10 Sleep-Disordered Breathing and Bariatric Surgery

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Introduction

Obesity has become increasingly prevalent globally. According to WHO research, the number of obese individuals has *doubled* since 1980. It has been estimated that roughly 1/3 of North Americans are categorized as obese, defined as having a BMI > 30 [1]. Worldwide, it is estimated that roughly 10% of the population meets this definition [2, 3]. Obesity is a chronic disease with systemic consequences. The respiratory system is no exception.

There are variety of mechanisms by which obesity can impact respiratory function. Furthermore, there is emerging evidence that the prognosis and natural history of some disease processes, including asthma, COPD, and several kinds of cancer, are influenced by obesity [2]. It increases the risk for pulmonary embolism as well as for aspiration pneumonia. Obesity, in the absence of other conditions, can cause respiratory complications in the perioperative setting. Bag-mask ventilation, endotracheal intubation, and operative oxygenation can all be compromised by excess adiposity. The focus of this chapter will be on sleep-related complications in bariatric surgery. The reader should be aware that excess adipose tissue can have a variety of adverse effects beyond its deleterious effects on sleep.

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Pulmonary Complications of Obesity

Several mechanical and physiologic alterations are present in the obese. Excess weight increases oxygen consumption and carbon dioxide production and mechanically disadvantages the respiratory system [4]. These changes can be slowly progressive and not perceived by the patient. Symptoms, when present, typically occur with exertion. In extreme cases, obesity heightens ventilatory demand, increases the work of breathing, mechanically disadvantages the respiratory muscles, and decreases the compliance (stiffens) the respiratory system [2].

Subdivisions of Lung Volumes and Pulmonary Function Tests

A grasp of pulmonary function testing, including spirometry, plethysmography, the determination of lung volumes, and the measurement of diffusion capacity, is essential to understanding the effects of obesity on respiration. Figure 10.1 demonstrates the subdivision of lung volumes. The abbreviations used for the various lung volumes and spirometric parameters are shown in Table 10.1.

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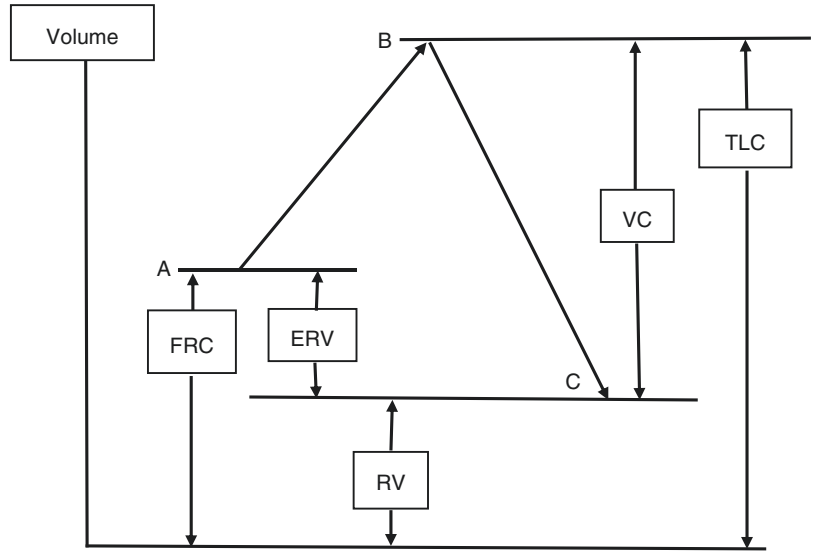

Fig. 10.1 Subdivision of lung volumes

Table 10.1 Common spirometric and subdivision of lung volume terms

FEV_1	Forced expiratory volume in 1 second
FVC	Forced vital capacity
FEV_1/FVC	Ratio of FEV_1 and FVC
TV	Tidal volume
FRC	Functional residual capacity
RV	Residual volume
ERV	Expiratory reserve volume
VC	Vital capacity
TLC	Total lung capacity

Functional residual capacity (FRC) (A) is the lung volume at the end of a passive expiration and is the point where the tendency of the lung to collapse is balanced by the equal and opposite recoil of the chest wall. If one makes a maximal inspiratory effort, the inspiratory limit is the total lung capacity (TLC) (B) or maximum lung volume. If one then makes a maximal expiratory effort, the expired volume is the vital capacity (VC), and the remaining air in the lungs is the residual volume (RV) (C). The volume difference between the FRC and the RV is called the expiratory reserve volume (ERV).

Obese patients will commonly have preserved total lung capacity [2, 4–6]. However, there are some changes that are considered typical for the obese patient. In obese patients, the increased weight compresses the chest wall reducing the lung volume where the recoil of the chest wall is balanced by the tendency of the lung to collapse reducing both FRC and ERV but RV remains relatively preserved. ERV decreases exponentially with increasing BMI (Fig. 10.2) [5, 7, 8]. The ERV may be reduced over 40% in those with a BMI of 30 kg/m^2–55 kg/m^2. In patients with a BMI $\geq$ 60 kg/m^2, the ERV can be reduced by 80%. Figure 10.3 demonstrates the effects of severe obesity on lung volumes.

