HAL
open science

How can I manage anaesthesia in obese patients?

Audrey de Jong, Amélie Rolle, François-Régis Souche, Olfa Yengui, Daniel Verzilli, Gerald Chanques, David Nocca, Emmanuel Futier, Samir Jaber

To cite this version:

Audrey de Jong, Amélie Rolle, François-Régis Souche, Olfa Yengui, Daniel Verzilli, et al.. How can I manage anaesthesia in obese patients?. Anaesthesia Critical Care & Pain Medicine, 2020, 39 (2), pp.229-238. 10.1016/j.accpm.2019.12.009 . hal-02483123

HAL Id: hal-02483123

https://hal.science/hal-02483123v1

Submitted on 2 Jul 2020

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

How can I manage anaesthesia in obese patients?

Audrey De Jong [a,*], Amélie Rollé [b], François-Régis Souche [c], Olfa Yengui [b], Daniel Verzilli [b], Gérald Chanques [a], David Nocca [c], Emmanuel Futier [d], Samir Jaber [a]

[a] *Inserm, CNRS, PhyMedExp, département d'anesthésie-réanimation, CHU Montpellier, university of Montpellier, hôpital Saint-Eloi, 80, avenue Augustin-Fliche, 34295 Montpellier cedex, France*
[b] *Département d'anesthésie-réanimation, hôpital Saint-Eloi, 80, avenue Augustin-Fliche, 34295 Montpellier cedex, France*
[c] *Département de chirurgie digestive (A), mini-invasive et oncologique, hôpital Saint-Eloi, 80, avenue Augustin-Fliche, 34295 Montpellier cedex, France*
[d] *Département de médecine périopératoire, anesthésie-réanimation, hôpital Estaing, CHU de Clermont-Ferrand, 63000 Clermont-Ferrand, France*

Keywords:
Anaesthesia
Obese
Obesity
Airway
Preoxygenation

A B S T R A C T

The obese patient is at risk of perioperative complications including difficult airway access (intubation, difficult or impossible ventilation), and postextubation acute respiratory failure due to the formation of atelectases or to airway obstruction. The association of obstructive sleep apnoea syndrome (OSA) with obesity is very common, and induces a high risk of per and postoperative complications. Preoperative OSA screening is crucial in the obese patient, as well as its specific management: use of continuous positive pre-, per- and postoperative pressure. For any obese patient, the implementation of difficult intubation protocols and the use of protective ventilation (low tidal volume 6–8 mL/kg of ideal body weight, moderate positive end-expiratory pressure of 10 cmH20, recruitment manoeuvres in absence of contra-indications), with morphine sparing and semi-seated positioning as much as possible are recommended, associated with a close postoperative monitoring. The dosage of anaesthetic drugs is usually based on the ideal body weight or the adjusted body weight and then titrated, except for succinylcholine that is dosed according to the total body weight. Monitoring of neuromuscular blockers should be used where appropriate, as well as monitoring of the depth of anaesthesia, especially when total intravenous anaesthesia is used in association with neuromuscular blockers. The occurrence of intraoperative awareness is indeed more frequent in the obese patient than in the non-obese patient. Appropriate prophylaxis against venous thromboembolism and early mobilisation are recommended, if possible included in an early rehabilitation protocol, to further reduce postoperative complications.

Contents

1. Introduction . . . 230
2. Pathophysiological consequences of obesity . . . 230
   2.1. Respiratory . . . 230
   2.2. Cardiovascular . . . 230
   2.3. Endocrine and metabolic systems . . . 230
3. The prevention of perioperative complications . . . 231
   3.1. Respiratory management . . . 231
      3.1.1. Preoperative preparation . . . 231
      3.1.2. Setting of ventilatory parameters . . . 233
      3.1.3. Extubation . . . 233
      3.1.4. Postoperative oxygenation/ventilation . . . 233
      3.1.5. Early rehabilitation . . . 233

* Corresponding author. Intensive care unit, anaesthesia and critical Care department, Saint-Eloi teaching hospital, university Montpellier 1, 80, avenue Augustin-Fliche, 34295 Montpellier cedex 5, France.
E-mail address: audreydejong@hotmail.fr (A. De Jong).

3.2. Cardiovascular management . . . 234
3.3. Metabolic-endocrine management . . . 234
3.4. Gastro-intestinal management . . . 234
3.5. Drug management . . . 235
3.5.1. Antibioprophylaxis and antibiotherapy . . . 235
3.5.2. Choice of anaesthetic technique . . . 235
3.6. Screening of bariatric complications . . . 235
4. Criteria for outpatient surgery or planned postoperative intensive care unit admissions . . . 235
5. Conclusion . . . 236
References . . . 236

1. Introduction

During the last thirty years, a significant increase in obesity prevalence has been observed around the world [1]. Overweight and obesity are defined as abnormal or excessive fat accumulation presenting an increased health risk. A crude population measure of obesity is the body mass index (BMI), a person's weight (in kilograms) divided by the square of his or her height (in meters), as defined by the World Health Organization (WHO) [2]. A person with a BMI of 30 or more is considered obese, of 40 or more morbidly obese, of 50 or more super-obese and of 60 or more super-super-obese [3,4]. A person with a BMI equal to or more than 25 is considered to be overweight [5]. In 2016, 39% of women and 39% of men aged 18 and over were overweight and 11% of men and 15% of women were obese [2].

Obese patients are at higher risk of perioperative complications than non-obese patients, mainly those related to the respiratory function [6]. Comorbidities and type of surgery are crucial for assessing the perioperative risk of these patients [7]. Preoperative SpO2, at least one preoperative respiratory symptom, preoperative chronic liver disease, history of congestive heart failure, open intrathoracic or upper abdominal surgery, surgical procedure lasting at least 2 h, and emergency surgery were associated with postoperative respiratory failure [8]. Intraoperatively, colloid administration and blood transfusion were also found to be associated with postoperative pneumonia [9]. Super-obese and super-super-obese patients are high-risk surgical patients with reported higher perioperative mortality and morbidity compared to overweight and obese patients [4,10].

Airway management can be challenging, with increased incidence of difficult mask ventilation and intubation [11,12]. The use of analgesic and sedative drugs in the immediate postoperative period increases the risk of respiratory depression [13]. Careful preparation, preoperative risk assessment, adequate anaesthetic management, blood pressure and glycaemic control, strict prevention of thromboembolic events, and effective control of postoperative pain may help reduce the risk associated with surgery in obese patients.

There are specific complications due to pathophysiologic state and a specific management of obese patients [14,15]. Two types of surgery should be considered in obese patients: bariatric and non-bariatric surgeries, with different outcomes and specificities [6]. The objective of this review is to present the perioperative peculiarities of anaesthesia of obese patients in order to offer optimised perioperative medical management, according to the type of surgery performed, bariatric or non-bariatric, aimed at reducing perioperative complications.

2. Pathophysiological consequences of obesity

2.1. Respiratory

In obese patients, intra-abdominal pressure is increased, and total lung capacity, vital capacity and functional residual capacity (FRC) are decreased, which favours the formation of atelectases [16,17]. In addition, oxygen consumption and respiratory work are increased [18], which leads to a decrease in oxygenation [16]. During anaesthesia, decreased pulmonary compliance is responsible for an increase in transpulmonary pressure (difference between the alveolar pressure and the pleural pressure) [19]. This transpulmonary pressure must be differentiated from the transthoracic pressure (difference between pleural pressure and atmospheric pressure, due to compression of the thoracic cavity by the abdomen). Extra pressure, whether transpulmonary or transthoracic, leads to an increase in ventilation pressures [19], which can be associated with haemodynamic compromise. Prolonged increased transpulmonary pressures are associated with an increased rate of postoperative pulmonary complications [20].

Obesity predisposes to and potentiates obstructive sleep apnoea syndrome (OSA) [21,22]. Moreover, the atelectasis and lung volume reduction are more severe in obese patients than in non-obese patients and sustained for more than 24 h, even after minor surgery, causing hypoxemia during and after surgery [23]. Increased abdominal volume with reduction of pulmonary volumes including FRC participates to the higher incidence of atelectases in obese patients [12]. Obese patients are therefore at increased risk of postoperative respiratory complications, including Acute Respiratory Distress Syndrome (ARDS) [14,24,25].

2.2. Cardiovascular

Obesity causes high blood pressure, increases cardiac output and cardiac work, as well as the incidence of coronary heart disease and arrhythmia [26]. The risk of atrial fibrillation is 1.5 times greater in the obese patient than in the non-obese patient [27]. The occurrence of long QT is also more common in obese patients [28].

