



Atelectasis in Bariatric Surgery: Review Analysis and Key Practical Recommendations

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Abstract

Obesity is a condition that affects multiple organ systems, particularly the cardiovascular and respiratory system. In recent years, bariatric surgery has been reported to be the gold standard in the treatment of morbid obesity. Body mass index alone is insufficient to predict risks related to anaesthesia and surgery. Obesity contributes to significant postoperative atelectasis and is considered an independent risk factor for postoperative atelectasis owing to decreased functional residual capacity. The treatment and reversibility of atelectasis developed in obese patients undergoing bariatric surgery are challenging. Therefore, an optimisation of pulmonary functions before surgery, lung-sparing ventilation during the perioperative period, awareness of potential postoperative complications and knowledge about preventive measures and therapeutic approaches have become increasingly important in bariatric surgery. The aim of this review was to aid clinicians in the management of atelectasis in patients undergoing bariatric surgery during the perioperative and postoperative period.

Keywords: Atelectasis, bariatric surgery, obesity

Introduction

According to the World Health Organization (WHO), obesity is defined as an excessive or abnormal fat accumulation that poses a risk to health. Based on the body mass index (BMI) calculation by WHO, a BMI ≥ 30 kg m⁻² indicates obesity. Obesity is divided into four categories according to BMI as follows: Class I obesity, BMI: 30.00-34.99; Class II obesity, BMI: 35.00-39.99; and Class III obesity (morbidly obese), BMI ≥ 40.00 (1).

The obesity prevalence has increased by three-fold worldwide since 1975. In 2016, 39% of adults aged ≥ 18 years (39% of men and 40% of women) has become overweight. Overall, 13% of the world population (11% of men and 15% of women) was in 2016. It is estimated that approximately one-half of the world population will become overweight or obese by 2030 (2).

In developed countries, the mortality of obesity is 0.1%, and obesity and related diseases account for 5%-10% of total healthcare expenses in the United States. In the European Union, the annual cost related to obesity is estimated to be 70 billion euros. The European Association for the Study of Obesity found that obesity is directly accountable for 1.5%-4.6% of healthcare expenses in France, whereas it is up to 7% in Spain (3).

In recent years, bariatric surgery has been reported to be the gold standard in the treatment of morbid obesity, as an effective treatment modality to achieve permanent weight loss and provide protection against polypharmacy, resulting in reduced healthcare costs in the long-term. According to the International Federation of Surgery, a to-

tal number of bariatric/metabolic surgeries were 593,792 in 2014. In the United Kingdom, it has been reported that the number of bariatric procedures has increased by thirty-fold in the past decade. Morbidity is less than 1% in surgical procedures in experienced centres. It has been postulated that this is due to a better understanding of short- and long-term surgical complications of bariatric interventions (4).

Obesity is a condition affecting multiple organ systems, particularly the cardiovascular and respiratory systems. An obese patient may have several comorbidities, such as hypertension, dyslipidaemia, ischaemic heart diseases, diabetes mellitus, osteoarthritis and asthma. Also, these patients may present with a restrictive pattern in the lung function evaluation (usually with a decreased functional residual capacity), and obstructive sleep apnoea syndrome is common among morbidly obese patients. Obesity-hypoventilation syndrome is a disorder increasing in frequency that could be responsible for respiratory failure (5).

Body mass index alone is insufficient to predict anaesthesia- and surgery-related risks. The waist or neck circumference shows a better correlation with cardiovascular disorders when compared to BMI. The android type fat distribution makes abdominal surgery challenging and is associated with an increased fat accumulation at the neck and around the airways. Therefore, a preoperative assessment is of importance in these patients. In addition, postoperative complications such as atelectasis, aspiration pneumonia, pleural effusion, pulmonary embolism, or adult respiratory distress syndrome (ARDS) can be experienced more commonly in obese patients (6).

Atelectasis is defined as the volume reduction in a one or more lobes or segments of the lung. With thoracoabdominal surgeries, the occurrence and extension of postoperative atelectasis depend on the type of anaesthesia and surgery, the patient positioning during the surgery, the agents used for anaesthetic management, and the patient's baseline preoperative pulmonary functions (7). Particularly, in patients undergoing bariatric surgery, obesity contributes to significant postoperative atelectasis and is considered an independent risk factor for postoperative atelectasis owing to decreased functional residual capacity. The treatment and reversibility of atelectasis developed in obese patients undergoing bariatric surgeries are challenging (8). Therefore, an optimisation of pulmonary functions before surgery, lung protective ventilation during the perioperative period, awareness of potential postoperative complications and knowledge about preventive measures (such as effective pain control and early mobilisation of patients) and therapeutic approaches have become increasingly important in bariatric surgery.

