

Objective evidence that bariatric surgery improves obesity-related obstructive sleep apnea

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Background. Obstructive sleep apnea (OSA) is associated with obesity. Our aim in this study is to report objective improvement of obesity-related OSA and sleep quality after bariatric surgery.

Methods. Prospective bariatric patients were referred for polysomnography if they scored ≥ 6 on the Epworth Sleepiness Scale. The severity of OSA was categorized by the respiratory disturbance index (RDI) as follows: absent, 0 to 5; mild, 6 to 20; moderate, 21 to 40; and severe, > 40 . Patients were referred for repeat polysomnography 6 to 12 months after bariatric surgery or when weight loss exceeded 75 lbs. Means were compared using paired *t* tests. Chi-square tests and linear regression models were used to assess associations between clinical parameters and RDI; $P < .05$ was considered statistically significant.

Results. Of 349 patients referred for polysomnography, 289 patients had severe (33%), moderate (18%), and mild (32%) OSA; 17% had no OSA. At a median of 11 months (6 to 42 months) after bariatric surgery, mean body mass index (BMI) was 38 ± 1 kg/m² ($P < .01$ vs 56 ± 1 kg/m² preoperatively) and the mean RDI decreased to 15 ± 2 ($P < .01$ vs 51 ± 4 preoperatively) in 101 patients who underwent postoperative polysomnography. In addition, minimum oxygen saturation, sleep efficiency, and rapid eye movement latency improved, and the requirement for continuous positive airway pressure was reduced ($P \leq .025$). Male gender and increasing BMI correlated with increasing RDI ($P < .01$) by chi-square analysis. In a multivariate linear regression model adjusted for age and gender, preoperative BMI correlated with preoperative RDI ($r = 0.27$; $P < .01$).

Conclusions. OSA is prevalent in at least 45% of bariatric surgery patients. Preoperative BMI correlates with the severity of OSA. Surgically induced weight loss significantly improves obesity-related OSA and parameters of sleep quality. (Surgery 2007;141:354-8.)

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OBSTRUCTIVE SLEEP APNEA (OSA) is highly prevalent in bariatric surgery patients and patients with clinically significant obesity.¹⁻⁴ OSA increases the burden of clinically significant obesity, because it induces alveolar hypoventilation and respiratory insufficiency, and contributes other cardiopulmonary consequences of pulmonary hypoventilation, such as pulmonary hypertension and cor pulmonale.⁵ Because OSA increases the likelihood of adverse events in the postoperative period, preop-

erative recognition and treatment are essential especially in bariatric patients.

Sporadic evidence documenting improvement of OSA after surgically induced weight loss has been reported in very small cohorts of patients.^{2,6-10} This study was undertaken to provide objective evidence that surgically induced weight loss improves obesity-related OSA and corrects sleep hygiene parameters in patients undergoing bariatric surgery for clinically important obesity.

METHODS

This study was approved by the Institutional Review Board at the University of South Florida Health Science Center and was conducted in compliance with HIPAA guidelines and regulations.

Patient population. Prospectively collected data on all patients who underwent Roux-en-Y gastric

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bypass (RYGB) for clinically significant obesity from September 1998 to March 2005 were analyzed. All patients who were evaluated for bariatric surgery were administered a health questionnaire that included the Epworth Sleepiness Scale (ESS). The ESS screens for daytime sleepiness and somnolence and has been previously validated.¹¹ Patients who scored ≥ 6 on the ESS were referred for a consultation with a pulmonologist and subsequently to the Sleep Laboratory for standard polysomnography.^{1,2}

Evaluation of sleep hygiene. Polysomnography was performed using a split-night protocol with an initial baseline segment followed by titration of continuous positive airway pressure (CPAP) or bi-level therapy (Bi-level Positive Airway Pressure [BiPAP], Respironics, Inc., Pittsburgh, PA). Measurements included the respiratory disturbance index (RDI), sleep efficiency, rapid eye movement (REM) latency, and minimum oxygen saturation (SpO_2). Severity of OSA was categorized by RDI (absent, 0 to 5; mild, 6 to 20; moderate, 21 to 40; and severe, >40). Preoperative CPAP or BiPAP was prescribed to all patients diagnosed with moderate-severe OSA and to the majority of patients diagnosed with mild OSA. All patients with OSA were referred for polysomnography 6 to 9 months postoperatively or when weight loss exceeded 75 lbs.² Patients completed follow-up ESS questionnaires during routine postoperative visits.

Bariatric procedure. Prospective bariatric patients were evaluated by an interdisciplinary team according to the criteria for bariatric surgery established by the National Institutes of Health. During the study period, Roux-en-Y gastric bypass was used as the operation of choice both for primary and revisional procedures. At the outset of the study, open RYGB was predominant; however, currently more than 90% of RYGB procedures are done laparoscopically (overall mix: 50% laparoscopic, 50% open). Fiber optic-assisted endotracheal intubation was used in the majority of patients with moderate-severe OSA and at the anesthesiologist's discretion. All patients were extubated in the operating room at the conclusion of the procedure.

