

# Obstructive Sleep-Related Breathing Disorders in Patients Evaluated for Bariatric Surgery

William C. Frey, MD, FCCP; John Pilcher, MD, FACS

Department of Pulmonary, Critical Care and Sleep Medicine, Brooke Army Medical Center, Fort Sam Houston, TX, USA

**Background:** Obesity is a well known risk factor for obstructive sleep apnea (OSA). Medical therapy is not effective for morbid obesity. Bariatric surgery is therefore a reasonable option for weight reduction for patients with clinically severe obesity. Unrecognized OSA, especially in those patients receiving abdominal surgery, has influenced perioperative morbidity and mortality. The incidence of OSA for patients being evaluated for bariatric surgery has not been previously defined.

**Methods:** 40 consecutive patients being evaluated for bariatric surgery were examined with a history, physical examination and laboratory data. Polysomnography (PSG) was conducted in all patients regardless of symptoms.

**Results:** An obstructive sleep-related breathing disorder (OSRBD) was present in 88% of the patients. OSA was present in 29 of 41 (71%) and upper airway resistance syndrome (UARS) in 7 of 41 (17%). The mean low oxygen desaturation was 84% and continuous positive airway pressure (CPAP) was 10 cm H<sub>2</sub>O pressure. The majority of the patients were women and mean BMI was 47 kg/m<sup>2</sup>. Patient characteristics failed to predict the severity of OSRBD.

**Conclusions:** This population of clinically severe obese patients being evaluated for bariatric surgery had an 88% incidence of an OSRBD, 71% with OSA. Appropriate therapy with CPAP perioperatively would theoretically prevent hypoxic complications associated with OSRBD. Providers should have a low threshold for ordering a PSG as part of the preoperative evaluation for bariatric surgery. Empiric CPAP at 10 cm H<sub>2</sub>O should be considered for those patients who cannot complete a PSG before surgery.

**Key words:** Morbid obesity, bariatric surgery, gastric bypass, obstructive sleep apnea syndromes

## Introduction

The obstructive sleep apnea-hypopnea syndrome (OSA) is a disorder characterized by repetitive partial or complete obstruction of the upper airway, often with associated oxygen desaturations and arousals from sleep. The classic daytime manifestations of OSA arise from fragmented sleep and include excessive daytime sleepiness (EDS) and decreased concentration. The patient or people familiar with the patient's sleep may report cessation of breathing, choking, gasping, frequent awakenings and unrefreshing sleep. Features associated with OSA include snoring, systemic and pulmonary hypertension, nocturnal angina, sleep-related cardiac dysrhythmias, gastroesophageal reflux, insomnia and impaired quality of life.<sup>1</sup> Upper airway resistance syndrome (UARS) shares the daytime symptoms with OSA, but patients do not have apneas or hypopneas on polysomnography (PSG). UARS patients have respiratory effort-related arousals (RERA) or a pattern of progressively negative intrathoracic pressure, terminating in an arousal from sleep.<sup>2</sup> OSA and UARS are the obstructive sleep-related breathing disorders (OSRBD). Other sleep-related breathing disorders include central sleep apnea syndrome, central alveolar hypoventilation syndrome, primary snoring, altitude insomnia, sleep-related laryngospasm, sleep choking syndrome and sleep disorders associated with other medical conditions such as asthma and chronic obstructive pulmonary disease.<sup>3</sup> The incidence of

---

Presented as a poster at the Annual Meeting of the American Thoracic Society, Toronto, Canada, May 5-10, 2002.

Reprint requests to: William C. Frey, MD, Dept. of Pulmonary, Critical Care and Sleep Medicine, Brooke Army Medical Center, 3851 Roger Brooke Dr., Fort Sam Houston, TX 78234, USA. Fax: 210-916-0709; e-mail: william.c.frey@us.army.mil

The opinions expressed herein are those of the authors and should not to be construed as official or reflecting the policy of either the Department of the Army or Department of Defense.