Clinical effects of bariatric surgery on respiratory system

The calorie intake and energy expenditure are higher in obese patients. The basal metabolism rate should be considered according to the body surface area. However, the body surface area is enlarged with increasing body weight; therefore, the absolute basal metabolism rate is increased when compared to lean individuals, resulting in a higher oxygen consumption and carbon dioxide production in obese individuals (9). Hypoxemia tendency in obese patients becomes more prominent in supine position and anaesthesia during bariatric surgery because of diaphragm function impairment. The apnoea period should be very short at the anaesthesia induction because there is a tendency of rapid desaturation progression. In addition, the lung compliance is decreased due to an increased pulmonary blood volume and an excessive weight of the fat tissue surrounding the chest wall. Total lung compliance may be decreased by up to 35%. Small airways are closed, and the functional residual capacity is significantly reduced by cephalad displacement of abdominal content and an increased intrathoracic blood volume. In non-obese adults, the functional residual capacity (FRC) is decreased by 0.7-0.8 L in the sitting position. However, FRC may drop below 1 L in obese patients with a BMI >40 kg m⁻². In morbidly obese patients, the forced vital capacity, forced expiratory volume in one second (FEV1) and mid-expiratory flow are also decreased (10).

Similarly, the alveolar-arterial (A-a) oxygen gradient increases with increased BMI. Therefore, the FRC approximates the closing volume during normal tidal ventilation, resulting in airway closure, ventilation-perfusion mismatch and atelectasis. The incidence of intrapulmonary shunt is also increased. A reduction in the compliance becomes marked by pneumoperitoneum during laparoscopic surgery. Although there are reports suggesting that the reverse Trendelenburg position provides partial recovery in the FRC loss, to the best of our knowledge, there are no studies indicating that it decreases postoperative atelectasis. The airway resistance is increased when the patient is shifted to supine from a sitting position. As a result of these factors, breathing workload is increased in morbid obesity. Mild preoperative (A-a) O₂ gradient and shunt fraction may markedly worsen during the anaesthesia induction, and the patient may require high FiO₂ and positive end-expiratory pressure (PEEP) for adequate oxygenation. Reduced chest wall compliance and diaphragm tone, and an increased risk for atelectasis and secretions will contribute to rapid desaturation during hypoventilation or apnoea, which might be sustained during the postoperative period. Supplemental oxygen alone may be inadequate or even may worsen atelectasis (11). Rivas et al. (12) showed that in obese patients undergoing bariatric surgery, the pulmonary shunt and the ventilation-perfusion ratio and subsequent pulmonary gas exchange can significantly improve after surgery.

Obesity also increases the risk of obstructive sleep apnoea (OSA). The OSA syndrome is characterised by apnoea episodes resulting from pharyngeal collapse during sleep, and it may be obstructive, central or mixed type. The incidence of OSA increases with an advanced age and obesity. It has been reported that its prevalence is 2%-24% and that BMI is $>40 \text{ kg m}^{-2}$ in almost one-half of patients with OSA. In a meta-analysis, it has been reported that OSA is associated with higher postoperative respiratory failure, cardiac event and ICU admission rates. Sleep studies are needed for diagnosis of OSA. These patients may be more vulnerable to depressive effects of anaesthetic agents, such as postoperative oxygen desaturation, hypercapnia or hypoxaemia. Respiratory control is affected in obesity hypoventilation syndrome with observation of diurnal alterations in ventilation and an increased PaCO_2 level. Depressants including many anaesthetic and analgesic drugs make these effects more prominent (13).

Obesity and surgery-related atelectasis

Atelectasis is a common complication that may be observed in all patient groups undergoing general anaesthesia. Atelectasis may develop immediately after the induction and cause decreased lung compliance and the ventilation/perfusion (V/Q) ratio. Unlike non-obese patients, the risk for atelectasis may continue up to 48 h following extubation in obese patients (14). In a meta-analysis involving more than 13,000 patients who underwent bariatric surgery, atelectasis was reported as the fourth most common cause (11%) of postoperative respiratory failure and related deaths (15). In a study by Baltieri et al. (16), it has been found that the atelectasis prevalence was 37% in patients who underwent bariatric surgery, although patients with complications such as obstructive sleep apnoea syndrome (OSAS), asthma, bleeding or fistulas, or those with conditions that may prolong a hospital stay due to continuous positive airway pressure (CPAP), bilevel positive airway pressure (BIPAP) or non-standard O_2 in ICU were excluded. In a study, Eichenberger et al. (17) have compared morbidly obese and lean patients regarding the resorption of atelectasis after general anaesthesia. Authors have suggested that the amount of atelectasis is higher in obese patients than in lean patients, even before induction; that the extent of increase in atelectasis is significantly higher in obese patients after induction; and that postoperative atelectasis remains significant 24 h post-surgery in obese patients, while it is completely resolved in lean patients. Therefore, the atelectasis risk should be taken into account in obese patients and appropriate measures should be taken.

