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Research progress in perioperative ventilator-induced lung injury

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ABSTRACT Lung-protective ventilation (such as low tidal volume and application of positive end-expiratory pressure) is beneficial for patients with acute lung injury or acute respiratory distress syndrome (ARDS) and has become the standard treatment in intensive care unit (ICU). However, some experts now question whether the protective ventilation strategy for ARDS patients in the ICU is equally beneficial for patients after surgery, especially for most patients without any pre-existing lung lesions. This review will discuss preoperative, intraoperative, and postoperative lung protection strategies to reduce the risk of complications associated with anesthesia.

KEY WORDS mechanical ventilation; ventilator-induced lung injury; lung protection; tidal volume; positive end-expiratory pressure

围手术期呼吸机诱导的肺损伤研究进展

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[摘要] 肺保护性通气(如低潮气量、应用呼气末正压等)对急性肺损伤或急性呼吸窘迫综合征患者有益，已成为重症监护病房的标准治疗方法。然而，现在一些专家对重症监护病房急性呼吸窘迫综合征患者的保护性通气策略是否同样有益于手术后患者提出质疑，特别是那些大多数术前无肺部病变者。本综述将讨论术前、术中和术后的肺保护策略，以期降低麻醉相关并发症的发生风险。

[关键词] 机械通气；呼吸机诱导的肺损伤；肺保护；潮气量；呼气末正压

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Mechanical ventilation (MV) is used during general anesthesia for patients with endotracheal tubes or supraglottic airways in place. A further understanding of how MV leads to ventilation-induced lung injury (VILI) unveils the need for a lung-protective ventilation strategy for patients with lung diseases such as acute respiratory distress syndrome (ARDS)[1-2]. Clinically, confirming MV as the precise cause of lung injury can be very difficult. Therefore, the term ventilator-associated lung injury is used when lung injury and MV occur simultaneously, but the lung injury is not necessarily caused by MV[3]. Recently, numerous clinical trials[4-6] have suggested that certain lung-protective strategies could also benefit patients without pre-existing lung diseases, such as those who receive intraoperative MV for surgery. Protective strategies include using low tidal volume (TV) to prevent alveolar over-distension, applying adequate positive end-expiratory pressure (PEEP) to decrease repeated alveolar opening and closing, and adjusting fraction of inspired oxygen (FIO_2) to prevent oxygen toxicity.

VILI may occur through either barotrauma caused by elevated transpulmonary pressures, volutrauma caused by high TV, or atelectrauma caused by the cyclic opening and closing of lung units under low TV at low airway pressure[3, 7-8]. More recently, biotrauma has also been described to contribute to VILI; MV can trigger the release of pro-inflammatory mediators[9]. This biological interaction in response to mechanical forces involves the alveolar epithelium, vascular endothelium, neutrophils, macrophages, and mediator release through autocrine and paracrine signaling transduction. Mechano-transduction is the key link between physical forces (such as stress and strain) imposed on the lung and intracellular signaling pathways leading to the production of cytokines[10]. Degree, pattern, and magnitude of lung stretch are important in activating an inflammatory cascade.

Collective clinical evidence and our laboratory results have shown that VILI can exacerbate existing lung injury or sensitize the lung to further injury, a term called the "two-hit lung injury model" which is significantly associated with sepsis, large transfusions, cardiopulmonary bypass, and lung ischemia–reperfusion injury[11]. Particularly, we found that both interleukin (IL)-33 and WNT1-inducible signaling pathway protein 1 (WISP1) play important roles in the VILI process. Using a mechanical stretch model in murine respiratory epithelial cells, we found that lung epithelial cells could release IL-33 through the HMGB1/TLR-4 signaling pathway[12]. In a two-hit mouse lung injury model, we found that the IL-33-ST2 pathway contributes to VILI in septic mice in a TV-dependent manner[13]. We also demonstrated that WISP1, identified using a genome-wide approach, acts as a cellular accessory molecule that leads to VILI in mice[14]. Furthermore, our most recent study revealed that non-injurious MV can exacerbate sepsis-induced acute lung injury (ALI), which is partly dependent on the WISP1-TLR4-integrin β5 pathway[15].

