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REVIEW

Mechanical ventilation in the operating room: Adjusting VT, PEEP, and FiO_2

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S U M M A R Y

Perioperative ventilatory management is a key component of patient outcome. Ventilation with physiological tidal volume decreases pulmonary inflammatory response, intubation time, risk of re-intubation, and postoperative pulmonary complications (PPC). Atelectasis is closely related to anaesthesia, produces hypoxaemia, and increases the risk of ventilator-induced lung injury (VILI). Perioperative prevention and treatment of atelectasis, with recruitment manoeuvres and the application of positive end-expiratory pressure, improves respiratory mechanics (Csr) and decreases the risks of ventilation/perfusion mismatch and VILI. Many anaesthesiologists only increase inspiratory oxygen fraction (FiO_2) as a first step for the treatment of perioperative hypoxaemia; however, the preventative perioperative use of high FiO_2 may decrease the risk of surgical site infections and postoperative nausea and vomiting.

1. Introduction

Perioperative ventilatory management has evolved dramatically in recent years—from ventilation with high tidal volumes (VT) of >15 ml/kg with no positive end-expiratory pressure (PEEP) to the use of more physiologic tidal volumes (<10 ml/kg) with PEEP in almost any patient and any surgery. Guidelines for ventilatory management are exclusive to patients with acute lung injury (ALI) or acute respiratory distress syndrome (ARDS); therefore, there are no consensus criteria for ventilatory management in patients without ALI/ARDS. However, evidence in the literature demonstrates that using lung-protective ventilation in patients without ALI/ARDS has benefits, including decreasing the risk of ventilator-induced lung injury (VILI).

The present review provides an overview of the current literature addressing tidal volume size, the level of PEEP, the application of recruitment manoeuvres, and the inspiratory oxygen fraction (FiO_2) in patients without ALI/ARDS during the intraoperative period.

2. Tidal volume

There is a paucity of evidence from randomised controlled trials on the best way to ventilate patients without ALI/ARDS. There are several justifications for using ventilation with lower tidal volumes (VT < 10 ml/kg). Firstly, they are more physiological; so-called lower tidal volumes are in fact "normal" tidal volumes.[1] Secondly, available evidence suggests a causal relationship between the use of large tidal volumes and the development of VILI.[2–5] Furthermore, it is well-known that VILI development can aggravate previous lung injury or sensitise the lung to further injury in the "two-hit model", which often occurs after lung ischaemia-reperfusion injury, large transfusions, cardiopulmonary bypass, infections, low perfusion states, and prolonged mechanical ventilation. VILI involves a complex interaction of overdistension (volutrauma), increased transpulmonary pressure (barotrauma), cyclic opening and closing of alveoli (atelectrauma), and production of inflammatory mediators (biotrauma).

2.1. Tidal volume and clinical outcomes

The relationship between ventilation using lower tidal volumes and mortality has not been investigated in patients without ALI/ARDS. However, previous studies, most of them in cardiac and thoracic surgery, have demonstrated that large perioperative VT (>8–10 ml/kg) leads to increased inflammatory response, prolonged intubation, and increased risks of re-intubation, postoperative pulmonary complications (PPC), and prolonged ICU stay.

In patients undergoing abdominal surgery, Lee et al. observed a lower incidence of infections and earlier extubation in the patients who received 6 ml/kg of VT compared to 12 ml/kg.[6] In patients undergoing planned esophagectomy, Michelet et al. showed that lung protective ventilation (VT of 5 ml/kg with PEEP) decreased the systemic inflammatory response, improved oxygenation (better PaO_2/FiO_2 ratio) and lung function, and resulted in earlier extubation compared with conventional ventilation (VT of 9 ml/kg with zero end expiratory pressure, ZEEP).[5] Fernandez-Pérez et al. performed

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a retrospective study in patients who underwent elective pneumonectomy, and showed that perioperative large tidal volumes (mean of 8.3 ml/kg) were associated with development of PPC compared with low VT (mean of 6.3 ml/kg).[2] Lellouche et al. recently conducted a prospective study in 3434 cardiac surgery patients, and showed that large tidal volumes (>10 ml/kg) are an independent risk factor for organ failure and prolonged ICU stay.[7] Furthermore, in cardiac surgery patients, Sundar et al. showed that use of 6 ml/kg of VT shortens the time to extubation and decreases the re-intubation risk when compared to 10 ml/kg of VT.[8]

During one-lung ventilation (OLV), Kalz et al. reported that larger tidal volumes produced the highest PaO_2.[9] In contrast, several more recent studies on OLV did not find any difference when comparing larger vs. lower tidal volumes with PEEP.[10,11] However, many other studies of OLV have shown that ventilation with a large VT, and therefore high airway pressure, increases the development of ALI.[3–5,12,13]