OSA in the United States has been estimated at 2% of women and 4% of men. The incidence of asymptomatic OSRBD has been estimated at 9% of women and 24% of men.<sup>4</sup>

The three most common sleep-related breathing disorders in obese patients are snoring, OSA and the obesity hypoventilation syndrome.<sup>5</sup>

Clinically severe obesity is defined as a body mass index (BMI) >40 kg/m<sup>2</sup> or a BMI 35-40 kg/m<sup>2</sup> with comorbid conditions such as cardiopulmonary disease, diabetes and "obesity-induced physical problems that interfere with lifestyle".<sup>6</sup> Overall, the population of patients with a BMI >40 kg/m<sup>2</sup> is growing.<sup>7</sup> The National Health and Nutrition Examination Survey (NHANES) III reported that between the years 1988-1994, 2.9% of adults were extremely obese (BMI >40 kg/m<sup>2</sup>). NHANES 1999-2000 revealed that the extremely obese population had increased by 1.8% to 4.7%. Subgroup analysis of the 1999-2000 survey revealed that non-Hispanic black women had the highest prevalence of extreme obesity, 15.1%.

Obesity is a well known risk factor for OSA.<sup>8,9</sup> Medical therapy for morbid obesity is usually ineffective.<sup>6</sup> Bariatric surgery for patients who fail medical therapy has been found to be effective and to decrease morbidity and mortality.<sup>6,10,11</sup> Thus, bariatric surgery is becoming more frequent for the treatment of obesity. OSA has the potential risk of increasing perioperative morbidity and mortality based on the relationship between obesity and OSA. The incidence of OSRBD has not been previously described for patients undergoing evaluation for bariatric surgery.

## Methods

Patients were enrolled between February 1996 and June 1999 and included 40 consecutive outpatients being evaluated for bariatric surgery and one patient that had a postoperative ambulatory inpatient PSG at Wilford Hall Medical Center (WHMC). A total of 38 patients were referred to the WHMC Sleep Disorders Center for a comprehensive evaluation. The comprehensive evaluation included a detailed sleep questionnaire describing sleep schedule and hygiene, screening for insomnia, movement disor-

ders, parasomnias, excessive sleepiness, and respiratory patterns during sleep. The questionnaire also covered general medical and surgical history, family and psychological history, medications, and social history to include use of alcohol and tobacco. Patients were encouraged to complete the questionnaire with someone who was familiar with their sleeping patterns. A clinic visit included a physician sleep specialist's review of the questionnaire with additional history taken as necessary, a physical examination, and laboratory work (thyroid-stimulating hormone level in all, other lab studies ordered at physician's discretion). The clinic visit also included patient education on what to expect from their sleep lab experience and PSG. Two patients received a PSG at another institution and only a summary report was available for interpretation. The one postoperative patient was evaluated by the sleep inpatient service, and a physician sleep specialist interpreted the PSG.

The patients receiving a comprehensive evaluation underwent a diagnostic protocol PSG (Table 1) at the WHMC sleep laboratory. PSG recordings were conducted in the standard fashion<sup>12</sup> and included: electroencephalogram (EEG), electrocardiogram (ECG), submental, intercostal and pretibial electromyography (EMG), electrooculogram (EOC), and pulse oximetry. Respiratory effort was meas-

**Table 1. WHMC Diagnostic Protocol**

### Criteria to initiate CPAP

- Apnea-hypopnea index (AHI) >20/hour in supine position
- AHI >10/hour in lateral or prone position
- Persistent oxyhemoglobin desaturation <85% with AHI <5/hour
- Repetitive oxyhemoglobin desaturation <75% with AHI <5/hour
- Repetitive oxyhemoglobin desaturation <80% with ventricular tachycardia or frequent premature ventricular contractions with AHI >5/hour
- Patient allowed to sleep through first rapid-eye movement (REM) period
- Above criteria and no REM within the first 3 hours

### Goals of CPAP titration

- Eliminate sleep-related breathing disorder
- Eliminate snoring
- Decrease the arousal index (ARI) <15/hour
- Above in supine position and during REM

ured with thoracic and abdominal inductance plethysmography. Airflow was measured with a nasal thermistor. A microphone detected snoring. All studies were conducted overnight under the supervision of a sleep technician. If the patient met diagnostic criteria for continuous positive airway pressure (CPAP) during the initial PSG, the technician titrated CPAP during the remainder of the study (split-night PSG). Patients with OSRBD that did not have an adequate CPAP titration during the split-night study were invited back for a second PSG for repeat CPAP titration. Patients with OSRBD that did not fulfill the diagnostic criteria for a split-night study on the initial PSG were also invited back for a PSG with CPAP titration.

