

Case Scenario: Management of Intraoperative Hypoxemia during One-lung Ventilation

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HYPOXEMIA that may reasonably be defined by an arterial hemoglobin oxygen saturation of less than 90% occurs in 5–10% of patients during one-lung ventilation (OLV).¹ The pathophysiology of hypoxemia is complex, and the management of intraoperative hypoxemia during OLV remains a challenge for anesthesiologists. In life-threatening hypoxemia, correct oxygenation should be restored rapidly. This may require discontinuing surgery to eliminate reversible causes of hypoxemia. Several strategies can then be applied to prevent and correct hypoxemia during OLV.

Case Reports

A 50-yr-old man undergoing resection of the upper right lung for neoplasia developed hypoxemia 15 min after the start of OLV. Preoperative examination revealed satisfactory cardiopulmonary status. Preoperative rest and dobutamine stress echocardiography revealed normal left ventricular function without any wall motion abnormalities. Doppler ultrasound examination of the carotid arteries was normal. Preoperative pulmonary function tests were normal without any obstructive disease (forced expired volume in 1 s was 3.7 l

and forced expired volume in 1 s/vital capacity was 73%). Blood electrolytes, hemoglobin concentration, and renal function were normal. There was no history of bronchopulmonary infection, and arterial blood gas values in room air were PaO₂ 83 mmHg, PaCO₂ 38 mmHg, pH 7.40, and HCO₃ 25 mm. Electrocardiogram showed regular sinus rhythm, and blood pressure on the morning of surgery was 128/65 mmHg. The patient refused epidural anesthesia for management of postoperative pain. Selective bronchial intubation of the left main-stem bronchus was performed with a 39F left-sided double lumen tube (DLT) (Broncho-part; Rüsch, Kernen, Germany). The correct position of the tube was immediately confirmed with a fiberoptic bronchoscopy, and the patient was then placed in the lateral position. Volume-controlled ventilation, including a 7 ml/kg tidal volume (V_T) of predicted body weight under 100% FIO₂, a 12 cycles/min respiratory rate, and a 5 cm H₂O positive end-expiratory pressure (PEEP), was used. End-tidal carbon dioxide and plateau pressure were 31 mmHg and 19 cm H₂O, respectively. No intrinsic PEEP was observed. OLV was initiated without any change in ventilator settings. Fifteen minutes later, the patient exhibited profound hypoxemia with a significant decrease in pulse oxymetry from 94% to 88%. Arterial blood gas values were pH 7.41, PaO₂ 52 mmHg (100% FIO₂), PaCO₂ 40 mmHg, HCO₃ 24.8 mM, and 87% SaO₂. The patient's hemodynamics and electrocardiogram remained stable. Expiratory flow and expiratory V_T were unchanged, and no leak was noticed. The correct position of the DLT was immediately confirmed by fiberoptic inspection. End-expiratory flow was not interrupted by the next insufflation and reached zero before the next respiratory cycle, therefore no dynamic hyperinflation or intrinsic PEEP (iPEEP) was observed. Once the surgeon was informed, the nondependent lung (nonventilated lung) was expanded manually by administration of pure oxygen, and a continuous positive airway pressure (CPAP) at 5 cm H₂O was subsequently applied. This strategy allowed rapid improvement of the patient's oxygenation, and oxymetry pulse could be maintained above 95% throughout the surgical procedure under OLV. During the manual expansion of the nondependent (nonventilated) lung, hemodynamics remained stable.

