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## Pathophysiology

Obstructive sleep apnea (OSA) is the periodic reduction (hypopnea) or cessation (apnea) of breathing due to narrowing or occlusion of the upper airway during sleep.

The altered breathing pattern results in hypoxia and sleep disturbance preventing restful sleep. Although some patients may be asymptomatic, loud snoring, restlessness and periods of apnea while sleeping, daytime somnolence, feeling unrefreshed after sleep, and the ability to easily fall asleep, are classic symptoms for this condition.

OSA is thought to affect between 17 and 24 percent of North American adults<sup>1</sup> and severely affect 2-6%. The incidence of OSA in the morbidly obese population is increased, and has been reported between 38% and 88%.<sup>2-5</sup> OSA has been linked to premature death<sup>6</sup>, traffic accidents<sup>7</sup>, hypertension<sup>8</sup>, ischemic heart disease, stroke<sup>9</sup>, type II diabetes<sup>10</sup>, increased neck circumference<sup>11,12</sup>, and visceral obesity<sup>13</sup>. It has been associated with a significantly increased incidence of sudden death from cardiac causes<sup>14</sup> as well as an increased incidence of nocturnal cardiac arrhythmias<sup>15</sup> and chronic or acute adverse cardiovascular events.<sup>16</sup>

The actual mechanism of OSA is believed to be secondary to increased tissue thickness of the structures of the tongue and soft tissues in the pharyngeal cavity which decrease the relative passageway for air to the trachea.<sup>17</sup> The protective muscles of the oropharynx have increased tone during the day to protect a patent airway. They relax during sleep, resulting in relative obstruction of the airway. When excessive soft tissue in the surrounding structures is present, the obstruction can lead to sleep-disordered breathing if mild, or obstructive apnea if more severe. Decrease in the volume of these

soft tissue structures after weight loss is hypothesized to produce improvement of OSA. Increased levels of inflammatory cytokines and decreased expression of inflammatory regulators have also been observed among obese patients with OSA, although this  
55 mechanism has yet to be fully elucidated.<sup>12,13</sup>

Diagnosis of OSA based on physical examination and review of past medical history is difficult. Several screening tools for sleepiness have been developed, such as the Epworth Sleepiness Score, the Maintenance of Wakefulness Test, the Berlin  
60 Questionnaire and the STOP-BANG Questionnaire, with variable sensitivities and specificities.<sup>18</sup> The standard method of diagnosing OSA is via polysomnography (PSG), which requires an overnight stay in a sleep laboratory. During each hour of sleep, the number of apneas, defined as complete cessation of airflow, and hypopneas, defined as a 50-90% decrease in airflow and at least a 4% drop in oxygen saturation for >10 seconds,  
65 are recorded. An “apnea hypopnea index” (AHI), or “respiratory disturbance index” (RDI) is used to quantitate these hypopneas and classify the degree of sleep disturbance. In general, an AHI of less than 5 is normal, 5-15 mild sleep apnea, > 15 moderate sleep apnea, and  $\geq 30$  severe sleep apnea.<sup>19</sup> The role of the sleep laboratory is to substantiate the diagnosis and degree of sleep apnea, determine its cause if possible, and make  
70 recommendations for treatment (requirement for and level of continuous positive airway pressure (CPAP) or bi-level positive airway pressure (Bi-level PAP).

An associated pulmonary condition, in addition to OSA, seen in patients with extreme morbid obesity while awake is the Obesity Hypoventilation Syndrome. This syndrome, similar to the Pickwickian syndrome of medical texts, involves significant  
75 abnormalities of pulmonary physiology from longstanding severe obesity, pulmonary

hypertension, right-sided cardiac failure, and abnormalities of arterial gas exchange at rest. These patients have high resting arterial pCO<sub>2</sub> levels, depressed arterial pO<sub>2</sub> levels, elevated hematocrits, and dyspnea at minimal exertion or at rest. They are at high risk for pulmonary complications from any surgical intervention, and have an increased risk for venous thromboembolic complications as well.<sup>20,21</sup> OSA has been associated with an increased rate of 30-day mortality, venous thromboembolism, need for reintervention and a longer length of hospital stay.<sup>22</sup> Preoperative blood gas levels, ICU or step-down continuous monitoring postoperatively, and consideration of pulmonary artery catheter monitoring in the postoperative setting are all appropriate for obesity hypoventilation syndrome.

