Editorial

Pro: Low Tidal Volume Is Indicated During One-Lung Ventilation

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Since the introduction of one-lung ventilation (OLV) as a standard practice during thoracic surgery in the first half of the last century, anesthesiologists have had to deal with the problem of maintaining adequate intraoperative arterial oxygenation while ventilating only one of the patient's lungs. Evolution in this management has been one of the great success stories in the practice of anesthesiology, as the incidence of hypoxemia during OLV has declined from 20%–25% in the 1970s (1) to <1% today (2). This can be attributed to several advances, including the use of fiberoptic bronchoscopy for double-lumen endobronchial tubes and bronchial blocker positioning and the use of newer volatile anesthetics that cause less inhibition of hypoxic pulmonary vasoconstriction and less shunt during OLV than older volatile anesthetics (3). Oxygenation with ≤1 MAC of the newer volatile anesthetics is equivalent to that seen during total IV anesthesia (4). Further success with management of OLV can be attributed to ventilation strategies that minimize atelectasis. One prevalent approach is ventilation with large tidal volumes. Common teaching in fact has been to use similar tidal volumes (10–12 mL/kg) for both two-lung ventilation and OLV (5).

The incidence of hypoxemia during routine OLV has become so infrequent that the question arises whether these large tidal volumes may injure the lung. Acute lung injury (ALI) after pulmonary resection has been described since the use of OLV for thoracic surgery became routine. The most publicized report is a compilation of 10 cases after pneumonectomy published in 1984 (6), which focused on the role of IV overhydration as a cause of post-pneumonectomy pulmonary edema. Subsequently, there have been several reviews of this topic identifying a variety of other potentially causative factors for ALI, such as the administration of fresh-frozen plasma, mediastinal lymphatic damage, inflammation, and oxygen toxicity (7). The most thorough study to date (8) is a retrospective survey of 806 pneumonectomies that found 21 cases (2.5%) of post-pneumonectomy pulmonary edema, which represents one of the lowest incidences reported of this complication. There were no differences in perioperative fluid balance between post-pneumonectomy ALI (positive fluid balance at 24 h: 10 mL/kg) versus matched pneumonectomy controls (positive fluid balance at 24 h: 13 mL/kg). These authors used rigorous fluid restriction compared with other reports (9) (e.g., 24-h positive balance: 21 ± 9 mL/kg), suggesting that limiting intraoperative fluids might decrease but not eliminate ALI. Further reports demonstrate improved survival from post-pneumonectomy pulmonary edema, likely as a result of improved postoperative management of established cases (10).

Post-pneumonectomy ALI (11) has been found to have a bimodal distribution of onset. Late cases (10/37, 27%) presented 3–10 days postoperatively and were secondary to obvious causes such as bronchopneumonia and aspiration. "Primary" ALI (27/37, 73% of cases) presented on

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postoperative days 0–3. Four factors were independent significant predictors of primary ALI: high intraoperative ventilation pressures, excessive IV volume replacement, pneumonectomy, and preoperative alcohol abuse. The known facts about ALI after lung surgery, thus, include an incidence of 2%–4% after pneumonectomy (<1% post-lobectomy); increased frequency in right versus left pneumonectomies; symptomatic onset 1–3 days after surgery; frequent associated mortality (25%–50%); and resistance to standard therapies.

Although there is some association between postoperative ALI with excessive intravascular volume, the finding of low/normal pulmonary artery wedge pressures and high-protein edema fluid in affected patients suggests a role of endothelial damage (low-pressure pulmonary edema). The finding of postoperative increases in lung capillary permeability of the non-operated lung is primarily after pneumonectomy but not lobectomy (12). This capillary-leak injury may be attributable to an inflammatory cascade affecting even the non-operative lung that is triggered by lung resection and is proportional to the amount of lung tissue resected (13,14). Free oxygen radical generation in lung cancer patients is related to the duration of OLV (15). Nonetheless, there is no single mechanism that can fully explain ALI after lung resection and its etiology is likely multifactorial. A unifying hypothesis is that post-pneumonectomy pulmonary edema is one end of a spectrum of ALI that occurs during all lung resections. The more extensive the resection the more likely there is to be a postoperative injury. The increased dissection and trauma associated with extrapleural pneumonectomy places these patients at high risk to develop postoperative ALI (16).