Several reasons have been postulated for the development of atelectasis. Regardless of the anaesthetic agent used in general anaesthesia practices, FRC is decreased by 0.4-0.5L due to a diminished tone in respiratory muscles and an impaired

balance between the elastic recoil in forces of the lung and chest wall. In addition, it is shown that anaesthesia dominates over the stabilising features of surfactant. A decreased FRC results in an early closure of terminal airways, particularly during expiration. As a result, atelectasis develops due to the resorption of gases distal to the obstructed airway. FRC is particularly decreased in obesity-hypoventilation syndrome in obese patients. Pulmonary compression caused by an upward movement of diaphragm due to relaxation by anaesthesia in muscles worsens atelectasis. Such effects of general anaesthesia are more prominent in obese individuals (18, 19). The risk of decrease in FRC and associated postoperative pulmonary complications are higher in thorax and upper abdominal surgeries than surgeries in other regions. In bariatric surgery, surgical procedures negatively affect abdominal, thoracic and diaphragmatic muscle strength. In lungs with predisposition to atelectasis, laparoscopy enhances these effects. Lung compliance is decreased by pneumoperitoneum required for bariatric surgery, which creates difficulties and challenges related to ventilation (20).

During the postoperative period, pain contributes to a decreased FRC by preventing cough. A prolonged surgery time is another factor that decreases FRC in upper abdominal surgeries such as bariatric procedures (21).

Diaphragmatic dysfunction plays a critical role in the development of pulmonary complications, particularly in patients undergoing upper abdominal or thoracic surgery. Defects in diaphragmatic contraction may continue up to a week after surgery in some patients. An increased expiratory muscle activity often occurs during both anaesthesia and the postoperative period. These mechanisms together with a reduction in FRC may escalate the atelectasis severity (22).

In general anaesthesia, absorption atelectasis may be seen due to ventilation with 100% O_2 during induction and extubation. In a study by Edmark et al. (23), it has been shown that a reduced FiO_2 level used during the induction significantly prevents atelectasis. Therefore, patients are being ventilated by 100% O_2 during induction and recovery.

Atelectasis may be related to intraoperative ventilation strategies. It has been shown that a high tidal volume, together with a high plateau pressure causes ventilator-related lung injury during pneumoperitoneum. Therefore, a tidal volume should be calculated based on ideal body weight in obese patients. It is recommended to use pressure-controlled mechanical ventilation. In pressure-controlled modes, the intraabdominal pressure should be monitored closely since unexpectedly high tidal volumes may occur if the intraabdominal pressure is decreased due to leakage from ports or pneumoperitoneum is discontinued, resulting in volutrauma.

It has been reported that atelectasis is observed in 17% of OSAS patients after bariatric surgery and that OSA prevalence varies from 48% to 91% in sleep studies performed before bariatric surgery (24, 25).

Schumann et al. (26) have investigated the effects of the type and duration of bariatric surgery on postoperative pulmonary complications, such as pneumonia, atelectasis, pleural effusion, pneumothorax, ARDS, or acute respiratory failure. In that study, it has been found that postoperative pulmonary complications occurred in 1.3% of gastric bypass procedures, 0.3% of gastric band procedures, 0.84% of gastric sleeve procedures and 1.9% of other procedures, indicating significant differences among procedures. Of bariatric surgeries, 5.4% (4.9% open and 0.5% conversion to open surgeries) were performed as open surgery, while 94.6% were performed by using minimal invasive techniques. It has been reported that patients undergoing open procedures have a significantly higher number of postoperative pulmonary complications when compared to those undergoing minimal invasive surgeries. It has been also shown that duration is significantly longer in patients with postoperative complications in all procedure types. It is recommended to choose the fastest procedure (less than 3 h) to offer a possible elimination of postoperative complications. In addition, a history of smoking and impaired mucociliary clearance caused by chronic obstructive pulmonary disease (COPD) or chronic bronchitis also contributes to atelectasis. Age, gender, Acetylsalicylic acid (ASA) use, metabolic syndrome (MetS), asthma and congestive heart failure have been defined as independent risk factors for postoperative pulmonary complications (27).

