# Preoperative Screening for Obstructive Sleep Apnea in Morbidly Obese Patients

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#### Health Consequences of Obesity

In the western world, obesity is considered a key health problem. It has been estimated that more than two thirds of North Americans are overweight or obese and almost 34% of adults and 15% to 20% of children and adolescents of the US population are obese.<sup>1</sup> Obesity has long been recognized as a predecessor of morbidity and premature mortality. It is frequently associated with a higher incidence of hypertension, lethal cardiac dysrhythmia, ischemic heart disease, and premature coronary death. Other related medical problems include hyperglycemia, diabetes mellitus, dyslipidemia, gall bladder disease, nonalcoholic fatty liver disease, stroke, degenerative joint disease, and obstructive sleep apnea (OSA).<sup>2,3</sup> In addition to a poor quality of life; obesity is associated with hormonal changes that can lead to an increased likelihood of malignant tumors. Uterine and breast tumors have been documented in obese females. Similarly, a higher incidence of renal and esophageal carcinoma is reported in obese males compared to the normal weight population.<sup>4</sup> Data showed that obesity was responsible for 365,000 preventable deaths in 2000.<sup>1</sup> Given the increasing risk with comorbidity, the standard definition of morbid obesity was modified to incorporate patients of body mass index  $(BMI) \ge 35 \text{ kg/m}^2$  with concomitant obesity-related disease.<sup>5</sup> One of the major health care expenditures in the coming 2 decades will be the treatment of metabolic disease associated with obesity.<sup>6</sup> With the increased life expectancy and

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evolution in medicine, an increasing number of morbidly obese patients are subjected daily to surgical procedures.

## What is OSA?

OSA is a common, potentially serious sleep condition characterized by repetitive episodes of apnea and hypopnea. Subjects experience frequent pauses of 10 seconds or more in breathing (apnea) during sleep despite the presence of respiratory effort. OSA is frequently accompanied by hypoventilation (reduction of airflow by >30%), oxygen desaturation, sympathetic arousal, and awakening.<sup>7</sup> The most common diagnostic features for OSA are excessive daytime sleepiness, loud snoring, and restless sleep observed by one's partner. Other reported symptoms may include fatigue, nonrefreshing sleep, hyperacidity secondary to gastroesophageal reflux, dry mouth upon awakening, impaired memory and decreased performance at work, and morning headache due to carbon dioxide (CO<sub>2</sub>) retention. In addition, attention deficit, irritability, anxiety, change of mood particularly depression, frequent nocturia, nighttime diaphoresis, unexplained weight gain, and decreased sexual drive are also symptoms of OSA.

OSA is usually associated with increasing incidence of other medical conditions. Lavie et al<sup>8</sup> reported a strong positive correlation between high blood pressure and severity of OSA which is independent of all other risk factors including obesity. Cardiac arrhythmias in the form of atrial fibrillation, nonsustained ventricular tachycardia, sinus brady-cardia, sinus arrest, complex ventricular ectopy, and second-degree atrioventricular block is more common in patients with sleep-disordered breathing.<sup>9,10</sup> It had been estimated that nocturnal cardiac dysrhythmia is 2- to 4-fold higher in these patients compared to the general population.<sup>9</sup> Many researchers report an association between OSA with cerebrovascular disease, metabolic syndrome, diabetes, and insulin resistance.<sup>11,12</sup> Respiratory complications of long-standing OSA may be manifested in the form of hypoxemia, hypercapnia, polycythemia, and cor pulmonale.<sup>11</sup> OSA patients may remain symptom free for years until they develop severe complications of the disease.

OSA in the morbidly obese patients should be differentiated from obesity hypoventilation syndrome (OHS). The syndrome includes obesity, daytime hypoventilation, and sleep-disordered breathing with no other obvious cause. It had been estimated that OHS does exists in 10% to 20% of OSA patients.<sup>13</sup> One study showed that OHS is present in 22% of morbidly obese undergoing bariatric surgery.<sup>14</sup> Awake hyper-capnia ( $P_{aCO_2} \ge 45 \text{ mm Hg}$ ) is the main feature of the OHS, however, it is associated with low oxygen saturation (SPO<sub>2</sub> < 90%) and elevated serum bicarbonate (HCO<sub>3</sub>) level.<sup>13</sup>