Moreover, obesity is a prothrombotic condition associated with the occurrence of myocardial infarction, stroke and venous thromboembolism [15,29]. Postoperative incidence of venous thrombosis is ten times higher in obese women than in non-obese women [30]. A hypercoagulable condition may persist for two months, necessitating prolonged postoperative prophylaxis of thromboembolic venous disease depending on the type of surgery and the patient's BMI [31].

2.3. Endocrine and metabolic systems

Patients with centrally dispensed or "android" fat are at higher perioperative risk than those with peripheral fat or "gynoid shape" [32].

Insulin resistance and diabetes are common in obese patients [33]. Poor glycaemic control in the perioperative period is associated with increased morbidity [34]. In bariatric surgery, bypass gastric surgery results in a neurohumoral response, responsible for a rapid and significant reduction in insulin requirements, which begins immediately after surgery. In this type of patients, reintroduction of anti-diabetic drugs should be

careful and frequent blood glucose control should be performed postoperatively [35].

3. The prevention of perioperative complications

The experience of the anaesthetist and the surgeon with this type of patient is important, and an experienced team should be mandatory in obese patients at very high perioperative risk.

The weight limit for operating tables must be checked before any bariatric surgery, and appropriate tables installed. The installation of the bariatric patient is very important, in a room spacious enough to allow for unencumbered transfer of the morbidly obese patient to and from the operating theatre table. The supine position with the legs abducted on straight leg boards is largely used ("beach chair position").

Table 1 presents the main perioperative complications in obese patients and some key points to prevent them.

3.1. Respiratory management

3.1.1. Preoperative preparation

Whatever the type of surgery, and more particularly in bariatric surgery, a preoperative regimen may improve operating conditions [36]. A reasonable weight loss is recommended to reduce perioperative respiratory and cardiovascular complications.

Smoking cessation should be encouraged preoperatively. It appears that a smoking abstinence period of 6 to 8 weeks reduces the risk of postoperative respiratory complications to that of non-smokers [37]. However, even shorter delays are still significant to minimise the risks associated with hyper reactivity and lack of tissue oxygenation by decreasing carboxyhaemoglobin. As a consequence, it is advised to stop smoking preoperatively, whatever the delay [37].

In the case of associated OSA, an observational study reports a decrease in postoperative severe complications (i.e., cardiac events, respiratory complications) when applying CPAP preoperatively compared to the absence of CPAP [38]. Preoperative initiation of CPAP should therefore be considered, especially if OSA is severe. Preoperative use of mandibular advancement devices is advised when it is effective.

The identification of an already diagnosed OSA or the screening for an OSA not yet diagnosed is the main corner of the management of the obese patient. Comorbidities associated with increased incidence of OSA should be sought [39], such as high blood pressure [40], a history of stroke [41], of myocardial infarction [42], diabetes [43] or facial dysmorphia [44]. Any complication that has occurred during a previous procedure, such as difficult intubation or postoperative respiratory distress, should also be sought. Snoring, episodes of nocturnal apnoea, frequent awakenings during sleep (for example, vocalisation, changes of position, extremity movements), morning headaches and daytime sleepiness will suggest an OSA. Epworth score [45] assesses the propensity to doze in 8 different situations of everyday life. A score greater than or equal to 15 indicates excessive daytime sleepiness.

STOP-BANG score [46] is more and more used (Table 2) and validated in the obese patient [46]. The reference exam to confirm the diagnosis is polysomnography, or respiratory polygraphy. OSA is defined by the combination of daytime sleepiness and polysomnographic criteria (apnoea hypopnea index [AHI] ≥ 5). The severity of OSA depends on clinical symptoms and AHI:

Table 1
Prevention of perioperative complications in the obese patient.

Complications	Prevention
Respiratory	
Difficult mask ventilation	Loco-regional anaesthesia if possible
Difficult intubation	Rapid sequence induction if gastro-oesophageal reflux disease
Per- and postoperative atelectases	Preoxygenation with NIV ± HFNO
	PEEP and alveolar recruitment manoeuvres during invasive mechanical ventilation
	Postoperative (prophylactic) NIV if high risk of respiratory complications
	Early mobilisation
	Active respiratory physiotherapy
Cardiovascular	
Difficult perfusion	Central venous access, ultrasound++
Difficult monitoring (blood pressure+++)	Appropriate blood pressure cuff or invasive monitoring
Heart disease, pulmonary arterial hypertension	Specific monitoring, right pulmonary catheterisation, heart ultrasound
Excessive fluid loading	Fluid restriction, ideal body weight calculation for fluid loading
Related to anaesthetic drugs	
Prolonged action of anaesthetic drugs (opioid+++)	Balanced anaesthesia, opioid sparing
Residual use of neuromuscular blockers	Fast-acting opioid
	Neuromuscular blockers based on ideal body weight
Pneumoperitoneum	
Cardiopulmonary repercussion	Intraperitoneal pressure < 15 mmHg
Perioperative renal dysfunction	No excessive fluid loading
Position, neuropathy	
	Appropriate material
	Checking points of support after mobilisation
	Training of the health care team
Thromboembolic risk	
	Mechanic means (compression stockings, intermittent compression++)
	Prophylactic anticoagulation
	Early postoperative mobilisation
Postoperative analgesia	
Opioid-induced respiratory depression	Loco-regional analgesia
	Continuous monitoring, pain assessment++
	No continuous flow if patient-controlled morphine pump
	Non-opioid analgesia
	Standardised protocols

NIV: non-invasive ventilation; PEEP: positive end-expiratory pressure.

Table 2
STOP-BANG score.

S	Snoring
T	Tired
O	Observed apnoea
P	Pressure (high blood pressure treated or untreated)
B	Body mass index > 35 kg/m^2
A	Age > 50 years old
N	Neck circumference > 40 cm
G	Gender male
STOP-BANG (one point by item): clinical diagnosis of obstructive apnoea syndrome if ≥ 3	

- clinical symptoms: the severity of daytime sleepiness is assessed on the impact it has on the activities of social or professional life: mild (low impact), moderate (moderate repercussion), severe (significant disruption);
- an AHI from 5 to 15 events/h define a mild OSA, 15 to 29 a moderate OSA and ≥ 30 a severe OSA [47].

Continuous positive airway pressure (CPAP) equipment is indicated in patients with severe OSA (AHI greater than 30 obstructive events/h). For lower IAH values, CPAP at night will be indicated in case of severe cardiovascular comorbidity. CPAP is proposed as first-line in these indications. In order to decrease daytime sleepiness, treatment may also be offered to patients with OSA with an AHI less than 30 and with no associated serious cardiovascular comorbidity. Mandibular Advancement Orthosis is recommended as first-line therapy for this indication.

Regardless of the association or not with an OSA, the obesity-hypoventilation syndrome is defined by:

- chronic alveolar hypoventilation: PaCO2 > 45 mmHg (PaO2 < 70 mmHg);
- obesity (BMI ≥ 30 kg/m^2);
- the absence of associated respiratory disease.

In the case of associated OSA and moderate hypercapnia between 45 and 55 mmHg, CPAP equipment will be offered as first-line, followed by non-invasive ventilation (NIV) equipment in case of failure, allowing ventilation at two pressure levels. In case of a history of respiratory decompensation with acute hypercapnic respiratory insufficiency, hypercapnia greater than 55 mmHg and/or in the absence of associated OSA, a NIV device will be proposed.

Obesity and Chronic Obstructive Pulmonary Disease (COPD) are often associated. Asthma is also more common in obese patients. In addition, obesity by itself can generate an obstructive syndrome [48]. The anaesthetist should be aware that a bronchospasm is likely to occur per and postoperatively. OSA and obesity, and a fortiori the combination of both, are risk factors for difficult intubation [12]. Any intubation in an obese patient should be considered potentially difficult, and adequate preparation following a difficult intubation algorithm should be mandatory.

3.1.1.1. Airway management. OSA associated with obesity is a risk factor for difficult ventilation [49]. Patients with a beard should ideally shave to decrease the risk of poor ventilation. Similarly, tracheal intubation is more difficult in patients with OSA, reaching 15 to 20% (*versus* 2 to 5% in the general population), especially if OSA is severe [50,51]. Obesity probably contributes to the difficulty of intubation in patients with OSA. A recent study [12] showed an increase in the incidence of difficult intubation in obese patients. Moreover, in this same study [12], a high Mallampati score, a reduction in cervical mobility and the presence of OSA have been associated with difficult intubation in obese patients, suggesting the potential involvement of specific anatomical factors in obese patients predisposing to difficult intubation [52]. Both difficult mask ventilation and difficult tracheal intubation are risk factors for hypoxemia at induction of general anaesthesia [53], suggesting that techniques improving preoxygenation should be implemented in daily practice in obese patients.