Diagnosis

Postoperative atelectasis may manifest with tachypnoea and hypoxaemia; however, it may also be asymptomatic. Hypoxaemia generally becomes symptomatic after discharge from recovery room. The atelectasis risk increases on postoperative 48 hours and continues up to Days 4 and 5. Hypoxaemia is the major laboratory finding of atelectasis and is diagnosed by arterial blood gases. Carbon dioxide levels may be normal or low due to increased ventilation (28).

Findings of the lobar collapse may be observed on chest radiographs or a computed tomography scan. On sonography, atelectasis appears as homogeneous hypoechoic areas. Other diagnostic modalities including bronchoscopy, thoracoscopy and open surgery are generally used in management rather than diagnosis (16, 29). The STOP-BANG questionnaire relying on criteria such as snoring, daytime sleepiness, witnessed apnoea, neck circumference >40 cm, hypertension, BMI >35, age >50 and gender was developed to help diagnosis in patients with OSAS. Similarly, there are other surveys, including the Berlin questionnaire, Epworth sleepiness scale,

Stanford sleepiness scale and Pittsburgh Sleep Quality Index. In recent years, several studies have been performed about the prediction of postoperative pulmonary complications using these questionnaires, particularly the STOP-BANG questionnaire (30).

Consequences of Atelectasis

Atelectasis leads to severe hypoxaemia due to decreased lung compliance, ventilation-perfusion mismatch, shunts, impaired gas exchange and increased pulmonary vascular resistance. In addition, it may cause atelectrauma due to an acute lung injury resulting from impaired oxygenation and increased pulmonary permeability. Again, it may lead to an infection due to increased bacterial proliferation and translocation to circulation, as well as the failure of antibiotic to penetrate the lung tissue (31).

ARDS is characterised by rapid-onset bilateral pulmonary infiltrates and non-cardiac pulmonary oedema. Several pathophysiological phenomena are implied as the ARDS causes following bariatric surgery, which include volutrauma, barotrauma, biotrauma and atelectrauma, reflecting the complexity of acute respiratory distress syndrome. In the postsurgical setting, the term *atelectrauma* is used to characterise the lung injury mechanisms triggered by atelectasis. There are case reports suggesting that a post-aspiration ARDS develops following laparoscopic adjustable gastric banding (32, 33).

The rate of postoperative pneumonia and respiratory failure morbidity was reported as 20% after prolonged atelectasis. It is one of the causes that increase the 30-day mortality following bariatric surgery (34). In addition to pneumonia, antibiotic penetration to lung tissue is also impaired with atelectasis. Since the atelectasis-related perioperative ventilation-perfusion mismatch hampers the distribution of antibiotics in the lung tissue, prophylactic and/or therapeutic antibiotics may be ineffective in the treatment of the respiratory tract infection and/or pneumonia (35).

Postoperative respiratory failure may become a severe, life-threatening condition after bariatric surgery and is defined as the presence of acute respiratory distress or pulmonary failure. In a study on 304, 515 patients who underwent bariatric surgery over 3 years, the incidence of postoperative respiratory failure has been found as 1.35%. In-hospital mortality (5.69%) has been found to be significantly higher among patients with postoperative respiratory failure when compared to those without it (36).

Differential diagnosis

Respiratory distress developing in a postoperative recovery unit suggests hypoventilation due to anaesthetic effects and upper airway obstruction due to airway oedema. Second, it

may develop due to an accumulation of pharyngeal secretions, posterior prolapses of tongue or tongue oedema due to surgical manipulation or allergic reactions. Pulmonary embolism, pulmonary oedema and infections should be kept in mind if hypoxaemia, tachypnoea and dyspnoea develop during the postoperative period. Pain following upper abdomen surgeries, distention and pain at chest wall or upper abdomen can present similar clinical findings; therefore, they should be considered in the differential diagnosis of atelectasis (37).

Management

It has been reported that atelectasis is usually resolved within 2 days after laparoscopy or major surgery in obese patients and within 24 h in non-obese patients. Atelectasis may be prolonged in morbidly obese patients, resulting in relapsing hypoxemia and postoperative pulmonary complications. It is important to recognise the risk factors and to take appropriate measures to prevent such complications. Preoperative measures include quitting smoking, incentive spirometry, deep breathing exercises, coughing and pursed-lip respiration exercises, postural drainage, aerosol therapy and application of positive pressure. Early mobilisation in the postoperative period is the key to avoid respiratory complications. Preoperative patient education is strongly recommended as outcomes are better in high-risk patients if preoperative education is provided (38).

Good postoperative analgesia will help to prevent under-ventilation to institute manoeuvres to open collapsed alveoli, such as incentive spirometry and deep breathing exercises. In addition, it is helpful to relieve neuroendocrine stress response, which may lead to postoperative pulmonary complications and organ dysfunction. For both, it was shown that epidural analgesia and patient-controlled analgesia are superior to on-demand analgesics (39).