Total lung capacity (TLC) is only minimally decreased, even in cases of morbid obesity [9–11] (Fig. 10.3). Any reductions in TLC are likely on the basis of increasing diaphragmatic impedance and intercostal adiposity. The chest wall, despite the increased load, is only minimally affected. Tidal volumes are generally (but not universally) reduced. The morbidly obese (BMI > 40 kg/m^2) may have an increased respiratory rate, as well as an increased minute ventilation [12].

Spirometry is the most frequently used test for assessing pulmonary function and measures vital capacity and expiratory flow rates (Fig. 10.4). The most commonly used flow rate is the FEV_1. Reductions in FEV_1 with relative preservation of FVC are seen in obstructive lung diseases including asthma and COPD. In restrictive diseases such as interstitial lung disease and chest wall disorders including morbid obesity, both FEV_1 and FVC are reduced in a similar proportion. There is an inverse relationship between weight and FEV_1 and FVC. These changes are particularly apparent

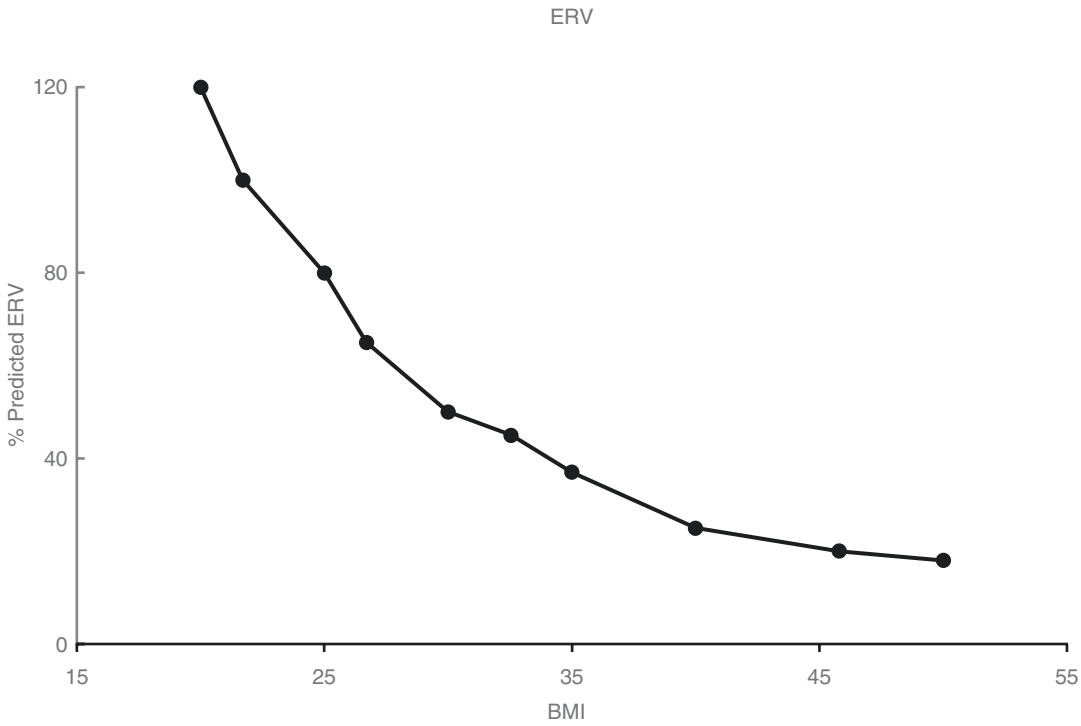

Fig. 10.2 Predicted ERV decreases exponentially with increasing BMI

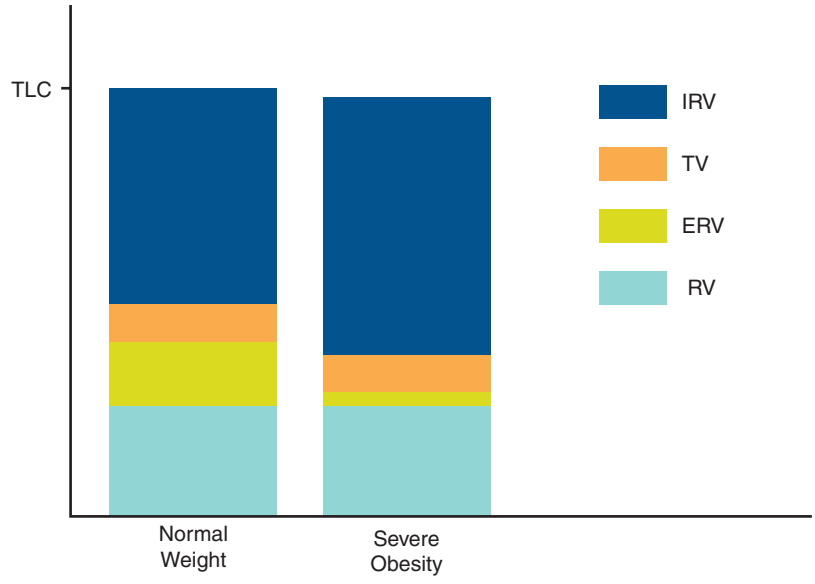

Fig. 10.3 The effect of severe obesity on lung volumes. The reduction in FRC is due to a decrease in ERV. The TLC is minimally affected

once the BMI exceeds 40 kg/m^2 [13]. A cross-sectional study of 1674 adults demonstrated that a 1 cm increase in waist circumference resulted in 13 ml/second and 11 ml average reductions in FEV_1 and FVC, respectively [6].

