A tailor made approach to obstructive sleep apnea
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Assessment of the Effect of Bariatric Surgery on Obstructive Sleep Apnea at Two Postoperative Intervals.

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N. de Vries

Submitted
ABSTRACT

BACKGROUND:
Small-scale studies have reported significant improvement of obstructive sleep apnea (OSA) in obese patients after bariatric surgery (BS). Weight loss following BS is rapid in the first few months, but it can take at least 1 year or more to reach the final result.

OBJECTIVES:
Measure effect of BS on OSA at two postoperative intervals.

SETTING:
General Teaching Hospital, the Netherlands

METHODS:
Prospectively all patients being evaluated for BS underwent a polysomnography (PSG). Patients diagnosed with OSA preoperatively were invited to undergo a PSG at least 6 months postoperatively and if OSA persisted, again at least 12 months postoperatively.

RESULTS:
A hundred and ten patients underwent a 1st postoperative PSG 7.7 months after surgery. The mean apnea hypopnea index (AHI) significantly decreased from 39.5 to 15.6/hour and the mean body mass index (BMI) from 45.4 to 36.3kg/m². In 58.2% the AHI was reduced to below 10 and in 25.5% to below 5. Fifty patients underwent a first PSG 7.1 months and a second PSG 16.9 months after surgery. The mean AHI decreased from 49.1/hour to 22.7/hour to 17.4/hour (p < .001, p < .001, p = .003) and the mean BMI from 45.0 kg/m² to 36.7 kg/m² to 35.1 kg/m² following BS (p < .001, p < .001, p = .001).

CONCLUSIONS:
This study clearly objectifies the significant, marked improvement and even remission of OSA following BS in obese patients, as measured by PSG. BS initiates dramatic improvement of clinical and sleep parameters during the first 7 months, which continues at a slower rate over the next 10 months. We recommend a follow-up PSG at 6 and in the case of persistent disease, again at 12 months after surgery to check for residual disease and if necessary, retribution of continuous positive airway pressure (CPAP), which may lead to higher treatment compliance.
INTRODUCTION

Obesity is a significant risk factor for obstructive sleep apnea (OSA), the most prevalent sleep disordered breathing problem. OSA affects 2 - 4% of the general population. Amongst patients suffering from morbid obesity, OSA is a lot more common. Not everyone with OSA is obese, but most people with obesity have OSA.

As reported previously by our group there is a high prevalence of OSA in patients undergoing bariatric surgery (BS). Of 279 consecutive patients undergoing BS, 69.9% were diagnosed with OSA preoperatively, specifically 40% from severe OSA.

Small-scale studies have reported significant improvement of OSA in obese patients after BS (range 3 months-12 years). Studies have shown that weight loss following BS, is rapid in the first few months, but it can take at least 1 year or more to reach the final result. Bearing this in mind we were interested to measure the effect of BS on OSA at two postoperative intervals: at least 6 and 12 months after surgery. To the best of our knowledge, this is the first report describing the effect of BS on OSA at two intervals postoperatively.

We report a large, prospective, follow-up study on a previously published paper, designed to assess the effect of significant weight loss after BS on various respiratory and sleep parameters of OSA measured by PSG 6 and 12 months after surgery.

MATERIALS AND METHODS

PATIENTS

We performed a prospective, multidisciplinary, single-center, observational study involving a consecutive series of patients who had been evaluated for BS in our clinic. Patients meeting the international IFSO criteria were eligible for BS. Specifically, patients aged 18 - 65, with a body mass index (BMI) > 40, or BMI > 35 with comorbidity (e.g. hypertension, diabetes, OSA or joint problems). Secondly patients were required to have made sufficient attempts at weight loss using conservative measures and had to...
be motivated for dietary and behavior modification. There was flexibility in these guidelines. Some patients with a BMI < 35 were also included if comorbid disease was present. A few exceptions were also made concerning the age restriction. Various BS methods were available in our clinic: laparoscopic adjustable gastric banding (LAGB), type Swedish adjustable gastric banding, laparoscopic gastric bypass (LRYGB) and sleeve gastrectomy (SG). All patients eligible for BS underwent a mandatory preoperative screening for OSA in addition to our routine preoperative work-up. Apart from patients with OSA previously diagnosed elsewhere, preoperatively patients visited the ear, nose and throat (ENT) out-patient department. Information was gained using patient history, ENT- and general examination and a full overnight PSG. Weight and length (BMI) were measured. The following BMI grading system was implemented: normal range (BMI 18.5 - 24.9kg/m$^2$), overweight (BMI 25 - 29.9kg/m$^2$), obese (BMI: 30 - 34.9kg/m$^2$), severely obese (BMI 35 - 39.9kg/m$^2$), morbidly obese (BMI 40 - 49.9kg/m$^2$), super obese (BMI > 50kg/m$^2$).