Follow-up. Current follow-up was obtained from office visits and/or phone interviews. Postoperative polysomnography performed at other sleep labs were reviewed, and those centers were contacted for further details of the sleep study, as needed.

Statistical analysis. The means of parametric continuous data were compared using the paired *t* test. Correlations and chi-square analyses were conducted to assess levels of associations among covariates. Analysis of variance (ANOVA) and lin-

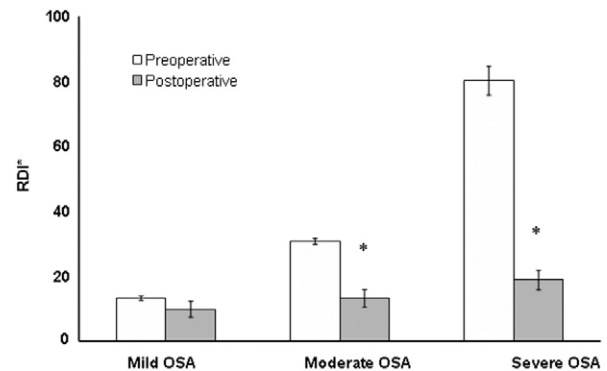


Figure. Improvement in respiratory disturbance index (RDI) as documented by polysomnography in 101 patients who underwent bariatric surgery (median follow-up, 11 months; range, 4 to 62 months). Patients were grouped according to the preoperative severity of obstructive sleep apnea (OSA) and RDI. The most dramatic improvement was in patients with severe OSA. * $P < .001$ vs preoperative.

ear regression were used to assess the relative impact of independent variables (age, body mass index [BMI], and ESS scores) on the dependent variable (RDI). Patients were stratified into groups according to BMI (<40 , 40 to 49, 50 to 59, ≥ 60 kg/m²) or the severity of OSA (absent, mild, moderate, severe) for chi-square analyses; $P < .05$ was considered significant.

RESULTS

Prevalence of OSA in bariatric patients. A total of 597 consecutive bariatric surgery patients completed the ESS during the study period (Figure); 447 patients who scored ≥ 6 on the ESS were referred for consultation with a pulmonologist. On further consultation with the pulmonologist, 98 of the 447 patients did not undergo polysomnography, because their symptoms were mild and not attributed to airway obstruction. The remaining 349 patients underwent polysomnography for evaluation and documentation of OSA. This group was composed of 321 women and 68 men; mean age was 45 ± 1 years; mean BMI was 52 ± 1 kg/m²; and mean ESS score was 10 ± 1 .

Severity of OSA. Of the 349 patients who underwent polysomnography, 289 patients had evidence of OSA; mean RDI was 32 ± 1 (normal, 0 to 5); 116 patients (33%) were diagnosed with severe OSA, 63 patients (18%) were diagnosed with moderate OSA, and 110 patients (32%) with mild OSA. The remaining 60 patients (17%) did not have polysomnographic evidence of OSA (Figure).

Preoperative treatment of OSA. All patients with moderate-severe OSA, as well as the majority of

Table. Preoperative and postoperative clinical parameters and polysomnography findings in 101 patients at a median of 11 months (6 to 42 months) after bariatric surgery

| | <i>n</i> | <i>Preoperative</i> | <i>Postoperative</i> | <i>P</i> |
|-------------------------------|----------|---------------------|----------------------|----------|
| BMI (kg/m ²) | 101 | 56 ± 1 | 38 ± 1 | <.001 |
| RDI | 101 | 51 ± 4 | 15 ± 2 | <.001 |
| SpO ₂ (%) | 54 | 77 ± 2 | 86 ± 1 | <.001 |
| Sleep efficiency (%) | 50 | 75 ± 2 | 87 ± 1 | <.001 |
| REM latency (min) | 32 | 193 ± 17 | 136 ± 17 | 0.025 |
| CPAP no. of patients | | 83 | 31 | |
| Pressure (cmH ₂ O) | | 11 ± 1 | 7 ± 1 | <.001 |
| BiPAP no. of patients | | 13 | 0 | |
| Pressure (cmH ₂ O) | | I = 15, E = 11 | n/a | n/a |

BMI, Body mass index; *RDI*, respiratory distress index; *SpO₂*, minimum oxygen saturation; *REM*, rapid eye movement; *CPAP*, continuous positive airway pressure; *BiPAP*, bi-level positive airway pressure; *I*, inspiratory positive airway pressure; *E*, expiratory positive airway pressure; *n/a*, not applicable. Data are mean ± SEM; *P* values are paired *t* test.

patients with mild OSA, were prescribed CPAP or BiPAP preoperatively. CPAP/BiPAP did not correct sleep parameters during polysomnography in 20 patients (6%) with mild OSA, and therefore they were not prescribed to those patients (Figure). Other than 2 patients who had previously undergone a tracheostomy prior to evaluation for bariatric surgery, we did not perform tracheostomies for the treatment of OSA in this cohort. Routine arterial blood gas measurement was done in all patients with OSA during the preoperative evaluation visit 1 week prior to RGYB.

