of sleep quality also decreased significantly ($p < 0.001$). There was a positive correlation between the changes in anthropometric measurements and the decrease in arousals. In a study by Ju et al., PAP treatment has significantly increased SWA, and there was a shift of sleep time from light stage 1 sleep to deeper stage 3 sleep, with the decrease in the frequency of arousals [40]. It is clear that we should insist on advocating weight loss in all our patients, regardless of the severity of their OSAS or adherence to our other therapies. The benefits of weight loss are unquestionable [13]. All of these findings support the idea of breaking the vicious cycle of OSAS–obesity either by conventional or novel treatment modalities that will treat OSAS and its complications.

Conclusion

In conclusion, almost all the parameters associated with sleep-disordered breathing showed improvement because of weight loss after LSG in the patients with severe obesity and OSAS. The results of this study underline the success of LSG in the treatment of OSAS. Weight loss, either by lifestyle changes or pharmacological or surgical therapies, must be encouraged, and treatment options should be applied by a stepwise approach from the least risky to the most. If behavioral and/or medical weight loss methods are unsuccessful, then bariatric surgery must strongly be recommended for patients with severe obesity and OSAS. We believe that weight loss strategies, especially bariatric surgery, will possibly be considered as the first-line therapy in the treatment of OSAS and obesity in the future.

Acknowledgments We want to thank our polysomnography technicians Salih Öz, Ömer Kılıç, and Ayşe Erdoğan Demirkan for their participation in the trial.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

References

1. Shah N, Roux F. The relationship of obesity and obstructive sleep apnea. Clin Chest Med. 2009;30(3):455–65.
2. Reid M, Maras JE, Shea S, et al. Association between diet quality and sleep apnea in the Multi-Ethnic Study of Atherosclerosis. Sleep. 2019;42(1):zsy194.
3. Ogden CL, Carroll MD, Kit BK, et al. Prevalence of childhood and adult obesity in the United States, 2011-2012. JAMA. 2014;311(8):806–14.
4. Busetto L, Enzi G, Inelmen EM, et al. Obstructive sleep apnea syndrome in morbid obesity: effects of intragastric balloon. Chest. 2005;128(2):618–23.
5. Peppard PE, Young T, Palta M, et al. Prospective study of the association between sleep-disordered breathing and hypertension. N Engl J Med. 2000;342(19):1378–84.
6. Garvey WT, Mechanick JI, Brett EM, et al. American Association of Clinical Endocrinologists and American College of Endocrinology comprehensive clinical practice guidelines for medical care of patients with obesity executive summary. Endocr Pract. 2016;22(7):842–84.
7. Kurono T, Sakuma T, Shinozaki T, et al. A case of obstructive sleep apnea syndrome remarkably improved by gastric restriction surgery. Nihon Kyobu Shikkan Gakkai Zasshi. 1990;28(5):767–72.
8. Ravesloot MJ, van Maanen JP, Hilgevoord AA, et al. Obstructive sleep apnea is underrecognized and underdiagnosed in patients undergoing bariatric surgery. Eur Arch Otorhinolaryngol. 2012;269(7):1865–71.
9. O'Keeffe T, Patterson EJ. Evidence supporting routine polysomnography before bariatric surgery. Obes Surg. 2004;14(1):23–6.
10. Xanthopoulos MS, Berkowitz RI, Tapia IE. Effects of obesity therapies on sleep disorders. Metabolism. 2018;84:109–17.
11. Lojander J, Maasilta P, Partinen M, et al. Nasal-CPAP, surgery, and conservative management for treatment of obstructive sleep apnea syndrome. A randomized study. Chest. 1996;110(1):114–9.
12. López-Padrós C, Salord N, Alves C, et al. Effectiveness of an intensive weight-loss program for severe OSAS in patients undergoing CPAP treatment: a randomized controlled trial. J Clin Sleep Med. 2020;16(4):503–14.
13. Ainge-Allen HW, Yee BJ. A moment on the lips, a lifetime on the mask: weight-loss programs and sleep apnea. J Clin Sleep Med. 2020;16(4):471–2.
14. Schauer PR, Bhatt DL, Kirwan JP, et al. Bariatric surgery versus intensive medical therapy for diabetes - 5-year outcomes. N Engl J Med. 2017;376(7):641–51.
15. Wolfe BM, Kvach E, Eckel RH. Treatment of obesity: weight loss and bariatric surgery. Circ Res. 2016;118(11):1844–55.
16. Papandreou C, Hatzis CM, Fragkiadakis GA. Effects of different weight loss percentages on moderate to severe obstructive sleep apnoea syndrome. Chron Respir Dis. 2015;12(3):276–8.
17. Doff MH, Hoekema A, Wijkstra PJ, et al. Oral appliance versus continuous positive airway pressure in obstructive sleep apnea syndrome: a 2-year follow-up. Sleep. 2013;36(9):1289–96.
18. Veasey SC, Rosen IM. Obstructive sleep apnea in adults. N Engl J Med. 2019;380(15):1442–9.
19. Lynch MK, Elliott LC, Avis KT, et al. Quality of life in youth with obstructive sleep apnea syndrome (OSAS) treated with continuous positive airway pressure (CPAP) therapy. Behav Sleep Med. 2019;17(3):238–45.
20. Taşbakan MS, Kaçmaz Başoğlu Ö, Yıldırım Şimşir I, et al. Obstructive sleep apnea characteristics in patients with well-controlled acromegaly and their compliance with positive airway pressure therapy. Turk J Med Sci. 2017;47(5):1348–53.
21. Schoch OD, Baty F, Niedermann J, et al. Baseline predictors of adherence to positive airway pressure therapy for sleep apnea: a 10-year single-center observational cohort study. Respiration. 2014;87(2):121–8.
22. Zozula R, Rosen R. Compliance with continuous positive airway pressure therapy: assessing and improving treatment outcomes. Curr Opin Pulm Med. 2001;7(6):391–8.
23. Zhu D, Wu M, Cao Y, et al. Heated humidification did not improve compliance of positive airway pressure and subjective daytime sleepiness in obstructive sleep apnea syndrome: a meta-analysis. PLoS One. 2018;13(12):e0207994.
24. Sanches E, Timmermans M, Topal B, et al. Cardiac remodeling in obesity and after bariatric and metabolic surgery; is there a role for gastro-intestinal hormones? Expert Rev Cardiovasc Ther. 2019;17(11):771–90.
25. Pujol Rafols J, Al Abbas AI, Devriendt S, et al. Roux-en-Y gastric bypass, sleeve gastrectomy, or one anastomosis gastric bypass as rescue therapy after failed adjustable gastric banding: a multicenter comparative study. Surg Obes Relat Dis. 2018;14(11):1659–66.
26. Bianco P, Rizzuto A, Velotti N, et al. Results following laparoscopic sleeve gastrectomy in elderly obese patients: a single center experience with follow-up at three years. Minerva Chir. 2020;75(2):77–82.
27. Del Genio G, Limongelli P, Del Genio F, et al. Sleeve gastrectomy improves obstructive sleep apnea syndrome (OSAS): 5 year longitudinal study. Surg Obes Relat Dis. 2016;12(1):70–4.
28. Maciejewski ML, Arterburn DE, Van Scoyoc L, et al. Bariatric surgery and long-term durability of weight loss. JAMA Surg. 2016;151(11):1046–55.
29. Manning S, Pucci A, Carter NC, et al. Early postoperative weight loss predicts maximal weight loss after sleeve gastrectomy and Roux-en-Y gastric bypass. Surg Endosc. 2015;29(6):1484–91.
30. Gadiot RP, Biter LU, van Mil S, et al. Long-term results of laparoscopic sleeve gastrectomy for morbid obesity: 5 to 8-year results. Obes Surg. 2017;27(1):59–63.
31. Neagoe R, Muresan M, Timofte D, et al. Long-term outcomes of laparoscopic sleeve gastrectomy - a single-center prospective observational study. Wideochir Inne Tech Maloinwazyjne. 2019;14(2):242–8.
32. Duarte RL, Magalhães-da-Silveira FJ. Factors predictive of obstructive sleep apnea in patients undergoing pre-operative evaluation for bariatric surgery and referred to a sleep laboratory for polysomnography. J Bras Pneumol. 2015;41(5):440–8.

