 Review article

Perioperative respiratory care in obese patients undergoing bariatric surgery: Implications for clinical practice

Sjaak Pouwels a, b, *, Frank W.J.M. Smeenk d, e, Loes Manschot c, Bianca Lascaris c, Simon Nienhuijs a, R. Arthur Bouwman c, Marc P. Buis c

ab Department of Surgery, Catharina Hospital, Eindhoven, The Netherlands
d Department of Epidemiology, CAPHRI Research School, Maastricht University, The Netherlands
e Department of Anaesthesiology, Intensive Care and Pain Medicine Catharina Hospital, Eindhoven, The Netherlands
f Department of Respiratory Medicine, Catharina Hospital, Eindhoven, The Netherlands
g SHE School of Health Professions Education, Maastricht University, The Netherlands

A B S T R A C T

Obesity is an increasing problem worldwide. The number of people with obesity doubled since the 1980's to affect an estimated 671 million people worldwide. Obese patients in general have an altered respiratory physiology and can have an impaired lung function, which leads to an increased risk of developing pulmonary complications during anaesthesia and after bariatric surgery (approximately 8%). Therefore the respiratory management of the bariatric surgical patient provides a number of challenges. This review will focus on the perioperative respiratory care in bariatric surgical patients discussing respiratory physiology in the obese and perioperative respiratory care in bariatric surgery. Finally the value of preoperative pulmonary function testing and preoperative OSAS screening will be discussed.

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* Corresponding author. Department of Surgery, Catharina Hospital, Michelangeloalaan 2, P.O. Box 1350, 5602 ZA, Eindhoven, The Netherlands.
E-mail address: sjaakpws@gmail.com (S. Pouwels).

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1. Introduction

Obesity is an increasing problem worldwide. The number of people with obesity doubled since the 1980’s to affect an estimated 671 million people worldwide [1,2]. Among these, the number of people with the highest Body Mass Index (BMI > 40 kg/m²) grew twice as fast as the group of people with a BMI of 30–40 kg/m² [2,3]. In the Netherlands, 48.3% of the people aged 19 years and older have overweight (defined as a BMI > 25 kg/m²) [2].

The only treatment with a longstanding effect is bariatric surgery [4]. With the increasing prevalence of obesity, the worldwide numbers of bariatric surgical procedures are also increasing [4]. With obesity affecting many organ systems, under which the respiratory system, it is therefore not surprising that the respiratory management of obese subjects undergoing bariatric surgery represents a growing challenge.

This review will focus on the perioperative respiratory care in bariatric surgical patients discussing the following subjects:

- Respiratory physiology in the obese patients
- Perioperative respiratory care in bariatric surgery
- The value of preoperative pulmonary function testing and OSAS screening

2. Respiratory physiology in the obese

Obesity negatively affects many organ systems, including the respiratory system. It is associated with an altered lung function, characterised by a reduction of lung volumes, mostly a restrictive pattern. The pathogenesis behind this is multifactorial, but an increased truncal fat load is one of the possible mechanisms [5]. A restrictive pattern is seen when both the vital capacity (VC) and the total lung capacity (TLC) is below 80% of their predicted value and the Tiffeneau index (forced expiratory volume in 1 s (FEV1)/VC) is < 0.7.

Because of affected respiratory physiological parameters such as compliance, neuromuscular strength, work of breathing (WOB), lung volumes and spirometric measurements [6,7], obese subjects are prone to develop pulmonary complications after bariatric surgery.

2.1. Relationship between anthropometric variables and lung function

Soriano et al. [8] investigated over 3000 people in Spain and found a restrictive pattern in 12.7%. A higher BMI (>30 kg/m²) was independently associated with a restrictive spirometry [8]. This was confirmed by Mannino et al. [9] who measured spirometry in different locations around the world. He found that a BMI below or above the reference categories (18.5–24 kg/m²) was a significant risk factor for a restrictive pattern [9]. Less is known about the prevalence of a restrictive lung function among obese people. Most research was done with obese people who were a candidate for bariatric surgery. Groups were small, varying between 20 and 150 patients and the prevalence varied between 6 and 50% [10–13].