Intrathoracic positive pressure application is the most important approach in the prevention and management of atelectasis. It prevents collapse in the airway and alveoli, increases FRC, decreases interstitial oedema and improves gas exchange. In addition, afterload is decreased with an improved diaphragm activity and reduced respiratory workload by intrathoracic positive pressure. Non-invasive mechanical ventilation (NIMV) is the best-established method for intrathoracic positive pressure application. The underlying mechanism is an increased intrathoracic pressure for the treatment of postoperative pulmonary complications (40).

Non-invasive mechanical ventilation can recover obesity-related comorbidities; relieve the upper respiratory tract obstruction; improve alveolar ventilation and solve atelectasis; improve gas exchange and respiratory function; relieve dyspnoea; and reduce the perioperative respiratory effort in obese

patients. NIMV is a safe treatment to optimise management and improve postoperative course in obese patients; therefore, it should be considered in the perioperative management (41).

Non-invasive mechanical ventilation can be used for either prophylactic or curative purposes. Prophylactic NIMV is employed in patients with severe risk factors for postoperative respiratory failure, such as obesity, comorbid COPD or cardiovascular diseases. Curative NIMV aims to treat a postoperative respiratory failure avoiding intubation. In both instances, it can be applied as CPAP or BIPAP. In CPAP, a fixed and continuous positive pressure is applied throughout the entire respiratory cycle, while BIPAP provides a pressure differential with a higher inspiratory airway pressure and lower expiratory airway pressure (EPAP) during ventilation. In studies on prophylactic CPAP after upper abdominal surgeries, it was shown that atelectasis was significantly prevented with a rapid improvement in the lung volume; in addition, significant improvements were observed in oxygenation and the length of ICU and hospital stay (42). It was reported that the implementation of CPAP immediately after extubation was associated with a greater improvement in spirometry values when compared to ICU implementation. In a randomised-controlled study on 209 patients who underwent NIMV for curative purposes, it was shown that CPAP in the management of postoperative hypoxemia following abdominal surgery was associated with marked improvement in intubation, pneumonia and sepsis rates, as well as the length of ICU stay (43). In a study by Conti et al. (44), it has been shown that NIMV improved oxygenation and the respiration rate in patients who developed postoperative respiratory failure after abdominal surgeries. Intubation was avoided in 66% of patients by NIMV provided with pressure-support ventilation (PSV) values that progressively escalated from 5-8 cmH₂O until achieving an 8-10 mL kg⁻¹ exhaled tidal volume and a PEEP of 4-8 cmH₂O.

Baltieri et al. (45) have conducted a study to identify whether the preoperative, intraoperative, or immediate postoperative period is the best period for the application of appropriate positive pressure to prevent postoperative atelectasis. They have indicated that in patients undergoing bariatric surgeries, the optimal time of application of non-invasive positive pressure ventilation is in the immediate postoperative period, immediately after extubation, because during that period, NIMV can significantly reduce the incidence of atelectasis by preserving the FRC and preventing significant losses in the expiratory reserve volume.

In the literature, PSV, pressure-controlled ventilation, volume-controlled ventilation, use of high tidal volume and PEEP alveolar recruitment manoeuvres are the most intensively characterised ventilation strategies in obese patients.

These strategies aim to improve oxygenation and to prevent undesired effects such as atelectasis, hemodynamic instability and, in rare instances, barotrauma (46). Recent data provide strong evidence that suggest lung protective mechanical ventilation using a lower tidal volume (6-8 mL kg⁻¹) estimated body weight, a moderate PEEP (6-8 cmH₂O) and recruitment manoeuvres are associated with postoperative functional or clinical outcomes in patients underwent upper abdominal surgery. Low tidal volume ventilation, a sufficient PEEP and repeated recruitment manoeuvres can decrease postoperative pulmonary complications in high-risk patients undergoing major abdominal surgery (47).

Novel methods have been developed to be used when classical therapies fail. High-flow, nasal oxygen therapy (HFNOT) was introduced in early 2000s. In hypoxaemic patients, a non-invasive high-flow respiratory support uses humidified oxygen. A novel method, HFNOT, has become increasingly popular in the perioperative management of high-risk patients. This method is used in the prophylaxis and management of hypoxaemic respiratory failure, cardiac pulmonary oedema, stabilisation of auto-PEEP and postoperative respiratory failure. It is capable of producing a low positive pressure with delivery of high oxygen concentration, while maintaining the ability of the patient to have effective cough and clear secretions. The gas pressure is reduced to 8 Psi from 50 Psi and given to patients via nasal cannula after humidification. The device is easy to use and better tolerated when compared to a traditional oxygen cannula (48).