33. Modena DAO, Cazzo E, Cândido EC, et al. Obstructive sleep apnea syndrome among obese individuals: a cross-sectional study. Rev Assoc Med Bras (1992). 2017;63(10):862–8.
34. Temirbekov D, Güneş S, Yazıcı ZM, et al. The ignored parameter in the diagnosis of obstructive sleep apnea syndrome: the oxygen desaturation index. Turk Arch Otorhinolaryngol. 2018;56(1):1–6.
35. Khan MT, Franco RA. Complex sleep apnea syndrome. Sleep Disord. 2014;2014:798487.
36. Borsini E, Blanco M, Schonfeld S, et al. Performance of Epworth Sleepiness Scale and tiredness symptom used with simplified diagnostic tests for the identification of sleep apnea. Sleep Sci. 2019;12(4):287–94.
37. Velázquez-Fernández D, Sánchez H, Monraz F, et al. Development of an interactive outcome estimation tool for laparoscopic Roux-en-Y gastric bypass in Mexico based on a cohort of 1002 patients. Obes Surg. 2019;29(9):2878–85.
38. Morselli LL, Temple KA, Leproult R, et al. Determinants of slow-wave activity in overweight and obese adults: roles of sex, obstructive sleep apnea and testosterone levels. Front Endocrinol. 2018;9:377.
39. Van Cauter E, Spiegel K, Tasali E, et al. Metabolic consequences of sleep and sleep loss. Sleep Med. 2008;9(Suppl 1(0 1)):S23–8.
40. Ju YS, Zangrilli MA, Finn MB, et al. Obstructive sleep apnea treatment, slow wave activity, and amyloid-β. Ann Neurol. 2019;85(2):291–5.