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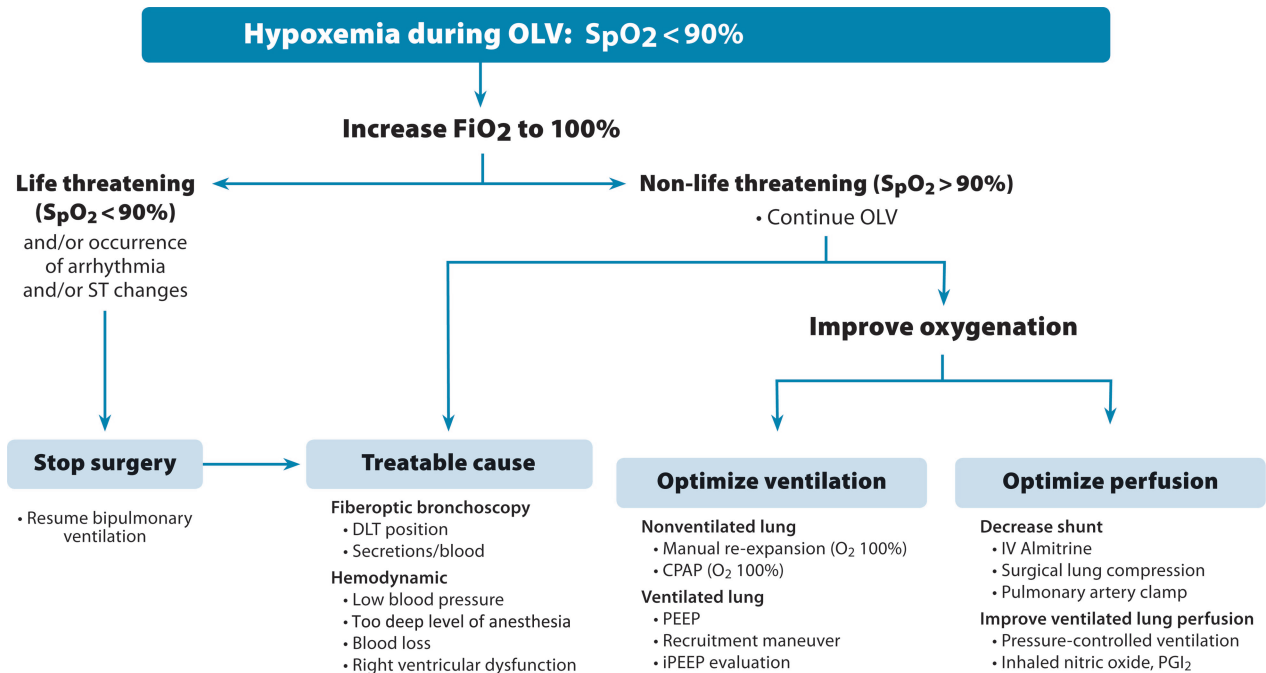


Fig. 1. Algorithm recommended to be performed in case of hypoxemia during one-lung ventilation (OLV). CPAP = continuous positive airway pressure; DLT = double lumen tube; FiO_2 = inspired fraction of oxygen; PEEP = positive end-expiratory pressure; PGI_2 = prostacyclin; ST = ST segment.

Because the surgical procedure was a right open thoracotomy, it was not impeded by the application of CPAP to the nondependent lung, which was stopped at the end of OLV. The postoperative course of this patient was uneventful. He did not exhibit any hypoxemia after tracheal extubation.

Discussion

Management of Intraoperative Hypoxemia

The first step when a patient suffers from hypoxemia (*i.e.*, a drop in pulse oxymetry less than 90%) during OLV is to stop surgery, increase the inspired oxygen fraction to 100%, and rapidly restore two-lung ventilation (TLV). That can be performed with a manual reexpansion of the lung using the hand bag with pure oxygen flow and an airway pressure limited valve between 20 and 30 cm H_2O ; the reexpansion of the deflated lung is visual. Once acceptable oxygenation has been restored, two simultaneous actions must be performed: (1) a reversible cause of hypoxemia must be ruled out before restarting surgery and (2) oxygenation of the patient must be improved to prevent the recurrence of hypoxemia. Because hypoxemia during OLV is mainly related to ventilator or perfusion mismatch, strategies to restore oxygenation should have two objectives: to improve alveolar ventilation and pulmonary perfusion. The first consists of restoring alveolar ventilation of the nondependent lung by applying oxygen inflation with CPAP and of the dependent lung by performing a recruitment maneuver and applying PEEP. Perfusion, on the other hand, involves strategies to limit intrapulmonary shunt: positioning of the patient (strict lateral decubitus po-

sition on the side of the dependent lung provides better oxygenation than semilateral or even supine position), surgical lung compression or clamping the pulmonary artery of the nondependent lung, limitation of airways pressure of the ventilated lung including limitation of intrinsic PEEP, and the use of moderate V_T (6–8 ml/kg) or pressure-controlled ventilation. Some pharmacological treatments, such as nitric oxide and almitrine, can also be used to decrease intrapulmonary shunt. An algorithm for the treatment of hypoxemia during OLV is summarized in figure 1.