Postoperative management of OSA. All patients who were prescribed CPAP/BiPAP preoperatively were instructed to bring their equipment to the hospital during the index bariatric procedure. CPAP/BiPAP was administered first in the recovery room, continued as tolerated during the day, and required during day or night sleep. Routine admissions to the intensive care unit (ICU) for respiratory care were rare in our program and did not exceed 1%. All patients with OSA were managed routinely in an acute care (non-ICU) setting with the help of pulmonologists and respiratory therapists. Specifically, patients with severe OSA were not monitored routinely by pulse oximetry or telemetry except in rare instances where intravenous insulin or β -blockers were used. Postoperative pulmonary complications included respiratory failure (1.2%) and pneumonia (1%).

Outpatient postoperative management of OSA. All patients were referred to a pulmonologist for CPAP/BiPAP monitoring and titration between 6 to 12 months after bariatric surgery or when weight loss exceeded 75 lbs. Repeat polysomnography was used to document resolution of OSA and to determine the need for continuing CPAP/BiPAP.

Postoperative polysomnography. At a median of 11 months postoperatively (range, 6 to 42 months),

101 patients with severe (50%), moderate (22%), and mild (28%) OSA underwent repeat polysomnography. The clinical characteristics and polysomnographic findings are summarized in the Table. Mean BMI decreased from 56 ± 1 to 38 ± 1 kg/m² ($P < .001$); RDI decreased from 51 ± 4 to 15 ± 2 postoperatively ($P < .001$). Similarly, other parameters of sleep quality (SpO₂, sleep efficiency, and REM latency) were improved postoperatively ($P \leq .025$).

Data on preoperative and postoperative use of CPAP/BiPAP was complete and interpretable in 96 of the 101 patients. The number of patients who were using CPAP/BiPAP decreased from 83 patients to 31 patients postoperatively, and the CPAP pressure settings were decreased to 7 ± 1 from 11 ± 1 cmH₂O preoperatively ($P < .001$, Table). BiPAP was discontinued in all 13 patients who were using it preoperatively.

The Figure depicts improvement in RDI of 101 patients who underwent postoperative polysomnography as stratified by preoperative OSA severity. There was decreased RDI in patients with moderate OSA (13 ± 3 vs 31 ± 1, $P < .001$ vs preoperative) and dramatic improvement in patients with severe OSA (19 ± 3 vs 80 ± 4, $P < .001$ vs preoperative). There was a modest degree of improvement in patients with mild OSA (RDI: 10 ± 2 vs 13 ± 1, $P > .05$ vs preoperative).

Follow-up. The remaining 168 patients who were diagnosed with preoperative OSA but did not undergo postoperative polysomnography were either still within the first 6 months after bariatric surgery and therefore have not been referred for polysomnography yet ($n = 89$ patients), refused to undergo follow-up polysomnography, or were denied insurance authorization for a follow-up study by third-party payers ($n = 77$ patients). Additionally, 2 patients died in the immediate postoperative pe-

riod, one from an anastomotic leak and the other from a pulmonary embolism.

Improvement in the ESS. Complete and interpretable data on preoperative and postoperative ESS were available in 516 patients. There was significant and progressive reduction in ESS scores within 3 months after bariatric surgery (6 ± 1 vs 10 ± 1 , $P < .001$ vs preoperative, paired t test; $n = 391$ patients) and in patients with follow-up at 2 years (4 ± 1 vs 10 ± 1 , $P < .0001$ vs preoperative; paired t test; $n = 138$ patients).

Predictors of the severity of OSA. The relationship between clinical parameters (age, gender, preoperative BMI, and preoperative ESS) and the severity of OSA as measured by RDI was tested. Increasing BMI or male gender was associated with an increased likelihood of higher RDI and more severe OSA ($P < .001$, chi-square analysis).

Linear regression models were used to further assess the association between preoperative BMI and RDI. BMI correlated with RDI ($r = 0.27$; $P < .001$) in univariate analysis as well as a multivariate analysis adjusted for age and gender ($P < .01$). Preoperative ESS scores did not correlate with the severity of OSA ($P > .05$).