If the apnea hypopnea index (AHI) was greater than 15/hour, continuous positive airway pressure (CPAP) was prescribed. Patients were instructed to bring their CPAP appliance with them when being admitted for surgery. Postoperatively on the recovery ward, CPAP was administered as well as when the patient was sleeping, both during the day and night. Patients with a preoperative AHI greater than 30/hour were routinely admitted to the intensive care ward for 24 hours postoperatively. Morphinomimetic painkillers were not prescribed in patients with OSA.

All patients diagnosed with OSA preoperatively received an invitation for a PSG 6 months postoperatively (t1). Patients with persistent OSA at t1 were invited for a second PSG 12 months postoperatively.

**POLYSOMNOGRAPHY**

Besides patients with OSA previously diagnosed elsewhere, all patients underwent a full-night comprehensive sleep study using a digital Embla recorder (Flaga Medical devices, Reykjavik, Iceland). This records sleep architecture (derived from electroencephalogram, electrooculogram and submental electromyogram), respiration (thoracic and abdominal
measurement), movements of limbs, body position (trunk measurement), nasal airflow and the intensity of the snoring (the last two measured by pressure sensor). Pulse oximetry was used to monitor oxygen saturation \( (\text{SaO}_2) \) and heart rate.\(^2\) Due to financial and capacity restrictions, 13 patients who underwent a second postoperative PSG, underwent a home polygraphy using a digital Embla titanium recorder (Flaga Medical devices, Reykjavik, Iceland). The same parameters are recorded except for the sleep architecture.

The severity of OSA is expressed in the AHI. Obstructive apneas were defined as cessation of airflow for at least 10 s. Hypopneas were defined as periods of reduction of > 30% oronasal airflow for at least 10 s and a ≥ 4% decrease in oxygen saturation. Arousals were not scored as hypopneas. The AHI was calculated as the sum of total events (apneas and hypopneas) per hour of sleep. An AHI of 5 - 15/hour is mild OSA, an AHI of 15 - 30/hour is moderate and AHI > 30/hour is severe OSA, as assessed by PSG.\(^1\),\(^2\)

**STATISTICAL ANALYSIS**

Statistical analysis was performed using Microsoft Excel and SPSS statistical software (version 18, SPSS Inc, Chicago USA). The distribution of recorded variables was characterized by calculating the mean and standard deviation. Since some parameters (especially the AHI) were expected to be non-normally distributed also the median and range were calculated. Pre- and postoperative quantitative data were compared using the paired Student’s \( t \) test with additional application of the Wilcoxon signed-rank test. A p-value of less than .05 was considered statistically significant.

The prevalence of OSA and OSA severity was subdivided for obesity severity subgroups. To identify independent predictors of OSA cure (AHI < 5) or surgical success (Sher’s criteria, a postoperative reduction of the AHI by 50% and to below 20), we used logistic multiple regression.

**RESULTS**

279 patients who underwent a preoperative PSG were included in the study. Based on the PSG results, 195 (69.9%) patients were diagnosed with OSA.\(^2\) Of the 195 patients diagnosed with OSA preoperatively,
22 patients withdrew themselves from the waiting list and did not undergo surgery, two are still on the waiting list for surgery due to unforeseen circumstances. 171 patients with OSA underwent surgery. The mean duration between the preoperative PSG and the date of surgery was 8.4 months. Eighty-five patients did not undergo a first postoperative PSG. The majority of patients (n = 48) refused a postoperative evaluation or did not show up for their scheduled sleep study. Ten patients underwent BS but are being treated for their sleep apnea in a different medical center; unfortunately we were unable to gather postoperative PSG results for these patients. Additionally one patient was pregnant in the postoperative period and 2 patients died, one from cardiac complications following surgery and the other from lung cancer.

Fifty patients underwent a second postoperative PSG. Of the 110 patients who underwent a first postoperative PSG, an AHI < 5 was objectified in 29 patients. Thirty-one refused a second follow-up sleep study or did not show up for their scheduled sleep study. These results are depicted graphically in Figure 1.