Preventive measures at postoperative period

There are several preventive measures that could be taken to avoid the formation of postoperative atelectasis in upper abdominal surgeries such as bariatric surgery: incentive spirometry, deep breath exercises, cough, postural drainage, percussion and vibration, suctioning and ambulation and nasal CPAP, all which enable pulmonary expansion/re-expansion. Nasal high-flow oxygen therapy is easily implemented, well-tolerated and is effective for improving postoperative hypoxaemia. For pain management, employing regional techniques and patient-controlled analgesic devices rather than potent analgesics have become important in the prevention of atelectasis by avoiding undesired side effects of opiates such as respiratory depression. In addition, the prevention of residual curarisation and proving the elimination of neuromuscular blockade at the end of surgery are also recommended. In this group of patients, monitoring of peripheral oxygen saturation under oxygen supplementation can cause overlooking of hypoventilation. Therefore, routine oxygen therapy should be considered only in prolonged hypoxaemia. Therefore, patients should be positioned in a semi-sitting position with head elevated at 45°, enabling the diaphragm movements, and respiratory exercises should be initiated and mobilisation should be achieved in the early period (49, 50).

Conclusion

In bariatric surgery, an optimisation of pulmonary functions before the operation, lung protective ventilation during the perioperative period, awareness of potential postoperative complications, and knowledge about preventive measures and therapeutic approaches are extremely important. An improved understanding and experiences of clinicians are the most effective methods in preventing potential complications.

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References

1. Global Health Observatory (GHO) data. Available from: URL: http://www.who.int/gho/ncd/risk_factors/overweight_text/en/. (2017).
2. Hruby A, Hu FB. The Epidemiology of Obesity: A Big Picture. *Pharmacoeconomics* 2015; 33: 673-89. [CrossRef]
3. The University of Reading (UK), Research and Enterprise Services; European Commission Eatwell Report: Available from: http://cordis.europa.eu/result/rcn/53206_en.html
4. Khwaja H, Coelho AJ, Mazzarella M, Miller K, Nimeri A, Ponce J, et al. The IFSO Website (www.ifso.com): The online gateway to obesity and metabolic disorders for bariatric surgery professionals and patients: on behalf of the IFSO communications committee. *Obes Surg* 2015; 25: 2176-9. [CrossRef]
5. Abdelaal M, le Roux CW, Docherty NG. Morbidity and mortality associated with obesity. *Ann Transl Med* 2017; 5: 161. [CrossRef]
6. Shetty S, Parthasarathy S. Obesity hypoventilation syndrome. *Curr Pulmonol Rep* 2015; 4: 42-55. [CrossRef]
7. Degani-Costa LH, Faresin SM, dos Reis Falcão LF. Preoperative evaluation of the patient with pulmonary disease. *Braz J Anesthesiol* 2014; 64: 22-34. [CrossRef]
8. Zammit C, Liddicoat H, Moonsie I, Makker H. Obesity and respiratory diseases. *Int J Gen Med* 2010; 3: 335-43. [CrossRef]
9. Carneiro IP, Elliott SA, Siervo M, Padwal R, Bertoli S, Battezzati A, et al. Is Obesity Associated with Altered Energy Expenditure? *Adv Nutr* 2016; 7: 476-87. [CrossRef]
10. O'Donnell DE, Deesomchok A, Lam YM, Guenette JA, Amornputtisathaporn N, Forkert L, et al. Effects of BMI on static lung volumes in patients with airway obstruction. *Chest* 2011; 140: 461-8. [CrossRef]