# Diagnosis of OSA

Polysomnography (PSG) is considered the gold standard diagnosis of OSA. The Apnea Hyponea Index (AHI), which is the sum of apnea and/or hypopnea episodes per hour, is determined with AHI > 5confirming the diagnosis of OSA.<sup>15</sup> There is a general agreement between clinicians that AHI is used to classify the severity of the syndrome. Classically, an AHI of 5 to 15 indicates mild OSA, AHI 15 to 30 specifies moderate OSA, and AHI > 30 points to severe OSA. However, the Canadian Thoracic Society diagnoses OSA if the patient has an AHI > 5 on PSG, and either (1) excessive daytime sleepiness that is not explained or (2) at least 2 other OSA symptoms such as choking and/or gasping during sleep, recurrent awakenings from sleep, unrefreshing sleep, daytime fatigue, or impaired concentration.<sup>15</sup> OSA is diagnosed by the United States Medicare guidelines with AHI > 15, or an AHI of 5 with 2 comorbidities that includes cardiovascular disease, stroke, metabolic syndrome, obesity, and gastroesophageal reflux. Other factors that may be used to determine the severity of OSA include the duration and the rate of oxygen desaturation, level of arousal threshold, and adequacy of ventilation recovery.<sup>15</sup>

The pathophysiology of OSA involves upper airway obstruction secondary to a bulky tongue and excessive airway soft tissue that increases the external pressure surrounding the pharynx. Partial or complete cessation of breathing occurs when negative pressure of inspiratory muscles exceeds the dilatation capacity of the upper airway muscles during sleep.<sup>15</sup> An alternative mechanism has been described in obese patients where lung volume decreases due to accumulation of visceral fat which leads to increased pharyngeal wall collapsibility.<sup>16</sup> As a result of the frequent sleep interruption at night, increased sympathetic vasoconstrictor tone, and intermittent nocturnal hypoxia, patients may develop serious health conditions which can be the initial presentation of OSA.

# Prevalence of OSA

The OSA syndrome usually affects the middle age and elderly population of both sexes. In the general population, it has been estimated that moderate to severe OSA is present in 11.4% of men and 4.7% of women.<sup>17</sup> The prevalence may be in the range of 1 in 4 males and 1 in 10 females.<sup>11</sup> It is more common in females after menopause without hormone replacement therapy because of the protective effect of progesterone.<sup>17</sup> The prevalence of OSA in obese patients is higher than in the general population, ranging between 70% and 95%.<sup>4,18–20</sup> Lopez and colleagues in a retrospective study reported an overall prevalence of OSA of 78% in bariatric patients. They further subdivided the patients according to the BMI and showed a 71.4% prevalence of

OSA in patients with BMI of 35 to 39.9 kg/m<sup>2</sup>, 74% with BMI of 40 to 49.9 kg/m<sup>2</sup>, 77% in patients with BMI of 50 to 59.9 kg/m<sup>2</sup>, and 95% in patient with BMI >  $60 \text{ kg/m}^{2.18}$  Rao et al<sup>19</sup> reported that the incidence of OSA in Asian patients undergoing laparoscopic gastric bypass surgery was 46% for patients with moderate OSA and 33% for patients with severe OSA with an overall incidence of 73% in the studied population. Another cross-sectional study of 170 morbidly obese patients screened for OSA before bariatric surgery showed a prevalence of 77%. However, the authors failed to report any correlation between BMI and OSA severity and they suggested that all morbidly obese patients should be considered at risk for the syndrome.<sup>20</sup> Bein and Scholz<sup>4</sup> reported that a BMI increase of  $4 \text{ kg/m}^2$  is associated with a 4-fold increased risk of sleep-associated breathing disorder and that almost 70% of OSA patients are obese. Dempsey et  $al^{21}$  showed that a BMI > 28 kg/m<sup>2</sup> increases the possibility of moderate to severe sleep apnea by 5-fold. Another study by Sharkey et al<sup>22</sup> retrospectively studied 296 women who had PSG in preparation for bariatric surgery and reported a prevalence of OSA of 86%. A recent study of 342 patients undergoing bariatric surgery reported an OSA prevalence of 77.2% irrespective of the severity of the symptoms, age, sex, BMI, and menopausal status.<sup>23</sup>