![Flowchart of subjects included in the study.](image)

**Figure 1:** Flowchart of subjects included in the study.

*AHI: apnea hypopnea index; BS: bariatric surgery; OSA: obstructive sleep apnea; PSG: polysomnography*
STUDY POPULATION WITH ONE POSTOPERATIVE PSG (N = 110)

PREOPERATIVE RESULTS

Of the 110 patients who underwent a first postoperative PSG, 73 (60.8%) were women and 37 (30.8%) men. Preoperatively, one (0.9%) patient was obese, 30 (27.3%) severely obese, 48 (43.6%) morbidly obese, 31 (28.2%) super obese. 35 (31.8%) patients underwent LAGB (average BMI: 41.0 ± standard deviation (SD) 4.6 kg/m$^2$), 70 (63.6%) LRYGB (average BMI: 47 ± SD 7.1 kg/m$^2$) and 5 (4.5%) SG (average BMI: 53.4 ± SD 9.5 kg/m$^2$). Patient baseline characteristics are shown in Table 1. Preoperatively, based on the PSG results, 28 (25.5%) patients were diagnosed with mild OSA, 30 (27.3%) with moderate OSA and 52 (47.3%) with severe OSA.

POSTOPERATIVE RESULTS

At the first postoperative visit, 7.7 ± 2.4 months after surgery, the mean AHI significantly decreased from 39.5 ± SD 31.7/hour to 15.6 ± SD 17.4/hour, the mean BMI significantly decreased from 45.4 ± SD 7.3 kg/m$^2$ to 36.3 ± SD 6.1 kg/m$^2$ and the mean weight significantly decreased from 132.5 ± SD 26.9 kg to 106.4 ± SD 23.8 kg. Pre- and postoperative clinical parameters are summarized in Table 1.

Postoperatively, one patient (0.9%) had a normal weight, 12 (10.9%) were overweight, 38 (34.5%) obese, 31 (28.2%) severely obese, 24 (21.8%) morbidly obese and 4 (3.6%) super obese. 28 (25.5%) were cured of their OSA. 48 (43.6%) still suffered from mild disease, 17 (15.5%) moderate disease, 17 (15.5%) severe disease.

Similarly, repeat testing showed a statistically significant decrease in AI and DI and a significant increase in mean and minimum oxygen saturation and sleep efficiency (Table 1).

Patients with preoperative mild disease were more likely to be “cured” than those with a preoperative severe disease (53.6% vs. 17.9%) (Figure 2). The mean AHI after BS in patients (n = 82) with residual disease was
20.2 ± SD 18.1/hour. The mean AHI, in the severe OSA group decreased from 66.1 ± SD 26.9/hour to 25.4 ± SD 20.8/hour \((p < .001)\), in the moderate OSA group from 21.2 ± SD 4.4 to 8.9 ± SD 6.5 per hour \((p < .001)\) and in the mild OSA group from 9.6 ± SD 2.6 to 4.8 ± SD 3.2 per hour \((p < .001)\) (Figure 3). Supplementary Figure 1 shows the percentage decrease in AHI following BS, stratified per preoperative OSA severity grade.

66.4% of the patients could be considered successfully treated, when applying Sher’s surgical success criteria (50% reduction in AHI and/or ≤ 20). The mean percentage weight loss after treatment in the responder group was higher than in the non-responder group \((22.4 ± SD 9.3 \% \text{ versus } 15.4 ± SD 9.8 \% \text{ respectively, unpaired } t \text{ test, } p < .001)\).

Supplementary Figure 2 shows a scatter plot of the preoperative and postoperative AHI data (with a linear regression line). On examination, the scatterplot showed 2 distinguishable clusters of subjects. Data points were either clustered around the \(x=y\) line (little to no effect of surgical weight loss on the AHI), or below the regression line (good effect of surgical weight loss on the AHI).