Diffusion capacity is a measure of the gas exchange ability of the lungs and is reduced in diseases of lung parenchyma such as emphysema and interstitial lung diseases. It is generally pre-

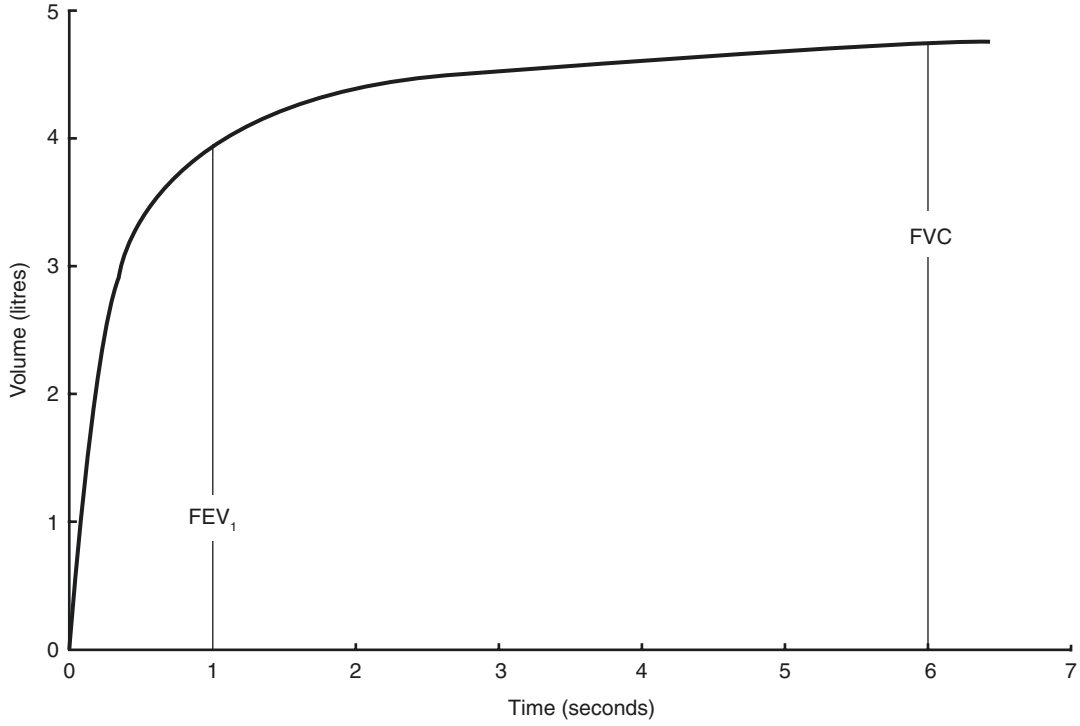

Fig. 10.4 The expiratory spirogram. The FEV1 is the volume of air forcefully expired in 1 second. The FVC is the total volume of air that can forcefully be expired. Though decreases in both are is seen in obesity, changes are most apparent when BMI exceeds 40 kg/m^2

served in obese patients in the absence of underlying lung disease.

Weight loss after bariatric surgery is the most effective intervention to improve pulmonary function. It is associated primarily with an increase in ERV, but improvements may also be seen in RV, FRC, and TLC [14]. However, it should be noted that pulmonary function improvements are smaller than expected when compared to never-obese patients of similar height and ethnicity [13].

Distribution of Obesity

In obese patients, the respiratory system is also affected by adipose tissue distribution. Fat distributed around the lower thorax and upper abdomen, both in subcutaneous tissue and around the viscera, impedes chest wall motion. This is referred to as central obesity. Peripheral obesity, where fat distribution is concentrated on the hips and extremities, has less of an effect on chest wall mechanics. An increased waist-to-hip ratio (WHR), a surrogate for central obesity, predicts this respiratory impairment better than BMI [2, 6]. Other markers of central obesity include waist circumference, abdominal height, and subscapular skinfold thickness [4]. Weight gain affects respiratory function more in men since they have a greater propensity for central obesity.

Physiologic Changes and Gas Exchange in Obesity

With increasing obesity, the ERV is reduced, and the patient breathes at a lower absolute lung volume. Breathing at lower lung volumes may cause airway closure. In turn, atelectasis may develop in the dependent lung zones. Total respiratory compliance may be reduced both by chest wall restriction and atelectasis [7]. The relative contributions of each are variable and likely patient specific [15–19]. These processes are exacerbated by abdominal pressure on the

diaphragm in the supine position, a concern during abdominal surgery. In extreme obesity, tidal volumes (TV) are reduced with a compensatory increase in the respiratory rate that is particularly evident during exercise [4, 20].

Even in the absence of lung disease, gas exchange may be compromised in the morbidly obese. Airway closure and atelectasis will reduce ventilation to the dependent lung zones where perfusion is greatest. The resulting ventilation/perfusion mismatch may cause hypoxemia.