### **Incidence of Obstructive Sleep Apnea and Preoperative Screening**

OSA is a common condition in the general population with increasing prevalence in the obese and morbidly obese populations. The general prevalence of obstructive sleep apnea with daytime somnolence is 3-7% of adult men and 2-5% of adult women.<sup>23-25</sup> The prevalence of OSA is affected by age, gender, ethnicity, associated conditions (pregnancy, hypothyroidism), smoking, alcohol and most strikingly, excess weight and obesity. The overall incidence of OSA in the bariatric population is believed to be 12 to 30 fold higher.<sup>26,27</sup> Preoperative sleep studies prior to bariatric surgery demonstrate an overall prevalence rate ranging from 48 to 91%.<sup>3,4,28,29,30</sup> These studies also show that OSA continues to be under-diagnosed in patients with morbid obesity and that a lower threshold for diagnosis, and intervention may be warranted.<sup>3</sup>

While some reports in the literature recommend routine screening for OSA prior  
100 to bariatric surgery<sup>3</sup>, other reports suggest clinical screening only does not result in any  
increase in postoperative pulmonary complications after laparoscopic RYGB.<sup>31</sup> Most  
current surgical practices refer patients with clinical symptoms of OSA for  
polysomnography, but do not make this a routine preoperative test prior to bariatric  
surgery.

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### **Obstructive Sleep Apnea: Medical and Other Non-Bariatric Surgical Therapies**

#### Medical Treatment

CPAP is the mainstay treatment for moderate to severe OSA and has been shown  
110 to improve objective and subjective measures of OSA.<sup>32,33</sup> Although the optimal form of  
CPAP delivery has not yet been determined, a variety of devices are available that may  
be useful alternatives when a patient is unable to tolerate conventional nasal masks. A  
face mask should be considered if nasal obstruction or dryness limits the use of a nasal  
mask.<sup>32,33</sup> Poor adherence to CPAP is widely recognized as a significant limiting factor  
115 for OSA treatment.<sup>34,35</sup>

CPAP has been shown to be a highly effective treatment for OSA if appropriately  
used. There is evidence that CPAP may lead to an improvement in hypertension,  
especially for patients with moderate to severe OSA.<sup>32,33,36</sup>

CPAP has been shown to improve sleepiness in 2-3 weeks.<sup>37,38</sup> There is,  
120 however, no data in the literature regarding the optimal or minimum amount of time a  
patient with newly diagnosed OSA should be on CPAP therapy prior to surgery to

decrease the risk of perioperative complications. Therefore, while no specific recommendations can be made regarding the optimal duration of CPAP therapy prior to surgery for a patient with newly diagnosed OSA, it is reasonable to incorporate some  
125 time for adaptation to the device into the preoperative period.

The risk of anastomotic complications is not increased by CPAP use in the immediate postoperative period following Roux-en-Y gastric bypass based on the existing literature.<sup>39</sup> It has also been suggested that postoperative CPAP and Bi-level PAP can be safely omitted following gastric bypass if they are observed in a monitored setting  
130 and their pulmonary status is optimized by aggressive incentive spirometry and early ambulation.<sup>31</sup> Postoperative use of CPAP should not be viewed as potentially adverse to outcomes following bariatric surgery due to any such concerns, and its use should be employed by bariatric surgeons based on the patient's pulmonary status postoperatively.