11. Hodgson LE, Murphy PB, Hart N. Respiratory management of the obese patient undergoing surgery. *J Thorac Dis* 2015; 7: 943-52.
12. Rivas E, Arismendi E, Agustí A, Sanchez M, Delgado S, Gistau C, et al. Ventilation/Perfusion distribution abnormalities in morbidly obese subjects before and after bariatric surgery. *Chest* 2015; 147: 1127-34. [\[CrossRef\]](#)
13. Mandal S, Hart N. Respiratory complications of obesity. *Clin Med* 2012; 12: 75-8. [\[CrossRef\]](#)
14. Sebbane M, El Kamel M, Millot A, Jung B, Lefebvre S, Rubenovitch J, et al. Effect of weight loss on postural changes in pulmonary function in obese subjects: a longitudinal study. *Respir Care* 2015; 60: 992-9. [\[CrossRef\]](#)
15. Esquinas Rodriguez AM, Papadakos PJ, Carron M, Cosentini R, Chiumello D. Clinical Review: Helmet and non-invasive mechanical ventilation in critically ill patients. *Crit Care* 2013; 17: 223. [\[CrossRef\]](#)
16. Baltieri L, Peixoto-Souza FS, Rasera-Junior I, Montebelo MI, Costa D, Pazzianotto-Forti EM. Analysis of the prevalence of atelectasis in patients undergoing bariatric surgery. *Braz J Anesthesiol* 2016; 66: 577-82. [\[CrossRef\]](#)
17. Eichenberger A, Proietti S, Wicky S, Frascarolo P, Suter M, Spahn DR, et al. Morbid obesity and postoperative pulmonary atelectasis: an underestimated problem. *Anesth Analg* 2002; 95: 1788-92. [\[CrossRef\]](#)
18. Pedoto A. Lung physiology and obesity: anesthetic implications for thoracic procedures. *Anesthesiol Res Pract* 2012; 154208. [\[CrossRef\]](#)
19. Magnusson L, Spahn DR. New concepts of atelectasis during general anaesthesia. *Br J Anaesth* 2003; 91: 61-72. [\[CrossRef\]](#)
20. Hu XY. Effective ventilation strategies for obese patients undergoing bariatric surgery: a literature review. *AANA J* 2016; 84: 35-45.
21. Elrazek AE, Elbanna AE, Bilasy SE. Medical management of patients after bariatric surgery: Principles and guidelines. *World J Gastrointest Surg* 2014; 6: 220-8. [\[CrossRef\]](#)
22. Hedenstierna G, Edmark L. Mechanisms of atelectasis in the perioperative period. *Best Practice Res Clin Anaesthesiol* 2010; 24: 157-69. [\[CrossRef\]](#)
23. Edmark L, Auner U, Enlund M, Ostberg E, Hedenstierna G. Oxygen concentration and characteristics of progressive atelectasis formation during anaesthesia. *Acta Anaesthesiol Scand* 2011; 55: 75-81. [\[CrossRef\]](#)
24. Jaber S, Coisel Y, Chanques G, Futier E, Constantin JM, Michelet P, et al. A multicenter observational study of intraoperative ventilator management during general anaesthesia: tidal volumes and relation to body weight. *Anaesthesia* 2012; 67: 999-1008. [\[CrossRef\]](#)
25. Martí-Valeri C, Sabaté A, Masdevall C, Dalmau A. Improvement of associated respiratory problems in morbidly obese patients after open Roux-en-Y gastric bypass. *Obes Surg* 2007; 17: 1102-10. [\[CrossRef\]](#)
26. Schumann R, Shikora SA, Sigl JC, Kelley SD. Association of metabolic syndrome and surgical factors with pulmonary adverse events, and longitudinal mortality in bariatric surgery. *Br J Anaesth* 2015; 114: 83-90. [\[CrossRef\]](#)
27. Atilla N, Arpag H, Bozkus F, Kahraman H, Cengiz E, Bulbuloglu E, et al. Can we predict the perioperative pulmonary complications before laparoscopic sleeve gastrectomy: original research. *Obes Surg* 2017; 27: 1524-8. [\[CrossRef\]](#)
28. Malbouisson LM, Humberto F, Rodrigues Rdos R, Carmona MJ, Auler JO. Atelectasis during anesthesia: pathophysiology and treatment. *Rev Bras Anesthesiol* 2008; 58: 73-83. [\[CrossRef\]](#)
29. Lichtenstein D A, Lascols N, Prin S, Mezière G. The “lung pulse”: an early ultrasound sign of complete atelectasis. *Intensive Care Med* 2003; 29: 2187-92. [\[CrossRef\]](#)
30. Nagappa M, Patra J, Wong J, Subramani Y, Singh M, Ho G, et al. Association of STOP-Bang Questionnaire as a Screening Tool for Sleep Apnea and Postoperative Complications: A Systematic Review and Bayesian Meta-analysis of Prospective and Retrospective Cohort Studies. *Anesth Analg* 2017; 125: 1301-8. [\[CrossRef\]](#)
31. Rama-Maceiras P. Peri-operative atelectasis and alveolar recruitment manoeuvres. *Arch Bronconeumol* 2010; 46: 317-24. [\[CrossRef\]](#)
32. Avriel A, Warner E, Avinoach E, Avnon LS, Shteinberg M, Shteinberg D, et al. Major respiratory adverse events after laparoscopic gastric banding surgery for morbid obesity. *Respir Med* 2012; 106: 1192-8. [\[CrossRef\]](#)
33. Rubenfeld GD, Caldwell E, Peabody E, Weaver J, Martin DP, Neff M, et al. Incidence and outcomes of acute lung injury. *N Engl J Med* 2005; 353: 1685-93. [\[CrossRef\]](#)
34. Guimarães MM, El Dib R, Smith AF, Matos D. Incentive spirometry for prevention of postoperative pulmonary complications in upper abdominal surgery. *Cochrane Database Syst Rev* 2009; 8: CD006058. [\[CrossRef\]](#)
35. Hutschala D, Kinstner C, Skhirtladze K, Mayer-Helm BX, Zeitlinger M, Wisser W, et al. The impact of perioperative atelectasis on antibiotic penetration into lung tissue: an in vivo microdialysis study. *Intensive Care Med* 2008; 34: 1827-34. [\[CrossRef\]](#)
36. Masoomi H, Reavis KM, Smith BR, Kim H, Stamos MJ, Nguyen NT. Risk factors for acute respiratory failure in bariatric surgery: data from the Nationwide Inpatient Sample, 2006-2008. *Surg Obes Relat Dis* 2013; 9: 277-81. [\[CrossRef\]](#)
37. Bajwa SS, Kulshrestha A. Diagnosis, prevention and management of postoperative pulmonary edema. *Ann Med Health Sci Res* 2012; 2: 180-5. [\[CrossRef\]](#)
38. Gross JB, Bachenberg KL, Benumof JL, Caplan RA, Connis RT, Coté CJ, et al. Practice guidelines for the perioperative management of patients with obstructive sleep apnea: a report by the American Society of Anesthesiologists Task Force on perioperative management of patients with obstructive sleep apnea. *Anesthesiology* 2006; 104: 1081-93. [\[CrossRef\]](#)
39. Alaparathi GK, Augustine AJ, Anand R, Mahale A. Comparison of diaphragmatic breathing exercise, volume and flow incentive spirometry, on diaphragm excursion and pulmonary function in patients undergoing laparoscopic surgery: a randomized controlled trial. *Minim Invasive Surg* 2016: 1967532. [\[CrossRef\]](#)
40. Karcz M, Papadakos PJ. Respiratory complications in the postanesthesia care unit: A review of pathophysiological mechanisms. *Can J Respir Ther* 2013; 49: 21-9.
41. Carron M, Zarantonello F, Ieppariello G, Ori C. Obesity and perioperative noninvasive ventilation in bariatric surgery. *Minerva Chir* 2017; 72: 248-64.
42. Jaber S, De Jong A, Castagnoli A, Futier E, Chanques G. Non-invasive ventilation after surgery. *Ann Fr Anesth Reanim* 2014; 33: 487-91. [\[CrossRef\]](#)