A Technician scored and a Board Certified Physician Sleep Specialist reviewed and interpreted all PSGs. Scoring was by standard procedure.<sup>12</sup> OSA was defined as an apnea-hypopnea index (AHI) of  $\geq 5$  per hour. Severity of OSA was classified as mild (AHI 5-15/hour), moderate (AHI 16-30 per hour) and severe (AHI  $>30$  per hour).<sup>1</sup> UARS was defined as an AHI  $<5$  per hour, an Epworth Sleepiness Scale (Epworth)<sup>13</sup>  $\geq 8$  of 24 (a measure of EDS), an arousal index (ARI)  $\geq 10$  per hour, and the arousals were associated with increasing respiratory effort by intercostal EMG or crescendo snoring. Patients with UARS did not have another diagnosis that would account for the EDS. This definition of UARS differs from Guilleminault's<sup>2</sup> original study by not using an esophageal manometer to measure respiratory effort. Optimal CPAP was determined by the physician sleep specialist interpreting the PSG and was documented in the PSG summary. All patients were invited back to the Sleep Disorders Center for a follow-up clinic visit to discuss the PSG results and prescribe CPAP when indicated.

## Results

The majority of the patients were women, 34 of 41 (83%). The mean age of the women was 43 years and for the men 50 years old. The average BMI for men and women was similar, 47 kg/m<sup>2</sup> (range 41-58 kg/m<sup>2</sup>) and 48 kg/m<sup>2</sup> (range 37-58 kg/m<sup>2</sup>) respectively. Systemic hypertension was present in 49% of the patients, all of the men and 41% of the women.

Physical examination included the Mallampati airway classification for 24 patients with a mean of 3 (range 1 to 4). Hypothyroidism was not detected in the 39 patients evaluated at WHMC. The serum bicarbonate level mean was 27 for 34 patients. None of the patients had an elevated bicarbonate level to suggest compensation for chronic hypercarbia or hypoventilation. The Epworth mean was 13 (range 8-18) for seven of eight men and 11 (range 2-22) for 31 of 35 women who completed the questionnaire. The patient population is characterized in Table 2.

An OSRBD was evident in 36 of 41 patients (88%). The majority of the OSRBD was OSA, 29 of 41 (71%). UARS was present in 7 of 41 patients (17%). All patients were snorers and five of 41 patients (12%) had normal PSGs. All the men studied had OSRBD, six (86%) with OSA and one (14%) with UARS. OSRBD was present in 29 of 34 women (85%), 23 with OSA (68%) and six with UARS (18%). Table 3 includes the results from the PSGs.

**Table 2. Patient characteristics**

	Women	Men
n	34	7
Age (years)	43 (25-60)	50 (19-58)
Weight (kg)	128 (93-198)	145 (127-181)
BMI (kg/m <sup>2</sup> )	48 (37-90)	47 (38-58)
Epworth Scale	11 (2-22)	13 (8-18)
Hypertension	14/34 (41%)	7/7 (100%)

BMI= Body mass index.

Age, Weight, BMI and Epworth data presented as mean (range).

**Table 3. Polysomnography Data**

	Women	Men
Apnea-hypopnea Index	16 (1-70)	22 (4-75)
Arousal Index	27 (7-85)	34 (7-97)
Baseline oxyhemoglobin saturation	95 (90-99)	94 (90-98)
Low oxyhemoglobin saturation	83 (63-93)	79 (68-94)

Data presented as mean (range).

