ejpmr, 2018,5(7), 116-124

SJIF Impact Factor 4.897

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Review Article

ISSN 2394-3211

EJPMR

ANAESTHETIC CHALLENGES IN ADULT LAPAROSCOPIC BARIATRIC SURGERY

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Article Received on 13/05/2018 **Article Revised on 03/06/2018** **Article Accepted on 24/06/2018**

INTRODUCTION

Obesity represents a significant and growing problem around the globe. Aside from the impairment of an individual patient, the negative consequences impose a significant economic burden for many health care systems.[1]

For over a decade, bariatric surgical procedures have established themselves as a way to achieve a permanent weight reduction for a large number of patients.[2]

This patient population represents a particular challenge for the anesthetist, with multiple publications offering a wide range of recommendations on the matter. Additionally, there are also large differences in terms of the data that outline the perioperative process times. All of this results in difficulty to establish such a program and predict its trajectory at the early stages.[3]

The physiology and anatomy of bariatric patients demand a tailored approach from both the anaesthesiologist and the perioperative team. The interaction of a multi-disciplinary team is key to achieving good outcomes and a low rate of complications.[4]

RECOMMENDATIONS

1. Every hospital should nominate an anaesthetic lead for obesity.
2. Operating lists should include the patients' weight and body mass index (BMI).
3. Experienced anaesthetic and surgical staff should manage obese patients.
4. Additional specialised equipment is necessary.
5. Central obesity and metabolic syndrome should be identified as risk factors.
6. Sleep-disordered breathing and its consequences should always be considered in the obese.
7. Anaesthetising the patient in the operating theatre should be considered.
8. Regional anaesthesia is recommended as desirable but is often technically difficult and may be impossible to achieve.
9. A robust airway strategy must be planned and discussed, as desaturation occurs quickly in the obese patient and airway management can be difficult.
10. Use of the ramped or sitting position is recommended as an aid to induction and recovery.
11. Drug dosing should generally be based upon lean body weight and titrated to effect, rather than dosed to total body weight.
12. Caution is required with the use of long-acting opioids and sedatives.
13. Neuromuscular monitoring should always be used whenever neuromuscular blocking drugs are used.
14. Depth of anaesthesia monitoring should be considered, especially when total intravenous anaesthesia is used in conjunction with neuromuscular blocking drugs.
15. Appropriate prophylaxis against venous thromboembolism (VTE) and early mobilisation are recommended since the incidence of venous thromboembolism is increased in the obese.
16. Postoperative intensive care support should be considered, but is determined more by co-morbidities and surgery than by obesity.

DEFINITION

Obesity is a chronic, multifactorial condition that results from accumulation of fat, regionally located throughout the body, due to the positive difference between food consumption and energy expenditure.[5]

Obesity is an independent risk factor for cardio vascular diseases (CVD). Obesity is associated with an increased risk of morbidity and mortality as well as reduced life expectancy. The last two decades of the previous century have witnessed dramatic increase in health care costs due to obesity and related issues among children and adolescents.[6]

CLASSIFICATION

The body mass index (BMI) is the most practical way to evaluate the degree of overweight. It is calculated from the height and weight as follows:

The World Health Organization (WHO) uses a class system to define obesity (Table 1). Statistics for 2013 from the UK, Health and Social Care Information Centre show that in adults, 24% of men and 25% of women are classified as obese and over 3% have class-3 obesity.[7] For an average UK district general hospital serving an adult population of 200 000, this equates to 52 000 obese and over 6000 class-3 obese patients.[8]

Table 1: World Health Organization classification of obesity.

Body mass index; kg.m2	Classification
< 18.5	Underweight
18.5–24.9	Normal
25.0–29.9	Overweight
30.0–34.9	Obese 1
35.0–39.9	Obese 2
> 40.0	Obese 3 (previously 'morbid obesity')

PHYSIOLOGICAL CHANGES IN OBESITY

Increasing obesity leads to respiratory and cardiovascular changes that impact the delivery of anesthesia and perioperative analgesia.