Effect of Obesity on Sleep

One of the major consequences of obesity on the respiratory system, sleep-disordered breathing (SDB), is often unnoticed and undiagnosed [21, 22]. SDB encompasses a variety of conditions, including upper airways resistance syndrome (UARS), obstructive sleep apnea (OSA), central sleep apnea (CSA), Cheyne-Stokes respiration, and obesity hypoventilation syndrome (OHS). These conditions are associated with a variety of adverse clinical outcomes, including poor neurocognitive performance, psychiatric disturbances, increased risk of motor vehicle accidents, and cardiovascular morbidities and hypertension, stroke, and coronary artery disease. SDB also represents a significant risk factor for perioperative complications, including cardiorespiratory failure and prolonged intubation. These conditions are associated with significant socioeconomic burden. Among these conditions, OSA is the most clinically relevant to the bariatric surgeon. The other disorders of breathing are briefly reviewed below.

Central Sleep Apnea and Cheyne-Stokes Respiration

CSA is characterized by the cessation of airflow and of ventilatory effort. The condition can be idiopathic or secondary to another process (i.e., Cheyne-Stokes respiration).

Cheyne-Stokes respiration is a cyclical breathing pattern characterized by apnea followed by rapid frequency crescendo-decrescendo tidal volumes terminating with another apnea. Cheyne-Stokes respiration is commonly associated with congestive heart failure, neurologic disease, and sedative medications. It is unrelated to obesity.

Obesity Hypoventilation Syndrome

OHS is a severe form of sleep-disordered breathing. It is defined by the presence of chronic daytime hypercarbia ($PaC0_2 > 45$ mmHg) not caused by other conditions such as severe lung disease, neurological disease, or sedative or narcotic medication, in patients with obesity (BMI > 30 kg/m^2). It is often referred to as obesity-related respiratory insufficiency in the literature. It often occurs in association with OSA; 80% of those with OHS have OSA. It is estimated that 10–20% of those with OSA have OHS, with higher prevalence among the morbidly obese [23]. The pathogenesis is incompletely understood. Several different proposed mechanisms, including mechanical impedance due to excessive adipose tissue, an impaired central response to hypercarbia and hypoxemia, as well as neurohormonal disturbances, are thought to play a role [23]. It has a similar clinical presentation to OSA but is often more severe. It is associated with cognitive deficits, pulmonary hypertension and consequent right heart failure, and endothelial dysfunction with resultant adverse cardiovascular effects. It is also a significant risk factor for perioperative complications. Bariatric surgery may be effective treating this condition [24]. A small study of 31 patients who underwent bariatric surgery demonstrated significant improvements in $Pa0_2$ and $PaC0_2$ 1 year after surgery. However, in 12 of these patients, both $Pa0_2$ and $PaC0_2$ had worsened 5 years later in the absence of weight gain [25]. This indicates that SDB, including OSA, can recur despite successful surgery. Patients with OHS have an increased risk of perioperative death due to higher rates of postoperative respiratory failure and venous thromboembolism [26]. All patients with OHS should undergo polysomnography prior to surgery. OHS should be optimized medically prior to surgery with continuous positive airway pressure (CPAP).

Obstructive Sleep Apnea

OSA is characterized by cyclical cessation or reduction of airflow due to upper airway obstruction that occurs during sleep [9]. Obesity is its major risk factor. For every 10 kg increase in body weight, the risk of OSA increases twofold. An increase in BMI of 6 kg/m^2, or an increase in abdominal/hip girth of 13–15 cm, is associated with a fourfold increase in the risk OSA [27]. Other risk factors include craniofacial abnormalities, acromegaly, hypothyroidism, and increased neck circumference [28]. When defined by an apnea-hypopnea index (AHI) of ≥5, its prevalence ranges from 15 to 30% in males and 5 to 15% in females in the North American population [27]. The vast majority of patients are both undiagnosed and untreated [29].

Pathophysiology

It is a common misconception that the pathogenesis of OSA is simply due to fat and soft tissue directly obstructing the airway. There are multiple mechanisms postulated to contribute to upper airway obstruction. Anatomical factors, including enlarged soft tissue structures such as the tongue, tonsils, soft palate, and uvula surrounding the airway, can reduce airway patency. In the recumbent position, diaphragm excursion is reduced in the obese resulting in decreased intrathoracic pressure and lung volumes. This reduces the "tug" on the trachea applied indirectly through traction from the mediastinal structures [9]. Thickening the walls of the lateral pharyngeal walls and narrowing the airway also play a role [9]. Airway edema due to cephalad displacement of fluid from the lower extremities occurs in patients with OSA while recumbent. Neurohormonal influences on the thalamus and peripheral inflammation may also contribute [9].

Even in extreme obesity, the airway itself is rarely compromised during wakefulness. This phenomenon, known as the "wakeful stimuli," highlights a key aspect of OSA's pathogenesis [9]. In the obese, nocturnal pharyngeal collapsibility directly contributes to OSA pathogenesis. More than 20 skeletal muscles, known as pharyngeal dilation muscles, ensure airway patency in humans. When an individual falls asleep, the activity of these pharyngeal dilator muscles declines, as has been demonstrated by electromyography (EMG). The decreased activity is proportionally greater in the pharyngeal muscles than the reductions in other respiratory muscles, including the diaphragm [30]. As sleep deepens, pharyngeal muscle activity progressively lessens, making the upper airway more compliant and vulnerable to collapse. When inspiration reduces intraluminal airway pressure below the tissue pressure (applied by pharyngeal muscles, submucosal fat, and edema), the compliant pharyngeal tube will obstruct. This effect is exaggerated when the patient enters rapid eye movement (REM) sleep, a state where the accessory respiratory and pharyngeal muscles are effectively paralyzed [9].