The impact of obesity on the respiratory system may vary from patient to patient and cannot be predicted from weight and/or BMI measurements alone. While assessment of BMI gives an impression about overall nutritional status, it does not differentiate lean mass, fat mass and the distribution of adipose tissue. This distinction might be important as lean- and fat mass have an opposite effect on lung function and the distribution of fat seems more relevant than total body fat per se. Body fat percentage was associated with decreased FVC, FEV1 ratio and FEV1/VC ratios in men and women respectively [14–18]. Moreover, others showed an adverse relation with waist to hip ratios and the respiratory system. High waist to hip ratios were associated with reduced lung function and poorer gas-exchange [15,16,18,19].

Most studies use spirometry to assess lung function. Babb et al. [19] took a different approach. They state that the End-Expiratory Lung Volume (EELV or residual volume (RV)) is very sensitive to changes in static compliance of the lung and chest wall. Deposits of fat on the chest wall thus specifically alter the EELV [19]. They measured absolute and relative fat mass with MRI and compared it to the End-Expiratory Lung Volume (EELV). They found that fat distribution is relatively similar between lean and obese men and women, and that therefore the increase in chest wall fat distribution is proportional to the increase in obesity [19]. This means that measurements of overall obesity are significantly related to lung function as measured with EELV. So far, the books are not yet closed on which measurement should be used to assess the effect of obesity on lung function, but a combination of Waist/Hip (W/H) ratio and BMI seems recommended.

2.2. Respiratory compliance

Respiratory compliance is the ability of the respiratory system to stretch during a change in volume relative to an applied change in pressure [6]. Total respiratory compliance (e.g. the compliance of the pulmonary and extrapulmonary structures in the thoracic cage) can be reduced in obesity and in patients with the obesity-hypventilation syndrome to as little as one-third of the normal values [6,20]. This is mainly the result of reduced distensibility of extrapulmonary structures due to excess truncal fat [6,20]. Secondly, the increase in pulmonary blood volume and increased closure of dependant airways may also contribute to the low lung compliance seen in obese people [21]. These physiological changes are even more pronounced during recumbency in obese subjects (as compared to normal weight subjects), due to increased gravitational effects of the abdomen [22].

2.3. Lung volumes

The most consistent indicator of obesity is a reduction in expiratory reserve volume (ERV). This is due to a displacement of the diaphragm more cranially into the thoracic cage by the ‘obese’ abdomen and the increased chest wall mass [6,23,24]. This
association is seen in modest obesity, but the ERV rapidly decreases with increasing BMI [6,25,26]. The ERV decreases because the ‘obese’ abdomen inhibits the diaphragm to extend in caudally.

On the other hand, obesity has modest effects on residual volume (RV) and total lung capacity (TLC), but a relatively larger effect in reducing functional residual capacity (FRC) [26–28]. In some studies the reduction of the FRC is so marked that it approaches RV [27]. When the reduced FRC is equal to or lower than the closing volume, thoracic gas trapping may take place in obese subjects, as indicated by an elevated RV/TLC ratio [28–30]. The effects of obesity on lung volumes are shown in Fig. 1.

2.4. Work of breathing

To compensate the reduced respiratory compliance, (severely) obese subjects may breathe rapidly and shallowly, to maintain eucapnia [6,23,31–33]. Therefore people with obesity have a higher Work of Breathing (WOB). Because of a restrictive pattern, breathing takes place at a less compliant part of the pressure-volume curve. Thus they can develop a rapid, shallow breathing pattern, which comes with a higher oxygen cost [6,23,24]. The oxygen cost of breathing is an index that represents the energy required to breathe [21]. This index shows the oxygen consumed by the respiratory muscles per litre of ventilation [21]. Kress et al. [34] investigated eighteen severely obese patients and found a 16% reduction in oxygen consumption after elective intubation, mechanical ventilation and anaesthesia from the baseline values, as compared to <1% reduction among controls. This (relative) respiratory inefficiency among the obese suggests a decreased ventilatory reserve and a (possible) predisposition to respiratory failure [6,23,24,34].

2.5. Respiratory muscle strength

Regarding respiratory muscle strength, different results are being reported in the current literature. Several studies indicate that obese people have greater risk of developing respiratory muscle inefficiency, in terms of less capability to generate normal maximal inspiratory pressure and expiratory pressures [6]. Other studies showed that there was no difference in inspiratory pressures between obese individuals and non-obese controls [35–37]. A possible explanation for impaired respiratory muscle function in obesity can be the increased elastic load that the respiratory muscles are required to overcome during inspiration [6,38].