All calculations for men based on 7 patients.

Apnea-hypopnea index calculation for women based on 34 patients.

Arousal index calculation for women based on 31 patients.

Baseline oxyhemoglobin saturation for women based on 32 patients.

Low oxyhemoglobin saturation for women based on 33 patients.

For patients with OSA, the severity was mild in 11 of 29 (38%), moderate in 12 of 29 (41%) and severe in 6 of 29 (21%). Figures 1, 2 and 3 reveal that the severity of OSA did not correlate with the BMI, the Epworth or the Mallampati airway classification. The patients with OSA had a mean total AHI of 23 per hour (range 5-75) and a mean low oxyhemoglobin saturation of 84% (range 63-94%). The mean arousal index was 32 per hour (range 7-97) and the mean Epworth was 11 (range 2-22). The mean AHI in the six men with OSA was 22 per hour (range 6-75) with a mean low oxyhemoglobin desaturation of 79% (range 68-94%). The mean AHI of the 23 women with OSA was 16 per hour (range 5-67) with a mean low oxyhemoglobin desaturation of 83% (range 63-92%).

The six women and one man with UARS had a mean total AHI of 3 per hour (range 2-4). These patients, however, did have an increase in SRBD during Rapid Eye Movement sleep (REM) with a mean REM AHI of 9 per hour (range 0-16). These patients had a mean low oxyhemoglobin saturation of 84% (range 80-89%), mean ARI of 17 per hour (range 11-30) and a mean Epworth 11 (range 8-18).

Electrocardiogram evaluation was unremarkable in 37/38 (97%) of patients studied with a diagnostic protocol PSG. One woman with UARS had frequent premature ventricular contractions.

An adequate CPAP titration was performed in 23 of the 36 patients with OSRBD with an average CPAP of 10 cm H<sub>2</sub>O (range 5-14). CPAP titration for 13 patients was accomplished during a split-night study. The remaining 10 patients required a second night CPAP titration. Two patients did not tolerate CPAP during their overnight titration during a split-night study. They were encouraged to return to the Sleep Laboratory for daytime "desensitization" to CPAP before a second night titration, but declined the invitation. The other 11 patients were encouraged to return for repeat CPAP titration, but failed to comply with these recommendations.

Although the majority of the patients underwent a diagnostic protocol PSG, three other patients had alternative evaluations. OSA was diagnosed in two women having a sleep evaluation at another facility. The first patient had severe OSA with an AHI of 47 per hour and low oxyhemoglobin saturation of 81%. The other patient had moderate OSA with an AHI of 26 per hour and a low oxyhemoglobin saturation of

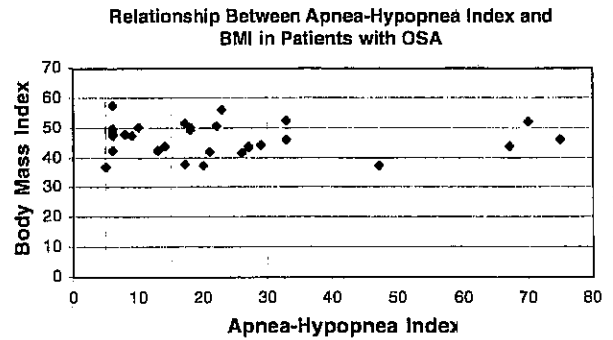


Figure 1. Solid lines indicate cutoff for OSA severity classification: Apnea-hypopnea index  $\geq$  5/hr - 15/hr, mild;  $\geq$  15/hr - 30/hr, moderate;  $\geq$  30/hr, severe.