An apneic event occurs when the upper airway obstructs, resulting in hypoventilation. The resultant hypercarbia and hypoxemia cause an arousal, associated with an adrenergic surge that restores upper airway patency by lessening (or fragmenting) sleep with an associated increase in pharyngeal muscle tone. Catecholamines and other hormones are released. The cycle occurs repetitively with subsequent sleep fragmentation that is associated with daytime symptoms including (but are not limited to) excessive daytime sleepiness, unrefreshing sleep, and snoring [29, 31, 32]. Neurocognitive complaints, including memory impairment, decreased occupational performance, depression, anxiety, and decreased sexual drive, are commonly described [29]. The large negative intrapleural pressures generated during episodes of airway obstruction may result in gastroesophageal reflux [32]. Biochemical disturbances can become apparent. Hypoventilation and consequent hypercarbia may cause respiratory acidosis. The kidneys respond to the respiratory acidosis by increasing retention of bicarbonate ($HC0_3$). The result is a chronic metabolic alkalosis. Chronic hypoxemia stimulates renal production of erythropoietin, increasing bone marrow production of

erythrocytes to increase oxygen-carrying capacity. This may manifest as (secondary) polycythemia, which may be associated with increased risk of venous thromboembolism [33]. Severe OSA is associated with insulin resistance and impaired glucose tolerance, conditions that are harbingers for diabetes. Additional hormonal effects include elevated serum leptin, which is associated with weight and satiety regulation, as well as respiratory control [9, 34]. OSA is associated with an increased risk of workplace accidents and motor vehicle collisions [35, 36]. Special attention should be paid to patients in high-risk occupations, particularly those in transportation or heavy industry, where accidents due to excessive sleepiness can have severe consequences both for the patient and public.

OSA has been linked with a variety of cardiovascular risk factors and endothelial dysfunction. It is associated with increased risks of congestive heart failure and cerebrovascular disease [37]. In severe cases, patients can develop cor pulmonale. There is a strong correlation between the severity of OSA and hypertension that is independent of obesity [38, 39]. An increase in the apnea-hypopnea index (AHI) (see definition below) by one event/hour increases the odds of hypertension by 1%. [38] OSA is associated with cardiac arrhythmias, particularly atrial fibrillation and supraventricular tachyarrhythmias [40–43]. Whether OSA is independently associated with ischemic heart disease remains controversial [44]. However, there is evidence that OSA, particularly when more severe, is associated with coronary artery disease and decreased survival (Fig. 10.5) [45–48].