Understanding that lung endothelial injury occurs after lung resection supports management principles similar to other conditions associated with ALI and acute respiratory distress syndrome (17). Studies in the latter group of patients demonstrate that ALI is exacerbated by the use of large tidal volumes and that lung-protective ventilation strategies with small tidal volumes and positive end-expiratory pressure (PEEP) are less injurious (18). The most important factor in the etiology of ventilator-induced lung injury is the end-inspiratory lung volume (19). Many patients, particularly those with emphysema, develop auto-PEEP during OLV (20), thus beginning inspiration at a lung volume above functional residual capacity. It is conceivable that routine use of a large tidal-volume (10–12 mL/kg) during OLV in such patients produces end-inspiratory lung volumes close to levels that contribute to ALI.

One must acknowledge that changes in respiratory function during OLV in the lateral position with an exposed nondependent hemithorax are complex. Initial studies of the application of PEEP during OLV suggested that it led to a deterioration of arterial oxygenation (21). It is now appreciated that the effects of applied PEEP during OLV depend on the lung mechanics of the individual patient. Most patients with chronic obstructive pulmonary disease develop auto-PEEP during OLV and thus external PEEP leads to hyperinflation and increased shunt (22). However, patients with normal lung parenchyma, or those with restrictive lung diseases, tend to fall below their functional residual capacity at end-expiration during OLV and may benefit from applied external PEEP (23).

Regardless, a central question in the debate of "low" versus "high" tidal volume ventilation during OLV is whether there is evidence that the routine use of the latter traditional approach may cause ALI in patients with healthy lungs. There is an accumulated experience of OLV in thoracic surgery over the past 60 yr, in fact, supporting the safety of OLV as currently practiced. A study of either large (12–15 mL/kg) or small (6 mL/kg plus added PEEP 10 cm H_2O) tidal volume ventilation during OLV could not demonstrate differences in inflammatory cytokines in serum or tracheal aspirates (24). This study was not powered to evaluate more important outcomes, leaving open the question of the effects of either method on ALI.

However, there is evidence that when an element of lung injury is added, large tidal-volume ventilation during OLV contributes to ALI. In a rabbit model of OLV during isolated perfusion, large tidal-volume (8 mL/kg) ventilation produced a picture of ALI absent in animals randomized to a lung-protective ventilation pattern (4 mL/kg plus PEEP). Another consideration is management of patients who have received preoperative chemotherapy with drugs such as cisplatinum and gemcitabine that may affect respiratory function and may increase the risk of postoperative respiratory complications, including ALI in some patients (25). Finally, larger lung resections (e.g., pneumonectomy or bi-lobectomy) are associated with some degree of ALI. ALI, diagnosed radiographically, was reported in 42% of pneumonectomy patients who had been ventilated with peak airway pressures >40 cm H_2O (26). A recent retrospective study found that post-pneumonectomy respiratory failure was associated with the use of higher intraoperative tidal volumes (8.3 mL/kg versus 6.7 mL/kg in pneumonectomy patients who did not develop respiratory failure) (27).

Because it is not always possible to predict which patient with a planned lobectomy may require a pneumonectomy for complete tumor resection, the routine use of several strategies during OLV seems logical. Over-inflation of the non-operated lung should be avoided by using lung-protective ventilation (5–6 mL/kg) adding PEEP to those patients without auto-PEEP and limiting plateau and peak inspiratory pressures to <25 cm H_2O and < 35 cm H_2O, respectively. Minimizing pulmonary capillary pressures by avoiding over-hydration for patients undergoing pneumonectomy is reasonable, even

though not all increases in pulmonary artery pressures perioperatively are the result of intravascular volume replacement. Other factors, such as hypercarbia, hypoxemia, and pain, can all increase pulmonary pressures and must be treated. Finally, it must be appreciated that not all hyperinflation of the residual lung occurs in the operating room. Over-expansion of the remaining lung after a pneumonectomy may occur postoperatively either with or without a chest drain in place. The use of a balanced chest drainage system to keep the mediastinum in a neutral position and avoid hyperinflation of the residual lung after a pneumonectomy has been suggested to contribute to a marked decline in this complication in some centers (28).

In conclusion, ALI may occur in selected patients, particularly after pneumonectomy. Its occurrence is unpredictable, its cause is likely multifactorial, and its development is associated with frequent operative morbidity and mortality. Given the low risks, lung-protective ventilation using low-tidal volumes and the selective use of PEEP would seem to be a logical choice for management of OLV for thoracic surgery in the era of infrequent hypoxemia and continuous arterial oxygen saturation monitoring.

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