43. Squadrone V, Cocha M, Cerutti E, Schellino MM, Biolino P, Occella P, et al. Continuous positive airway pressure for reatment of postoperative hypoxemia: a randomised controlled trial. *JAMA* 2005; 293: 589-95. [\[CrossRef\]](#)
44. Conti G, Cavaliere F, Costa R, Craba A, Catarci S, Festa V, et al. Non-invasive positive pressure ventilation with different interfaces in patients with respiratory failure after abdominal surgery: a matched controlled study. *Respir Care* 2007; 52: 1463-71.
45. Baltieri L, Santos LA, Rasera I Jr, Montebelo MI, Pazzianotto-Forti EM. Use of positive pressure in the bariatric surgery and effects on pulmonary function and prevalence of atelectasis: randomized and blinded clinical trial. *Arq Bras Cir Dig* 2014; 27: 26-30. [\[CrossRef\]](#)
46. Aldenkortt M, Lysakowski C, Elia N, Brochard L, Tramèr MR. Ventilation strategies in obese patients undergoing surgery: a quantitative systematic review and meta-analysis. *Br J Anaesth* 2012; 109: 493-502. [\[CrossRef\]](#)
47. Fernandez-Bustamante A, Hashimoto S, Serpa Neto A, Moine P, Vidal Melo MF, Repine JE. Perioperative lung protective ventilation in obese patients. *BMC Anesthesiol* 2015; 15: 56. [\[CrossRef\]](#)
48. Porhomayon J, Pourafkari L, El-Solh A, Nader ND. Novel therapies for perioperative respiratory complications. *J Cardiovasc Thorac Res* 2017; 9: 121-6. [\[CrossRef\]](#)
49. Sudré EC, de Batista PR, Castiglia YM. Longer Immediate Recovery Time After Anesthesia Increases Risk of Respiratory Complications After Laparotomy for Bariatric Surgery: A Randomized Clinical Trial and a Cohort Study. *Obes Surg* 2015; 25: 2205-12. [\[CrossRef\]](#)
50. Karcz M, Papadakos PJ. Respiratory complications in the postanesthesia care unit: A review of pathophysiological mechanisms. *Can J Respir Ther* 2013; 49: 21-9.