Despite a positive relation, the correlation between the percentage and absolute reduction in AHI and the percentage and absolute reduction in weight and BMI is poor \((R^2 \text{ ranging between 0.095 and 0.211})\).
Table 1: Effect of bariatric surgery on clinical and sleep parameters, 7.7 months after bariatric surgery

<table>
<thead>
<tr>
<th></th>
<th>Preoperative (t0)</th>
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<td>44</td>
</tr>
<tr>
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<tr>
<td>AI (per hour)</td>
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<tr>
<td>Mean SaO₂ (%)</td>
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<td>Minimum SaO₂ (%)</td>
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<tr>
<td>Desaturation Index (DI)</td>
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<td>Arousal index (per hour)</td>
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<td>Sleep efficiency (%)</td>
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<td>82.4 ± SD 13.4</td>
<td>85.9</td>
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</tbody>
</table>

AI: apnea index; AHI: apnea hypopnea index; BMI: body mass index, ESS: Epworth Sleepiness Scale; OSA: obstructive sleep apnea; SaO₂: oxygen saturation; SD: standard deviation.

p-values are paired t test. * parametric paired t test † non-parametric Wilcoxon signed-rank test.
Figure 2: Percentage of patients with a postoperative AHI < 5, stratified per preoperative OSA severity group. Patients with preoperative mild disease were more likely to be “cured” than those with a preoperative severe disease (53.6% vs. 17.9%).

AHI: apnea hypopnea index; OSA: obstructive sleep apnea
Effect of BS on OSA at Two Postoperative Intervals

**Figure 3:** Improvement in mean AHI, stratified per OSA severity group. In the severe OSA group the mean AHI decreased 66.1 ± SD 26.9 to 25.4 ± SD 20.8 per hour (p < .001), in the moderate OSA group from 21.2 ± SD 4.4 to 8.9 ± SD 6.5 per hour (p < .001) and in the mild OSA group from 9.6 ± SD 2.6 to 4.8 ± SD 3.2 per hour (p < .001).

AHI: apnea hypopnea index; OSA: obstructive sleep apnea

**STUDY GROUP WITH TWO POSTOPERATIVE PSGS (N = 50)**

**PREOPERATIVE RESULTS**

Of the 50 patients who underwent two postoperative PSGs, 33 (66%) were women and 17 (34%) men. Preoperatively, 1 (2%) patient was obese, 15 patients (19%) severely obese, 19 (38%) morbidly obese, 15 (30%) super obese. 18 (36%) patients underwent LAGB (average BMI: 40.9 ± SD 4.9 kg/m²), 29 (58%) LRYGB (average BMI: 46.7 ± SD 7.9 kg/m²) and 3 (6.0%) SG (average BMI: 53.9 ± SD 12.9 kg/m²). Patient baseline characteristics are shown in **Table 2**. Preoperatively, based on the PSG results, 5 (10%) patients were diagnosed with mild OSA, 15 (30%) with moderate OSA and 30 (60%) with severe OSA.
Table 2: Clinical parameters and polysomnographic results of the study population who underwent a 1st PSG 7.1 months and a 2nd PSG 16.9 months after bariatric surgery.

<table>
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<tr>
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<th>2nd postoperative visit 16.9 ± SD 4.3 months</th>
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<td></td>
<td>N</td>
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<tr>
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<td>Mean (SD)</td>
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<tr>
<td>Median</td>
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<td>Median</td>
<td>Median</td>
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<td>Range</td>
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<td>Range</td>
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<tr>
<td>BMI (kg/m²)</td>
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<td>50</td>
<td>42</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>50</td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>AHI (per hour)</td>
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<td>50</td>
<td>50</td>
</tr>
<tr>
<td>AI (per hour)</td>
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<td>Mean SaO₂ (%)</td>
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<tr>
<td>Minimum SaO₂ (%)</td>
<td>47</td>
<td>47</td>
<td>49</td>
</tr>
<tr>
<td>Desaturation Index (DI)</td>
<td>44</td>
<td>44</td>
<td>49</td>
</tr>
<tr>
<td>Arousal index (per hour)</td>
<td>47</td>
<td>47</td>
<td>50</td>
</tr>
<tr>
<td>Sleep efficiency (%)</td>
<td>44</td>
<td>44</td>
<td>50</td>
</tr>
</tbody>
</table>

AI: apnea index; AHI: apnea hypopnea index; BMI: body mass index; OSA: obstructive sleep apnea; SaO₂: oxygen saturation; SD: standard deviation. p-values are paired t test.
POSTOPERATIVE RESULTS