DISCUSSION

This study confirms that surgically induced weight loss results in significant improvement of obesity-related OSA in bariatric patients. Moreover, this is the largest series that documents objective improvement of obesity-related OSA as measured by polysomnography after bariatric surgery.

OSA is prevalent in obese patients and in patients seeking bariatric surgery.¹⁻⁴ This study confirms our earlier reports,^{1,2} that at least 45% of bariatric patients who were screened for OSA were confirmed subsequently to have OSA by polysomnography. Nevertheless, the prevalence of OSA may be greater because we do not routinely refer patients for pulmonary consultation if their ESS score is < 6 . Indeed, others have documented evidence of OSA or OSA-related disordered breathing in 77% to 88% of patients seeking bariatric surgery.^{3,4}

We have chosen an arbitrary cutoff for routine referral of asymptomatic patients based on the self-administered ESS (score ≥ 6).^{1,2} This approach was used throughout the study to ensure consistency and establish a predictive value for the ESS.

In our previous studies, we did not find a correlation between BMI, ESS, and RDI in a small cohort.^{1,2} This was likely due to Type II error because, in this much larger cohort, we were able to estab-

lish a relationship between male gender, increasing BMI, and severity of OSA as measured by RDI.

Screening and treating OSA should be an integral part of preoperative and postoperative care of the bariatric surgical patient because of the high prevalence of OSA in bariatric patients and the consequences of undiagnosed and untreated OSA in the postoperative period.^{5,12} Nonetheless, in our study, 22% of patients (98/447) who were referred to a pulmonologist had symptoms that were mild and not attributable to airway obstruction; an additional 17% (60/349 patients) who scored ≥ 6 on the ESS did not have polysomnographic evidence of OSA. These findings are important in the cost-containment environment of contemporary medical practices and warrant further investigation to identify screening methodology that has a higher predictive value.

Until that time, we recommend screening asymptomatic patients with the ESS and referral for consultation with a pulmonologist if patients score ≥ 6 on the ESS. A formal polysomnographic study remains the gold standard for diagnosing OSA, and titrating CPAP/BiPAP treatment should be undertaken if there is a high index of suspicion for OSA, regardless of ESS scores.

Secondly, we recommend an aggressive approach to treatment of OSA with CPAP/BiPAP. We allow at least 4 to 6 weeks of treatment with CPAP/BiPAP prior to the proposed bariatric procedure to maximize nightly application of the facial apparatus, and, more importantly, to maximize recruitment of alveoli and reverse alveolar hypoventilation. Additionally, we reinstitute CPAP/BiPAP treatment in the recovery room and during day- and night-time sleep during the immediate postoperative period. This concerted effort has minimized the incidence of primary respiratory failure in our cohort (1%). Although others¹³ have expressed concerns that CPAP/BiPAP may precipitate massive bowel distention and anastomotic leaks after RYGB, we and other researchers¹⁴ have not observed any untoward effects such as an increase in intestinal intraluminal air and ileus from wide application of CPAP/BiPAP.

Finally, we recommend postoperative monitoring of sleep quality and utilization of CPAP/BiPAP as weight loss progresses. We found that most patients discontinue the use of CPAP/BiPAP within the first 3 months after bariatric surgery because of dramatic symptomatic improvement and sometimes because of intolerance to "high" pressure settings. Therefore, we recommend formal postoperative consultation with a pulmonologist for titration of CPAP/BiPAP pressure settings to document

resolution of OSA by polysomnography and to determine the clinical significance of the mild degree of OSA after weight loss, as suggested by our data.

This study has several weaknesses. The true incidence and prevalence of OSA in bariatric patients may be underestimated by our data due to the exclusion of patients whose ESS scores were <6. Similarly, and based on our empiric observations at the onset of our study 7 years ago, repeat polysomnography was arranged within 6 to 12 months postoperatively or after 75 lbs of weight loss because many patients report significant symptomatic improvement during that period. However, patient compliance and insurance companies' restrictions negatively affected our ability to obtain postoperative polysomnography in all patients with OSA. In addition, because we used patients' weights at the time of postoperative polysomnography and not the weight at the most recent follow-up, the reported postoperative BMI does not reflect the long-term weight loss after gastric bypass.

By the same token, the persistence of a mild degree of OSA in this cohort may be a reflection of the timing of polysomnography relative to weight loss. This confounding variable may be addressed in future studies using devices that titrate automatically CPAP/BiPAP settings as weight loss progresses, thereby eliminating the need for repeat polysomnography.

Notwithstanding, these data are clear and objective evidence that OSA and other parameters of sleep quality improve after bariatric surgery. These findings, which were gathered from the largest cohort yet reported, also are in accordance with reports of small cohorts that document improvement in RDI, sleep quality, respiratory mechanics, and OSA-associated pulmonary hypertension.^{6-9,15-17}

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