Preoxygenation in spontaneous ventilation with a face mask is followed by rapid desaturation after induction in obese patients, despite a high-inspired oxygen fraction (FiO2) (3 min on average, sometimes less than 1 min in case of very severe obesity) [21,54]. It has also been shown [55] that the lung volume at the end of expiration was reduced by 69% after anaesthetic induction in the supine position. Five minutes of preoxygenation in NIV (inspiratory support [AI] + positive expiratory pressure [PEP]) allowed to obtain an expired fraction of oxygen (FeO2) > 90% more rapidly [56]. In another study [57], the use of NIV limited the decrease of lung volume and improved oxygenation compared to a conventional preoxygenation with the face l mask.

High-Flow Nasal Cannula Oxygen Therapy (HFNO), which provides a high-flow rate, heated and humidified air through a nasal cannula at a 100% fraction of inspired oxygen (FiO2) and a maximum flow rate of 60 L/min, can allow the passage of the orotracheal tube through the mouth. In patients with acute hypoxemic respiratory failure, several mechanisms for improving oxygenation with HFNO have recently been identified. [58]: reduction of patient effort and minute ventilation required to achieve a physiological rate of arterial carbon dioxide, increase in end-expiratory lung volume, improvement of dynamic compliance, positive transpulmonary pressure and homogenisation of ventilation. Apnoea oxygenation is a physiological phenomenon in which the difference between the alveolar rates of oxygen removal and carbon dioxide excretion generates a negative pressure gradient of up to 20 cmH2O. This negative pressure gradient allows the entry of oxygen into the lungs, provided there is airway permeability between the lungs and the atmosphere. A first study [59] evaluated the influence of nasal oxygen administration on the duration of peripheral oxygen saturation (SpO2) ≥ 95% in simulated difficult laryngoscopy in obese patients. Nasal administration of O2 was associated with a significant increase in the frequency and duration of SpO2 ≥ 95% and a higher minimal SpO2 in prolonged laryngoscopy in obese patients. Another observational prospective study [60] focused on the apnoea time of 25 patients with difficult planned intubation and undergoing general anaesthesia for hypopharyngeal or laryngotracheal surgery. HFNO was administered continuously, for preoxygenation and continued after intravenous induction of anaesthesia and neuromuscular blockade, until airway safety was ensured. Twenty-five patients were included, including 12 obese patients. The median apnoea time was 14 (5–65) min. No arterial oxygen desaturation was observed during the apnoea period. However, apnoea oxygenation has to be differentiated from preoxygenation. If apnoea oxygenation using HFNO can help to decrease the occurrence of low oxygen saturation during the apnoea, HFNO should not be used as a method of preoxygenation in the obese patient. In a recent single centre study [61], preoxygenation with HFNO in obese patients provided lower end-tidal oxygen concentration (EtO2) after intubation and a higher rate of desaturation < 95%, compared to preoxygenation with NIV.

After prolonged preoxygenation, positive pressure and semi-sitting position [62,63], the anaesthesiologist has to decide whether to carry out a rapid sequence induction or not. This consists in the administration of a hypnotic and a neuromuscular blocker of rapid onset action, allowing to not ventilate the patient before intubation. In obese patients, gastroesophageal reflux is common, with hypotonia of the lower oesophageal sphincter [64]. Although bronchial inhalation during the induction of anaesthesia is rare, it is an important cause of anaesthesia-related mortality. Even if this induction technique is appropriate in obese

patients with symptomatic gastroesophageal reflux and/or full stomach, the benefit/risk balance of the rapid sequence induction should be evaluated in the absence of other risk factors for added inhalation. In an unscheduled situation, the rapid sequence induction will be almost systematic. Intubation awakened with a fiberscope should be considered if there is a very high risk of difficult intubation and mask ventilation [65]. The use of a videolaryngoscope is the method of choice in case of difficult intubation of obese patients [65,66], predicted (when the mouth opening is more than 2 cm to allow the videolaryngoscope introduction) or unpredicted. The insertion of a laryngeal mask is strongly recommended in case of difficult intubation and/or difficult mask ventilation [65].

3.1.2. Setting of ventilatory parameters

The French [20], multicentre, randomised, double blind IMPROVE study, compared an "optimised" ventilation strategy called "protective ventilation" (tidal volume TV 6–8 mL/kg IBW, PEEP 6–8 cmH2O, systematic alveolar recruitment manoeuvre every 30 min) to a traditional strategy called "non-protective ventilation" (TV 10–12 mL/kg IBW without PEEP or alveolar recruitment) in major abdominal surgery. Protective ventilation reduced the overall complication rate from 27.5 to 10.5% and reduced the length of hospital stay by 2 days. Patients with BMI $\geq$ 35 kg/m^2 were excluded. The results of this study nevertheless seem generalisable to any obese patient. In both obese and non-obese patients, the optimal TV seems to be between 6 and 8 mL/kg IBW by associating a PEP to avoid atelectasis by alveolar closure (derecruitment). The setting of the VT should be guided by the size of the patient and not by measured weight. The easiest IBW calculation formula to remember is: IBW (in kg) = height (in cm) – 100 in men and height (in cm) – 110 for women. However, a recent study [67] showed that obese patients were often ventilated with a very high TV (> 10 mL/kg) peroperatively.

Respiratory rate should be increased in obese patients. In fact, the obese patient has a hyperproduction of CO2, secondary to high oxygen consumption (VO2), associated with an increase in respiratory work [68,69]. The ventilation settings can therefore be guided by repeated arterial blood gases. Capnography has limitation in obese patients, because of a greater expired CO2/arterial CO2 gradient than in healthy subjects [19].

Obese patients are more sensitive to atelectasis and therefore to the absence of PEEP. In obese patients, gas exchange is improved by the application of a PEEP, as well as respiratory mechanics and alveolar recruitment (decrease of inspiratory resistance and improvement of compliance) [16,70].

However, when the alveoli have been collapsed, the isolated application of a PEEP alone will not reopen them, when recruitment manoeuvres will allow them to be reopened (recruitment). It is therefore recommended to apply at the beginning of the mechanical ventilation and during the whole ventilation period a PEEP of approximately 10 cmH2O, associated with a TV of 6 to 8 mL/kg of IBW [57,71] and regular recruitment manoeuvres [57]. Recruitment manoeuvre using bag squeezing should be avoided, as it was recently found to be associated with postoperative pulmonary complications [72].

However, all obese patients are not the same, and individual monitoring should be used whenever possible. In a recent study [73], an intraoperative mechanical ventilation strategy with a higher level of PEEP and alveolar recruitment manoeuvres, compared with a strategy with a lower level of PEEP, did not reduce postoperative pulmonary complications in non-selected obese patients. However, use of vasopressors was higher in the interventional group. Optimisation of fluid loading should always be checked before applying a strategy of "open lung". Oesophageal pressure monitoring can also help PEEP titration in obese patients, by setting the minimal PEEP allowing to obtain a positive transpulmonary pressure, in order to limit the occurrence of atelectases (PEEP level too low) or overdistension (PEEP level too high) [74]. Nevertheless, the haemodynamic effects of the application of high pressures (high PEEP and/or recruitment manoeuvres) should always be assessed: risk of decreased arterial oxygenation and hypotension due to repercussions on cardiac output [75]. Driving pressure (plateau pressure – PEEP) may not be an appropriated parameter in obese ARDS patients [76]. Further studies are needed in obese non-ARDS patients.

Some teams advocate the controlled pressure mode, as the decelerating flow would allow a better distribution of the airflow in the alveoli. However, the studies comparing the two ventilatory modes report contradictory data: the divergences can be explained by different criteria of judgment between the studies and methodological limits [77]. In practice, the advantages and disadvantages of each of both modes should be considered and the most appropriate ventilation mode should be used.

3.1.3. Extubation

If possible, extubation will be performed in a lateral or semi-sitting position, but not lying down. Extubation should be performed as soon as possible, avoiding prolonged mechanical ventilation [78]. If rocuronium was used during the surgery, antagonisation of rocuronium by suggamadex should be considered [79], as recently advised by the French recommendations about neuromuscular blockers use [80].

