EDITORIAL

PREOXYGENATION OF THE MORBIDLY OBESE WITH OBSTRUCTIVE SLEEP APNEA

Morbid obesity is associated with a more rapid decrease in oxygen saturation during apnea following induction of anesthesia, compared to patients who have normal weight[1]. This is particularly hazardous as morbid obesity, complicated by obstructive sleep apnea, may be associated with an increased risk of difficult tracheal intubation and difficult face mask ventilation[2]. These morbidly obese patients, with a body mass index >35 kg/m2, a history of obstructive sleep apnea, a neck circumference >17 inch, a short thyromental distance, and a Mallampati class III, suggest difficult mask ventilation, difficult tracheal intubation, as well as rapid oxyhemoglobin desatruation during apnea (Fig. 1).

The more rapid hemoglobin desaturation may be attributed to increased oxygen consumption, associated with a decreased functional residual capacity of the lung (FRC), which is the main oxygen store. In addition, the supine position further decreases the FRC due to the cephalad displacement of the diaphragm by the abdominal content. Also, induction of general anesthesia will result in an additional reduction of the FRC. Whereas the FRC of the non-obese patients decreases by approximately 20% following induction of anesthesia, it decreases by approximately 50% in the morbidly obese patients. Thus, the tidal volume of the morbidly obese patient may fall within the closing capacity, resulting in microatelectasis and ventilation-perfusion (V/Q) mismatch, with a subsequent increase of the alveolar-arterial oxygen gradient associated with 10-20% intrapulmonary shunt, compared to 2-5% in the non obese patients.

In the morbidly obese patient, the time taken for the oxygen saturation to fall to 90% during apnea following standard preoxygenation by tidal volume breathing of oxygen for 3 minutes, is significantly reduced compared to the time taken in non-obese patients. The head-up position has been recommended to optimize preoxygenation in non-obese patients[3], as well as in morbidly obese patients[4]. The head-up position during preoxygenation in the morbidly obese patients has been shown to prolong the mean time of desaturation by about 50 seconds. The application of continuous positive airway pressure (CPAP) during preoxygenation has been suggested to optimize preoxygenation in the morbidly obese on the assumption that CPAP will increase the FRC[5]. However, CPAP only resulted in nonsignificant increase of the mean

Fig. 1
A morbidly obese male patient, with a body mass index (BMI) 35 kg/m2, a history of obstructive sleep apnea, a neck circumference 17 inch, a short thyromental distance, and a Mallampati score class III, suggesting difficult mask ventilation, and difficult tracheal intubation, as well as rapid oxyhemoglobin desaturation during apnea

time to desaturation to 90%, as the FRC will return to pre-CPAP levels once the patient is anesthetized and the CPAP mask is removed.

Recently, it has been shown in the morbidly obese patients, that nasopharyngeal oxygen insufflation following preoxygenation delays the onset of oxyhemoglobin desaturation during the subsequent apnea by apneic diffusion oxygenation[6]. In contrast, preoxygenation without subsequent nasopharyngeal oxygen insufflation in the morbidly obese patients, is followed by a more rapid desaturation during the subsequent apnea, associated with a significant negative correlation between the body mass index and the time to oxyhemoglobin desaturation[7].

In the critically ill morbidly obese patient suffering from respiratory failure, traditional preoxygenation without or even with subsequent nasopharyngeal oxygen insufflation may not significantly increase the FRC oxygen store, and improve the oxygen saturation before, during or after tracheal intubation. This may be attributed to the significant atelectasis and decrease of the FRC in these patients which results in marked intrapulmonary shunting associated with a significant increase of the alveolar-arterial oxygen gradient. In this situation, the use of noninvasive bilevel positive airway pressure (BiPAP) during preoxygenation can improve alveolar recruitment and decrease derecruitment with a consequent decrease of the significant ventilation-perfusion mismatch and the alveolar-arterial oxygen gradient. BiPAP preoxygenation can achieve a notable increase of oxyhemoglobin saturation associated with less hypercarbia, as compared to the traditional technique of preoxygenation[8].

Baillard et al showed in the critically ill patients that noninvasive ventilation (provided in the form of pressure support ventilation) in the intensive care unit setting ensured improved oxygen saturation before, during and after endotracheal intubation as compared with the standard preoxygenation technique; Baillard showed that a significant linear correlation exists between SpO_2 at the end of preoxygenation and the minimal SpO_2 during tracheal intubation[9]. Also, Baraka et al have demonstrated with different techniques of preoxygenation, that rapid oxyhemoglobin desaturation during the subsequent apnea will occur whenever SpO_2 decreases below 99%[10,11].

In conclusion, traditional preoxygenation is adequate in the non obese patient. However, nasopharyngeal oxygen insufflation following preoxygenation is indicated in the morbidly obese patients. In the critically ill morbidly obese patient, complicated with respiratory failure, BiPAP preoxygenation is indicated to recruit the atelectatic alveoli and decrease the significant ventilation-perfusion mismatch, with a subsequent increase of SpO_2 before, during and after tracheal intubation.

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