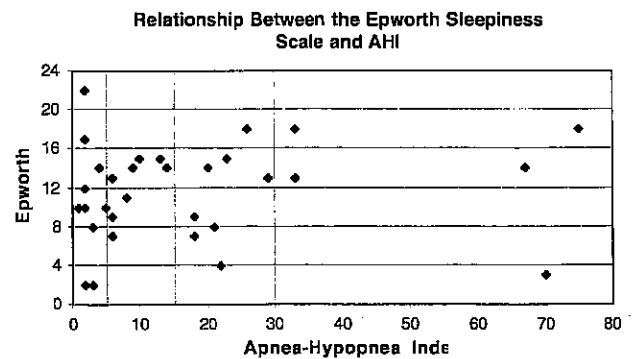


Figure 2. An Epworth  $\geq$  8 is consistent with excessive daytime somnolence.<sup>13</sup> An Epworth and AHI was available for 34 patients.

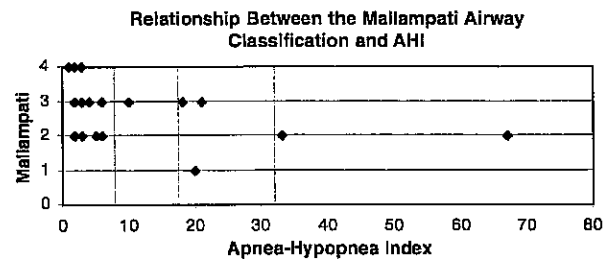


Figure 3. Mallampati airway classification and AHI was available for 24 patients.

84%. The woman evaluated with a postoperative ambulatory study also had moderate OSA with an AHI of 17 and a low oxyhemoglobin saturation of 71%. This patient was started on CPAP at an empiric level of 12 cm H<sub>2</sub>O.

Although five patients had a normal PSG and did not have excessive daytime somnolence, three of the five had transient oxyhemoglobin desaturations below 90% with a range from 74% to 88%.

## Discussion

Obesity is a well-known risk factor for OSRBD. Our bariatric surgery patient population with an average BMI of 47 kg/m<sup>2</sup> had a very high incidence of OSRBD (88%). The majority (71%) of these patients had OSA with a mean low oxyhemoglobin saturation of 84%. Even the patients that did not meet criteria for OSA, but had UARS, had a low oxyhemoglobin saturation of 84%. Transient nocturnal oxyhemoglobin desaturations occurred in three of five patients with a normal PSG.

Hemodynamic complication in the postoperative period can lead to significant morbidity and mortality. The acute hemodynamic consequences of OSA include arrhythmias, systemic hypertension, pulmonary hypertension and decrease in cardiac output. The most common cardiac arrhythmia is a cyclic bradycardia that is present in 80% of patients with OSA. The degree of bradycardia is proportional to the level of oxygen desaturation and occurs during the initial portion of the apnea. Bradycardia is secondary to an increase in vagal parasympathetic tone and can be prevented with atropine or supplemental oxygen.<sup>14,15</sup> The bradycardias are so common that they are typically not commented upon by the PSG technician or the sleep specialist reviewing the PSG at our institution.

In 400 patients evaluated for cardiac arrhythmias associated with OSA, 20% had frequent premature ventricular contractions (more than 2 per minute) and 4% had ventricular tachycardia. In that same patient population, sinus arrest (2.5 to 13 second pauses) occurred in 11%. Sudden death is a rare but documented complication of OSA.<sup>16</sup> Block et al<sup>17</sup> reported three patients that developed heart block after bariatric surgery. All three patients were diagnosed with OSA. For two of these patients, OSA was a new perioperative diagnosis. The hemodynamic manifestations and cardiac arrhythmias of OSA often resolve with appropriate therapy in an otherwise structurally normal heart.<sup>16</sup> Our population had only one patient with frequent premature ventricular contractions (PVCs), defined as more than 6 PVCs per minute.

Bariatric surgery is a safe and effective therapy for the treatment for clinically severe obesity. In 1992,

Mason et al<sup>18</sup> reported the 5-year experience of complications and postoperative hospital stay from the National Bariatric Surgery Registry. Only five deaths in 5,178 patients were reported. There were no postoperative complications in 89.7% of the patients. The median length of hospital stay in uncomplicated patients was 4 days. The most common complication was "respiratory" occurring in 4.47% with a median postoperative stay of 6 days (range 2-23 days). The specifics of the respiratory complications were not detailed. One patient had fatal pulmonary embolism and was reported separately from the other respiratory complications. It is conceivable that postoperative desaturations associated with OSA would be considered a postoperative respiratory complication.