I Preoperative lung protection strategies

Several preoperative interventions, including optimization of respiratory status, smoking cessation, and physiotherapy/rehabilitation, may reduce perioperative pulmonary complications[16].

Patients with chronic respiratory diseases like asthma and chronic obstructive pulmonary disease (COPD) should maintain at their baseline without deterioration before surgery[17]. Communication with primary care physicians or respiratory therapists can be helpful to determine the optimization level. Control of infection and airway hyperresponsiveness and use of anticholinergics in COPD patients and beta-2 adrenergic stimulants in asthma patients, as well as steroids for both COPD and asthma patients, will effectively limit acute exacerbations. In addition, the risk of pulmonary complications will be also reduced by preoperative administration of leukotriene antagonists[18].

Smoking increases perioperative complications, resulting in delayed postoperative recovery; thus, stopping smoking at some point during the preoperative period is meaningful[19]. In the cardiothoracic surgical population, longer periods of smoking cessation have been found to lead to a greater reduction of risk for postoperative pulmonary complications (PPCs). A period of at least four to eight weeks of cessation is recommended to decrease PPCs, including ARDS[20]. Cessation for two to three months will lead to decreased sputum production[20]. However, smoking cessation has also been found to possibly increase short-term pulmonary risk because of increased mucus production from improved mucociliary function and decreased coughing from less bronchial

irritation[16]. Though smoking cessation should always be encouraged, the best timing for this with respect to surgery remains controversial[21].

For patients undergoing preoperative rehabilitation or "pre-habilitation," the perioperative risk profile of patients with poor functional ability may be improved with strategies including chest physiotherapy, comprehensive programs of physical exercise, optimizing nutritional status, improving oral hygiene, and correcting anemia[22-23]. These programs help reduce the incidence of postoperative complications and all-cause mortality in major surgery[24]. In patients with COPD, preoperative exercise programs are known to improve pulmonary function before surgery, decrease in-hospital length of stay, and reduce the risk of PPCs during surgery for lung transplantation, lung cancer, and lung volume reduction[25-26], although some disparities are apparent due to different studies, risks of bias, and sample sizes[27]. Preoperative chest physiotherapy combined with lung expansion techniques, such as forced expiration techniques, incentive spirometry, and active breathing, were found to reduce PPCs by 50%–60% in those who performed these exercises for 20 minutes a day for at least two weeks before surgery[28]. A recent systematic review[29] also confirmed that preoperative respiratory muscle training programs reduced PPCs by 50% in patients undergoing upper abdominal and cardiothoracic surgery and respiratory muscle weakness was associated with PPCs.

2 Intraoperative lung-protective ventilation

2.1 Ventilating patients with lung injury

Several studies[11, 15] have shown that MV, by eliciting a pre-existing local inflammatory response, can induce postoperative ALI. According to the "two-hit model", the primary insult (first hit) predisposes the lung to MV injury (second hit). In patients with preexisting lung injury, further lung injury risk induced by the second insult, MV, is significantly greater. However, the lung injury caused by MV can be attenuated by using appropriate ventilation strategies. Because patients with preexisting lung injury may develop intraoperative hypoxemia or even ARDS with major surgery, a protective ventilation strategy must be applied to achieve optimal arterial oxygenation and reduce potential lung injury[11].

2.1.1 TV

Cumulative data support that low TV ventilation in patients with pre-existing lung injury like ARDS decreases PPCs and improves outcomes[30-31]. TV of 6–8 mL/kg to ideal body weight should be targeted, with a low plateau pressure [<16 cmH_2O (1 cmH_2O=0.098 kPa)] and preferably a low ΔP(<13 cmH_2O). TVs of 4–5 mL/kg should be targeted for one lung ventilation in conjunction with PEEP and intermittent recruitment maneuvers. Permissive hypercapnia in the absence of raised intracranial pressure or severe right heart failure should be allowed without having to normalize $PaCO_2$. The potential benefits of hypercapnia include an increase in oxygen release from erythrocytes by increased P_{50} values and rightward shift of the oxygen-hemoglobin dissociation curve, increased cardiac output, and anti-inflammatory effect.