Fifty patients underwent a first PSG 7.1 ± SD 1.3 months after surgery (t1) and a second PSG 16.9 ± SD 4.3 months after surgery (t2). The results are summarized in Table 2. The mean AHI decreased statistically significantly from 49.1/hour (t0) to 22.7/hour (t1) to 17.4/hour (t2) following BS (p < .001, p < .001, p = .003), the mean BMI from 45.0 kg/m² (t0) to 36.7 kg/m² (t1) to 35.1 kg/m² (t2) (p < .001, p < .001, p = .001) and the mean weight from 130.2 kg (t0) to 106.6 kg (t1) to 102.1 kg (t2) (p < .001, p < .001, p = .004) (Table 3 and Supplementary Figure 3). Furthermore, in comparison to the baseline parameters (t0), at both t1 and t2, there was a statistically significant decrease in AI and DI and a statistically significant increase in mean and minimum oxygen saturation and sleep efficiency. When comparing the PSG outcomes between t1 and t2, besides the statistically significant decrease in AHI, BMI and weight, there was a statistically significant increase in mean and minimum oxygen saturation.

Postoperatively at t2, 2 patients (4%) had a normal weight, 7 (14%) were overweight, 18 (26%) obese, 12 (24%) severely obese, 10 (20%) morbidly obese and 1 (2%) super obese. Twelve (24%) were cured of their OSA. Twenty-one (42%) still suffered from mild disease, 6 (12%) moderate disease, 11 (22%) severe disease. At t2 29 (58%) of the patients can be considered successfully treated, when applying Sher's surgical success criteria (50% reduction in AHI and ≤ 20). The mean AHI after BS in patients (n = 38) with residual disease was 22.1 ± SD17.5/hour. The mean AHI, in the severe OSA group decreased from 69.7 ± SD 28.3/hour to 24.1 ± SD 19.3/hour (p < .001), in the moderate OSA group from 21.3 ± SD 4.1 to 8.6 ± SD 6.3 per hour (p < .001) and in the mild OSA group from 8.6 ± SD 2.4 to 3.5 ± SD 3.1 per hour (p = .091).

58% of the patients could be considered successfully treated, when applying Sher’s surgical success criteria (50% reduction in AHI and/or ≤ 20). The mean percentage weight loss after treatment in the responder group was higher than in the non-responder group (21.7 ± SD 10.5% versus 19.0 ± SD 12.3% respectively, unpaired t test, p = .417)
Table 3: Comparison of pre- and postoperative quantitative data using the paired Student’s t test with additional application of the Wilcoxon signed-rank test of the study population who underwent a first polysomnography at a mean 7.0 months (t1) and a second polysomnography at a mean 16.9 months (t2) after bariatric surgery.

<table>
<thead>
<tr>
<th></th>
<th>Preoperative (t0)</th>
<th>1st postoperative PSG (t1) 7.1 ± SD 1.3 months</th>
<th>2nd postoperative PSG (t2) 16.9 ± SD 4.3 months</th>
<th>p t0t1</th>
<th>p t0t2</th>
<th>p t1t2</th>
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<tr>
<td>BMI (kg/m²)</td>
<td>45.0 ± SD 8.0</td>
<td>35.0 ± SD 6.2</td>
<td>35.0 ± SD 6.0</td>
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<td>&lt; .001</td>
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<tr>
<td>Weight (kg)</td>
<td>130.2 ± SD 27.7</td>
<td>106.6 ± SD 23.4</td>
<td>102.1 ± SD 20.1</td>
<td>&lt; .001</td>
<td>&lt; .001</td>
<td>= .004</td>
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<td>AHI (per hour)</td>
<td>49.1 ± SD 33.8</td>
<td>22.7 ± SD 16.3</td>
<td>17.4 ± SD 17.4</td>
<td>&lt; .001</td>
<td>&lt; .001</td>
<td>= .003</td>
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<td>AI (per hour)</td>
<td>30.7 ± SD 34.2</td>
<td>10.7 ± SD 10.8</td>
<td>9.7 ± SD 12.2</td>
<td>&lt; .001</td>
<td>&lt; .001</td>
<td>= .478</td>
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<td>Mean SaO₂ (%)</td>
<td>91.5 ± SD 3.8</td>
<td>93.8 ± SD 1.9</td>
<td>94.6 ± SD 1.8</td>
<td>&lt; .001</td>
<td>&lt; .001</td>
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<tr>
<td>Minimum SaO₂ (%)</td>
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<td>81.5 ± SD 5.3</td>
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<td>&lt; .001</td>
<td>= .006</td>
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<td>Desaturation Index (DI)</td>
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<td>Sleep efficiency (%)</td>
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<td>88.4 ± SD 9.3</td>
<td>= .001</td>
<td>= .009</td>
<td>= .436</td>
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</table>