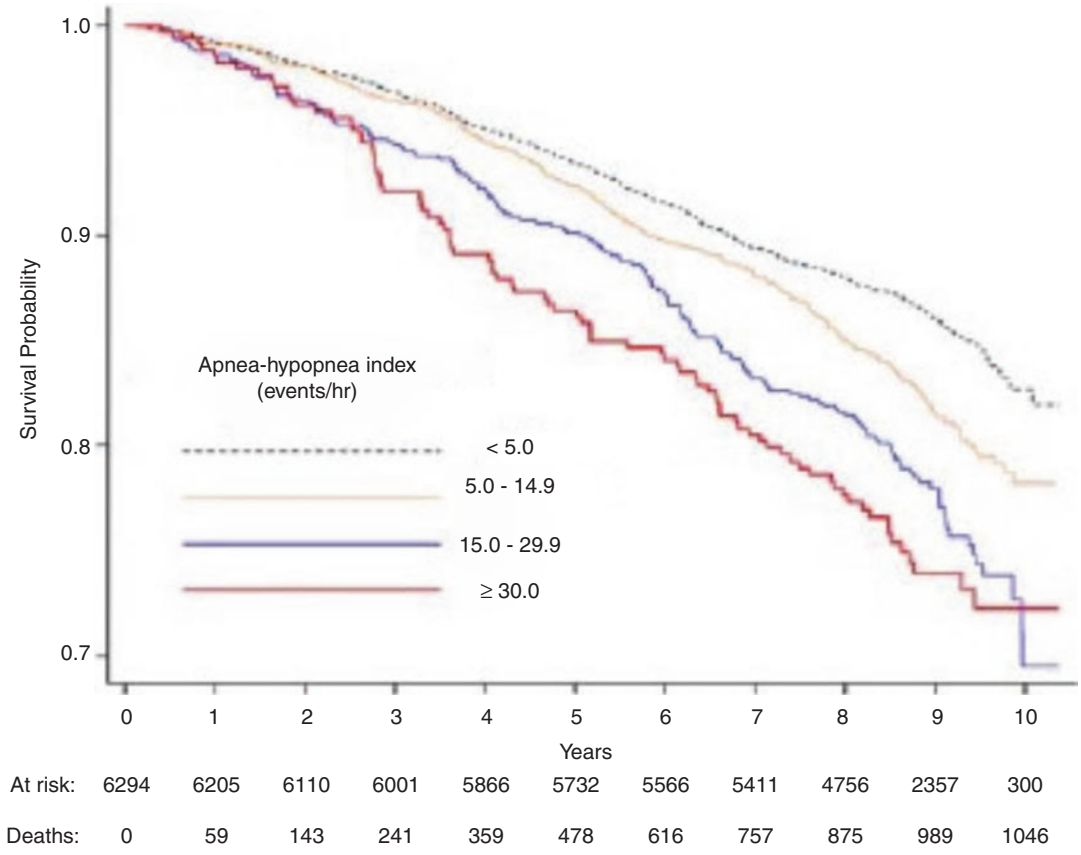

Fig. 10.5 Probability of survival in patients with different severities of OSA as measured by AHI. (Reproduced with permission from [48])

Diagnosis and Classification of OSA

The criterion for diagnosis and severity of OSA most often used is the AHI. An apnea is cessation (≥90% reduction) of airflow for a minimum of 10 seconds. An event is considered obstructive if there is continuing respiratory effort during the apnea. A hypopnea has variable definitions. This chapter will use the definition suggested by the American Academy of Sleep Medicine; a hypopnea is a decrease in airflow by 30% for ≥10 seconds associated with arterial oxygen desaturation of ≥3% measured by oximetry [49].

An AHI of ≥5 is considered abnormal. An AHI ≥ 5 associated with symptoms of obstructive sleep apnea is diagnostic of OSA (Table 10.2). Severe OSA is most often defined as an AHI >30 (Table 10.3); however there is some controversy surrounding this definition [50, 51]. The reader should be aware that other markers of severity include, but are not limited to, the nadir arterial oxygen saturation, as well as length and pattern of desaturations. The study of OSA is a rapidly changing field, and these definitions are likely to change as our understanding increases.

Numerous screening tools have been developed to predict OSA in individuals referred for evaluation of SDB. However, a diagnosis of SDB, particularly OSA, should be suspected in every patient referred for bariatric surgery. Several studies have demonstrated that the prevalence of OSA is between 70% and 80% in the bariatric surgery population [52–54].

Table 10.2 Symptoms of OSA

Symptoms of OSA
Excessive daytime sleepiness Choking or gasping during sleep
Unrefreshing sleep
Recurrent awakenings from sleep
Witnessed apneas
Daytime fatigue
Impaired concentration
Cognitive deficits
Depression/mood changes
GERD
Morning headaches

Table 10.3 Severity of OSA as determined by AHI

Severity of OSA	
Classification	AHI
Normal	<5
Mild	5–15
Moderate	16–30
Severe	>30

The evaluation for OSA should begin with a detailed clinical and sleep history. A patient should be interrogated about snoring, witnessed apneas, and nocturnal gasping. Often, it is helpful to ask the patient's bed partner these questions as many patients themselves may be unaware. Psychiatric and cognitive disturbances, such as depression and decreased ability to concentrate or remember, are classic symptoms of OSA. A comprehensive evaluation should include an estimate of total sleep time, questions about insomnia, and reasons for waking (e.g., nocturia). Daytime sleepiness should be quantified by a standardized tool, such as the Epworth Sleepiness Scale, seen below (Fig. 10.6) [29]. A score of 11 or more indicates abnormal daytime sleepiness. A history of conditions associated with OSA including hypertension, cerebrovascular disease, and motor vehicle accidents should be sought.

Physical examination is also important in the assessment. Increased neck circumference (≥16 inches in women, ≥17 inches in men) also increases the risk of OSA. Oropharyngeal crowding due to obesity, macroglossia, an oversized uvula, or tonsillar hypertrophy increases the risk of OSA. This is reflected in the Mallampati score, which has been shown to be an independent predictor of both presence and severity of OSA [55]. Retrognathia, micrognathia, and nasal abnormalities, including polyps, septal deviation, or turbinate hypertrophy, are also implicated. However, these conditions do not improve with bariatric surgery.

Those at high risk of OSA should have objective confirmation of disease presence and severity. The American Academy of Sleep Medicine (AASM) recommends that objective determination of OSA severity is necessary to determine appropriate management [29]. Two types of testing are endorsed: the polysomnogram (PSG) and the home sleep apnea test (HSAT), also known as the portable monitor (PM). The PSG is an in-laboratory, supervised overnight study where the

How Sleepy Are You?

How likely are you to doze off or fall asleep in the following situations? You should rate your chances of dozing off, not just feeling tired. Even if you have not done some of these things recently try to determine how they would have affected you. For each situation, decide whether or not you would have:

- No chance of dozing =0
- Slight chance of dozing =1
- Moderate chance of dozing =2
- High chance of dozing =3

Write down the number corresponding to your choice in the right hand column. Total your score below.

Situation	Chance of Dozing
Sitting and reading	•
Watching TV	•
Sitting inactive in a public place (e.g., a theater or a meeting)	•
As a passenger in a car for an hour without a break	•
Lying down to rest in the afternoon when circumstances permit	•
Sitting and talking to someone	•
Sitting quietly after a lunch without alcohol	•
In a car, while stopped for a few minutes in traffic	•

Total Score = _____

Fig. 10.6 Epworth sleepiness scale

subject is observed sleeping by a certified sleep technician. It is considered the gold standard for diagnosis [29, 56].