## OSA is a Perioperative Risk Factor for Morbidly Obese Patients

Long-standing OSA is considered an independent risk factor for postoperative complications. Flum et al<sup>24</sup> reported that the 30-day mortality rate of morbidly obese patients undergoing a Roux-en-Y gastric bypass or laparoscopic adjustable gastric banding was significantly higher in patients with preexisting OSA. Kaw et al<sup>25</sup> documented that OSA is a major contributor to poor pulmonary outcomes in the obese patient and that elevated head position increases the stability of the upper airway, hence decreasing the incidence of postoperative complications. Ballantyne et al<sup>26</sup> studied hospital record of 311 patients who underwent Roux-en-Y gastric bypass. The studies showed that OSA is a predictor of prolonged hospital stay. In addition, a cost analysis study showed that OSA contributes significantly to increased hospital cost after gastric bypass surgery.<sup>27</sup>

The increased incidence of postoperative respiratory problems in patients with OSA could be explained by the depressive effects of narcotics as well as other anesthetic drugs on the function of the upper airway muscles. Anesthesia may also attenuate the ventilatory response to airway obstruction and abolish normal response to hypoxia and hypercapnia.<sup>28</sup> The American Society of Anesthesiologists' (ASA) published practice guidelines for the management of OSA patients is accompanied by a suggested scoring system that allows the caregiver to

determine the perioperative risk.<sup>7</sup> The scoring system is based upon the measurement of (1) severity of OSA based on formal sleep study or screening questionnaires (0 to 3 points); (2) invasiveness of surgery and anesthesia (0 to 3 points); and (3) the need for postoperative opioid, either low or high dose, administered through oral, parenteral, or neuraxial route (0 to 3 points). The overall score is between 0 and 6 by sum of the score item 1 plus the higher score of either items 2 or 3. Patients who scored  $\geq 5$  points were considered at a significantly higher perioperative risk from OSA.

### Methods of Screening for OSA

The preoperative diagnosis of OSA is crucial before bariatric surgery as the syndrome has a deleterious effect on many of the organ systems especially the cardiorespiratory system. The diagnosis of OSA and its consequences can have an impact on the anesthetic management, level of postoperative monitoring, use of postoperative continuous positive airway pressure (CPAP)/bilevel positive airway pressure, and suitability for ambulatory care. As a part of the routine preoperative preparation before bariatric surgery, many institutions perform a formal sleep study (PSG). However, PSG may not always be available because of the limited number of sleep laboratories and long wait-time. In addition, PSG is associated with high cost that is an added burden for many health care systems. Alternatively, portable home-based monitoring devices and validated screening questionnaires could be used to diagnose and determine the severity of OSA.

#### **Screening Questionnaires**

Historically, screening questionnaires were proposed to forecast the patients who needed a formal sleep study. There are numerous questionnaires developed to diagnose OSA. The most commonly used questionnaires are: Wisconsin sleep questionnaire, Apnea score, Haraldsson questionnaire, Sleep Apnea scale of the Sleep Disorders Questionnaire, Epworth sleepiness scale, 4 variables screening tool, Berlin questionnaire.<sup>29,30</sup> Perioperative sleep apnea prediction score (P-SAP) and Sleep apnea clinical score have also been developed.<sup>31,32</sup> For preoperative diagnosis tool of OSA only 4 questionnaires, namely the STOP-Bang questionnaire, Berlin questionnaire, American Society of Anesthesiologists' checklist, and P-SAP score, have been validated in surgical patients.

The Berlin Questionnaire is one of the most frequently used questionnaires. It was developed in 1996 in Berlin and consists of 9 items in 3 categories related to snoring, witnessed apneic episodes (category 1), daytime fatigue and/or sleepiness (category 2), falling asleep while driving, and high blood pressure and BMI >  $35 \text{ kg/m}^2$  (category 3). If patients are positive in at least 2 symptom categories, they are considered at high risk for sleep apnea. If patients deny having persistent symptoms, or have only 1 positive symptom category, they are considered at a lower risk for OSA.<sup>33</sup> The validation of the Berlin Questionnaire against overnight laboratory PSG in 177 surgical patients showed a sensitivity of 68.9% and a specificity of 56.4% at AHI > 5. In patients with severe OSA (AHI > 30) the Berlin Questionnaire has 72.2% sensitivity and 46.4% specificity.<sup>33</sup>