Diagnosis of a Treatable Cause of Hypoxemia

Because dislodgment of the DLT is a common cause of hypoxemia, the first step is to check that the DLT is in the correct position and to clear secretions or blood from the tube or the respiratory tract. Initially, blinded bronchoaspiration can be performed on both sides of the DLT. It is sometimes sufficient to clear proximal secretions and blood. Clinical examination of the correct position of the DLT with auscultation and inspection of the DLT is necessary but sometimes fails to identify DLT malpositioning. Consequently, correct positioning of the DLT must be systematically confirmed by fiberoptic.^{1,2} Fiberoptic bronchoscopy must be repeated once hypoxemia occurs or in the presence of increased airways pressure. The DLT can be misplaced leading to impaired oxygenation and inadequate lung separation with high airway pressure or significant leak.² Hemodynamic conditions also need to be assessed. Although patients undergoing lung resection are vulnerable to fluid overhydration, the maintenance of cardiac output, mainly by

fluid loading, is required for that oxygen delivery to meet patient metabolic needs. As OLV induces increase in right ventricular overload, intraoperative assessment of ventricular function may be useful.³ Right ventricular dysfunction may also be related to volume overload or intrinsic myocardial depression.³ Despite some limitations in the analysis of right ventricular function, transesophageal echocardiography is probably the best intraoperative monitoring technique to use during OLV.³ The two main signs that must be sought are the right ventricular dilatation as well as paradoxical septal motion.⁴ Moreover, maintenance of an adequate level of anesthesia allows limiting hemodynamic disturbances related to a too-deep level of anesthesia.

How Can Ventilation of the Nondependent Lung be Improved?

Application of CPAP. Although it is frequently applied, increasing F_{IO_2} in the ventilated lung is often not sufficient to improve oxygenation because OLV is responsible for an important shunt-like effect of approximately 30%.⁵ Administration of oxygen to the nondependent lung is usually used to treat hypoxemia during OLV, but it may also be used to prevent it. Oxygen may be administered with or without CPAP of 5–10 cm H_2O to the nondependent lung. If no CPAP valve is available, the hand bag with airway pressure limited valve can be used with a concentration of 5–10 cm H_2O . The oxygen flow and airway pressure limited valve must be adapted to obtain inflation without interfering with surgery. After informing the surgeon, transitory reinflation of the collapsed lung is necessary by applying a higher airway pressure between 20 and 30 cm H_2O and then using CPAP to keep the lung at a constant level of inflation; the reexpansion of the lung is visual. Oxygen flow and CPAP are very effective at improving oxygenation during OLV. A CPAP concentration of 5–10 cm H_2O is usually sufficient and does not seem to impede open-chest surgery.^{6,7} Only rarely does hypoxemia during OLV persist when CPAP is applied. In this case, surgery should be transitorily stopped, and a switch to TLV should be performed. However, OLV should be maintained when hypoxemia exists preoperatively or when emergency thoracic surgery is required (hemoptysis or postoperative anastomosis leakage).

How Can Ventilation of the Dependent Lung Be Improved?

Application of PEEP. Conflicting results have been reported concerning the application of PEEP to the dependent lung during OLV.^{8,9} In some patients, the application of PEEP to the dependent (ventilated) lung might be beneficial by restoring functional residual capacity to normal values,¹⁰ thus preventing atelectasis when its value is titrated along the static compliance curve.¹¹ However, PEEP may also paradoxically impair oxygenation by shifting pulmonary blood flow to the nondependent lung, thereby increasing intrapulmonary shunt. It was commonly thought that a similar tidal