A mechanical disadvantage exists because of an overstretched diaphragm, leading to decreased inspiratory muscle strength and efficiency [6,39]. Also some studies indicate decreased levels of skeletal muscle glycogen synthase activity in obese subjects that can be a contributing factor in the decrease in isokinetic skeletal muscle endurance [40,41].

2.6. Clinical relevance of pulmonary function changes

The pulmonary function changes due to obesity can roughly be summarised in three parts I) a restrictive effect of mass on the chest wall, II) a tendency to breathe at low lung volumes and III) the effect of fat distribution on pleural pressure [28,42–47]. The physiological changes are heightened during sleep in supine position, due to a negative impact pulmonary mechanics of diaphragm impedance by the abdomen with a change in lung volume. This leads to intolerance for apnoeic episodes and to early desaturation. These pulmonary mechanics changes in obesity lead to a lower FRC, FVC and FEV₁ [28,42–47].

2.7. Obstructive Sleep Apnoea Syndrome (OSAS) and Obesity Hypoventilation Syndrome (OHS)

Research in patients with the Obesity Hypoventilation Syndrome (OHS: the existence of alveolar hypoventilation which cannot be explained by an other neuromuscular or pulmonary disorder) demonstrated that these patients exhibit a lowered neuromuscular response to hypercapnia and hypoxemia, caused by a blunted central drive [6,23,31–33]. It is unclear, however, what causes this blunted central drive. A lowered chemosensitivity could be genetic, but other factors such as a distorted sleep-breathing pattern (Obstructive Sleep Apnoea Syndrome (OSAS)) and neurohormonal factors could also play a role. The fact that effective treatment of the obstructed breathing may partly correct nocturnal hypoxemia and awake hypcapnia, seems to prove that upper airway obstruction and flow limitation are important factors in the development of OHS [6,23,24].

But few patients, even among those with severe OSAS, develop daytime hypercapnia. Possibly, the severity of the nocturnal hypoxemia is important [6,26,31–33]. Despite a similar BMI, severity of the apnoea/hypopnea index, arousal indices and sleep architecture, OHS patients exhibit a more severe desaturation compared to the eucapnic obese group [23].

Animal research showed that nocturnal hypoxemia lowers the hypoxic ventilatory drive and raises the arousal threshold, possibly because of an effect on the synthesis and turnover of neurotransmitters [23,48–51]. Because of this, patients fail to compensate adequately after an episode of hypoventilation. This causes decreased ventilation in between episodes for a given CO₂-load, combined with a relatively shorter time-span between episodes considering the duration of apnoeic episodes [48,50,51]. Norman et al. [52] hypothesizes that repeated nocturnal CO₂-accumulations cause bicarbonate retention by the kidneys. Most patients have

Abbreviations: ERV = Expiratory Reserve Volume, FRC = Functional Residual Capacity, TLC = Total Lung Capacity, RV = Residual Volume

Fig. 1. Lung function parameters before and after surgical weight loss (Adapted from Thomas et al. [93]).
enough time to compensate for this brief raise in CO2. But if patients compensate insufficiently after an event, chronic bicarbonate retention develops, raising the threshold for hypercapnia [23,52]. What causes this diminished response is yet to be discovered. Possible actors might be leptin and IL-GF-1 because of their stimulating effect on central respiratory centres [23].

3. Perioperative respiratory care in bariatric surgery

Because the percentage of obese people in the population increases [2,3], more people are at risk for an altered lung function due to obesity. As a consequence, the risk of respiratory complications under general anaesthesia might increase as well. This can result in hypoxemia, hypercapnia and increased formation of atelectasis.

3.1. Induction and airway management

Obesity increases the risk of a difficult airway during induction, because of an altered upper airway anatomy [6,7,23]. Heinrich et al. [53] found that 6% of cases of a difficult laryngoscopy over a 6-year period occurred in patients with a BMI ≥ 35 kg/m². The incidence of a difficult intubation is increased from 5.8% in the general population and 15.8% in people with a BMI >30 kg/m² [54]. The restricted lung function leads to a greater risk of desaturation during induction. The lowered FRC can approach the closing capacity, which can lead to closing of airseways during tidal breathing [6,54,55]. This leads to shunting and a ventilation/perfusion mismatch. This is exaggerated by a supine position, in which the abdominal pressure and the pulmonary blood volume are increased and recruitment of dependent alveoli exists, and by an increased oxygen demand [55].