3.1.4. Postoperative oxygenation/ventilation

Oxygen supplementation should be administered continuously to all patients with OSA until they are able to maintain baseline oxygen saturation in ambient air. If possible, CPAP or NIV (with or without oxygen supplementation) can be used continuously in patients who use these modalities preoperatively, unless they are contraindicated by the surgical procedure. A randomised controlled trial [81] showed an improvement in ventilatory function when comparing postoperative CPAP to the absence of postoperative CPAP. Compliance with CPAP or NIV could be improved if patients bring their own equipment to the hospital. In the case of well-tolerated equipment and in the absence of other postoperative complications requiring monitoring in intensive care units, the patient may be admitted to a surgery ward. If possible, lying position should be avoided in patients with or at risk of OSA. In case of frequent and/or severe hypoxemia, initiation of CPAP or NIV sessions should be considered. The prophylactic application of a NIV after extubation could help to reduce the risk of acute respiratory failure by 16% and the length of stay. In addition, in obese hypercapnic patients [82], the use of postextubation NIV is associated with a decrease in mortality. In the case of major respiratory comorbidities, prophylactic NIV should be almost systematic in the recovery room and then in the intensive care unit.

Curative NIV can also be applied in case of acute respiratory failure to avoid intubation [83–85]. In obese patients with hypercapnia, higher levels of PEEP should be used over a longer period of time to reduce hypercapnia below 50 mmHg [14].

Postoperative HFNO has recently been investigated and compared with the use of postoperative NIV for cardiac and thoracic surgery, both preventive and curative, in a large population [86] including obese patients [87]. The non-inferiority of HFNO compared to the NIV was reached, suggesting effectiveness of postoperative HFNO therapy to prevent or avoid intubation.

3.1.5. Early rehabilitation

An improved rehabilitation protocol with early mobilisation after surgery, especially bariatric, is essential [88] to prevent respiratory complications. If possible, early removal of a urinary

catheter, infusions or other devices should be encouraged. Chest physiotherapy could be an important part of postoperative respiratory management improving respiratory functions, regulating arterial blood gases, increasing oxygen saturation, functional capacity and quality of life, and decreasing dyspnea levels [89].

3.2. Cardiovascular management

Morbidly obese patients often have very limited mobility and may be asymptomatic even when they have cardiovascular diseases. Physical examination is also difficult.

Obese patients should be evaluated in the same way as other patients, with a stress test, with appropriate equipment, in case of clinical suspicion of coronary heart disease.

Before the surgery, the preparation includes an adequate venous access, possibly using ultrasound guidance [90]. Monitoring of blood pressure can be particularly problematic in these patients. If the (blood pressure) cuff is too small, the blood pressure may be overestimated. The forearm can be used if the arm is too wide or cylindrical. In some cases, an arterial catheter may be necessary, and will also allow close monitoring of arterial blood gases.

Obese patients are particularly at risk for postoperative cardiovascular complications, including atrial fibrillation rhythm disorders [27]. Haemodynamic optimisation, especially avoiding overfilling or underfilling [91], by measurement of cardiac output by intra-oesophageal Doppler or transpulmonary thermodilution with monitoring of the pulse wave, could help to limit such perioperative complications.

Obesity is a risk factor for venous thromboembolism. Strategies to reduce the risk of venous thromboembolic complications include: early postoperative mobilisation, intermittent pneumatic compression, compression stockings and anticoagulant medications. Evidence of the effectiveness of simple compression stockings is limited. When used, it is essential that they be properly adjusted to prevent vascular occlusion. There is no argument in the literature for the routine use of vena cava filters in the obese population [92]. Oral anticoagulants such as rivaroxaban and dabigatran can be used for BMI ≤ 40 kg/m^2. Dose adjustment is not recommended at this time. In case of BMI > 40 kg/m^2, monitoring by dosage is recommended. When using low molecular weight heparins (LMWH), several protocols of adjustment of doses exist [91]. The Association of Anaesthetists of Great Britain and Ireland Society for Obesity and Bariatric Anaesthesia proposed the following recommendations [91]: If the patient is between 50 and 100 kg, 4000 IU/day of enoxaparine or equivalent could be used. If the patient is between 100 and 150 kg, 4000 IU twice a day of enoxaparine or equivalent could be considered, and if the patient is > 150 kg, 6000 IU twice a day of enoxaparine or equivalent could be used [91]. The European guidelines on perioperative venous thromboembolism prophylaxis for obese patient surgery [93] recommend low-dose low molecular weight heparin (3000 to 4000 anti-Xa IU/12 h subcutaneously) for obese patients with a lower risk of venous thromboembolism, and a higher dose of low molecular weight heparin (4000 to 6000 anti-Xa IU/12 h subcutaneously) for obese patients with a higher risk of venous thromboembolism. They underlined that higher doses of anticoagulants could be proposed for obese patients with a BMI more than 40 kg/m^2.

3.3. Metabolic-endocrine management

Screening for metabolic and nutritional abnormalities should be considered, especially in patients scheduled for bariatric surgery. The preoperative evaluation will focus on the presence of diabetes, anti-diabetic drugs and their possible perioperative relay. In general, these medications will not be stopped in case of outpatient surgery. In minor or major surgery, oral anti-diabetic medications will not be taken in the morning. Metformin will also not be taken the night before [94]. Vitamin and nutritional deficits can lead to postoperative polyneuropathy, known as acute neuropathy postgastric reduction, a nutritional disease characterised by postoperative vomiting, hyporeflexia and muscle weakness [95]. Special attention to the dosing and monitoring of the neuromuscular blockers is recommended in case of suspicion or diagnosis of neuropathy. Chronic vitamin K deficiency, in case of previous obesity surgery, may lead to coagulation abnormalities, necessitating the administration of vitamin K or fresh frozen plasma.

3.4. Gastro-intestinal management

The frequency of gastroesophageal reflux is strongly correlated with the increase in BMI [96]. Obesity is also associated with increased intra-abdominal pressure, gastric volumes, hiatal hernia, as well as decreased pH and fatty infiltration of the liver. An antacid premedication can be used before the procedure.

Table 3
Dosages adjustments of some medications.

Weight	Definition	Antibiotics (initial dosing)	Others
Total body weight (real weight)	Real weight of the patient	Beta-lactam[a]	Succinylcholine
		Vancomycin (loading dose) Daptomycin (monitor CPK)	
Ideal body weight	Lorentz formula. Men: ideal body weight (kg) = height (cm) – 100 – ((Height (cm) – 150)/4). Women: ideal body weight (kg) = Height (cm) – 100 – ((Height (cm) – 150)/2,5); = about height (cm) – 100 for men, height (cm) – 110 for women	Linezolid (standard doses 600 mg*2/j, continuous infusion may be useful, increasing doses according to CMI)	Neuromuscular blockers
		Macrolides	Opioids
Adjusted weight (closed to lean body weight)	Takes into account the increase in lean mass and in volume of distribution: ideal body weight + 0,4 (total body weight – ideal body weight)	Beta-lactam[a]	Lidocaine
		Vancomycin (maintenance dose) Aminoglycoside Fluoroquinolones	Ketamine Propofol

CPK: creatinine phosphokinase. This table is given as an indication and remains to be completed and modified according to the practices of each centre and the new results reported in the literature. In all cases, during prolonged treatments, a dosage is recommended in order to better adapt the doses administered.

[a] The dose of beta-lactam will depend on the minimum inhibitory concentration (MIC) and toxicity. For amoxicillin and oxacillin, adjusted weight or actual weight may be used. For piperacillin-tazobactam, we can reach 20 to 24 g/24 h [115]. The doses of imipenem should theoretically be increased but the risk of toxicity including neurological will be increased, so it is better not to exceed 3 g/d, despite the risk of underdosing. For meropenem, the maximum dose of 2 g/8 h can be used. In prophylaxis, in patients over 100 kg and/or body mass index > 35 kg/m^2, even outside bariatric surgery, doses of beta-lactam should be twice those recommended for non-obese patients.

Patients with adjustable gastric rings (rings placed around the upper part of the stomach, defining a small pocket) have peculiarities. Indeed, these patients are at high risk of pulmonary inhalation during the induction of general anaesthesia due to a disorder of oesophageal motility and dilation above the ring. A rapid sequence induction should thus be used in this patient, and sedation without intubation in endoscopic procedures (gastroscopy, colonoscopy) should be avoided.

3.5. Drug management

3.5.1. Antibioprophylaxis and antibiotherapy

The latest French Society of Anaesthesia and Intensive Care Medicine (SFAR) expert recommendations on perioperative antibiotic prophylaxis [97] have defined the antibiotic prophylaxis of the obese patient. In obese patients (patients over 100 kg and body mass index > 35 kg/m^2), doses of beta-lactams should be twice those recommended for non-obese patients. For vancomycin and gentamicin, doses of antibiotic prophylaxis are calculated on the actual weight.