Respiratory physiology

Obesity-related respiratory changes occur as a consequence of physical impingement of lung volumes and chest movement as well as the increased metabolic requirements of excess tissue; these in turn lead to increased work of breathing, increased oxygen (O_2) consumption, and disordered ventilation to perfusion matching.[9]

As a consequence, respiratory rates are increased, and functional residual capacity (FRC) and expiratory reserve volume (ERV) are decreased, even in mild obesity **(10)**. FRC may be sufficiently reduced such that small airways and alveoli remain closed during spontaneous ventilation, leading to ventilation-perfusion mismatch and right to left shunting. Lung volumes and intrapulmonary shunt worsen with the induction of general anesthesia in all patients, but to a much greater degree in obese patients. Supine position and obstructive sleep apnea (OSA) increase the magnitude of these effects.[10]

Consequences of these changes of concern to anaesthesiologists include

- Decreased time to desaturation during apnea.
- Increased O_2 requirements.
- Hypoventilation with supine spontaneous ventilation.[10]

Cardiovascular physiologic changes in obesity include

- Increased circulating blood volume, although it is a lower proportion of total weight (50 mL/kg as compared with 75 mL/kg) compared with patients with normal BMI.
- Decreased systemic vascular resistance.
- Increased cardiac output by 20 to 30 mL per kilogram of excess body fat. Stroke index, cardiac index, and heart rate remain normal; the increased cardiac output occurs by means of expanded stroke volume.
- Left ventricular hypertrophy, related to the duration of obesity. The increased cardiac output can lead to either left ventricular failure (especially when associated with hypertension), or right heart failure (especially when associated with the hypoxia and hypercapnia of OSA).

TYPES OF LAPAROSCOPIC BARIATRIC SURGERIES

The incidence of bariatric surgery is increasing with safely as evidenced by low complication rates. Extremely obese patient experiences significant weight loss and remission of type 2 diabetes mellitus after Roux-en-Y gastric bypass with improvements in insulin resistance, beta-cell function, and cardiovascular risk factors.[11] *table(2).*

The goal of bariatric surgery is to reduce the patient's caloric intake by restricting the amount of food that is able to be consumed (gastric restriction procedure) or, by a dual mechanism, to restrict and decrease the amount of absorption from the gastrointestinal tract (mixed restrictive and malabsorptive procedure). Operative mortality (30 days postoperatively) is estimated to be approximately 0.1% for restrictive procedures and 1% for mixed restrictive and malabsorptive procedures.[12]

Table 2: Percentage of Excess Body Weight Lost by Type of Bariatric Surgery within the First Year.

Adjustable gastric banding	25% - 80%
Sleeve gastrectomy	65% - 75%
Vertical gastric banding	50% - 60%
Roux en Y gastric bypass	70% - 70%
Biliopancreatic diversion with duodenal switch	65%- 75%

PHYSIOLOGIC EFFECTS OF LAPAROSCOPIC SURGERY

The changes during laparoscopy are variable and dynamic. These effects are generally well tolerated by healthy patients. However, significant intraoperative changes can occur in older patients, the changes during laparoscopy can be summarized as follows:

A) Cardiovascular changes
B) Pulmonary changes
C) Regional circulatory changes **(13)**

A) Cardiovascular changes

Table 3: Cardiovascular changes during laparoscopy.[14]

Parameters	Change	Causes
Systemic vascular resistance and mean arterial pressure	Increased	▪ Hypercarbia ▪ Neuroendocrine response (ie, increased catecholamines, vasopressin, and cortisol) ▪ Mechanical factors (ie, direct compression of aorta)
Cardiac filling pressures	Increased	Interaction among: ▪ Increased intravascular volume resulting from compression of liver and spleen ▪ Reduced preload and venous return ▪ Positioning ▪ Patient's preexisting status
Cardiac index	Variable; decreased or no change	Interaction among: ▪ Increased afterload ▪ Decreased venous return ▪ Increased cardiac filling ▪ Increased intravascular volume ▪ Positioning ▪ Patient's preexisting status
Cardiac rhythm	Bradyarrhythmias Tachyarrhythmias	Peritoneal stretch – vagal ▪ Hypercarbia ▪ Hypoxia ▪ Capnothorax ▪ Pulmonary embolism