2.1.2 Positive end expiratory pressure

PEEP can prevent VILI and improve pulmonary function in animals with injured lungs[32]. Choosing optimal PEEP to balance beneficial and harmful effects is challenging because of heterogeneous lung units, particularly in pre-existing lung injury where PEEP may be beneficial in some lung units, but unfavorable in other lung units. Appropriate PEEP prevents alveolar collapse at the end of expiration to improve pulmonary compliance and better oxygenation, which minimizes the sheer forces and biotrauma experienced in an unevenly atelectatic and affected lung. Applying a PEEP of 10 cmH_2O at the start of ventilation and adjusting optimal PEEP level according to oxygenation may be reasonable[31].

2.1.3 Recruitment maneuvers

Recruitment maneuvers should not be routine, particularly with severe ARDS, because the condition may be associated with significant hemodynamic instability due to effects on right ventricular preload and afterload. Recent practice recommendations[33-34] support personalizing patient care and avoiding unnecessary attempts to expand lung units to worsen lung injury due to biotrauma and atelectrauma, as the benefit of recruitment maneuvers on patient outcomes remains inconclusive.

2.2 Ventilating patients without lung injury

Although lung-protective ventilation has played a favorable role in affected lung patient groups, definitive evidence of its benefit for the unaffected lung in the general surgical population is still lacking.

2.2.1 TV

Recent studies[4-5, 35-36] have shown that protective ventilation targeting TV at 6–8 mL/kg to predicted body weight (PBW) reduces the occurrence of PPCs and improves clinical outcomes compared with the conventional ventilation settings. TV should be calculated based on PBW rather than on actual body weight, because the latter leads to potential harmful ventilatory settings associated with adverse pulmonary effects, although at present, protective ventilation settings don't seem to provide abundant evidence to modify the mortality rate.

2.2.2 PEEP

Each patient's optimal PEEP level for MV is unknown because of heterogeneous lung units and the patient physiological status (e. g. volume status and body mass index). Thus, optimal PEEP levels vary and should be individualized.

Low PEEP levels ($\leqslant$5 cmH_2O) may not reliably reverse atelectasis or improve arterial oxygenation. In addition, zero or minimal PEEP leads to increased lung inflammation and is even associated with an increased risk of 30-day mortality[37-38]. High PEEP may compromise circulation. Although high PEEP (>12 cmH_2O) reduces the incidence of intra-tidal recruitment/derecruitment and does not worsen overdistension compared with low PEEP ($\leqslant$2 cmH_2O) in randomized controlled trials, high PEEP does not protect against PPCs[39-40].

In summary, protective TV should include optimal PEEP or recruitment maneuvers to reach beneficial effects to reduce PPCs. Use of MV incorporating at least 5 cmH_2O PEEP at starting level seems plausible for unaffected lungs.

2.2.3 Recruitment maneuvers

Recruitment maneuvers are often performed using the "bag-squeezing method," which consists of switching the ventilator to manual mode, setting a maximum pressure with the adjustable airway pressure-limiting valve of the anesthesia machine, and squeezing the bag. Recruitment maneuvers are effective rescue interventions to increase oxygenation and revert desaturation due to alveolar collapse; however, their routine preventive use is not recommended for unaffected lungs[35].

2.2.4 FIO_2

FIO_2 decreases after induction of general anesthesia. FIO_2 should be set at < 60% to ensure adequate oxygenation and decrease the risk of resorption atelectasis because higher values are associated with worse outcomes[41]. In one large, single-center, retrospective database study of 73 922 cases, median FIO_2 (79%) was associated with a dose-dependent increase in major respiratory complications (re-intubation, respiratory failure, pulmonary edema, and pneumonia) developed within 7 days after surgery and 30-day mortality across a range of low to high FIO_2 (0.31 to 0.79)[42].

2.2.5 Ventilation modes

Volume-controlled ventilation and pressure-controlled ventilation are widely used in intraoperative assistance; no strong evidence reveals either one of them can improve patients' outcomes[43-44].