When curative antibiotic therapy is required, the initial dosage will most often be based on actual weight or adjusted weight. The dose of aminoglycosides will be calculated on the adjusted weight. Then, repeated dosing is needed to adjust the dose of antibiotics performed. Table 3 presents the recommended dose adjustments for some antibiotics.

3.5.2. Choice of anaesthetic technique

Loco-regional anaesthesia should always be preferred over general anaesthesia if possible. If general anaesthesia is required, the use of easily reversible and fast-acting drugs is the agent of choice for induction in obese patients.

There is little pharmacological data on anaesthetic drugs used routinely in obese patients (Table 3). Fortunately, many anaesthetic agents can be titrated.

The fasciculation associated with suxamethonium increase the consumption of oxygen and have shown a reduction in apnoea time without desaturation [98]. A dose of rocuronium based on total weight does not significantly shorten the duration of action, but significantly increases the duration of actionit [99]. The IBW is therefore recommended to adapt the doses of rocuronium. On the other hand, due to an increase in plasma cholinesterase activity, the total weight is appropriate for suxamethonium. The doses of neostigmine and suggamadex are related to the time and total dose of injected curare and will be titrated on efficacy. After rocuronium administration, suggamadex may provide faster recovery of neuromuscular function and prevent more postoperative residual curarisation in the morbidly obese patient compared to neostigmine [100]. Sugammadex is a hydrophilic molecule that distributes mainly in extracellular fluids and its effects should be only slightly affected by the volume of body fat [91,101]. Therefore, its dosage in morbidly obese patients should be based on weight lower than total body weight [91,102]. The recent recommendations are to use ideal body weight for dosing sugammadex [102] and then to monitor degree of neuromuscular blockade [80].

For maintenance of anaesthesia, short-acting anaesthetic agents should be used. Maintaining anaesthesia with propofol or halogenated agents should be initiated early after induction because of the increased risk of intraoperative awakening in the obese patient and thus intraoperative memory [103]. Airway reflexes are more quickly restored in case of desflurane anaesthesia compared to sevoflurane anaesthesia in obese patients [104].

Obese patients are very sensitive to the respiratory depressant effects of sedatives, opioids and inhaled anaesthetics. Benzodiazepines are to be avoided in these patients, even in premedication, because of their long half-life.

For opioids, the clinical effect is little related to plasma concentration [105]. The dose using the ideal weight is a starting point until the patient is awake and titration to effect is possible. For propofol, the Marsh and Schneider model is no longer reliable for patients over 140–150 kg [99]. This is why the pumps do not allow to enter weights > 150 kg using the Marsh model, or a BMI > 35 kg/m^2 (female) and 42 kg/m^2 (male) using the Schneider model.

3.6. Screening of bariatric complications

A rare but serious complication in the obese patient is rhabdomyolysis [106]. Predisposing risk factors, in addition to obesity, are: hypotension, immobility, long duration of surgery and insufficient fluid loading [107]. Rhabdomyolysis should be suspected in cases of deep postoperative pain, typically at the gluteal level. Blood creatinine phosphokinase (CPK) levels should be measured rapidly, and if increased, aggressive management may be required to prevent worsening of renal function [106].

Postoperative tachycardia may be the only sign of a postoperative surgical complication and should not be overlooked. The bleeding from the staple line and the early gastric fistula are the main complications of bariatric surgery.

3.6.1.1. Postoperative analgesia

Loco-regional anaesthesia techniques should be considered to reduce or eliminate the use of opioids in obese patients and therefore reduce postoperative complications. If patient-controlled opioid analgesia is used, continuous flow should be avoided or used with extreme caution. The administration of opioids should be all the more cautious that the obese subject may have an undiagnosed OSA. Oxygen administration should be continued in the ward until the patient regains his baseline SpO2 and no longer requires the use of opioids.

To reduce the use of opioids, non-steroidal anti-inflammatory agents or non-drug analgesia may be considered [108]. Different alternatives could reduce opioids use (Opioids Free Anaesthesia [OFA], ketamine and lidocaine infusion), as defined in the Enhanced Recovery After Bariatric Surgery (ERABS) Protocol [88]. Even if some observational studies showed encouraging results [88,108,109] with less opioid use, results of large randomised controlled trials are waited before concluding that such protocols are safe and efficient. It should be kept in mind that the concomitant administration of sedative agents (e.g. benzodiazepines, barbiturates) increases the risk of respiratory depression and airway obstruction.

4. Criteria for outpatient surgery or planned postoperative intensive care unit admissions

The recommendations of the French Health Authority (HAS) about outpatient surgery made in 2014 [110] state:

- in the obese patient: patients with a BMI < 35 kg/m^2 are eligible for outpatient surgery, except individual contra-indications; patients with a BMI > 40 kg/m^2 and an OSA associated, and all the more if it is severe and associated with other comorbidities (pulmonary hypertension, resistant hypertension, major damage to the coronary arteries, resistant heart failure treatment) do not seem eligible for outpatient surgery;
- in the patient with OSA: patients diagnosed with OSA are eligible for outpatient surgery if they have controlled comorbidities, if they are able to use nasal ventilation with CPAP or NIV and if surgery is not painful and does not require the use of opioids postoperatively. In the case of a choice of outpatient care, the intervention could be scheduled in the morning to allow monitoring the rest of the day.

After bariatric surgery, due to the high risk of postoperative bleeding the day following the surgery, benefit/risk of outpatient management should be carefully assessed [7]. Highly trained team have performed laparoscopic sleeve gastrectomy in a daily case manner without increasing the rate of complications [111,112]. It is worth noting that the rate of haemorrhage is greater in the gastric bypass group than in the laparoscopic sleeve gastrectomy group [113].

In case of moderate to severe OSA without preoperative CPAP, or in case of other premorbid comorbidities (mainly respiratory or cardiovascular, confer the chapter "the prevention of perioperative complications") [114], or in case of major surgery at high risk of postoperative complications [7], the patient will be postoperatively admitted to the intensive care unit (ICU).

5. Conclusion

Postoperative complications of the obese patient can be reduced by screening for OSA, involving continuation or initiation of preoperative CPAP or NIV, use of loco-regional anaesthesia rather than general anaesthesia when possible, anticipation of difficult mask ventilation and difficult intubation, reduction of hypnotics and morphine doses and perioperative protective ventilation. This is based on NIV preoxygenation in a semi-sitting position, small TV, moderate PEEP and careful intraoperative recruitment manoeuvres. The optimisation of pre- and peroperative management will be continued postoperatively, with extubation in a semi-seated position, prevention of thromboembolic complications and continuation or early onset of CPAP or NIV, prophylactically or in case of the occurrence of acute respiratory insufficiency.

Disclosure of interest

Pr. Jaber reports receiving consulting fees from Drager, Hamilton, Maquet, Medtronic, and Fisher & Paykel.

Dr De Jong reports personal fees from Baxter and Medtronic-Covidien, and travel reimbursements from Fresenius-Kabi, MSD France, Astellas, and Pfizer and Fisher Paykel.

Pr Chanques has consulted for and received honorarium from Orion Pharma, and Aspen Medical France.

Dr. Futier reports consulting fees from Edwards Lifesciences, Drager, GE Heathcare, and Orion Pharma; and lecture fees from Fresenius-Kabi, Fisher and Paykel Healthcare, and Baxter.

The other authors declare that they have no competing interest.

Funding

This work did not receive any grant from funding agencies in the public, commercial, or not-for-profit sectors.

Author contributions

All authors attest that they meet the current International Committee of Medical Journal Editors (ICMJE) criteria for Authorship.