Studies of hemodynamic events during laparoscopy in patients with significant cardiopulmonary disease have reported an increase in mean arterial pressure (MAP), systemic vascular resistance (SVR), and central venous pressure (CVP), with decreases in cardiac output (CO) and stroke volume (SV) during peritoneal insufflation. Compared with healthy patients, those with cardiopulmonary disease may require more pharmacologic interventions and more intensive monitoring to respond to these changes.[14] *table(3).*

B) Pulmonary changes

Table 4: Pulmonary changes during laparoscopic surgery.[14]

Parameter	Change	Causes
Lung volume (ie, functional residual capacity)	Decrease	Elevation of diaphragm Increased intraabdominal pressure Positioning
Lung compliance	Decreased Increased pleural pressure Increased airway pressure	Elevation of diaphragm Increased intraabdominal pressure
PCO_2	Increased, depending on ventilation	CO_2 absorption
PO_2	Variable	Interaction among: Atelectasis Hypoxic pulmonary vasoconstriction Preoperative pulmonary status
Tracheal position	Cephalad displacement, possible mainstem intubation	Increased intraabdominal pressure Trendelenburg position

PO_2: partial pressure of oxygen; PCO_2: partial pressure of carbon dioxide; CO_2: carbon dioxide.

Pneumoperitoneum with CO_2 and surgical positioning are associated with changes in pulmonary function and gas exchange. These changes can result from increased IAP with pneumoperitoneum and from absorption of CO_2. During laparoscopy, minute ventilation must be increased to compensate for absorption of CO_2. Hyperventilation may be difficult for patients with chronic obstructive pulmonary disease (COPD), asthma, and in morbidly obese patients, especially in Trendelenburg position. In patients with COPD and in older patients, end-tidal CO_2 ($ETCO_2$) may not accurately reflect arterial partial pressure of CO_2; in such patients, arterial blood gases may be required to monitor ventilation. The absorption and elimination of CO_2 in the morbidly obese appears to be similar to nonobese patients. Arterial oxygenation decreases and alveolar–arterial oxygen gradient increases in obese anesthetized patients when placed in Trendelenburg position (Table 4).[15]

C) Regional circulatory changes

1- Splanchnic blood flow

The mechanical and neuroendocrine effects of pneumoperitoneum can decrease splanchnic circulation, resulting in reduced total hepatic blood flow and bowel perfusion. However, hypercapnia can cause direct splanchnic vasodilatation. Thus, the overall effects on splanchnic circulation are not clinically significant.[16]

2- Renal blood flow

The creation of a pneumoperitoneum results in reduction in renal perfusion and urine output associated with renal parenchymal compression, reduced renal vein flow, and increased levels of vasopressin. When IAP is kept under 15 mmHg, renal function and urine output generally normalize soon after pneumoperitoneum deflation, without histologic evidence of pathologic changes.[17]

The effects of laparoscopy on renal function for patients with preexisting renal disease have not been studied. In most cases, we believe that the benefits of a minimally invasive surgical approach outweigh theoretical concerns about the effect of increased intraabdominal pressure on renal function.[18]

3- Cerebral blood flow

Increased intraabdominal and intrathoracic pressures, hypercarbia, and Trendelenburg positioning can all increase cerebral blood flow (CBF) and intracranial pressures (ICP). In healthy patients undergoing prolonged pneumoperitoneum and steep Trendelenburg position, cerebral oxygenation and cerebral perfusion remain within safe limits. In patients with intracranial mass lesions or significant cerebrovascular disorders (eg, carotid atherosclerosis and cerebral aneurysm), the increase in ICP may have clinical consequences. Therefore, in this patient population, we maintain strict normocapnia during laparoscopy.[19]