2.2. Tidal volume and inflammation

Different studies have investigated the relationship between perioperative VT and the inflammatory response through measurements of inflammatory markers. Most of these studies have shown that the use of larger VT increases the inflammatory response compared to lower VT. Wrigge et al. and Zupancich et al. observed in patients undergoing cardiopulmonary bypass that lower VT (6 and 8 ml/kg) decreased the inflammatory response, as shown by lower levels of cytokines and TNF than observed with 12 and 10–12 ml/kg of VT, respectively.[14,15] In patients scheduled for an elective surgical procedure, Choi et al. showed that mechanical ventilation with higher VT (12 ml/kg) without PEEP caused an increase in soluble thrombomodulin in lavage fluids and lower levels of bronchoalveolar activated protein C in comparison with those observed with lower VT (6 ml/kg) and 10 cmH_2O of PEEP.[16] These changes reflect pulmonary inflammation, as it is characterised by local procoagulant shift of the alveolar haemostatic balance that promotes fibrin depositions within the airways.[17] Choi et al. further demonstrated that mechanical ventilation with lower tidal volumes and PEEP can largely prevent these procoagulant changes. Schilling et al. studied patients undergoing OLV, and showed that 10 ml/kg of VT increases the inflammatory response (higher levels of IL-8 TNF, sICAM-1, and PMN in BAL) compared to 5 ml/kg.[4] This study differed from those mentioned above in that Schilling et al. used the same level of PEEP in both groups, and thus, the different inflammatory responses were exclusively related to the perioperative VT.

Only very few studies have not found differences in the inflammatory response between large and low perioperative VT during thoracic,[4] cardiac,[18] or abdominal surgery.[19,20] Different results from studies on pulmonary inflammatory response comparing high vs. low VT may be related to how inflammatory mediators are measured, duration of the study, and type of surgery. While direct measurements (in BAL) demonstrate clear differences, indirect measurements (in plasma) have produced controversial results. Levels of systemic inflammatory mediators depend on translocation through the alveolar-capillary membrane, and translocation in healthy lung is not produced in short periods (perioperative) but after a “first hit”, like during cardiopulmonary bypass. Thus, it seems clear that inflammatory response is related to the level of VT used, despite the reports of a few studies that did not detect such a difference by measuring markers in plasma.

3. PEEP and recruitment manoeuvres

There are many reports showing benefits of the use of positive end-expiratory pressure (PEEP) in terms of oxygenation, ventilation, time of intubation, and PPC. However, the last Cochrane review of the effects of PEEP during anaesthesia concluded that there is currently insufficient evidence to make conclusions about whether intraoperative PEEP alters the risks of postoperative mortality and respiratory complications among undifferentiated surgical patients.

General anaesthesia decreases functional residual capacity (FRC) and compliance of the respiratory system (Csr), both of which are closely related to atelectasis. Atelectasis appears in almost 90% of all patients who are anaesthetized,[21] developing immediately after induction, during either spontaneous breathing or positive pressure ventilation; it predominantly occurs in dependent lung areas and may persist for many days after surgery. The amount of atelectasis on computed tomography (CT) scan is correlated with the severity of intrapulmonary shunt.[22] Ventilation/perfusion mismatches, especially hypoxaemia, are often due to the development of intrapulmonary shunt. Mild to moderate hypoxaemia (arterial oxygen saturation of between 85 and 90%) occurs in about half of all patients undergoing anaesthesia and elective surgery, despite a FiO_2 of 0.3–0.4.[23] Therefore, it has been clearly shown that perioperative hypoxaemia is usually produced by atelectasis.

Atelectasis alters surfactant function, stimulating alveolar macrophages, increasing pro-inflammatory cytokines, altering the permeability of the alveolar capillary membrane, and potentially predisposing the patient to VILI. Atelectasis can be prevented and treated during the perioperative period through the open lung approach (OLA), which involves recruitment manoeuvres and the application of PEEP; this decreases the risk of developing VILI[24,25] and ventilation/perfusion mismatch, and improves respiratory mechanics (Csr).[26] Tusman et al. showed that OLA improves oxygenation during general anaesthesia compared to ZEEP or PEEP without recruitment manoeuvres.[27]

Many patients are particularly prone to benefit from the OLA, including laparoscopic surgery patients (especially during bariatric surgery) and patients undergoing OLV or cardiopulmonary bypass. While laparoscopic surgery has shown many clinical benefits, a physiopathological consequence is increased intrapulmonary shunt due to increased blood flow into pulmonary circulation and more frequent compression atelectasis in the dependent zones.[28] Maracajá-Neto et al. showed that the application of 10 cmH_2O of PEEP improves respiratory mechanics (higher Csr and lower Rsr) compared with ZEEP.[29] In laparoscopic bariatric surgery patients, Whalen et al. showed that OLA with 12 cmH_2O of PEEP improves ventilation/perfusion mismatch (higher PaO_2) compared with the application of 4 cmH_2O PEEP without recruitment manoeuvres.[30] Talab et al. showed that OLA with higher levels of PEEP (10 cmH_2O vs. 5 and 0 cmH_2O) improved peri and postoperative oxygenation, as shown by less atelectasis evidenced by CT scan and fewer PPC.[31] Similar results were obtained by Futier et al., who showed that OLA with 10 cmH_2O of PEEP increased the FRC and improved oxygenation.[32]

One-lung ventilation is necessary but not mandatory in many surgeries, and many anaesthesiologists use it in order to improve the surgical field. During surgery with a patient on lateral decubitus, both lungs are perfused. Perfusion of the non-dependent lung produces shunt, the atelectasis produced in the dependent lung increases the shunt effect, and both increase the risk of hypoxaemia.

There is some controversy over the benefit of external PEEP application to the dependent lung during OLV; various studies have demonstrated that PEEP results in an increase,[33] no change,[10] or a decrease[34] in oxygenation. Slinger et al. showed that PEEP is effective in preventing atelectasis; however, its value must be titrated in accordance with the static compliance curve to involve the oxygenation.[11] Michelet et al. observed that 5 and 10 cmH_2O of