AI: apnea index; AHI: apnea hypopnea index; BMI: body mass index, OSA: obstructive sleep apnea; PSG: polysomnography; SaO₂: oxygen saturation; SD: standard deviation.

p-values are paired t test.
Supplementary Figure 4 shows a scatterplot of the preoperative and postoperative AHI at t1 and t2 (with separate linear regression lines for each measurement interval). The difference in average AHI between the first and second postoperative recording was not explained by a difference in percentage of sleeping time in supine position.

Despite a positive relation, the correlation between the percentage and absolute reduction in AHI and the percentage and absolute reduction in weight and BMI is poor ($R^2$ ranging between 0.057 and 0.253).

Even though the mean pre- and postoperative AHI was higher in the study population who underwent 2 postoperative PSGs than in the study population who underwent 1 postoperative PSG, the difference in mean AHI reduction (42.75 ± SD 36.4% and 55.6 ± SD 33.7% respectively) was not statistically significant $t(158) = 2.18, p = .031$ (un-paired $t$ test) (see supplementary Figure 5).

IDENTIFICATION OF PREDICTORS

In logistic regression models age, pre-operative BMI and weight, were inadequate predictors of cure and surgical success (Sher’s criteria) at both measurement intervals (t1 and t2).

DISCUSSION

In this series of patients with OSA, BS initiates dramatic improvement of clinical and sleep parameters during the first 7 months, which continues at a slower rate over the next 10 months. To our best knowledge this is the first paper studying the effect of BS on OSA at 2 intervals postoperatively. Secondly, this study confirms the hypothesis that in patients with OSA and obesity, BS results in significant reduction in OSA severity. The gross majority of patients reported a significant decrease in AHI and other OSA parameters following BS, but the mean AHI after BS is consistent with moderately severe OSA. Greenburg et al in a meta-analysis identified 12 studies representing 342 patients, reporting results of PSGs performed before and at least 3 months after BS. The average study included 28.5 patients; the mean duration between surgery and the follow-up
PSG is not mentioned (range: 3 months – 12 years). The random-effects pooled mean AHI decreased by 38.2/hour (95% confidence interval (CI), 31.9 - 44.4) from 54.7 events/hour (95% CI, 49.0 -60.3) to 15.8/hour (95% CI, 12.6 - 19.0) and the mean BMI was from 53.3 kg/m² (95% CI, 53.5 - 57.1) to 37.7kg/m² (95% CI, 36.6 - 38.9). They conclude that BS significantly reduces the AHI, but the mean AHI after BS is consistent with moderately severe OSA.

Patients with persistent OSA at 7 months, underwent their second evaluation 16.9 ± 4.3 months after surgery. The effect of BS on weight is gradual. Weight loss in the first few months is rapid, but it can take at least 1 year or more to reach the final result.\(^\text{17}\) Weight loss continues albeit at a slower rate over the next 6 months.\(^\text{22}\) The same pattern can be observed when studying the effect of BS on OSA. BS initiates dramatic improvement of clinical and sleep parameters during the first 7 months, thereafter improvement continues albeit at a slower rate. Physicians should bear in mind that sleep and clinical parameters will most probably have improved significantly 6 months after surgery, but a continuation of reduction in the severity of OSA and improvement of the success rate can be expected thereafter. We recommend a follow-up PSG at 6 months and in the case of persistent disease, again 12 months after surgery.

It can be questioned whether some patients not only suffer from OSA but also from obesity hypoventilation syndrome (OHS). As we didn’t take an arterial blood gas in all patients, to measure the carbon dioxide level in the blood, we weren’t able to identify presence of OHS.