### 135 Surgical Treatment

Although various surgical procedures are available to increase the posterior airspace to treat OSA, no surgical treatment is 100% effective.<sup>40</sup> A variety of extirpative and reconstructive surgical procedures can be performed to address upper-airway soft tissue obstruction or abnormalities that contribute to OSA. Preoperative pharyngeal  
140 anatomy, severity of OSA and patient preference (recovery time, risk of prolonged facial paresthesias and malocclusion) all are factors determining surgical choice.

Tracheostomy may be used as a temporizing bypass approach. Soft tissue removal procedures include tonsillectomy, adenoidectomy, uvulopalatoplasty, and even partial glossectomy. Septoplasty has been employed, as have other skeletal and soft tissue

145 modifications. Although maxillomandibular advancement is the most effective  
craniofacial surgery for the treatment of OSA in adults (86% effective, 43% cure),  
uvulopalatopharyngoplasty (UPPP) is the most widely performed OSA pharyngeal  
surgical technique (50% effective, 16% cure rate).<sup>40,41</sup> However, UPPP has not been  
shown to be effective in patients with clinically severe obesity with a BMI > 35 kg/m<sup>2</sup>.

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### **Treatment of Obstructive Sleep Apnea with Bariatric Surgery**

OSA is associated with obesity and a positive correlation between increasing BMI and  
155 sleep apnea severity has been supported.<sup>1,23,42,43</sup> A ten percent increase in weight  
predicted a 6-fold increase in the odds of developing moderate-to-severe OSA.<sup>44</sup> Weight  
loss by any method, including that following bariatric surgery, is a well-documented  
treatment for OSA. In general, many patients after bariatric surgery will develop clinical  
improvement or resolution of symptoms of OSA, regardless of whether a normal BMI is  
160 ever achieved, as even a modest weight loss of 10-20% has been associated with  
improvement of symptoms and greater than 26% reduction in AHI.<sup>44</sup> It is apparent that  
not all causes of OSA are related to obesity, however, and it is likely that not all patients  
with OSA will resolve despite bariatric surgery. Bariatric surgery has been shown to  
reduce truncal obesity; whether or not there are comparable changes in the upper airway  
165 has not been well established.<sup>45</sup> In a recently published meta-analysis evaluating the  
effects of surgical weight loss on the apnea-hypopnea index, the reduction in AHI was  
found to be 71%.<sup>46</sup>

Data in the literature demonstrates subjective improvement in symptoms of OSA after bariatric surgery. Such benefits include improvement in self-reported postoperative sleep quality and the reduction in daytime sleepiness after Roux-en-Y gastric bypass (RYGB) and vertical banded gastroplasty (VBG)<sup>47</sup>, and laparoscopic adjustable gastric banding (LAGB).<sup>48,49</sup> Improvement in validated quality of life scores was shown with both LAGB and RYGB.<sup>50-52</sup> Improvement in Epworth Sleepiness Scores have been demonstrated after LAGB<sup>48-50</sup>, and RYGB.<sup>5,30,49,52</sup> Other still somewhat subjective parameters of decreased use of CPAP and decreased CPAP pressure requirements have been shown to occur in bariatric patients after LAGB<sup>48,50</sup> and after RYGB.<sup>30,51,52</sup>

Objective parameters regarding severity of OSA have been shown to improve after bariatric procedures and associated weight loss. These include RDI or AHI score improvement for LAGB<sup>48,50</sup>, VBG<sup>47,53</sup> and RYGB.<sup>5,30,47,51-54</sup> Patients who have significant weight loss after RYGB<sup>5,30,51</sup> and LAGB<sup>48</sup> have been shown to demonstrate improvements in postoperative versus preoperative oxygen saturation. Patients undergoing bariatric surgery with postoperative polysomnography testing have shown improved sleep efficiency and increased amounts of REM sleep after VBG<sup>47,53</sup>, LAGB<sup>48,50</sup>, and RYGB.<sup>5,30,47,53</sup> Severity of OSA is often also documented as being improved after these operations, although when strictly measured many patients do have mild OSA for which they take no treatment. In one meta-analysis, more than 62% of patients were found to have significant residual disease after bariatric surgery, with an AHI of more than 15.<sup>46</sup> The severity of preoperative OSA often influences the degree to which OSA improves or resolves after bariatric surgery.<sup>30,48</sup>