The incidence of OSRBD for patients being evaluated for bariatric surgery has not been clearly documented. In 1991, Rajala et al<sup>19</sup> published a study examining the breathing disturbances during sleep of morbidly obese patients. Their results from 27 patients revealed OSA in 40.7%. OSA was documented in 10 of 13 men (76.9%) and 1 of 14 women (7.1%). The lower incidence of OSA may be explained by the length and timing of PSG (a daytime 90 minute study) and OSA definition (an oxygen desaturation index of  $\geq 10$  per hour rather than AHI). In 1998, Dominguez-Cherit et al<sup>20</sup> reported on the anesthetic preoperative evaluation of 37 bariatric surgery patients. Of these patients, eight had PSGs and six were diagnosed with OSA (diagnostic criteria not reported). In 1999, van Boxem et al<sup>21</sup> reported a lower incidence of sleep-disordered breathing for patients referred from the Dutch Obesity Center for potential gastric banding. The incidence of OSA (AHI  $\geq 5$ ) was 19 of 48 (40%). A significant limitation to the study was that the portable PSG data recorder (Eden Trace<sup>®</sup>, II Recording System) did not include EEG, EOG and chin EMG. These PSG components are recommended as part of the minimum monitoring for PSG to evaluate SRBD.<sup>22</sup> More recently, Valencia-Flores et al<sup>23</sup> reported a high incidence of OSA for patients followed at the Obesity Clinic of the National Institute of Nutrition Salvador Zubiran (Mexico City). Patients referred for PSG included 52 "morbidly" obese patient with an average BMI of 51 kg/m<sup>2</sup> and symptoms of snoring and excessive daytime somnolence. Daytime hypercapnia was present

in 23% of these patients. A very high incidence of this cohort, 51 of 52 patients (98%), had an AHI  $\geq$  5/hr. Cardiac abnormalities were present in 31% of the patients. Patients with an AHI  $\geq$ 65 or oxygen desaturations below 65% had a higher incidence of cardiac abnormalities. This study described a different population of severely obese patients than our population, because it included patients with daytime hypercarbia (Pickwickian syndrome<sup>8</sup>) and did not mention if the patients were being evaluated for bariatric surgery. The very high incidence of OSA may be secondary to a selection bias. The patients were required to have symptoms of daytime sleepiness and snoring for entry into the study. Our study reveals a high incidence of OSRBD for patients being evaluated for bariatric surgery, with and without symptoms consistent with OSRBD.

Sleep is divided into two distinct forms, REM and non-REM (NREM) sleep. The most striking difference between REM and NREM is that REM is associated with muscle hypotonia or atonia in all muscles except extraocular muscles and the diaphragm.<sup>24</sup> The hypoxic ventilatory response and the arousal threshold in response to hypoxia are both decreased in REM.<sup>25</sup> Especially in obese patients, REM sleep is associated with apnea prolongation and severe oxygen desaturation secondary to decreased lung volumes and loss of chest wall muscle tone. These deep desaturations may have significant cardiovascular consequences.<sup>17,23,26</sup> The REM AHI was not only elevated in the OSA group, but also in the UARS patients. The majority of the desaturations in the UARS subset as well as the patients with normal PSGs occurred during REM.

The initial postoperative period is characterized by sleep deprivation and decreased or absent REM. Knill et al<sup>27</sup> reported the sleep stage patterns in patients undergoing abdominal surgery, cholecystectomy and gastroplasty. The gastroplasty patients had significant REM suppression on the operative day through postoperative day 2. On postoperative day 4, the gastroplasty patients had an increase in density and duration of REM or REM "rebound". REM rebound in the late postoperative period is associated with an increase in the number of oxyhemoglobin desaturation,<sup>28</sup> suggesting more apneas and hypopneas. The National Registry<sup>18</sup> reported that the median postoperative stay in the hospital was 4 days. With REM rebound occurring around

this time, it is possible for the patients with unrecognized OSRBD to have severe hypoxic complications while at home and not in a monitored setting.