volume should be used for both TLV and OLV. Nevertheless, use of a large tidal volume during OLV produces end-inspiratory lung volumes close to concentrations that contribute to acute lung injury.¹² When small V_T are used, especially during high F_{IO_2} , more atelectasis may occur, worsening oxygenation.¹³ Therefore, to avoid atelectasis, PEEP should be applied when V_T is small. Valenza *et al.*¹⁴ showed that oxygen responders to 10 cm H_2O PEEP during OLV were patients in whom forced expiratory volume in 1 s was more than 72%, and that these patients were less likely to develop iPEEP. In obstructive patients, the use of PEEP may sometimes interfere with iPEEP and decrease oxygenation.^{9,11,15} No adverse events have been reported in patients in whom a PEEP up to 10 cm H_2O was applied. However, when alveolar pressure is increased, the risk of barotrauma must be considered. Morbidly obese patients given anesthesia exhibit more severe impairment of respiratory mechanics and gas exchange than normal subjects.¹⁶ PEEP improves respiratory function and oxygenation in these patients, and this is significantly correlated with alveolar recruitment.^{16,17} Therefore, the use of PEEP has to be considered a useful tool to improve alveolar ventilation of the lung during OLV, but its effect on dependent lung perfusion, and therefore on oxygenation, has to be monitored by arterial blood gases.

Alveolar Recruitment Maneuvers

During OLV in the lateral position, atelectasis zones in the dependent lung contribute to ventilation/perfusion mismatch. These disturbances tend to increase shunt in the nondependent lung. Short-term application of unusually high airway pressure can restore near normal lung volumes at end-expiration and improve oxygenation. Tusman *et al.*¹⁸ examined the beneficial effects of an alveolar recruitment strategy with pressure-controlled ventilation (PCV) during OLV. Using alveolar recruitment, they reported that values of P_{aO_2} were almost as high as during TLV. As Rothen *et al.*¹⁹ pointed out, in anesthetized patients without lung injury, peak airway pressure of 40 cm H_2O is required to open atelectatic zones. Alveolar recruitment should be performed progressively and transiently with PCV to avoid or reduce hemodynamic adverse effects. Similarly to PEEP, increasing alveolar pressure may be responsible for barotrauma. This has not been described during OLV, but it is important to bear this complication in mind because a pneumothorax in the dependent lung during OLV with a patient in the lateral position is difficult to diagnose and treat rapidly.

Switch to PCV

PCV has been suggested to reduce peak airway pressure and intrapulmonary shunt, thereby improving oxygenation and limiting the risk of barotrauma.²⁰ The benefits of PCV can be explained by the reduction in dynamic airway pressure. Compression of small intraalveolar vessels during inflation of the alveoli produces increased resistance to pulmonary blood flow in the dependent lung and tends to divert blood to the

nonventilated alveoli of the nondependent lung. However, the latter is most closely related to the mean airway pressure or, more specifically, the mean alveolar pressure. Our group has recently demonstrated that peak airway pressure is modestly affected by a switch from volume-controlled ventilation to PCV during OLV when it was measured inside the main ventilated bronchus.²¹ Our results suggest that the beneficial effect of PCV during OLV is not mediated by a decrease in peak pressure. To date, the improvement in oxygenation induced by PCV compared with volume-controlled ventilation during OLV remains controversial.^{22–24}

How Can Pulmonary Perfusion be Improved?

Pharmacological Treatment to Limit Hypoxic Pulmonary Vasoconstriction. During OLV, it is possible to modulate hypoxic pulmonary vasoconstriction (HPV) using inhaled nitric oxide or almitrine. Almitrine is a peripheral chemoreceptor agonist that potentiates HPV. Inhaled nitric oxide only accesses the ventilated alveoli, where it causes localized vasodilation and increases perfusion to the adequately ventilated region of the lung. Almitrine, although delivered systemically, enhances constriction primarily under hypoxic conditions, and thus, at low doses, selectively enhances HPV in the nonventilated operative lung. Moutafis *et al.*²⁵ showed that inhaled nitric oxide alone (20 ppm) in patients undergoing OLV did not improve oxygenation. However, when inhaled nitric oxide was associated with intravenous almitrine ($16 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$), arterial partial oxygen pressure was significantly increased. Inhaled nitric oxide and PEEP could have a synergistic effect on oxygenation.²⁶ However, Michelet *et al.*²⁶ found that inhaled nitric oxide only improved oxygenation when PEEP was high (*i.e.*, 15 cm H₂O), and it was associated probably with overdistension. Intravenous almitrine can also be used alone.²⁷ A continuous infusion of $8 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ limits OLV-induced hypoxemia without causing any hemodynamic disturbances in patients without pulmonary hypertension.²⁷ An adverse effect of almitrine is elevation of pulmonary vascular resistance, which, if excessive, could potentially induce right ventricular dysfunction. It is possible to use almitrine as a treatment strategy in patients with hypoxemia during video-assisted thoracoscopy where CPAP and intermittent TLV are impossible to use because they will interfere with surgery. Almitrine is not approved for clinical use in North America because of the toxicity of the drug, which includes peripheral neuropathy.