References

[1] NCD-Risk. Trends in adult body-mass index in 200 countries from 1975 to 2014: a pooled analysis of 1698 population-based measurement studies with 19.2 million participants. Lancet 2016;387(10026):1377–96.
[2] Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organ Tech Rep Ser 2000;894:1–253 [i–xii, PMID: 11234459].
[3] Dapri G, Cadiere GB, Himpens J. Superobese and super-superobese patients: 2-step laparoscopic duodenal switch. Surg Obes Relat Dis 2011;7(6):703–8.
[4] Kakarla VR, Nandipati K, Lalla M, Castro A, Merola S. Are laparoscopic bariatric procedures safe in superobese (BMI >= 50 kg/m^2) patients? An NSQIP data analysis. Surg Obes Relat Dis 2011;7(4):452–8.
[5] Leykin Y, Pellis T, Del Mestro E, Marzano B, Fanti G, Brodsky JB. Anesthetic management of morbidly obese and super-morbidly obese patients undergoing bariatric operations: hospital course and outcomes. Obes Surg 2006;16(12):1563–9.
[6] Bazurro S, Ball L, Pelosi P. Perioperative management of obese patient. Curr Opin Crit Care 2018;24(6):560–7.
[7] De Jong A, Verzilli D, Geniez M, Chanques G, Nocca D, Jaber S. Pourquoi le patient obèse morbide est-il un patient à risque anesthésique élevé ? Presse Med 2018;47(5):453–63.
[8] Canet J, Sabate S, Mazo V, Gallart L, de Abreu MG, Belda J, et al. Development and validation of a score to predict postoperative respiratory failure in a multicentre European cohort: a prospective, observational study. Eur J Anaesthesiol 2015;32(7):458–70.
[9] Russotto V, Sabate S, Canet J. Development of a prediction model for postoperative pneumonia: a multicentre prospective observational study. Eur J Anaesthesiol 2019;36(2):93–104.
[10] Dupree A, El Gammal AT, Wolter S, Urbanek S, Sauer N, Mann O, et al. Perioperative short-term outcome in super-super-obese patients undergoing bariatric surgery. Obes Surg 2018;28(7):1895–901.
[11] Peterson GN, Domino KB, Caplan RA, Posner KL, Lee LA, Cheney FW. Management of the difficult airway: a closed claims analysis. Anesthesiology 2005;103(1):33–9.
[12] De Jong A, Molinari N, Pouzeratte Y, Verzilli D, Chanques G, Jung B, et al. Difficult intubation in obese patients: incidence, risk factors, and complications in the operating theatre and in intensive care units. Br J Anaesth 2015;114(2):297–306.
[13] Lee LA, Caplan RA, Stephens LS, Posner KL, Terman GW, Voepel-Lewis T, et al. Postoperative opioid-induced respiratory depression: a closed claims analysis. Anesthesiology 2015;122(3):659–65.
[14] De Jong A, Verzilli D, Jaber S. ARDS in obese patients: specificities and management. Crit Care 2019;23(1):74.
[15] Schetz M, De Jong A, Deane AM, Druml W, Hemelaar P, Pelosi P, et al. Obesity in the critically ill: a narrative review. Intensive Care Med 2019;45(6):757–69.
[16] Pelosi P, Croci M, Ravagnan I, Cerisara M, Vicardi P, Lissoni A, et al. Respiratory system mechanics in sedated, paralyzed, morbidly obese patients. J Appl Physiol Respir Environ Exerc Physiol 1997;82(3):811–8.
[17] Hedenstierna GLA. Influence of abdominal pressure on respiratory and abdominal organ function. Curr Opin Crit Care 2012;18(1):80–5.
[18] Kress JP, Pohlman AS, Alverdy J, Hall JB. The impact of morbid obesity on oxygen cost of breathing (VO[2RESP]) at rest. Am J Respir Crit Care Med 1999;160(3):883–6.
[19] De Jong A, Chanques G, Jaber S. Mechanical ventilation in obese ICU patients: from intubation to extubation. Crit Care 2017;21(1):63.
[20] Futier E, Constantin JM, Paugam-Burtz C, Pascal J, Eurin M, Neuschwander A, et al. A trial of intraoperative low-tidal-volume ventilation in abdominal surgery. N Engl J Med 2013;369(5):428–37.
[21] De Jong A, Futier E, Millot A, Coisel Y, Jung B, Chanques G, et al. How to preoxygenate in operative room: healthy subjects and situations "at risk". Ann Fr Anesth Reanim 2014;33(8):457–61.
[22] Romero-Corral A, Caples SM, Lopez-Jimenez F, Somers VK. Interactions between obesity and obstructive sleep apnea: implications for treatment. Chest 2010;137(3):711–9.
[23] Reinius H, Jonsson L, Gustafsson S, Sundbom M, Duvernoy O, Pelosi P, et al. Prevention of atelectasis in morbidly obese patients during general anesthesia and paralysis: a computerized tomography study. Anesthesiology 2009;111(5):979–87.
[24] De Jong A, Molinari N, Sebbane M, Prades A, Futier E, Jung B, et al. Feasibility and effectiveness of prone position in morbidly obese patients with ARDS: a case-control clinical study. Chest 2013;143(6):1554–61.
[25] De Jong A, Verzilli D, Sebbane M, Monnin M, Belafia F, Cisse M, et al. Medical versus surgical ICU obese patient outcome: a propensity-matched analysis to resolve clinical trial controversies. Crit Care Med 2018;46(4):e294–301.
[26] Hernandez AF, Whellan DJ, Stroud S, Sun JL, O'Connor CM, Jollis JG. Outcomes in heart failure patients after major noncardiac surgery. J Am Coll Cardiol 2004;44(7):1446–53.
[27] Peeters A, Barendregt JJ, Willekens F, Mackenbach JP, Al Mamun A, Bonneux L. Obesity in adulthood and its consequences for life expectancy: a life-table analysis. Ann Intern Med 2003;138(1):24–32.
[28] Lavie CJ, Milani RV, Ventura HO. Obesity and cardiovascular disease: risk factor, paradox, and impact of weight loss. J Am Coll Cardiol 2009;53(21):1925–32.
[29] Blokhin IO, Lentz SR. Mechanisms of thrombosis in obesity. Curr Opin Hematol 2013;20(5):437–44.
[30] Parkin L, Sweetland S, Balkwill A, Green J, Reeves G, Beral V. Body mass index, surgery, and risk of venous thromboembolism in middle-aged women: a cohort study. Circulation 2012;125(15):1897–904.
[31] Magee CJ, Barry J, Javed S, Macadam R, Kerrigan D. Extended thromboprophylaxis reduces incidence of postoperative venous thromboembolism in laparoscopic bariatric surgery. Surg Obes Relat Dis 2010;6(3):322–5.
[32] Glance LG, Wissler R, Mukamel DB, Li Y, Diachun CA, Salloum R, et al. Perioperative outcomes among patients with the modified metabolic syndrome who are undergoing noncardiac surgery. Anesthesiology 2010;113(4):859–72.
[33] Tasali E, Ip MS. Obstructive sleep apnea and metabolic syndrome: alterations in glucose metabolism and inflammation. Proc Am Thorac Soc 2008;5(2):207–17.
[34] Frisch A, Chandra P, Smiley D, Peng L, Rizzo M, Gatcliffe C, et al. Prevalence and clinical outcome of hyperglycemia in the perioperative period in