2.2.6 Driving pressure

Driving pressure is defined as the difference between plateau pressure and PEEP (Pplat-PEEP); it is the result of the interaction between the ventilator and the respiratory system[45]. Increasing driving pressure has been identified as a significant predictor of mortality and PPCs[46-47]. Furthermore, application of a lung-protective strategy can lead to a decrease in driving pressure, which then results in a reduction in the risk of PPCs. However, even with the use of a lung-protective strategy, the increases in driving pressure caused by an increase in PEEP were associated with a significant prediction of PPCs[47]. Because of the strong correlation between driving pressure and clinical outcomes such as mortality and PPCs, the driving pressure should be kept as low as possible.

2.2.7 Respiratory rate

Respiratory rate is determined by maintaining adequate carbon dioxide exchange and with the goal to achieve end-tidal CO_2 ($ETCO_2$) in the range of 4.7–6.0 kPa or 35–45 mmHg. Respiratory rate is usually set at 8 to 12 breaths per minute and then adjusted according to $ETCO_2$. So far, no studies have confirmed the exact impact of respiratory rate on VILI and PPCs; however, a high respiratory rate may be needed with the use of low TV in lung-protective ventilation to adequately clear CO_2, especially with CO_2 insufflation for laparoscopic surgery.

2.3 Individual strategies, or a combination of strategies for intraoperative lung-protective ventilation

The combined use of low TV, moderate PEEP, and recruitment maneuvers seems to reduce PPCs in patients without lung injury. A recent review by Guay et al[4]

included 19 studies, 776 participants without ALI in the low TV (6.0–8.1 mL/kg) group and 772 in the high TV (10–12 mL/kg) group. Other factors such as PEEP at any level and recruitment maneuvers were also considered in the review. Participants were scheduled for heart, abdominal, spinal, pulmonary thromboendarterectomy, and knee surgery. They found moderate-quality evidence that low TV decreases pneumonia and the need for postoperative ventilatory support (invasive and non-invasive). Furthermore, the effect of low volume ventilation on barotrauma (pneumothorax) was uncertain. Additionally, protective ventilation may not modify the overall mortality rate[5-6, 48-49].

Studies have also investigated the benefits of individual components of protective ventilation. The protective ventilation using high versus low positive end-expiratory pressure ("PROVHILO") trial[40] explored the benefits of PEEP and recruitment maneuvers with fixed TV and found that high PEEP and alveolar recruitment alone may not improve oxygenation. On the contrary, high PEEP is associated with a higher incidence of intra-operative hypotension[40]. The results with low TV ventilation alone were also somewhat conflicting. While two Meta-analyses of 4 700 cases reported a significantly lower rate of ALI with low TV alone, they did not standardize the protocol for PEEP and recruitment maneuver. When controlled for PEEP and recruitment maneuver, low TV did not reduce the rate of ALI[31, 50].

2.4 Additional considerations

2.4.1 Fluid administration and transfusion

Perioperative goal-directed fluid therapy, an optimized fluid management strategy that provides a better prognosis for perioperative patients, has become an important measure for perioperative care. Dynamic markers of fluid responsiveness such as stroke volume variation and pulse pressure variation are thought to be superior to static markers such as cardiac output. In addition, a restrictive transfusion strategy with the goal of targeting 7 g/dL in the postoperative intensive care unit (ICU) period should be used.

2.4.2 Anesthetic choice

Volatile anesthetics may provide protection in perioperative lung injury, as they have immune-modulatory properties by inhibiting the expression of pro-inflammatory mediators; however, they do have a negative effect on hypoxic pulmonary vasoconstriction.

3 Postoperative assistance

General anesthesia and postoperative analgesia impair the cough reflex, immune response, mucociliary clearance, and surfactant production. All these impairments must be promptly addressed with appropriate strategies; otherwise they will lead to PPCs[51-52].

3.1 Non-invasive respiratory support

Non-invasive ventilation (NIV), including continuous positive airway pressure (CPAP) and bi-level positive airway pressure (BPAP), can not only rapidly relieve dyspnea and reduce respiratory work, but also help to avoid re-intubation induced by potential and variable causes of respiratory failure. Therefore, NIV has been used frequently to prevent the occurrence of acute respiratory failure (ARF) or to treat ARF to avoid re-intubation[53]. The ability of NIV/CPAP to avoid further deterioration of respiratory function makes it an option for high-risk patients or patients with mild PPCs. However, there is still no strong evidence to recommend the routine use of NIV/CPAP, so further randomized trials are needed to prove their benefit[54-55].