Techniques that can be used to reduce this risk are:

- **Prediction difficult intubation**: indirect mirror laryngoscopy [56].
- **(Awake) videolaryngoscopy**: Moore et al. [57] found that 96% of 50 morbid obese patients undergoing bariatric surgery and classified as having a difficult airway were successfully intubated using awake videolaryngoscopy.
- **Head-up induction**: Several studies have investigated different positioning techniques to improve the laryngeal view in patients with morbid obesity. Lee et al. [58] found that the laryngeal view can be significantly improved when patients were put in a 25° head-up position when compared with the conventional supine position. Collins et al. [59] found a statistical significant better laryngeal view when morbidity obese patients were placed in the ‘ramped’ position, in which the external auditory meatus was at the same level as the sternal notch. Gupta et al. [60] compared rapid sequence induction (RSI) in semi-erect position with the investigator in front of the patient compared to RSI in supine position with the GlideScope videolaryngoscope of morbid obesity patients, but found no significant differences between the two groups when it comes to intubation parameters or patient safety.
- **Pre-oxygenation**: Dixon et al. [61] found obesity achieved higher oxygen tensions and therefore a clinically significant increase in the desaturation safety period in the 25° head-up position. Ramkumar et al. [62] investigated the 20° head up pre-oxygenation in non-obese individuals, and found that time to desaturation (<93%) was significantly prolonged as well.
- **Apnoeic oxygenation**: Ramachandran et al. [63] found that providing obese patients with additional nasal O2 during simulated difficult laryngoscopy was associated with a significant prolongation of SpO2 ≥ 95%, a significant increase in patients with SpO2 ≥ 95% apnoea at 6 min, and a significantly higher minimum SpO2. Resaturation times were no different.

3.2. Ventilation strategies in obese patients

General anaesthesia and paralysis negatively affect pulmonary gas exchange and respiratory mechanics (Fig. 2). When PEEP is not applied, atelectasis formation is present in 90% of patients under general anaesthesia [64,65].

Patients with morbid obesity have a higher risk of atelectasis formation, exhibit more profound changes in respiratory function and are at a higher risk for hypoxemia [6,7,66]. Pelosi et al. [66] found that under general anaesthesia, PaO2 is inversely related to BMI. The severity of atelectasis formation is related to body weight and extends longer in the postoperative phase when patients are severely overweight [67,68].

Reinis and colleagues [68] showed in obese patients under general anaesthesia that PEEP in combination with recruitment manoeuvres reduced atelectasis and improved PaO2/FiO2 ratio. This effect was still present 40 min later. The compliance was also improved [68]. Aldenkortt et al. [69] found similar results based on their performed meta-analysis on the effects of different ventilation strategies in obese patients. Also Aldenkortt et al. [69] compared pressure- and volume-controlled ventilation (PCV and VCV). In terms of intraoperative PaO2, FiO2 ratio and tidal volumes no significant differences were found between the both ventilation strategies [69].

3.2.1. **Positive airway pressure (BiPAP)**

Yu et al. [70] hypothesized that BiPAP ventilation may diminish the development of pulmonary shunt and may improve ventilation-perfusion mismatch when compared to standard IPPV, with or without PEEP when neuromuscular paralysis has been used during surgery. They found that BiPAP ventilation was beneficial in decreasing ventilation-perfusion mismatch and improving oxygenation when compared with conventional IPPV (with or without PEEP) [70].

Ebee et al. [71] investigated the effect of BiPAP on pulmonary function in obese patients following open gastric bypass surgery. This resulted in significantly higher VC and FEV1 12–24 h post-operative, but did not result in fewer hospital days or lower complication rates [71]. Joris et al. [72] found similar results in terms of postoperative pulmonary function, but no significant
differences were found between the BiPAP and control group in terms of peak expiratory flow rate [72].

3.2.2. Non-invasive ventilation strategies (CPAP and NIV)

Continuous positive airway pressure (CPAP) and NIV have been used to prevent and treat acute respiratory failure after surgery or to treat acute respiratory failure [73,74]. What is the most ideal ventilation strategy is to reduce postoperative morbidity is unclear. A meta-analysis [75] showed that CPAP following abdominal surgery significantly reduced postoperative pulmonary complications, atelectasis and pneumonia, which is in contrast with a recent Cochrane review [76] that stated the evidence is of low quality.