noncardiac surgery. Diab Care 2010;33(8):1783–8.
[35] Guidone C, Manco M, Valera-Mora E, Iaconelli A, Gniuli D, Mari A, et al. Mechanisms of recovery from type 2 diabetes after malabsorptive bariatric surgery. Diabetes 2006;55(7):2025–31.
[36] Edholm D, Kullberg J, Haenni A, Karlsson FA, Ahlstrom A, Hedberg J, et al. Preoperative 4-week low-calorie diet reduces liver volume and intrahepatic fat, and facilitates laparoscopic gastric bypass in morbidly obese. Obes Surg 2011;21(3):345–50.
[37] Société française d'anesthésie-réanimation. Recommandations sur la prise en charge du tabagisme en période péri-opératoire; 2016.
[38] Gupta RM, Parvizi J, Hanssen AD, Gay PC. Postoperative complications in patients with obstructive sleep apnea syndrome undergoing hip or knee replacement: a case-control study. Mayo Clinic Proc 2001;76(9):897–905.
[39] Kaw R, Pasupuleti V, Walker E, Ramaswamy A, Foldvary-Schafer N. Postoperative complications in patients with obstructive sleep apnea. Chest 2012;141(2):436–41.
[40] Lavie P, Herer P, Hoffstein V. Obstructive sleep apnoea syndrome as a risk factor for hypertension: population study. BMJ 2000;320(7233):479–82.
[41] Dyken ME, Somers VK, Yamada T, Ren ZY, Zimmerman MB. Investigating the relationship between stroke and obstructive sleep apnea. Stroke 1996;27(3):401–7.
[42] Hung J, Whitford EG, Parsons RW, Hillman DR. Association of sleep apnoea with myocardial infarction in men. Lancet 1990;336(8710):261–4.
[43] Katsumata K, Okada T, Miyao M, Katsumata Y. High incidence of sleep apnea syndrome in a male diabetic population. Diab Res Clin Pract 1991;13(2):45–51.
[44] Guilleminault C, Li K, Chen NH, Poyares D. Two-point palatal discrimination in patients with upper airway resistance syndrome, obstructive sleep apnea syndrome, and normal control subjects. Chest 2002;122(3):866–70.
[45] Rosenthal LD, Dolan DC. The Epworth sleepiness scale in the identification of obstructive sleep apnea. J Nerv Ment Dis 2008;196(5):429–31.
[46] Chung F, Yang Y, Liao P. Predictive performance of the STOP-Bang score for identifying obstructive sleep apnea in obese patients. Obes Surg 2013;23(12):2050–7.
[47] Bibbins-Domingo K, Grossman DC, Curry SJ, Davidson KW, Epling Jr JW, Garcia FA, et al. Screening for obstructive sleep apnea in adults: US preventive services task force recommendation statement. JAMA 2017;317(4):407–14.
[48] Ramirez-Molina VR, Gomez-de-Terreros FJ, Barca-Duran J, Masa JF. Non-invasive positive airway pressure in obesity hypoventilation syndrome and chronic obstructive pulmonary disease: present and future perspectives. COPD 2017;14(4):418–28.
[49] Langeron O, Masso E, Huraux C, Guggiari M, Bianchi A, Coriat P, et al. Prediction of difficult mask ventilation. Anesthesiology 2000;92(5):1229–36.
[50] Siyam MA, Benhamou D. Difficult endotracheal intubation in patients with sleep apnea syndrome. Anesth Analg 2002;95(4):1098–102 [Table of contents].
[51] Kim JA, Lee JJ. Preoperative predictors of difficult intubation in patients with obstructive sleep apnea syndrome. Can J Anaesth 2006;53(4):393–7.
[52] Brodsky JB, Lemmens HJM, Brock-Utne JG, Vierra M, Saidman LJ. Morbid obesity and tracheal intubation. Anesth Analg 2002;94(3):732–6.
[53] Baillard C, Boubaya M, Statescu E, Collet M, Solis A, Guezennec J, et al. Incidence and risk factors of hypoxaemia after preoxygenation at induction of anaesthesia. Br J Anaesth 2019;122(3):388–94.
[54] Baraka AS, Taha SK, El-Khatib MF, Massouh FM, Jabbour DG, Alameddine MM. Oxygenation using tidal volume breathing after maximal exhalation. Anesth Analg 2003;97(5):1533–5.
[55] Futier E, Constantin JM, Petit A, Jung B, Kwiatkowski F, Duclos M, et al. Positive end-expiratory pressure improves end-expiratory lung volume but not oxygenation after induction of anaesthesia. Eur J Anaesthesiol 2010;27(6):508–13.
[56] Delay JM, Sebbane M, Jung B, Nocca D, Verzilli D, Pouzeratte Y, et al. The effectiveness of noninvasive positive pressure ventilation to enhance preoxygenation in morbidly obese patients: a randomized controlled study. Anesth Analg 2008;107(5):1707–13.
[57] Futier E, Constantin JM, Pelosi P, Chanques G, Massone A, Petit A, et al. Noninvasive ventilation and alveolar recruitment maneuver improve respiratory function during and after intubation of morbidly obese patients: a randomized controlled study. Anesthesiology 2011;114(6):1354–63.
[58] Mauri T, Turrini C, Eronia N, Grasselli G, Volta CA, Bellani G, et al. Physiologic effects of high-flow nasal cannula in acute hypoxemic respiratory failure. Am J Respir Crit Care Med 2017;195(9):1207–15.
[59] Ramachandran SK, Cosnowski A, Shanks A, Turner CR. Apneic oxygenation during prolonged laryngoscopy in obese patients: a randomized, controlled trial of nasal oxygen administration. J Clin Anesth 2010;22(3):164–8.
[60] Patel A, Nouraei SA. Transnasal humidified rapid-insufflation ventilatory exchange (THRIVE): a physiological method of increasing apnoea time in patients with difficult airways. Anaesthesia 2015;70(3):323–9.
[61] Vourc'h M, Baud G, Feuillet F, Blanchard C, Mirallie E, Guitton C, et al. High-flow nasal cannulae versus non-invasive ventilation for preoxygenation of obese patients: the PREOPTIPOP randomized trial. E Clin Med 2019;13:112–9.
[62] Dixon BJ, Dixon JB, Carden JR, Burn AJ, Schachter LM, Playfair JM, et al. Preoxygenation is more effective in the 25 degrees head-up position than in the supine position in severely obese patients: a randomized controlled study. Anesthesiology 2005;102(6):1110–5 [Discussion 5A].
[63] Altermatt FR, Muñoz HR, Delfino AE, Cortínez LI. Pre-oxygenation in the obese patient: effects of position on tolerance to apnoea. Br J Anaesth 2005;95(5):706–9.
[64] Sabate JM, Jouet P, Merrouche M, Pouzoulet J, Maillard D, Harnois F, et al. Gastroesophageal reflux in patients with morbid obesity: a role of obstructive sleep apnea syndrome? Obes Surg 2008;18(11):1479–84.
[65] Langeron O, Bourgain JL, Francon D, Amour J, Baillard C, Bouroche G, et al. Difficult intubation and extubation in adult anaesthesia. Anaesth Crit Care Pain Med 2018;37(6):639–51.
[66] Andersen LH, Rovsing L, Olsen KS. GlideScope videolaryngoscope vs. Macintosh direct laryngoscope for intubation of morbidly obese patients: a randomized trial. Acta Anaesthesiol Scand 2011;55(9):1090–7.
[67] Jaber S, Coisel Y, Chanques G, Futier E, Constantin JM, Michelet P, et al. A multicentre observational study of intra-operative ventilatory management during general anaesthesia: tidal volumes and relation to body weight. Anaesthesia 2012;67(9):999–1008.
[68] Malhotra A, Hillman D. Obesity and the lung: 3. Obesity, respiration and intensive care. Thorax 2008;63(10):925–31.
[69] Pepin Jl BJ, Janssens JP. Obesity hypoventilation syndrome: an underdiagnosed and undertreated condition. Am J Respir Crit Care Med 2012;186(12):1205–7.
[70] Pelosi P, Croci M, Ravagnan I, Tredici S, Pedoto A, Lissoni A, et al. The effects of body mass on lung volumes, respiratory mechanics, and gas exchange during general anesthesia. Anesth Analg 1998;87(3):654–60.
[71] Talab HF, Zabani IA, Abdelrahman HS, Bukhari WL, Mamoun I, Ashour MA, et al. Intraoperative ventilatory strategies for prevention of pulmonary atelectasis in obese patients undergoing laparoscopic bariatric surgery. Anesth Analg 2009;109(5):1511–6.
[72] Ball L, Hemmes SNT, Serpa Neto A, Bluth T, Canet J, Hiesmayr M, et al. Intraoperative ventilation settings and their associations with postoperative pulmonary complications in obese patients. Br J Anaesth 2018;121(4):899–908.
[73] Bluth T, Serpa Neto A, Schultz MJ, Pelosi P, Gama de Abreu M. Effect of intraoperative high positive end-expiratory pressure (PEEP) with recruitment maneuvers vs low peep on postoperative pulmonary complications in obese patients: a randomized clinical trial. JAMA 2019;321(23):2292–305.
[74] Eichler L, Truskowska K, Dupree A, Busch P, Goetz AE, Zollner C. Intraoperative ventilation of morbidly obese patients guided by transpulmonary pressure. Obes Surg 2018;28(1):122–9.
[75] Nestler C, Simon P, Petroff D, Hammermuller S, Kamrath D, Wolf S, et al. Individualized positive end-expiratory pressure in obese patients during general anaesthesia: a randomized controlled clinical trial using electrical impedance tomography. Br J Anaesth 2017;119(6):1194–205.
[76] De Jong A, Cossic J, Verzilli D, Monet C, Carr J, Conseil M, et al. Impact of the driving pressure on mortality in obese and non-obese ARDS patients: a retrospective study of 362 cases. Intensive Care Med 2018;44(7):1106–14.
[77] Aldenkortt M, Lysakowski C, Elia N, Brochard L, Tramer MR. Ventilation strategies in obese patients undergoing surgery: a quantitative systematic review and meta-analysis. Br J Anaesth 2012;109(4):493–502.
[78] Jaber S, Petrof BJ, Jung B, Chanques G, Berthet J-P, Rabuel C, et al. Rapidly progressive diaphragmatic weakness and injury during mechanical ventilation in humans. Am J Respir Crit Care Med 2011;183(3):364–71.
[79] Suzuki T, Masaki G, Ogawa S. Neostigmine-induced reversal of vecuronium in normal weight, overweight and obese female patients. Br J Anaesth 2006;97(2):160–3.
[80] Société française d'anesthésie-réanimation (SFAR). Curarisation et décurarisation en anesthésie; 2018.
[81] Neligan PJ, Malhotra G, Fraser M, Williams N, Greenblatt EP, Cereda M, et al. Continuous positive airway pressure via the Boussignac system immediately after extubation improves lung function in morbidly obese patients with obstructive sleep apnea undergoing laparoscopic bariatric surgery. Anesthesiology 2009;110(4):878–84.
[82] Jaber S, De Jong A, Castagnoli A, Futier E, Chanques G. Non-invasive ventilation after surgery. Ann Fr Anesth Reanim 2014;33(8):487–91.
[83] Azoulay E, Kouatchet A, Jaber S, Lambert J, Meziani F, Schmidt M, et al. Noninvasive mechanical ventilation in patients having declined tracheal intubation. Intensive Care Med 2013;39(2):292–301.
[84] Jaber S, Michelet P, Chanques G. Role of non-invasive ventilation (NIV) in the perioperative period. Best Pract Res Clin Anaesthesiol 2010;24(2):253–65.
[85] Jaber S, Chanques G, Jung B. Postoperative noninvasive ventilation. Anesthesiology 2010;112(2):453–61.
[86] Stephan F, Barrucand B, Petit P, Rezaiguia-Delclaux S, Medard A, Delannoy B, et al. High-flow nasal oxygen vs noninvasive positive airway pressure in hypoxemic patients after cardiothoracic surgery: a randomized clinical trial. JAMA 2015;313(23):2331–9.
[87] Stephan F, Berard L, Rezaiguia-Delclaux S, Amaru P. High-flow nasal cannula therapy versus intermittent noninvasive ventilation in obese subjects after cardiothoracic surgery. Respir Care 2017;62(9):1193–202.
[88] Awad S, Carter S, Purkayastha S, Hakky S, Moorthy K, Cousins J, et al. Enhanced recovery after bariatric surgery (ERABS): clinical outcomes from a tertiary referral bariatric centre. Obes Surg 2014;24(5):753–8.
[89] Duymaz T, Karabay O, Ural IH. The effect of chest physiotherapy after bariatric surgery on pulmonary functions, functional capacity, and quality of life. Obes Surg 2019;30(1):189–94.
[90] Ueda K, Hussey P. Dynamic ultrasound-guided short-axis needle tip navigation technique for facilitating cannulation of peripheral veins in obese patients. Anesth Analg 2017;124(3):831–3.
[91] Nightingale CE, Margarson MP, Shearer E, Redman JW, Lucas DN, Cousins JM, et al. Peri-operative management of the obese surgical patient 2015: Association of Anaesthetists of Great Britain and Ireland Society for Obesity and Bariatric Anaesthesia. Anaesthesia 2015;70(7):859–76.