4- Intraocular pressure

Intraocular pressure (IOP) increases with pneumoperitoneum and increases further when the patient is positioned in Trendelenburg. A prospective observational study of IOP in patients who underwent robotic laparoscopic prostatectomy in steep Trendelenburg position found that IOP increased by an average of 13 mmHg from baseline at the end of the procedure (29 mmHg versus 16 mmHg, upper limit of normal 20 mmHg). The clinical implications of this degree of increase are unknown, though increased IOP may play a role in the rarely reported postoperative visual loss in patients with prolonged cases.[20]

COMPLICATION OF LAPORASCOPIC SURGERIES

The rate of serious complications associated specifically with a laparoscopic approach is overall low. Up to half of complications occur at the time of abdominal access for camera or port placement [(21)]. Complications can also arise from abdominal insufflation, tissue dissection, and hemostasis. Conversion to an open procedure may be needed to manage complications that have been identified intraoperatively, while others may not be recognized until the postoperative period. Severe complications such as vascular injury and bowel perforation can be catastrophic and are the main cause of procedure-specific morbidity and mortality related to laparoscopic surgery.[22]

INTRAOPERATIVE COMPLICATIONS

Complications during laparoscopy include those related to the physiologic effects of the laparoscopic approach (eg, hemodynamic compromise, respiratory decompensation), surgical maneuvers (eg, access-related injury; vascular, solid-organ, or bowel injury; carbon dioxide [CO_2] spread to subcutaneous and intrathoracic spaces; gas embolism), and patient positioning.[23] table(5,6,7,8)

Table 5: Differential diagnosis of hemodynamic collapse during laparoscopy.[14]

Decreased cardiac preload:
▪ Hemorrhage
▪ Positional blood pooling
▪ Gas embolism
▪ Excessive intraabdominal pressure
▪ Capnothorax
▪ Cardiac tamponade due to capnomediastinum or capnopericardium
Decreased cardiac contractility:
▪ Anaesthetic medication effect
▪ Myocardial ischemia or infarction
▪ Acidosis due to hypercarbia
Decreased SVR:
▪ Anaesthetic overdose
▪ Acidosis due to hypercarbia
▪ Anaphylaxis
▪ Sepsis
Bradycardia:
▪ Vagal stimulation

SVR: systemic vascular resistance.

Table 6: Differential diagnosis of hypercarbia during laparoscopic surgery.[14]

Increased CO_2 absorption
▪ CO_2 insufflation (extraperitoneal insufflation > intraperitoneal insufflation) ▪ Subcutaneous emphysema ▪ CO_2 embolism ▪ Capnothorax, capnomediastinum, capnopericardium
Decreased alveolar ventilation (ie, hypoventilation, V/Q mismatch)
▪ Endobronchial intubation ▪ Atelectasis ▪ Airway obstruction ▪ Reduced cardiac output
Increased CO_2 production
▪ Obesity ▪ Malignant hyperthermia ▪ Fever ▪ Thyrotoxicosis
CO_2 rebreathing
▪ Defective CO_2 absorber ▪ Malfunctioning breathing circuit valves

CO_2: carbon dioxide; V/Q: ventilation/perfusion.

Table 7: Causes of hypoxemia during laparoscopic surgery.[14]

Patient factors
▪ Preexisting cardiopulmonary dysfunction ▪ Morbid obesity
Intraoperative ventilation
▪ Low FiO_2 ▪ Hypoventilation
V/Q mismatch
▪ Endobronchial intubation ▪ Atelectasis ▪ Capno (pneumo) thorax ▪ Pulmonary embolism ▪ Patient position (eg, lateral decubitus)
Reduced cardiac output
▪ Inferior vena cava compression ▪ Dysrhythmias ▪ Myocardial depression (eg, anaesthetic drug effects) ▪ Hemorrhage
Anemia
▪ Pre-existing anemia ▪ Hemorrhage

FiO_2: fraction of inspired oxygen; V/Q: ventilation/perfusion.