The feasibility and safety of NIV use in the recovery room after various types of surgery has been demonstrated [77] and also a recent meta-analysis [78] showed that in perioperative bariatric care NIV is well tolerated and significantly reduces respiratory complications. However there is still lack of comparative studies to determine which ventilation strategy is superior in perioperative bariatric care.

3.3. Influence of metabolic syndrome and comorbidities on the perioperative period

Bariatric surgery is an acceptable and effective method to manage obesity-related comorbidities in morbidly obese patients [79,80]. Nearly four in five patients scheduled for bariatric surgery has metabolic syndrome [80]. In particular the presence of cardiac, pulmonary, metabolic and hepatic comorbidities may vary in patients scheduled for bariatric surgery, thus posing particular challenges to the anaesthesiologist [79]. Hypertension (both systemic and pulmonary), dyslipidaemia and hyperglycaemia respond to bariatric surgery [79–81]. A large retrospective study by Purnell et al. [80] showed that there was no significant difference in perioperative complications in patients with or without metabolic syndrome (Table 1 shows the correlation between anthropometric variables and pulmonary function parameters).

4. Is preoperative pulmonary function testing necessary in bariatric surgery?

The current body of literature is sparse regarding the effects of an impaired pulmonary function and its relation with the occurrence postoperative complications in bariatric surgery. A study conducted by van Huisstede et al. [82] investigated the relationship between pulmonary function parameters and the risk of postoperative complications in a 485 patients. They found 53 complications of which 8 of them were from pulmonary origin. Patients with complications had a significantly lower FEV1 (mean 86.9% of predicted) and FVC (mean 95.6% of predicted) compared to patients without complications (P < 0.05) [82]. A FEV1/FVC <70% and a ΔFEV1 ≥ 12% were found to be predictors for pulmonary complications [82]. In the study by Sood et al. [83] morbidly obese patients (with a BMI >40 kg/m²) have increased odds of developing pulmonary complications.

Questions arise whether it might be useful to perform preoperative pulmonary function testing in patients scheduled for bariatric surgery. And which BMI group is at risk for postoperative (pulmonary) complications and does this increased risk has a relation with the lung function. A second problem that may arise is the possible need for screening for OSAS, in this population. Because this co morbidity occurs frequently and if untreated, it might result in postoperative complications [84–87].

Lastly, we searched the literature for evidence for preoperative strategies that might be applied in obese patients with impaired pulmonary function to prevent postoperative complications.

4.1. Preoperative pulmonary function screening

The current body of literature regarding the clinical utility of preoperative pulmonary assessment in bariatric surgery remains questionable. Reasons to preoperatively screen patients is to identify those who are at high risk of developing postoperative pulmonary complications. To assess the utility and predictive value we must separate the literature regarding open and laparoscopic bariatric procedures.

Farina et al. [12] investigated the value of spirometry as a preoperative screening tool to identify patients scheduled for open biliopancreatic diversion at risk for postoperative pulmonary complications (PPC). They found a very low rate of PPC’s (7.5%) in patients with suspected restrictive pulmonary impairment [12]. Hamou et al. [88] investigated the usefulness of pulmonary function tests in predicting the overall risk for complications (including PPC’s). They found on multivariate analysis that age (p = 0.01) and a decreased VC (p = 0.0007) were significant predictors for postoperative pulmonary complications [88].

Catheline et al. [10] screened 77 patients prior to bariatric surgery on cardiac and pulmonary abnormalities and found no consequences for the management of the perioperative period. Notwithstanding their results they still found based on their clinical experience that cardiac and pulmonary screening essential prior to bariatric surgery [10].