The exact pathophysiology of OSA in obese patients remains poorly understood, but it is thought that in these patients local fatty tissue deposition in the neck results in reduction of the lumen of the upper airway thereby reducing airflow and inducing airway collapse.\(^\text{23, 24}\)

Even modest weight loss can be effective in reducing OSA severity.\(^\text{25-27}\) Unfortunately, losing weight is particularly tasking in patients with OSA. Daytime hypersomnolence, an important symptom of OSA, reduces the motivation for physical activity and dieting.\(^\text{3}\) Secondly, OSA is thought to induce weight gain. Sleep deprivation and intermittent hypoxia may
cause impaired glucose metabolism, hyperphagia and imbalances of leptin, ghrelin and orexin levels.\textsuperscript{28, 29}

Therefore, on the whole, conservative treatment fails more often in obese patients with OSA, in which case BS can be considered. BS is not only the most effective treatment modality in obese patients to lose weight, producing durable weight loss, it is also known to have a positive effect on comorbidities. BS is therefore becoming increasingly popular.\textsuperscript{22}

Various studies have reported improvement or remission of comorbidities after BS. A systematic review and meta-analysis published in 2004 reported a resolution of diabetes and hypertension in 78.6\% and 61.7\% of patients, respectively. Hyperlipidemia, hypertension and diabetes improved in 70\%, 78.5\% and 86\% of patients, respectively.\textsuperscript{30} Even though the percentage of patients cured of OSA isn’t comparable to the curative effect of BS on other obesity-related comorbidities, it is important to bear in mind that the effect on OSA by BS is comparable to other OSA treatment modalities. Studies have shown that a mean AHI < 5, for both sleep surgery and CPAP therapy, is rarely achievable.\textsuperscript{31} Despite being very efficacious, it is a clinical reality that the use of CPAP is often cumbersome and that CPAP compliance rates are poor. Weaver and Grunstein report in their review that 29\% to 83\% of patients are non-adherent and use their CPAP less than 4 hours per night.\textsuperscript{32} Sleep surgery on the other hand has a continuous effect, but success rates are poor. When the traditional surgical definition (50\% reduction in AHI and/or ≤ 20) is applied, the pooled success rate for Phase I procedures (soft palate surgery with or without adjuvant treatment targeting base of tongue obstruction) is 55\%, but with AHI ≤ 10 as a cut-off point, success rate decreases to 31.5\%; and at AHI ≤ 5, success is reduced to 13\%.\textsuperscript{33} Studies have shown that the success rates of sleep surgery are lower in patients with an increased BMI.\textsuperscript{34, 35} In this current study, the success rates of bariatric surgery decreases from 66.4\% to 54.5\% to 26.4\% respectively in the study population who underwent one postoperative PSG (n = 110).
The treatment of OSA is a stepwise approach. In patients with OSA and obesity, BS is an important treatment option, resulting in a decrease in AHI and if indicated, consequent less aggressive future OSA treatment plans. For example, as the AHI drops, so does the CPAP pressure needed, potentially improving tolerance and compliance.\(^{36}\) Patients with moderate to severe OSA should continue to lose weight if necessary and pursue CPAP treatment to alleviate symptoms and decrease the risk of cardiovascular disease.

Comparable to previous studies we did not find a linear relationship between the extent of weight loss and improvement in OSA. In a randomized controlled trial, the effect of conventional weight loss versus surgical weight loss with LAGB on OSA in obese patients was compared.\(^{37}\) Despite greater weight loss in the surgical weight loss group, there was no statistically significant difference in AHI between the 2 groups. There was substantial inter-individual variability in AHI changes. In our current study, we confirm that weight loss and reduction in BMI is associated with a decrease in AHI, but the correlation between the percentage and absolute reduction in AHI and the percentage and absolute reduction in weight and BMI is poor. Furthermore age, preoperative weight and BMI were inadequate predictors of cure and surgical success at both measurement intervals. One can conclude that the effect of BS on the AHI is difficult to predict.

Several limitations of this study need to be recognized. Ideally all patients who underwent a preoperative PSG and BS would have been invited for a PSG 6 months postoperatively regardless of the presence or absence of OSA preoperatively. Likewise regardless of the outcome of the first postoperative PSG, ideally all patients would have been invited for a second PSG 12 months postoperatively. It can be questioned whether patients with a preoperative AHI below 5, continue to maintain an AHI below 5 postoperatively. As for the patients who underwent a second PSG, the chance is greater that a patient will maintain an AHI greater than 5 at the first postoperative PSG, if the preoperative AHI was high. Therefore the mean AHI is relatively high at the three measurement intervals and the chance is greater that these patients will not be cured at
the 2nd postoperative PSG. Even though the mean pre- and postoperative AHI was higher in the study population who underwent 2 postoperative PSGs than in the study population who underwent 1 postoperative PSG the difference in mean percentage AHI reduction (42.75 ± SD 36.4% and 55.6 ± SD 33.7% respectively) was not statistically significant $t(158) = 2.18, p = .031$ (un-paired t test) (see Supplementary Figure 5).