Table 8: Causes of subcutaneous emphysema, capnothorax, and capnomediastinum During (laparoscopic) surgery.[14]

▪ Inadvertent peritoneal breach ▪ Retroperitoneal insufflation ▪ Misdirected Veress needle or peritoneal port (eg, damage to falciform ligament, subcutaneous needle or port placement) ▪ Specific to capnothorax and capnomediastinum: Gas tracked through fascial planes from neck and thorax into the mediastinum and pleural space Dissection around the diaphragm (eg, during Nissen fundoplication, gastric bypass surgery) Passage of gas through the pleuroperitoneal hiatus (ie, foramen of Bochdalek) Passage of gas through congenital defects (ie, foramen of Morgagni)

PHARMACOLOGY OF ANAESTHETIC DRUG IN OBESITY

There are changes in physiology in obese patients that affect, anaesthetic drug dosing in obesity, as it differs from patients with normal BMI.

Dosing Anesthetic Drugs

Drug dosing in obese patients may be based on total body weight (TBW), lean body weight (LBW), or ideal body weight (IBW), depending upon the agent chosen. When optimal dosing method for a specific drug is unknown, it is reasonable to base doses on LBW, except for highly lipophilic drugs for which TBW should be used**.** However, because of the complexities of pharmacokinetics and dynamics introduced by various types of obesity, the choice of the most appropriate dosing scalar for some drugs is debated. Improved dosing algorithms for target controlled infusion devices in obese patients (not available for use in the US) have been published.[24]

Ideal body weight (IBW), a weight that is believed to be maximally healthful for a person, based chiefly on height but modified by factors such as gender, age, build, and degree of muscular development.[25]

Adjusted body weight

This calculator uses an adjustment factor of 0.4, or 40%, to provide an adjusted body weight in patients who are more than 20% of their ideal body weight. This adjustment uses the following equation:[26]

$$AdjustedBW = IdealBW + (0.4 \times (ActualBW - IdealBW))$$

Table 9: Summary of Drug Doses Commonly used During Anaesthesia.

Drug	Dose	Comments
Acetaminophen IV	15 mg/kg (LBW)	Clearance is T, so dosing may need to be more frequent, max dose 4-6 g/day Check liver function.
Alfentanil	130-245 mcg/kg, and infusion 0.5-1.5 mcg/kg/ min (LBW)	
Antibiotics	TBW	Subcutaneous soft tissue penetration looks impaired in obese patients. Cefuroxime 1.5 g IV is sufficient against gram-positive but not gram-negative organisms.
Cisatracurium	0.15-0.2 mg/kg (IBW)	Duration of action prolonged when given based on TBW.
Dexmedetomidine	0.2 mcg/kg/h (TBW)	When used as infusion it † opioid use, nausea, and the PACU length of stay. But it does not affect late recovery (eg, bowel function) or improve overall quality of recovery.
Enoxaparin	0.5 mg/kg (TBW)	DVT prophylactic dose, divided into bid doses.
Etomidate	0.2 mg/kg (LBW)	Despite more hemodynamically stable characteristics, etomidate induction is associated with a substantially T risk for 30-day mortality, cardiovascular morbidity, and prolonged hospital stay, in a general population. A recent study suggested that etomidate can be dosed according to IBW in MO patients**.**
Fentanyl	2-3 mcg/kg (LBW)	Clearance significantly higher, and increases linearly with "pharmaco- kinetic mass," which is highly correlated to LBW.
Ketamine	1-2 mg/kg (LBW)	Might be used in hypotensive patient. Adding 1 mcg/kg/min to pro- pofol and remifentanil TIVA mixture was found to provide more hemodynamic stability, satisfactory recovery profile, and adequate postoperative pain relief**.**
Lidocaine	1.5 mg/kg bolus, and 2 mg/kg/h infusion both as CBW48	Bolus followed by infusion until end of surgical procedure with indicated doses shows lower opioid consumption and better quality of recovery
Midazolam	Premedication 0.15-0.35 mg/kg (TBW), max 5 mg. If used as continuous infusion, use IBW	Concomitant use with opioids can potentiate its respiratory AEs.
Morphine	0.05-0.10 mg/kg (LBW)	Better to avoid due to longer duration of action, particularly in patient with OSA or OHS.
Neostigmine	0.05 mg/kg (CBW)	Or 70 mcg/kg LBW; total dose ≤5 mg.
Propofol	1.5-2.5 mg/kg for induction (LBW), maintenance infusion	The most common induction agent used; its high lipophilicity and rapid distribution profile account for its short duration of action. Volume of distribution and clearance at steady state