In 2006, the ASA published its checklist for OSA diagnosis. The checklist is divided into 3 different categories with 12 questions, with 2 more items related to pediatric patients. The first category is related to the predisposing physical characteristics. The second category is related to the history of airway obstruction, whereas daytime somnolence items make up the third category. Patients are considered at high risk of OSA if  $\geq$  2 categories are scored positive.<sup>7</sup> The validation of the ASA checklist in surgical patients showed similar sensitivity and specificity to the Berlin Questionnaire as well as STOP questionnaire for mild, moderate, and severe OSA.<sup>33</sup>

Recently, the P-SAP was published.<sup>31</sup> It was derived from 43,576 patients of whom 3884 patients had an OSA diagnosis. The score consists of: (1) 3 demographic variable that include age (> 43 y), sex (male), and obesity; (2) 3 history variables that include history of snoring, type 2 diabetes mellitus, and hypertension; and (3) 3 airway measures that include large neck circumference, Mallampati class 3 or 4, and short thyromental distance. One point is assigned for each of the 9 variables with a total score between 0 and 9. A validation of the P-SAP score in patients with confirmed OSA diagnosis by formal sleep study was performed. It showed that for AHI  $\geq$  5, a score of  $\geq$  2 showed 94% sensitivity and 25% specificity, whereas a score of  $\geq$  6 showed 21% sensitivity and 91% specificity. Furthermore, For AHI  $\geq$  30, a score of  $\geq$  2 showed 98% sensitivity and 12% specificity, whereas a score of  $\geq$  6 showed 32% sensitivity and 85% specificity.<sup>31</sup>

The most frequently used test for preoperative screening of surgical patients for OSA is the STOP-Bang questionnaire (Table 1).<sup>34</sup> The STOP-Bang questionnaire has 8 yes/no items. Each yes answer will score 1 point, whereas no answers will count as 0 with a total score of between 0 and 8. The patient is considered at risk for OSA if the patient answers positively 3 questions for the STOP-Bang model.<sup>34</sup> The questionnaire was first published in 2008; it is self-administered, simple, and easy to use in a busy clinical setting.

The STOP-Bang model showed a high sensitivity (84% to 100%) and negative predictive value (61% to 100%) especially in patients with moderate to severe OSA. More recently, the association between the probability of having severe OSA and the score on STOP-Bang has been

 Table 1.
 STOP-Bang Questionnaire

- S = Snoring. Do you snore loudly (louder than talking or loud enough to be heard through closed doors)?
- T = Tiredness. Do you often feel tired, fatigued, or sleepy during daytime?
- O = Observed apnea. Has anyone observed you stop breathing during your sleep?
- P = Pressure. Do you or are you being treated for high blood pressure?
- $B = BMI > 35 \text{ kg/m}^2$
- A = Age > 50 y
- N = Neck circumference >40 cm
- G = Male sex (gender)

High risk of obstructive sleep apnea is considered if answering yes to  $\geq 3$  for STOP-Bang questionnaire.

Adapted from Chung et al.<sup>34</sup>

demonstrated. A study conducted over a period of 4 years by Chung et al<sup>35</sup> reported that a high STOP-Bang score indicates high probability for moderate and/or severe OSA. The results from 746 OSA patients showed that a score of <3 would allow the caregiver confidently to rule out OSA. In contrast, a score of  $\geq 5$  showed a high specificity to detect moderate and severe OSA. The study showed that the higher the score, the higher the predicative value as the odds ratio (OR) of moderate to severe OSA increases with the increase in the score (Table 2).<sup>35</sup> For a STOP-Bang score of 5, OR for moderate to severe OSA was 4.8 and for severe OSA was 10.4. For a STOP-Bang of 6, the OR for moderate to severe OSA was 6.3, whereas it was 11.6 for severe OSA. Moreover, a STOP-Bang score of 7 or 8 has an OR of 6.9 for moderate to severe OSA and 14.9 for severe OSA.35 This will help to classify patients with unrecognized OSA. Similarly, Farney et al<sup>36</sup> suggested that the STOP-Bang model can be used to categorize patients into no OSA, mild, moderate, and severe OSA as defined by AHI. In a recent work by Chung and colleagues, the addition of serum  $HCO_3$  level to a STOP-Bang score of  $\geq 3$  considerably enhanced the specificity of moderate to severe OSA prediction. A STOP-Bang score of  $\geq 3$  together with serum  $HCO_3 \ge 28 \text{ mmol/L}$  showed 81.7% specificity for patients with AHI > 15and 79.7% specificity for patients with  $AHI > 30.3^{\circ}$ 