[92] Rowland SP, Dharmarajah B, Moore HM, Lane TR, Cousins J, Ahmed AR, et al. Inferior vena cava filters for prevention of venous thromboembolism in obese patients undergoing bariatric surgery: a systematic review. Ann Surg 2015;261(1):35–45.
[93] Venclauskas L, Maleckas A, Arcelus JI. European guidelines on perioperative venous thromboembolism prophylaxis: surgery in the obese patient. Eur J Anaesthesiol 2018;35(2):147–53.
[94] Société française de diabétologie et d'anesthésie-réanimation. Fiches pratiques de prise en charge du patient diabétique en périopératoire. Elsevier Masson; 2017.
[95] Chang CG, Adams-Huet B, Provost DA. Acute post-gastric reduction surgery (APGARS) neuropathy. Obes Surg 2004;14(2):182–9.
[96] Jacobson BC, Somers SC, Fuchs CS, Kelly CP, Camargo Jr CA. Body-mass index and symptoms of gastroesophageal reflux in women. N Engl J Med 2006;354(22):2340–8.
[97] Martin C, Auboyer C, Boisson M, Dupont H, Gauzit R, Kitzis M, et al. Antibioprophylaxis in surgery and interventional medicine (adult patients) update 2017. Anaesth Crit Care Pain Med 2019;38(5):549–62.
[98] Hennis PJ, Meale PM, Hurst RA, O'Doherty AF, Otto J, Kuper M, et al. Cardiopulmonary exercise testing predicts postoperative outcome in patients undergoing gastric bypass surgery. Br J Anaesth 2012;109(4):566–71.
[99] Leykin Y, Pellis T, Lucca M, Lomangino G, Marzano B, Gullo A. The pharmacodynamic effects of rocuronium when dosed according to real body weight or ideal body weight in morbidly obese patients. Anesth Analg 2004;99(4):1086–9 [Table of contents].
[100] Gaszynski T, Szewczyk T, Gaszynski W. Randomized comparison of sugammadex and neostigmine for reversal of rocuronium-induced muscle relaxation in morbidly obese undergoing general anaesthesia. Br J Anaesth 2012;108(2):236–9.
[101] Gijsenbergh F, Ramael S, Houwing N, van Iersel T. First human exposure of Org 25969, a novel agent to reverse the action of rocuronium bromide. Anesthesiology 2005;103(4):695–703.
[102] Van Lancker P, Dillemans B, Bogaert T, Mulier JP, De Kock M, Haspeslagh M. Ideal versus corrected body weight for dosage of sugammadex in morbidly obese patients. Anaesthesia 2011;66(8):721–5.
[103] Pandit JJ, Andrade J, Bogod DG, Hitchman JM, Jonker WR, Lucas N, et al. 5th National Audit Project (NAP5) on accidental awareness during general anaesthesia: summary of main findings and risk factors. Br J Anaesth 2014;113(4):549–59.
[104] McKay RE, Malhotra A, Cakmakkaya OS, Hall KT, McKay WR, Apfel CC. Effect of increased body mass index and anaesthetic duration on recovery of protective airway reflexes after sevoflurane vs desflurane. Br J Anaesth 2010;104(2):175–82.
[105] Hammoud HA, Aymard G, Lechat P, Boccheciampe N, Riou B, Aubrun F. Relationships between plasma concentrations of morphine, morphine-3-glucuronide, morphine-6-glucuronide, and intravenous morphine titration outcomes in the postoperative period. Fundam Clin Pharmacol 2011;25(4):518–27.
[106] Wool DB, Lemmens HJ, Brodsky JB, Solomon H, Chong KP, Morton JM. Intraoperative fluid replacement and postoperative creatine phosphokinase levels in laparoscopic bariatric patients. Obes Surg 2010;20(6):698–701.
[107] Chavez LO, Leon M, Einav S, Varon J. Beyond muscle destruction: a systematic review of rhabdomyolysis for clinical practice. Crit Care 2016;20(1):135.
[108] King AB, Spann MD, Jablonski P, Wanderer JP, Sandberg WS, McEvoy MD. An enhanced recovery program for bariatric surgical patients significantly reduces perioperative opioid consumption and postoperative nausea. Surg Obes Relat Dis 2018;14(6):849–56.
[109] Mannaerts GH, van Mil SR, Stepaniak PS, Dunkelgrun M, de Quelerij M, Verbrugge SJ, et al. Results of implementing an enhanced recovery after bariatric surgery (ERABS) protocol. Obes Surg 2016;26(2):303–12.
[110] HAS. Prise en charge en chirurgie ambulatoire; 2014.
[111] Rebibo L, Dhahri A, Badaoui R, Dupont H, Regimbeau JM. Laparoscopic sleeve gastrectomy as day-case surgery (without overnight hospitalization). Surg Obes Relat Dis 2015;11(2):335–42.
[112] Rebibo L, Dhahri A, Badaoui R, Hubert V, Lorne E, Regimbeau JM. Laparoscopic sleeve gastrectomy as day-case surgery: a case-matched study. Surg Obes Relat Dis 2019;15(4):534–45.
[113] Lager CJ, Esfandiari NH, Subauste AR, Kraftson AT, Brown MB, Cassidy RB, et al. Roux-En-Y gastric bypass vs sleeve gastrectomy: balancing the risks of surgery with the benefits of weight loss. Obes Surg 2017;27(1):154–61.
[114] Morgan DJ, Ho KM, Armstrong J, Baker S. Incidence and risk factors for intensive care unit admission after bariatric surgery: a multicentre population-based cohort study. Br J Anaesth 2015;115(6):873–82.
[115] Jung B, Mahul M, Breilh D, Legeron R, Signe J, Jean-Pierre H, et al. Repeated piperacillin-tazobactam plasma concentration measurements in severely obese versus nonobese critically ill septic patients and the risk of under- and overdosing. Crit Care Med 2017;45(5):e470–8.