CPAP can be equipped with high flow systems with general purpose or dedicated fans. Stand-alone devices that operate as long as oxygen is delivered are available, but each type of device performs differently[56]. BPAP can be equipped with a dedicated or ICU ventilator. Clinicians should therefore have expertise with the various available interfaces and be familiar with the most common NIV settings to maximize patient compliance. The most common two-layer ventilation mode after surgery is based on pressure-supported ventilation (PSV), which has different names depending on the device vendor. PSV is flow-triggered, pressure limited, and flow-cycled; the ventilator provides a higher-pressure level when the patient's inspiratory force is detected, and when the inspiratory flow is below a certain value, the ventilator circulates toward the expiratory phase. In most ICU ventilators, PEEP and the drive pressure or inspiratory peak pressure are set, and the inspiratory stop criterion is usually set based on the percentage of the peak inspiratory flow. In a dedicated NIV ventilator, the operator must select the inspiratory and expiratory pressures

corresponding to the peak pressure of the ICU ventilator and PEEP. Only updated NIV fans allow the operator to manually set the cycle standard. Compared with standard oxygen therapy, NIV can reduce the rate of short-term re-intubation[57].

For patients with postoperative hypoxic respiratory failure, CPAP or BPAP should always be considered to avoid re-intubation. However, when hypercapnia is predominant or coexists with hypoxemia, BPAP is preferred. CPAP and BPAP setups are easy, and the development of new interfaces is improving their tolerance. In hospitals with adequately-trained staff, NIV should also be encouraged outside the ICU.

3.2 High-flow nasal oxygen

In recent years, the use of humidified high-flow nasal cannulas (HHFNCs) has become an effective method for the treatment of hypoxic respiratory failure. They are thought to be an intermediate step between traditional oxygen therapy and NIV, but their role in the perioperative period remains to be determined. These devices are usually tolerable. They can also be used in surgical wards because they can be operated with small, dedicated, humidified flow generators. In a study of patients undergoing cardiothoracic surgery, HHFNCs did not reduce re-intubation rates any more than NIV[58]. However, in an observation of postoperative patients after major abdominal surgery, HHFNCs were not superior to conventional oxygen therapy in preventing postoperative hypoxemia[59]. Since reversal of postoperative atelectasis is necessary for the recovery of respiratory function, the minimum positive pressure effect HHFNCs can achieve is now believed to be the main mechanism to explain HHFNCs' lack of postoperative effectiveness[60].

4 Conclusion

The use of low TV, moderate PEEP, and recruitment maneuvers have been shown to improve oxygenation and pulmonary physiology and reduce PPCs in at-risk patients. Although definitive evidence of its benefit for the general surgical population is lacking, we still suggest using lung-protective ventilation for ALL patients who receive MV during anesthesia or in other settings like ICUs. The primary goals of intraoperative ventilation are to provide non-harmful ventilation and open the alveoli and keep them open into the postoperative period. For most patients, we recommend the use of low TV (i.e., 6 to 8 mL/kg), an initial PEEP of 5 cmH_2O (10 cmH_2O during laparoscopy), and plateau pressures $\leqslant$16 mmHg, during anesthesia. Recruitment maneuvers should be performed to improve oxygenation when indicated in specific patient populations (e.g., obese patients, pediatric patients, during open abdominal surgery, and before, during, and after insufflation for laparoscopy and thoracoscopy), and in specific circumstances (e.g., after disconnecting from the ventilator for suctioning). Further studies to delineate the underlying mechanisms of VILI, particularly the IL-33-ST2 pathway and WISP1-TLR4-integrin β5 pathway, will guide our clinical practice to minimize VILI. Larger scale, clinical, practical human studies are needed to validate these findings and potentially contribute to a much lower incidence of mortality and morbidity associated with perioperative VILI.

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