190 A more controversial aspect of the effect of bariatric surgery on postoperative  
OSA and sleep quality is that most of the evidence fails to show a significant correlation  
between the BMI and percentage of excess weight loss after surgery to the change in the  
RDI or AHI. This has been reported for VBG<sup>53</sup>, LAGB<sup>48</sup>, and RYGB.<sup>5,52,53</sup> Only one  
study showed a correlation between self-reported symptoms of the patient's sleepiness  
195 and weight change<sup>49</sup>, while one study showed a correlation between BMI and RDI score.<sup>5</sup>  
Weight regain after both VBG and RYGB has been shown to be associated with a  
recurrence of OSA symptoms.<sup>47,53</sup>

These data suggest that although there is significant improvement of OSA after  
bariatric surgery, many patients may still need treatment of OSA based on their AHI.  
200 Since the absence of clinical symptoms does not necessarily correlate with normalization  
of AHI and/or severity of sleep apnea, surgeons should consider repeat PSG testing or a  
CPAP titration study after significant weight loss has occurred. There is currently no  
consensus regarding indications or timing for repeat PSG either in the general population  
or for those individuals that have undergone bariatric surgery. Consideration should be  
205 given to performing repeat PSG when evaluating patients who present after significant  
weight regain or reemergence of prior resolved conditions such as diabetes or  
hypertension, especially if reoperative surgery is being considered.

210 **Postoperative Monitoring and Care of Patients with OSA**  
**Undergoing Bariatric Surgery**

Data regarding the appropriate postoperative monitoring and care of patients with OSA undergoing bariatric surgery has increased greatly in the past decade, and is an evolving body of published experience. More recent reports contradict some older more stringent recommendations.

Published guidelines with more conservative and strict standards for postoperative monitoring include the American Society of Anesthesiology guidelines, which state bariatric patients should have continuous monitoring during and after surgical intervention, including continuous pulse oximetry and emergency airway equipment nearby. Continuous pulse oximetry (in a critical care or step-down unit or by a dedicated, appropriately trained professional observer in the patient's room) is felt to reduce the likelihood of complications among patients with OSA.<sup>55</sup> Intermittent pulse oximetry or continuous bedside oximetry without continuous observation is alleged, by these guidelines, to not provide the same level of patient safety.<sup>55</sup> Another report recommends continuous monitoring should be maintained for as long as patients remain at increased risk and for *at least* 3 hours beyond the standard observation time of their non-OSA counterparts.<sup>56</sup> That group also recommends that patients can be safely discharged from the monitored setting once oxygen saturation above 90% can be maintained on room air including during sleep, there is no hypoxemia/airway obstruction when the patient is left undisturbed, and patients no longer require parenteral narcotics. Such guidelines are clearly relevant for inpatient postoperative hospitalizations, but are not readily applicable to outpatient procedures such as LAGB, and they do not account for patients with undiagnosed sleep apnea.

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In contrast to these published recommendations, other reports in the literature have recently reviewed experiences where ICU type monitoring was not routinely used for patients with OSA. Jensen et al<sup>31</sup> reported an experience of over 1000 laparoscopic RYGB patients in which over 800 did not have clinically-proven OSA. A postoperative program of no routine use of CPAP, spirometry, and early ambulation, was associated with a very low incidence of pulmonary complications. Grover et al<sup>57</sup> reported that ICU monitoring was not used for their bariatric patients undergoing laparoscopic RYGB. Over 200 of 650 patients undergoing surgery had OSA, and that group had no increase in overall or pulmonary complications despite non-routine use of ICU in their postoperative care.