The Choice of Anesthetics

The influence of inhalational anesthetics *versus* intravenous anesthetic agents on HPV remains a large subject of controversy. Animal^{28,29} as well as human^{30,31} studies found that isoflurane is associated with increased intrapulmonary shunt fraction as a result of HPV inhibition during OLV. Conversely, inhalation anesthetics, by affecting cardiac output, may decrease perfusion of the nondependent lung, and thus

decrease shunt fraction. This could explain that HPV may be significantly affected by volatile anesthetics without any clinically relevant changes in oxygenation.³² Moreover, a recent clinical study suggests that sevoflurane attenuates the effect of OLV on the pulmonary inflammatory response and improves clinical outcome.³³

The Surgeon Can Help to Limit Hypoxemia during One-lung Ventilation

The surgeon has access to the perfusion of the nondependent lung. Consequently, he/she is able to reduce pulmonary blood flow, and therefore transpulmonary shunt. Ishikawa *et al.*³⁴ showed that repeated compression of the nondependent lung using a retractor during the surgical procedure may significantly improve oxygenation in patients undergoing OLV. These authors did not report whether compression of the pulmonary vessels induced complications. This strategy is almost never used in clinical practice and is certainly not feasible during a video-assisted thoracic procedure. Likewise, during pneumonectomy, the pulmonary artery of the nondependent lung may be rapidly ligated, and this will immediately improve oxygenation by significantly reducing the shunt-like effect.

The potential therapeutic target for improving oxygenation during one-lung ventilation is summarized in figure 2.

Basic Science

During OLV anesthesia in the lateral position, the pulmonary shunt-like effect ranges from 15% to 40% because of total collapse of the nondependent lung and an increase in atelectatic areas in the dependent lung.⁵ The consequence is a profound ventilation-perfusion mismatch resulting in arterial hypoxemia. PaO₂/FIO₂ ratio is often less than 200 and sometimes even less than 100.³⁵ Because transpulmonary venous shunting depends on the amount of cardiac output that is not oxygenated, the less the perfusion of the nonventilated lung and the more the perfusion of the ventilated lung, the higher the PaO₂ during OLV. One of the reasons why the pulmonary shunt-like effect during OLV is less than 50% is HPV. HPV is unique to the pulmonary circulation because most systemic beds dilate in response to hypoxia. The stimulus for HPV is primarily the alveolar partial pressure of oxygen, consistent with the localization of the oxygen sensor in the resistant pulmonary arteries.³⁶ The onset of HPV is rapid, occurring within 7 s in isolated lungs. Conversely, it resolves within minutes after normoxic ventilation is restored.³⁷ There is considerable variation in the strength of HPV, even at sea level. For example, many patients with previous high-altitude pulmonary edema have unusually strong HPV, which may contribute to their high-altitude pulmonary edema susceptibility.³⁸ In terms of genetic variability, there are individuals who are perfectly healthy but have weak HPV. This may explain why young patients without any comorbidity, undergoing thoracoscopy, sometimes develop life-threatening hypoxemia during OLV.