Narcotics are commonly used for analgesia in the postoperative period. Administration of 10 mg of morphine to normal subjects decreases the hypoxic and hypercapnic ventilatory responses by 60% and 40% respectively. Narcotics also decrease the upper airway tone and OSRBD can occur in normal patients subsequent to administration. The severity of apneas, hypopneas and oxyhemoglobin desaturations also can increase after narcotic administration in patients with untreated OSRBD.<sup>29</sup> The combination of REM rebound and narcotics could potentially increase the hypoxic complications seen in the postoperative untreated OSRBD patient.

Predicting the severity of OSA is difficult. Serafini et al<sup>30</sup> attempted to determine clinical predictors of sleep apnea in patients undergoing bariatric surgery. The Epworth Sleepiness Scale and BMI independently failed to predict the severity of sleep apnea. This was also true for our patient population (Figures 1 and 2). Figure 3 reveals that the Mallampati airway classification also does not predict severity of OSRBD.

The level of CPAP necessary to treat the OSRBD is very variable and should be individualized. An overnight PSG is a reliable study to determine the presence and severity of OSRBD and initiate corrective therapy with CPAP.<sup>22</sup> Despite patient education, 36% of our population did not comply with the recommendation of CPAP titration. The patients that did comply with therapy needed an average of 10 cm H<sub>2</sub>O pressure of CPAP. CPAP has been shown to be safe in the postoperative gastric bypass patient.<sup>31</sup>

## Summary

The incidence of OSRBD in our bariatric study population was very high. Cardiovascular consequences of OSRBD are well documented. These consequences may be increased in the postoperative period when the combination of REM rebound and narcotic analgesia increase oxyhemoglobin desaturations. Health-care providers evaluating patients for bariatric surgery should consider referral for a sleep

evaluation and PSG as part of the preoperative evaluation. Clinical evaluation with BMI, Epworth Sleepiness Scale and the Mallampati airway classification failed to predict the severity of OSRBD. Therapy for OSRBD should be initiated prior to surgery to minimize the hemodynamic complications of OSRBD and to familiarize the patient with CPAP. Patients should be educated about the importance of CPAP use to correct OSRBD. Continued use of CPAP in the postoperative period will theoretically decrease the potential morbidity and mortality of OSRBD in the hospital and after discharge from the hospital.

Special thanks to Ethan Emmons, MD for his technical expertise.

