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REVIEW Postoperative respiratory management of morbidly obese patient

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SUMMARY

Morbidly obese patients are at increased risk of respiratory complications, appropriate management of these patients prevents perioperative respiratory complications. Some aspects of respiratory physiology, posture, ventilatory management including mechanical ventilation and Non-invasive ventilation (NIV), drugs related to respiratory function, and chest physiotherapy will be reviewed to manage these patients properly.

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1. Introduction

Globesity is the term used by the World Health Organization (WHO) to describe the global problem of obesity affecting developed countries. The latest available data show that obese individuals (BMI > 30 kg/m²) make up more than 33% of the population in the United States, around 21% of the population in the UK, and 15% of the population in Mediterranean countries, such as Spain.¹ In the United States, obesity is the second leading cause of death (15.2% of overall mortality), exceeded only by tobacco.² The prevalence of obese patients in critical care units is 26%, 6.7% for morbidly obese.^{3–5} Morbid obesity is associated with multiple comorbidities, including respiratory diseases, cardiovascular diseases, and endocrine diseases. The ASA (American Society of Anaesthesiologists) classify morbidly obese patients as ASA III independent of any other comorbidity. However, obese patients in intensive care units (ICU) have lower mortality than non-obese patients; this is known as the obesity paradox. Morohunfolu et al.⁶ reported a statistical analysis of 65,000 patients, 15,000 of whom were obese; in this analysis obesity appears to protect against the risk of dying in the ICU (RR 0.86, 95% CI 0.81–0.91, P = 0.001, $I^2 = 0$). This result may have been due to the hypercatabolism and systemic inflammatory response habitual in critically ill patients being partially offset by the increase in fat reserves and immunomodulatory hormone secretion (leptin and interleukin-10).

The treatment of complications in these patients is a challenge that requires understanding the pathophysiology and appropriately managing the patient perioperatively to avoid complications. In this review, we focus on the respiratory problems experienced by

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2210-8440/\$ — see front matter \odot 2012 Elsevier Ltd. All rights reserved. http://dx.doi.org/10.1016/j.tacc.2012.12.002 morbidly obese patients after surgery, including laparoscopic surgery that is increasing day by day, and the existing evidence regarding their postoperative management.

2. Respiratory physiology (Table 1)

Knowing the respiratory physiology of morbidly obese patients can help the anaesthetist and other professionals in the management of these patients. The respiratory system of morbidly obese patients is mostly restrictive and associated with an obstructive pattern. The restrictive pattern is produced by the increase in adipose tissue in the chest wall, at the intrathoracic level, and in the abdominal cavity (which pushes the diaphragm up), leading to reduced compliance and lung volumes. In particular, the expiratory reserve volume (ERV) is reduced by 25%-53% according to the BMI,⁷ thereby reducing the functional residual capacity (FRC). The reduction in FRC can be so marked that it comes close to the residual volume.⁸ Moreover, decreased ERV reduces vital capacity (VC) and total lung capacity (TLC). All of these changes make it easier to reach the closing volume during tidal volume (VT) at rest, causing a collapse of the lower airway at the end of normal expiration, causing pulmonary atelectasis, favouring the appearance of a ventilated, but not perfused alveolus by the closure of the lower airway. This context causes ventilation-perfusion mismatch (shunt effect) with hypoxaemia.^{9,10}

Hypercarbia is also frequent in the morbidly obese. The resting VT decreases, but the minute volume is increased due to an increased respiratory rate to compensate for the increased oxygen consumption and CO_2 production in these patients.^{11–13} Consequently, the morbidly obese have up to 70% increased breathing effort compared with the non-obese, mainly due to an increased minute volume and the restrictive respiratory system. The obstructive pattern is produced by a narrowing of the airway due to

Table 1

Changes in respiratory physiology of morbidly obese patient.

- Restrictive pattern with reduced chest wall compliance and lung volumes (reduced ERV, VC, TLC, and FRC).
- Obstructive pattern with narrowing of the airway due to the increased fatty tissue (reduced FEV1).
- Frequent association with two respiratory diseases with clinical implications: OSA and Asthma.
- 70% increased breathing effort, with less respiratory muscle endurance, weakness and muscle inefficiency.
- Pulmonary atelectasis with hypoxaemia.

the increased fatty tissue, which compresses the lumen and infiltrates the smooth muscle, altering its function and predisposing the patient to bronchial hyper-reactivity and asthma. Obese individuals generate lower maximum inspiratory pressures (MIP) and have less respiratory muscle endurance compared to the non-obese. Therefore, weakness and muscle inefficiency in these patients also contributes to the reduced FEV1 (Forced Expiratory volume in the first second).¹⁴

Morbid obesity is often associated with two respiratory diseases that have clinical implications for management. One is asthma, which is increased by 50% in severity, prevalence, and incidence compared to non-obese patients.^{15,16} The other is obstructive sleep apnoea syndrome (OSA); 40–70% of patients undergoing bariatric surgery meet the criteria for OSA. Patients with OSA suffer frequent episodes of apnoea and desaturation due to a collapse of the upper airway. OSA is associated with an increased number of complications, with double the risk of suffering respiratory complications.^{17,18} All changes in the respiratory physiology in morbid obesity and the associated diseases favour possible respiratory failure, and the postoperative period is a crucial time for the appearance of respiratory complications. Thus, rapid preventive and therapeutic interventions are necessary to prevent complications.