#### PSG

PSG, also called type 1 PSG, is considered the gold standard for the evaluation of sleep-disordered breathing including OSA. It requires the subject to spend 1 full night at the sleep laboratory. PSG includes electroencephalogram, electrocardiogram (ECG), and electrooculography—a method used for measuring retina resting potential, oxygen saturation, and chest and abdominal wall strain gauges. The airflow pressure transducer is applied to measure respiratory airflow and degree of snoring. Furthermore, electromyography of chin and anterior tibialis is indispensable to detect arousals from sleep and to assess

- STOP-Bang Score	Odds Ratio for OSA at Different AHI Cutoffs		
	Mild OSA (AHI > 5)	Moderate/Severe OSA (AHI > 15)	Severe OSA (AHI > 30)
Score 3 vs. score 0-2	3.01	2.59	3.56
Score 4 vs. score 0-2	3.15	3.33	5.33
Score 5 vs. score 0-2	3.98	4.75	10.39
Score 6 vs. score 0-2	4.52	6.29	11.55
Score 7 and 8 vs. score 0-2	7.04	6.88	14.86

 Table 2.
 Odds Ratio of Different STOP-Bang Scores

AHI indicates Apnea Hyponea Index; OSA, obstructive sleep apnea. Adapted from Chung et al. $^{35}$ 

periodic limb movement that occurs with OSA.<sup>38</sup> The presence of a sleep technologist during the study is highly important to observe patient compliance and technical adequacy to have an accurate result. Nevertheless, PSG is not 100% accurate as the technique has its own limitations. Many patients may have different sleep quality and body position in the laboratory versus home. In addition, there is variability in AHI measures secondary to the differences in the available scoring method used by each laboratory. Similarly, human factors play a role as shown by the various result interpretations by different well-trained technicians using the same scoring method.<sup>38</sup>

Laboratory testing is a lengthy procedure and requires huge resources. Therefore, a long waiting list is present in most sleep laboratories. A study showed that patients could wait for 11.6 months to initiate medical treatment (CPAP), and 16.2 months to initiate surgical therapy for OSA.<sup>39</sup> The availability of sleep laboratories appeared to be the major restriction for the management of these patients.

To improve sleep evaluation in bariatric patients, limit health care spending and decrease the waiting time for surgery, a number of high-performance, simple, and portable sleep monitoring devices have been developed. Portable devices can be divided into comprehensive portable PSG (type 2), modified portable sleep apnea testing that consists of 4 channels, which include respiratory movement, airflow, ECG or heart rate, and oxygen saturation (type 3), and single-parameter or 2-parameter devices (type 4).<sup>40</sup> The portable devices can be used at the hospital bedside or at the patient's home. Chung et al<sup>41</sup> validated a type 2 portable sleep device (Embletta  $\times 100$ ) as an alternative to standard PSG; however, they reported the need for physical presence of a sleep technologist to accurately connect the device and read the score to get reliable results. Further work has been carried out to validate the accuracy of type 3 monitoring devices against conventional PSG.40 Collop<sup>42</sup> reported that unattended home-based OSA diagnostic testing showed feasibility and equivalent results compared to those of standard

PSG. These devices showed a reasonable sensitivity and specificity for the diagnosis of symptomatic patients suspected of severe OSA. However, poor accuracy was observed in patients with mild to moderate OSA, and a standard PSG may be necessary to completely rule out the condition.<sup>41</sup>

High-resolution nocturnal oximetry (type 4 monitor) has shown to be useful in helping the preoperative diagnosis of OSA patients.<sup>43</sup> The device has the ability to detect the variability of oxygen saturation caused by apnea and hypopnea during sleep through calculation of the Oxygen Desaturation Index (ODI). The index is defined as the number of hourly episodes of desaturation of 4% that last for 10 seconds of the previous 120 seconds. It has been reported that ODI is an accurate method for predicting AHI at cutoffs 5, 15, and 30 with sensitivity of 87, 84, and 93.3%, respectively, and ODI > 10 can detect patients with moderate and severe OSA with 93% sensitivity and 75% specificity.43 Because it is not able to monitor respiratory effort or nasal flow, highresolution oximetry is not able to distinguish central apnea from obstructive apnea.<sup>43</sup> The sensitivity of oximetry alone versus portable polysmonography (type 3 monitor) was studied as a screening technique for OSA in bariatric surgical patients.<sup>43</sup> The study reported that the use of nocturnal oximetry alone with 3% desaturation index was able to detect all patients with severe OSA. The authors suggest that bariatric patients with oxygen desaturation index of 10 to 30 should be referred to formal PSG for accurate diagnosis.<sup>44</sup>