Drug	Dose	Comments
	(TBW)	increases with T TBW.
Remifentanil	1 mcg/kg for endotracheal intubation, and 0.2-2 mcg/kg/min for infusion (LBW)	Its rapid onset of action (1 min) and ultra-short half-life (5-10 min) make it a good option for intraoperative pain management. Aids in maintaining adequate relaxation and reducing volatile anaesthetic or propofol requirements.
Rocuronium	0.6-1.2 mg/kg (IBW)	1.2 mg/kg has rapid onset. The use of rocuronium (1 mg/kg) reversed with sugammadex (16 mg/kg) was found superior to succinylcholine as it allows earlier re-establishment of spontaneous ventilation**.**
Succinylcholine	1-1.5 mg/kg (TBW), ≤150 mg total dose	As in MO patients, the amount of pseudocholinesterase and the extracellular fluid T; doses should be administered based on TBW. Postoperative myalgia seems to be more severe and problematic in obese patients; pretreatment with low-dose nondepolarizing NMBAs or sodium channel blockers such as lidocaine is highly recommended in this population It is a very useful and effective medication in the treatment of laryn- gospasm in obese patients.
Sufentanil	1-2 mcg/kg (LBW)	
Sugammadexa		A final consensus on optimal sugammadex dosing in obese patients has not been reached. Different dosing scalar have been suggested: 2 mg/kg (IBW + 40%)55 and 2 mg/kg (CBW)50; however, TBW seems to be the most safe and effective dosage regimen for complete reversal from NMB with sugammadex in MO patients. Caution: A case report has shown that the use of sugammadex does not guarantee absence of the risk for recurarization. Train-of-four still must be checked before extubation.
Thiopental sodium	3-5 mg/kg (LBW)	Very similar to propofol pharmacokinetics.
Vecuronium	0.1-0.12 mg/kg (IBW)	No rapid onset dose, has longer duration of action when given based on TBW.

T: increased; †: decreased
CBW = IBW + 0.4 × (TBW – IBW)
IBW in male = 50 kg + 2.3 kg for each inch over 5 ft; in female = 45.5 kg + 2.3 kg for each inch over 5 ft.
LBW in male = (1.10 x weight [kg]) – 128 × (weight2/[100 × height [m-2]); in female = (1.07 x weight [kg]) – 148 × (Weight2/(weight2/[100 ×height [m-2])
MO: either BMI >40 kg/m2, or BMI >35 kg/m2 with associated comorbidities such as diabetes mellitus, hypertension, OHS, OSA, pulmonary arterial hypertension, and RV and LV failure.

Lean body mass is a component of body composition, calculated by subtracting body fat weight from total body weight: total body weight is lean plus fat. In equations:

$$LBM = BW - BF$$

The percentage of total body mass that is lean is usually not quoted – it would typically be 60–90%. Instead, the body fat percentage, which is the complement, is computed, and is typically 10–40%. The lean body mass (LBM) has been described as an index superior to total body weight for prescribing proper levels of medications and for assessing metabolic disorders, as body fat is less relevant for metabolism.

Estimation

- LBM is usually estimated using mathematical formulas.

The following formula may be used

- For men: LBM = (0.32810 × W) + (0.33929 × H) − 29.5336
- For women: LBM = (0.29569 × W) + (0.41813 × H) − 43.2933 where W is body weight in kilograms and H is body height in centimeters.
- A nomogram based on height, weight and arm circumference may be used ***(Fuchs et al., 1978).***
- Lean body mass has 3 formulas to work it out