4.2. Preoperative OSAS screening

Nepomnayshy et al. [89] investigated the additive value of screening for sleep apnoea prior to laparoscopic bariatric surgery for predicting postoperative pulmonary complications and compared them with obese patients undergoing orthopaedic surgical procedures. As a result of screening, of 882 patients, 119 bariatric patients (25%) were newly diagnosed with OSAS. The orthopaedic surgery group had 17.3% (72 of 415 patients) with pre-existing OSAS. The unscreened orthopaedic patients had

| Table 1 Correlation between anthropometric variables and pulmonary function parameters. |

<table>
<thead>
<tr>
<th>Gender</th>
<th>Anthropometric variable</th>
<th>Pulmonary function variable</th>
<th>Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jung et al. [14]</td>
<td>Men</td>
<td>Body fat percentage</td>
<td>FVC and FEV1</td>
</tr>
<tr>
<td>Zavorsky et al. [15,16]</td>
<td>Women</td>
<td>Body fat percentage</td>
<td>FEV1/VC</td>
</tr>
<tr>
<td></td>
<td>Women</td>
<td>Waist/hip ratio</td>
<td>Gas exchange</td>
</tr>
<tr>
<td>Wehrmeister et al. [17]</td>
<td>Men</td>
<td>Waist circumference</td>
<td>FEV1 and FVC</td>
</tr>
<tr>
<td>Rossi et al. [18]</td>
<td>Men</td>
<td>Body fat percentage</td>
<td>FVC and FEV1</td>
</tr>
<tr>
<td>Woman</td>
<td>Body fat percentage</td>
<td>FVC and FEV1</td>
<td>Inverse</td>
</tr>
<tr>
<td>Babb et al. [19]</td>
<td>Men</td>
<td>Fat deposition thorax</td>
<td>RV</td>
</tr>
<tr>
<td>Woman</td>
<td>Fat deposition thorax</td>
<td>RV</td>
<td>Inverse</td>
</tr>
</tbody>
</table>

Abbreviations: FVC = Forced Vital Capacity, FEV1 = Forced expiratory volume in 1 s, RV = Residual Volume.
complication rate of 6.7% compared to 2.6% for the screened bariatic patients. This difference was not statistically significant after adjusting for age and comorbidity (p = 0.3383) [89].

Peromaa-Haavisto et al. [90] showed that in a population of 197 obese patients scheduled for bariatric surgery, there was a prevalence of OSAS was 71%, with a significantly higher prevalence in males (90%) compared with women (60%). According to their study results, they recommend OSAS screening preoperatively especially in obese men [90].

4.3. Preoperative pulmonary preparation

The current evidence around preoperative pulmonary preparation (in case of an impaired lung function found during screening) is lacking. Also the effects on postoperative complications are not clear. In a study by Barbalho-Moulim et al. [91] randomised 32 obese women undergoing elective open bariatric surgery to either an inspiratory muscle-training group or usual care group. Compared to the preoperative values, the MIP decreased significantly in both groups after surgery. However the reduction in MIP was 28% in the inspiratory muscle training group en 47% in the usual care group [91]. There was a significant reduction in postoperative complications in the inspiratory muscle-training group.

Van Huijstede et al. [82] concluded that the risk of pulmonary complications after laparoscopic bariatric surgery is low. However patients with abnormal spirometry test results have a threefold risk of complications after laparoscopic bariatric surgery [82]. In obese patients with asthma, van Huijstede et al. [92] recently showed that bariatric surgery has beneficial effects on lung function, in terms of small airway function, decreased systemic inflammation and the number of mast cells in the airways compared to obese patients without asthma. In both groups FEV1, FVC and TLC significantly improved, whereas FEV1/VC only improved in the obese patients with asthma [92]. Unfortunately it is unclear whether obesity/morbid obesity and preoperative asthma control has influence on the postoperative outcomes.

It can be hypothesized that perioperative respiratory physiotherapy (in bariatric surgery) might have a role in preventing PPC’s in patients with a (obesity induced) respiratory defect. Also for specific groups (asthma, OSA and OHS patients) pulmonary function tests may be useful and might bring clinical advantages.

5. Conclusion

The majority of the obese patients have an altered respiratory physiology and have an impaired lung function, which leads to an increased risk of developing pulmonary complications during anaesthesia and after bariatric surgery. Therefore the respiratory management of the bariatric surgical patient is challenging. There are a growing number of studies in particular around optimal ventilation strategies to minimize the risk of postoperative complications. Unfortunately, this is still a grey area because not one ventilation strategy has shown superiority in preventing postoperative atelectasis after bariatric surgery. Whether patients scheduled for bariatric surgery need to be screened for obesity related pulmonary function impairment (and OHS/OSAS) is still subject to discussion.

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References