The easily available high-resolution oximetry may be an alternate pathway to the screening of bariatric patients for OSA, streamlining the whole process. These surrogate investigations may help to identify the bariatric patients at higher risk of OSA-related complications, so that preoperative CPAP therapy may be considered and appropriate perioperative precautionary measures may be undertaken.

## Current Practice of Preoperative Screening of OSA Before Bariatric Surgery

There is a marked difference in opinion between experts regarding the screening of morbidly obese patients before weight reduction surgeries. Lopez et al<sup>18</sup> recommended that morbidly obese patients should be referred to sleep laboratory to have a formal sleep study before surgery. Similarly, an earlier study supported the routine use of PSG as a preoperative screening tool for OSA before bariatric surgery regardless of BMI based on the increasing incidence of surgical complications including anastomotic leaks if OSA was not diagnosed and treated preoperatively.<sup>20</sup> Furthermore, 2 other studies by Rao et al<sup>19</sup> and Sareli et al<sup>23</sup> recommended the routine use of PSG for all surgical bariatric patients. Sharkey et al<sup>22</sup> stated that the predictive models based on the clinical characteristics are not effective for predicting OSA in women planning bariatric surgery and suggested that these patients should be evaluated with PSG. More selectively, Catheline et al<sup>45</sup> recommended a preoperative evaluation by PSG for morbidly obese patients presenting for bariatric surgery with a history of cardiac or pulmonary diseases and/or abnormal preoperative ECG. Similarly, Schumann et al<sup>46</sup> recommended PSG in the presence of independent predictors of OSA that include sex, neck circumference, and waist-to-hip ratio.

In contrast, Kurrek and colleagues in a retrospective study of patients undergoing outpatient laparoscopic gastric banding found that the 30-day anesthesia-related morbidity was < 0.5%. In addition, the authors stated that oxygen desaturation in the recovery period is clinically insignificant and the benefits of preoperative screening for OSA remain undetermined.<sup>47</sup> Similarly, Loadsman<sup>48</sup> stated that we should stop trying to identify patients with OSA and use only personal judgment to determine the appropriate perioperative management. The advantage of preoperative PSG in all bariatric patients is not clear. A study by Weingarten et al<sup>49</sup> showed a negative correlation between the severity of OSA and the rate of perioperative complications in bariatric patients. However, this study was carried out in an academic tertiary care center where patients received perioperative positive airway pressure therapy and were monitored postoperatively with oximetry. Therefore, the results cannot be extrapolated to bariatric patients with unrecognized or untreated OSA.

As screening questionnaires have their own limitations, many experts have suggested that a combination of clinical tools could lead to a better diagnosis of OSA. In the authors' opinion, OSA diagnosis may be achieved by combining the STOP-Bang questionnaire with confirmatory testing with a portable PSG monitor. The morbidly obese patients who have an ODI score of >10 should be referred to formal sleep studies. Similarly, symptomatic morbidly obese patients with significant comobidities such as heart failure, pulmonary hypertension, and hypercapnia should also be directed to the sleep laboratory. Patients with OHS are distinct from mere morbid obesity and OSA, as indicated by the severe upper airway obstruction, restrictive chest physiology, blunted central respiratory drive, and pulmonary hypertension. These patients should have PSG preoperatively in order to receive treatment with positive airway pressure therapy before surgery.

In conclusion, OSA is highly prevalent in morbidly obese patients undergoing bariatric surgery. Although PSG is the gold standard for the diagnosis of OSA, it is costly and time consuming. A simple screening with the STOP-Bang questionnaire and the appropriate confirmatory testing with portable or laboratory PSG may be a better alternative. High-resolution nocturnal oximetry may be a simple and high-performance alternative method to stratify bariatric patients for OSA before surgery. The authors have no conflicts of interest to disclose.

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