The attended PSG is a complex and costly test that monitors several physiologic and mechanical parameters throughout sleep. Electroencephalography (EEG), electrooculography (EOG), muscle activity, airflow, oxygen saturation, respiratory effort, and cardiac rate and rhythm are all simultaneously monitored under direct observation by a trained sleep technologist. It is recommended that the results are summarized and interpreted by a certified sleep physician. SDB is a common concern, and many areas have limited PSG capacity. Wait lists for diagnosis of OSA can be lengthy. A Canadian study demonstrated that OSA patients may wait up to 11.6 months and 16.2 months for initiation of medical or surgical treatment, respectively [57].

HSAT is a useful alternative to the PSG for the diagnosis of suspected OSA, particularly when there is a high pretest probability of moderate-to-severe disease [29, 56]. The HSAT records fewer physiologic variables. At a minimum, it should record airflow, respiratory effort, and oxygen saturation. Its accuracy is affected by comorbid sleep disorders or major comorbid medical disorders. Exclusion of significant cardiopulmonary, vascular, and neurological comorbidities is necessary. These tests do not measure EEG and cannot reliably determine when the patient is sleeping. Consequently, they generally

underestimate the severity of SDB. They are not recommended for patients with moderate-to-severe pulmonary disease, neuromuscular disease, and congestive heart failure and those with a comorbid sleep disorder.

Treatment

OSA is a chronic disease and should be managed in a multidisciplinary manner. Therapy should always include behavioral and lifestyle modification. This includes diet, exercise, and weight loss. Patients should also be advised to avoid sedating medications and alcohol, particularly at nighttime. These interventions rarely lead to complete OSA remission but should always be promoted [58]. A systematic review and meta-analysis confirmed that lifestyle interventions are effective at reducing OSA severity as measured by AHI [59].

CPAP is considered the mainstay therapy for OSA. When used properly, CPAP can be curative. [29]. CPAP utilizes a pressurized hose and upper airway interface (facemask or nasal cannula) to pneumatically splint open and stabilize the airway throughout the respiratory cycle during sleep. Since CPAP became widely available in the 1980s, its major limitation has been patient compliance. A patient is considered to be compliant if they use their CPAP for ≥4 hours nightly for ≥70% of nights [60]. However, reality often falls far short of this goal. A review found that 29–83% do not use CPAP for 4 hours [61]. Twelve–25% of patients will entirely abandon CPAP within 3 years [62]. This has led to the development of alternative modalities of treatment. Alternatives and adjuncts include medications, positional therapies, exercise regimes to strengthen the upper airway, oral appliances, upper airway surgery, tracheostomy, and, sometimes as a last resort, bariatric surgery. Several experimental therapies are also in development, including a variety of upper airway muscle stimulators.

Bariatric surgery is an effective means to promote and maintain weight loss [63]. Consequently, it can be a very effective therapeutic option for the treatment of OSA. A 2004 meta-analysis of outcomes in bariatric surgery demonstrated that SDB is the most responsive obesity-related pathology [63]. Several reviews have demonstrated a marked reduction in nocturnal respiratory events following bariatric surgery [64, 65]. When compared to nonsurgical weight loss, bariatric surgery is more effective at reducing both BMI and AHI [66]. A systematic review of 13 900 patients with OSA who underwent various bariatric procedures demonstrated that 75% had an improvement in their AHI. However, only 4% had complete resolution of OSA (AHI < 5). For this reason, bariatric surgery is considered an *adjunctive* therapy [29]. Despite marked weight loss in patients following successful surgery, the body of evidence indicates that the majority have an elevated (but improved) AHI or RDI following recovery from bariatric surgery [63, 64]. A meta-analysis by Buchwald quotes resolution of OSA symptoms in 85.7% of bariatric surgery patients [63]. However, only the minority of studies included in this analysis objectively quantitated respiratory disturbances following surgery. PSG analysis following surgery demonstrates that 62% of patients have a residual AHI of ≥15, which would be characterized as moderately severe OSA. Although OSA may improve following surgery, patients should first be offered a noninvasive alternative. Most patients will still require CPAP following bariatric surgery [67]. This remains the case several years after surgery, despite the bariatric patient experiencing marked weight loss. A randomized controlled trial that compared conventional weight loss to bariatric surgery demonstrated that after 2 years, despite a significant sustained improvement in BMI in the surgical patients, the AHI were similar in two groups [68]. Another smaller trial had similar findings [69]. These studies highlight the importance of continuing conventional therapies for the management of OSA postoperatively, over the long term, regardless of weight loss. Following significant weight loss (≥10%), objective testing to determine OSA severity and for appropriateness of prescribed CPAP is necessary. The AASM recommends a follow-up sleep study be performed on any bariatric patient in whom moderate-to-severe OSA existed preoperatively. [29]

Perioperative Management of Sleep Apnea and Sleep-Disordered Breathing

Perioperative risk is higher in those with OSA compared to other subjects undergoing a wide range of surgical procedures [70–72]. The bariatrician's expectation should be that their patient has OSA of some severity. Screening tools for OSA, such as the Berlin Questionnaire, STOP-BANG score, and Sleep Apnea Clinical Score, were not validated in patient populations being evaluated for bariatric surgery [73–75]. Nonetheless, some experts suggest using them as a screening tool in bariatric populations [31]. There is a vigorous debate in which specific screening tests or specific preoperative care is necessary for SDB in patients referred for bariatric surgery.