Perioperative Anaesthetic Management

The perioperative management of obese and morbidly obese patients presents significant organizational and practical issues. The Association of Anaesthetists has recently produced a helpful guideline which can be used as the basis of a rational approach to provision of safe anaesthetic services. However, individual patients require a 'tailored' plan. A detailed anaesthetic assessment must be performed. Many morbidly obese patients have limited mobility and may therefore appear relatively asymptomatic, despite having significant

cardio-respiratory dysfunction. The drug history should note any amphetamine-based appetite suppressants as these contribute to increased perioperative cardiac risk. Symptoms and signs of cardiac failure and OSA should be sought actively. Many patients have been unable to lie flat for several years, and may routinely sleep sitting up in an armchair. An assessment of the ability to tolerate the supine position may reveal unexpected profound oxygen desaturation, airway obstruction, or respiratory embarrassment. Awake intubation in a sitting or semi-recumbent position is often better tolerated than supine induction of anaesthesia and asleep endotracheal intubation.[27]

Pre-operative management

The vast majority of obese patients presenting for surgery are relatively healthy and their peri-operative risk is similar to that of patients of normal weight. The patients at high risk of peri-operative complications are those with central obesity and metabolic syndrome, rather than those with isolated extreme obesity.

In bariatric surgery, it is routine to initiate a pre-operative 'liver shrinking' diet to reduce the size of the liver and make access to the stomach technically easier. There is evidence that 2–6 weeks of intense pre-operative dieting can improve respiratory function and facilitate laparoscopic surgery, and may be worth considering in the higher risk patients.

Intra-operative management

The specific peri-operative requirements of the obese patient should be included in the pre-operative team brief of the WHO surgical checklist to ensure the presence of appropriate equipment, including suitable operating tables, beds and trolleys, where possible, regional anaesthesia is preferred to general anaesthesia, although a plan for airway management is still mandatory. There is a higher risk of failure of regional techniques in the obese, and appropriate patient counselling/consent is advised. Induction of general anaesthesia. Easily reversible drugs, with fast onset and offset, are the agents of choice for obese patients. Anaesthetising the patient in the operating theatre has the advantages of avoiding the problems associated with transporting an obese anaesthetised patient, and will also reduce the risk of arterial desaturation and AAGA associated with disconnection of the breathing system during transfer.

Postoperative management

Full monitoring should be maintained in the post-anaesthesia care unit (PACU). The patient should be managed in the sitting position or with a 45° head-up tilt.

Oxygen therapy should be applied to maintain pre-operative levels of arterial oxygen saturation and should be continued until the patient is mobile postoperatively. If the patient was using CPAP therapy at home, it should be reinstated on return to the ward or even in the PACU if oxygen saturation levels cannot be maintained by the use of inhaled oxygen alone.

Before discharge from the PACU, all obese patients should be observed whilst unstimulated for signs of hypoventilation, specifically.

CONCLUSION

Bariatric anaesthesia comes with its unique challenges, but when approached in an thoughtful and interdisciplinary fashion it becomes safely manageable. In addition, the need for some urgent follow-up operations and appropriate postoperative monitoring capabilities have to be accounted for at all times.

Anaesthesia for morbidly obese patients is generally regarded as being associated with increased risks. Accordingly, there are various contributions on the anaesthetic and perioperative management in this patient group, including the preoperative assessment, especially with regards 2 to the airway management and monitoring strategies, the intra-operative phase, the emergence from anaesthesia and the postoperative phase.

A key factor for the perioperative course of those patients is the duration of anaesthesia, which in return consists mostly of the duration of surgery and the expertise of the surgeon.

The rate of intraoperative complications is described to be around 5%, of those roughly a fifth can be regarded as anaesthesia-related. During the procedure the adequate oxygenation of the patient is of main concern. Recruitment maneuvers and increased Peek End Expiratory Pressure (PEEP) levels are effective, but not of great sustainability.

The rate of postoperative ventilation requirement and the surveillance in either a 24-hour post anaesthesia care unit or an intensive care unit (ICU) varies considerably. Sleep apnea syndrome is considered to be an indication for postoperative admission to the ICU, in accordance to the American Society of Anaesthesiologists (*ASA* guidelines for postoperative monitoring in Obstructive Sleep apnea (OSA) patients. Pain management can be particularly challenging in bariatric surgery patients.

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