Postoperative monitoring of the bariatric surgical patient with OSA depends on a variety of factors including type of procedure, approach (laparoscopic vs. open), severity of sleep apnea, other patient comorbidities and individual facility capabilities. Minimum requirements include pulse oximetry or capnography in the post-anesthesia care unit and in the surgical ward. The majority of patients with OSA can be safely monitored in this fashion.<sup>57</sup> Select higher risk patients, including males, BMI >60, severe OSA, and age >50 may be monitored in an ICU setting at the discretion of the surgeon.<sup>58,59</sup>

Patients with diagnosed OSA who undergo bariatric surgery, and who routinely use CPAP or are prescribed to use it, should be prepared to use CPAP in the postoperative setting if ordered by the surgeon. It is often best if the patient brings their own CPAP mask, with or without their machine, with them to the hospital. This ensures the equipment fits the patient and is readily available for use in the postoperative period.

## Summary Recommendations

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Based on the evidence in the literature to date, the following guidelines regarding OSA in the bariatric surgery patient and its perioperative management are recommended:

1. OSA is highly prevalent in the bariatric patient population. The high prevalence demonstrated in some studies suggests that consideration be given to testing all patients, and especially those with any preoperative symptoms suggesting obstructive sleep apnea.
2. Untreated OSA is yet another comorbidity observed with high prevalence in the bariatric patient population that leads to increased mortality and increased medical disability from several cardiovascular diseases. These observations further emphasize the value of bariatric surgery as a potentially definitive treatment for OSA in severely obese patients.
3. Patients who have documented moderate to severe OSA should be strongly encouraged to accept treatment preoperatively with CPAP and to use it postoperatively until clinical evaluation demonstrates resolution of OSA.
4. These patients should also bring their CPAP machines, or at least their masks, with them at the time of surgery and use them following bariatric surgery at the discretion of the surgeon.
5. All commonly performed bariatric operations that have been assessed for impact on OSA have shown evidence for significant relief of subjective symptoms of

OSA and improvement of objective parameters of OSA that may not always correlate with amount of weight lost.

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6. Since bariatric surgery produces many improvements in the quality of life and other co-existing medical conditions for severely obese patients with OSA, it should be considered as the initial treatment of choice for OSA in this patient population as opposed to surgical procedures directed at the mandible or tissues of the palate.
  7. Routine pulse oximetry or capnography for postoperative monitoring of patients with OSA after bariatric surgery should be utilized, but the majority of these  
290 patients do not routinely require an ICU setting.
  8. No clear guidelines exist upon which to base recommendations for retesting for OSA following bariatric surgery. However, strong consideration should be given to retesting patients who present years after bariatric surgery with regain of weight, a history of previous OSA, and who are being reevaluated for appropriate  
295 medical and potential reoperative surgical therapy.

### **Peri-operative Management of Obstructive Sleep Apnea Position Statement and Standard of Care**

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These guidelines and recommendations are not intended to provide inflexible rules or requirements of practice and are not intended, nor should they be used, to state or establish a local, regional, or national legal standard of care. Ultimately, there are various

appropriate treatment modalities for each patient, and surgeons must use their judgment  
305 in selecting from among the different feasible treatment options.

The ASMBS cautions against the use of these guidelines and recommendations in  
litigation in which the clinical decisions of a physician are called into question. The  
ultimate judgment regarding appropriateness of any specific procedure or course of  
action must be made by the physician in light of all the circumstances presented. Thus,  
310 an approach that differs from these guidelines or recommendations, standing alone, does  
not necessarily imply that the approach was below the standard of care. To the contrary,  
a conscientious physician may responsibly adopt a course of action different from that set  
forth in the guidelines when, in the reasonable judgment of the physician, such course of  
action is indicated by the condition of the patient, limitations on available resources, or  
315 advances in knowledge or technology. All that should be expected is that the physician  
will follow a reasonable course of action based on current knowledge, available  
resources, and the needs of the patient in order to deliver effective and safe medical care.  
The sole purpose of these guidelines is to assist practitioners in achieving this objective.

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