3. The posture

The posture is also an important element that has clinical implications. Classical texts show that differences in FRC exist depending on the position in which a patient is maintained; a lower FRC can trigger perioperative respiratory complications.¹⁹ Patients achieve the largest FRC in a standing position. The FRC is reduced progressively as the patient moves from standing to sitting (90°) to a supine position (0°), with the lower FRC achieved in the Trendelenburg position.²⁰ Adults move from 3 L standing to 2 L in Trendelenburg (30°) ,²¹ and the FRC is reduced by 25% when moving from sitting to supine. Recent articles have shown that the change in FRC does not happen exactly like this in the morbidly obese, but these patients start with a markedly decreased FRC, and the decrease in FRC depends on the body mass index (BMI) more than the position. According to some authors^{22,23} position changes do not considerably change the FRC, finding that changes in the FRC at supine (0°) , $+30^\circ$, or -30° were not significant in obese patients. Differences in FRC only become significant when comparing these positions with the sitting position. Other authors, such as Pelosi et al.,²⁴ have shown that keeping obese patients in the supine position is not beneficial for them because the intra-abdominal pressure is too high compared to the non-obese, leading to reduced lung volume, atelectasis, and hypoxaemia. Consequently, keeping morbidly obese patients supine (0°) has no beneficial respiratory effects, but in a position of semi-sitting on the bed (60°) or even seating them fully seems useful, though the degree of improvement remains to be established. Therefore, the supine position (0°) should be avoided.

4. Drugs and respiratory function

Another important aspect are the drugs commonly used intraoperatively, that may impair postoperative respiratory function. Early extubation is indicated because it shortens the duration of mechanical ventilation, but it can be done when hypnotics, opioids, and muscle relaxants persist in the body of these patients. The residual effects of these three drug groups deserve special attention because they favour atelectasis with hypoxaemia, CO₂ retention, and the risk of aspiration with an increased risk of pneumonia. Therefore, increasing the risk of respiratory complications can lead to respiratory failure and require re-intubation.⁹

When we talk about neuromuscular relaxants, train of four (TOF) ratio $(T_4/T_1) > 0.7$ does not appear to affect respiratory variables like VT, vital capacity, and inspiratory and expiratory maximal pressures. However, neuromuscular relaxants and inhaled anaesthetics produce a residual inhibition of the chemoreceptor response to hypoxia, even with a TOF ratio of 0.7. Eriksson ²⁵ studied 30 healthy volunteers using non-depolarizing muscle relaxants (atracurium, pancuronium, and vecuronium) with a TOF ratio of 0.7 and found that the hypoxic ventilatory response was depressed by approximately 300%, but with a TOF > 0.9 no differences were found compared to baseline measures. Residual neuromuscular blockade inhibits genioglossus response in deep inspiration, facilitating postoperative obstruction of airflow in the pharynx, retropalatal and retroglossal areas, a mechanism reminiscent of the pathophysiology of OSA.^{26,27}

As for analgesia, parenteral opioids are some of the most effective drugs available to combat moderate to severe pain that occurs in the immediate postoperative period, but they are not exempt from side effects, especially respiratory effects. The morbidly obese are very sensitive to the respiratory depressant effects of opioids and their use should be limited as much as possible in this population.⁶⁶ Similarly, these drugs are recommended to be avoided in patients with OSA. Many morbidly obese patients have OSA; therefore, other drugs are preferable.²⁸ When large amounts of opioids are needed in these patients, loco-regional techniques are preferred, such as epidural. Epidural returns the FRC and closing capacity to pre-operative values.²⁹ Loco-regional techniques compared to opioids have beneficial effects by reducing the risk of atelectasis and re-intubation.³⁰

On the other hand, postoperative pain has three main components (nociceptor stimulation, tissue injury, and activation of the central pathways), and acting simultaneously on different mechanisms that cause pain seems more effective than acting on a single mechanism, which is known as multimodal analgesia.³¹ Multimodal analgesia includes the combined use of analgesics, such as acetaminophen, NSAIDs, local anaesthetics, and other drugs. Reducing the amount of an individual analgesic drug significantly reduces side effects. In relation to this topic, Feld et al.³² conducted a study of 30 morbidly obese patients undergoing gastric by-passes who were randomized into two groups. One group received intraoperative fentanyl analgesia and the other received a combination of non-opioid, multimodal analgesia, including ketorolac, clonidine, lidocaine, ketamine, magnesium sulphate, and methylprednisolone. Postoperative morphine requirements administered by a pca-pump (analgesic pump used on demand, controlled by the patient) were studied. Postoperative opioid requirements were lower in the multimodal analgesia group (5.2 \pm 2.6 mg/h vs. $7.8 \pm 3.3 \text{ mg/h}$, P = 0.04). This group was also less sedated than the group that used opioids during the intra-operative period. Therefore, we recommend early extubation but also to ensure thorough rinsing of the inhaled anaesthetic and complete reversal of muscle relaxation with a TOF ratio >0.9. Also, the use of multimodal analgesia is recommended in these patients to reduce the use of Download English Version:

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