Existing recommendations regarding perioperative care are largely based on expert opinion, which are variable. Some suggest that all patients being evaluated for bariatric surgery should be screened using an objective sleep study (PSG or HSAT) to determine presence and severity of OSA [76–81]. Others suggest the selective use of PSG only in circumstances where there is objective evidence of cardiac or pulmonary disease [82, 83]. Others disagree that every patient should be screened because of the relatively low perioperative complication rates seen in bariatric surgery in general. There is no evidence to suggest that preoperative PSG decreases the complication rate. A commonly seen complication, postoperative oxygen desaturation, may not be clinically significant [84]. What is not controversial is that those with significant comorbidities, such as right or left heart failure, pulmonary hypertension, and hypercapnia, should be studied with PSG preoperatively [31].

Patients with confirmed OSA awaiting bariatric surgery should have their SDB treatment optimized prior to surgery. Those with moderate-to-severe OSA are at highest risk for perioperative complications, especially if the patient is still symptomatic (daytime tiredness, hypercapnia) or if there has been excessive recent weight gain. Evaluation may include a repeat PSG, HSAT, or consultation with a sleep specialist. Patients should be screened for concomitant OHS, as daytime hypercapnia is associated with adverse perioperative outcomes. An arterial blood gas while awake can be used to screen for hypercarbia ($PaC0_2$ > 45 mmHg). A metabolic alkalosis ($HC0_3$ > 27 mmol) that is otherwise unexplained is also a relatively sensitive indicator of OHS [85].

The pathophysiology of OSA itself can be exacerbated at several stages during surgery. Medications, most notably sedatives, opioids, anesthetics, and paralytics, can reduce upper airway tone, increasing the likelihood and severity of apneic events. Furthermore, these agents depress central respiratory drive and may inhibit the protective arousal response. Complications from intubation, most notably laryngeal edema or tracheal stenosis, can further compromise an already tenuous airway. Positioning a patient supine, the position where OSA is worst, can also exacerbate OSA. Fluid administration during surgery can cause pharyngeal edema further compromising the upper airway. Lastly, many patients neglect to inform their clinicians that they have OSA and consequently forget to bring their therapeutic devices (CPAP, bi-level positive pressure, oral appliances) to hospital.

Surgical risk is also increased by conditions associated with OSA, such as pulmonary arterial hypertension, metabolic syndrome, arterial hypertension, and coronary artery disease. Hypoxemia due to OSA, particularly when untreated, can cause cardiac arrhythmias. A meta-analysis of 3942 patients showed that OSA patients are two to four times more likely to experience postoperative oxygen desaturations, respiratory failure, or adverse cardiac events or require ICU admission [70]. Not surprisingly, bariatric patients with OSA are at increased risk of prolonged hospitalization, with consequently increased healthcare costs [86]. They have an increased risk respiratory compromise during sedation. These concerns highlight why numerous experts continue to advocate for the need to screen bariatric patients to determine the severity of OSA. With respect to postoperative care, expert opinion advises continuous monitoring on

a designated surgical or medium care unit, particularly when patients are known to have moderate or severe OSA. In the absence of complications, routine ICU admission is not necessary [31].

Bariatric surgery patients represent a high-risk surgical group. However, it is important to note that OSA does not confer additional risk above that of the bariatric patient without a formal diagnosis of SDB [76, 77, 87]. A meta-analysis of 13 studies with 98 935 patients compared perioperative outcomes in bariatric surgery patients with confirmed OSA to those without a formal diagnosis. Morbidity rates ranged from 0 to 25% but were similar in each group. There was no increased risk of cardiopulmonary morbidity, intensive care utilization, mortality, or length of stay after bariatric surgery for those with a history of OSA [77]. It is important to note that this study did not analyze preoperative care; those with diagnosed OSA may have received preoperative optimization of SDB prior to surgery.

There has been debate on whether CPAP should be administered following bariatric surgery. Concerns of anastomotic complications by positive airway pressure causing digestive tract distention have been reported [88]. However, several reviews of large patient cohorts, particularly those undergoing Roux-en-Y gastrectomy, suggest that it is safe and advisable [89, 90]. If positive pressure is omitted, early ambulation and incentive spirometry are critical [91].

Our understanding of obesity and its consequences for the respiratory system has increased substantially. It is clear that further research is of paramount importance given the global epidemic of obesity. Bariatric surgery is one of the most effective means of reversing the deleterious effects of excess fat on pulmonary function and OSA. The pathophysiology of OSA is complex, requiring an understanding of physiology, upper airway mechanics, and neurohormonal interplay. Perioperative identification and optimization of patients with OSA is important prior to bariatric surgery. Though usually not curative, OSA can improve considerably after bariatric surgery. As bariatric surgery rates increase, it will be important to monitor the outcomes in this interesting patient group.

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