The second main limitation of this study was the loss of patients to follow-up. Despite conscientiously performing a preoperative PSG on all patients undergoing BS irrespective of history or clinical findings, 31.3% did not show up or refused the first postoperative evaluation. Patients undergoing BS have a reputation of non-compliance; our data supports this observation.\textsuperscript{38,39} Patients received a written invitation. If not accepted, patients were contacted by telephone and encouraged to accept the follow-up evaluation. Most patients reported that their symptoms had subsided; therefore they deemed a second evaluation unnecessary. Others, despite our best efforts, simply refused. We may therefore be under-estimating the success rate, as patients with residual symptoms were keener for re-evaluation. 64.5% of the tested patients maintained residual disease. Patients are blinded by the benefits of weight loss and note an allover improvement of their general health, while they may still suffer from OSA albeit less severe. On the contrary, patients who have not measured significant weight loss are often reluctant to continue follow-up. This is particularly true for patients undergoing gastric bypass or gastric sleeve surgery as nothing further can be done to induce weight loss besides dietary modification\textsuperscript{38} Plus, studies have shown that attendance of follow-up appointments is associated with better weight loss outcomes.\textsuperscript{39}

The third study limitation is absent data. Due to financial and capacity restrictions, a few of the patients invited for a second postoperative PSG, underwent a home respiratory polygraphy, which does not record sleep efficiency or the arousal index.

Lastly, we did not perform a CPAP washout period prior to the postoperative PSG. CPAP is thought to play a role in reducing edema
resulting from snoring-associated vibration and apnea-induced suction of the upper airway. The baseline AHI may be reduced by a fraction in chronic CPAP use. One could argue that we are over-estimating the effect of BS on OSA, even though research suggests that the effect is minimal.40 Advising patients not to use CPAP, leaving patients untreated for a period of time prior to a follow-up PSG, raised ethical concerns (traffic or professional accidents, cardiovascular complications) within our research group, especially since so many patients were diagnosed with extremely severe OSA preoperatively.

CONCLUSION

Nonetheless, these data clearly objectify the significant, marked improvement and even remission of OSA following BS in obese patients, as measured by PSG. BS initiates dramatic improvement of clinical and sleep parameters during the first 7 months, which continues at a slower rate over the next 10 months. It is of key importance to educate patients prior to BS on OSA: the high prevalence of OSA in the BS population, the risks of untreated OSA and need for appropriate treatment, especially since the effect of BS on the AHI isn’t predictable. One hopes patients will recognize the importance of preoperative and follow-up PSG, to ensure appropriate treatment. We recommend follow-up PSG at 6 months and in the case of persistent disease, again at 12 months after surgery to check for residual disease and if necessary retrimination of CPAP, which may lead to higher treatment compliance.
**SUPPLEMENTARY MATERIAL**

**Supplementary Figure 1:**  Mean percentage AHI decrease, stratified per OSA severity group. In the mild OSA group the AHI decreased by an average 46%, in the moderate OSA group by 58% and in the severe group by 59%.

*AHI:* apnea hypopnea index; *OSA:* obstructive sleep apnea
Supplementary Figure 2: Scatterplot of individual pre- and postoperative AHI of the study population who underwent one postoperative polysomnography (n = 110).

*AHI*: apnea hypopnea index
Supplementary Figure 3: Improvement in mean AHI and AI from baseline (t0), 7.1 ± SD 1.3 months (t1) and 16.9 ± SD 4.3 months (t2) after surgery. The mean AHI decreased significantly from 49.1 (t0) to 22.7 (t1) to 17.4 (t2) per hour (p < .001, p < .001, p = .003) and the AI from 30.7 (t0) to 10.7 (t1) to 9.7 (t2) per hour (p < .001, p < .001, p = .478).

AI: apnea index; AHI: apnea hypopnea index; CI: confidence interval
Supplementary Figure 4: Scatterplot of individual pre- and postoperative AHI of the study population who underwent two postoperative polysomnographies (n = 50).

AHI: apnea hypopnea index
Supplementary Figure 5: Decrease in mean AHI per study group at the various measurement intervals.

AHI: apnea hypopnea index; PSG: polysomnography

REFERENCES


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