## References

1. The report of an American Academy of Sleep Medicine Task Force. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. *Sleep* 1999; 22: 667-89.
2. Guilleminault C, Stoohs R, Clerk A et al. A cause of excessive daytime sleepiness. The upper airway resistance syndrome. *Chest* 1993; 104: 781-7.
3. The International Classification of Sleep Disorders, Revised: Diagnostic and Coding Manual. Rochester, Minnesota: American Sleep Disorders Association, 1997.
4. Young T, Palta M, Dempsey J et al. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993; 328: 1230-5.
5. Shahi B, Praglowski B, Deitel M. Sleep-related disorders in the obese. *Obes Surg* 1992; 2: 157-68.
6. National Institute of Health Consensus Development Conference Draft Statement. Gastrointestinal surgery for severe obesity 25-27 March 1991. *Obes Surg* 1991; 1: 257-66.
7. Flegal KM, Carroll MD, Ogden CL et al. Prevalence and trends in obesity among US adults, 1999-2000. *JAMA* 2002; 288: 1723-7.
8. Burwell C, Rabin E, Whaley R et al. Extreme obesity associated with alveolar hypoventilation-Pickwickian syndrome. *Am J Med* 1956; 21: 811-8.
9. Kryger M, Guesney L, Holder D et al. The sleep deprivation of the obese patient. *Am J Med* 1974; 56: 431-9.
10. Pories WJ. Why does the gastric bypass control type 2 diabetes mellitus? *Obes Surg* 1992; 2: 303-13.
11. Rasheid S, Banasiak M, Gallagher SF et al. Gastric bypass is an effective treatment for obstructive sleep apnea in patients with clinically significant obesity. *Obes Surg* 2003; 13: 58-61.
12. Rechtschaffen A, Kales A. A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects. Brain Information Service, Brain Research Institute, UCLA, Los Angeles, 1978.
13. Johns MW. A new method for measuring daytime sleepiness: The Epworth Sleepiness Scale. *Sleep* 1991; 14: 540-5.
14. Shepard JW. Cardiopulmonary consequences of obstructive sleep apnea. *Mayo Clin Proc* 1990; 65: 1250-9.
15. Shepard JW. Cardiorespiratory changes in obstructive sleep apnea. In: Principles and Practice of Sleep Medicine, 2nd Edn. WB Saunders Company, 1994: 657-66.
16. Gilleminault C, Connolly SJ, Winkle RA. Cardiac arrhythmia and conduction disturbances during sleep in 400 patients with sleep apnea syndrome. *Am J Cardiol* 1983; 52: 490-4.
17. Block M, Jacobson LB, Rabkin RA. Heart block in patients after bariatric surgery accompanying sleep Apnea. *Obes Surg* 2001; 11: 627-30.
18. Mason EE, Renquist KE, Jiang D. Perioperative risks and safety of surgery for severe obesity. *Am J Clin Nutr* 1992; 55 (Suppl): 573S-576S.
19. Rajala R, Partinen M, Sane T et al. Obstructive sleep apnea syndrome in morbidly obese patients. *J Intern Med* 1991; 230: 125-9.
20. Dominguez-Cherit G, Gonzalez R, Borunda D et al. Anesthesia for morbidly obese patients. *World J Surg* 1998; 22: 969-73.
21. Van Boxem TJM, de Groot GH. Prevalence and severity of sleep disordered breathing in a group of morbidly obese patients. *Neth J Med* 1999; 54: 202-6.
22. Chesson AL, Ferber RA, Fry JM et al. Practice parameters for the indications for polysomnography and related procedures. *Sleep* 1997; 20: 406-22.
23. Valencia-Flores M, Orea A, Castaño VA et al. Prevalence of sleep apnea and electrocardiographic disturbances in morbidly obese patients. *Obes Res* 2000; 8: 262-9.

24. Carskadon MA, Dement WC. Normal human sleep: an overview. In: Principles and Practice of Sleep Medicine, 3rd Edn. WB Saunders Company, 2000: 15-25.
25. Douglas NJ. Respiratory physiology: control of ventilation. In: Principles and Practice of Sleep Medicine, 3rd Edn. WB Saunders Company, 2000: 221-8.
26. Weiss JW, Launois SH, Anand A. Cardiorespiratory changes in sleep-disordered breathing. In: Principles and Practice of Sleep Medicine, 3rd Edn. WB Saunders Company, 2000: 859-67.
27. Knill RL, Moote CA, Skinner MI, et al. Anesthesia with abdominal surgery leads to intense rem sleep during the first postoperative week. *Anesthesiology* 1990; 73: 52-61.
28. Rosenberg J, Wildschiodtz G, Pedersen MH et al. Late postoperative nocturnal episodic hypoxaemia and associated sleep pattern. *Br J Anaesth* 1994; 72: 145-50.
29. Robinson RW, Zwillich CW. The effect of drugs on breathing during sleep. *Clin Chest Med* 1985; 6: 603-12.
30. Serafini FM, Anderson WM, Rosemurgy AS et al. Clinical predictors of sleep apnea in patients undergoing bariatric surgery. *Obes Surg* 2000; 11: 28-31.
31. Huert S, DeShields S, Shpiner R, et al. Safety and efficacy of postoperative continuous positive airway pressure to prevent pulmonary complication after Roux-en-Y gastric bypass. *J Gastrointest Surg* 2002; 6: 354-8.

*(Received January 31, 2003; accepted March 29, 2003)*