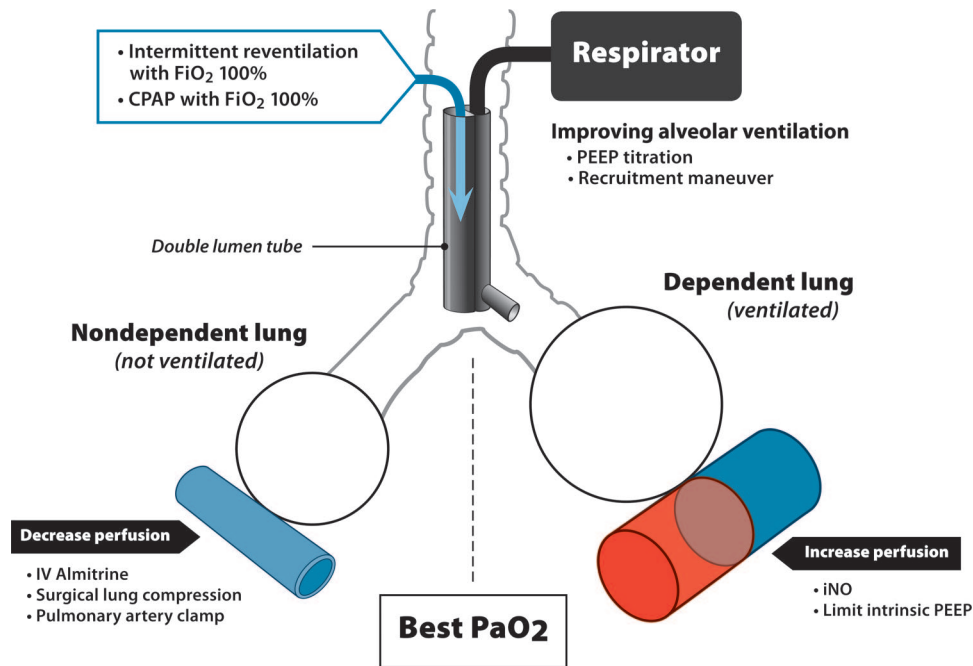


Fig. 2. Potential therapeutic target for improving oxygenation during one-lung ventilation (on this figure, the left lung is the ventilated lung). CPAP = continuous positive airway pressure; FiO_2 = inspired fraction of oxygen; HPV = hypoxic pulmonary vasoconstriction; iNO = inhaled nitric oxide; iPEEP = intrinsic PEEP; PaO_2 = arterial partial pressure of oxygen; PEEP = positive end-expiratory pressure.

Epidemiology and Risk Factors for Intraoperative Hypoxemia during OLV

In-hospital mortality is estimated to be approximately 6% after pneumonectomy. In a recent survey, an analysis of the European and French Thoracic Surgery Database identified the following independent risk factors for perioperative death: male sex, age, functional performance status, dyspnea score, American Society of Anesthesiologist score, and extent of resection.³⁹ During the procedure, depending on the definition, the rate of hypoxemia occurring during OLV has been estimated to be between 4% and 10%.^{35,40,41} However, prediction of intraoperative hypoxemia is much less accurate. The individual response to OLV is hard to predict because it relies on a complicated interaction between the patient's genetic background (in terms of HPV response),^{36,37} preoperative function tests,³⁵ underlying disease, anesthetic management, and surgical procedure.

Lung Function Abnormalities and Hypoxic Pulmonary Vasoconstriction

As reported previously by Slinger *et al.*,⁴² low intraoperative PaO_2 during TLV may predict hypoxemia during OLV. The distribution of perfusion between the two lungs can be measured preoperatively by perfusion scans and might be helpful to predict hypoxemia during OLV.⁴³ Ventilation and perfusion, and consequently shunt, will largely be influenced by the position and the size of the tumor. A large, central, compressing tumor will result in more progressive perfusion impairments because the lung is already partially preoperatively excluded from perfusion and, subsequently, a less intraoperative shunt-

like effect will occur once the lung with the tumor is not ventilated than a small, peripheral, noncompressing mass. Although other pulmonary function tests could be useful for predicting postoperative complications and outcome, their accuracy as reliable indicators of hypoxemia is unclear.

An inadequate response or failure of HPV will result in severe hypoxemia. The HPV response relies essentially on genetic background, but weak HPV should be expected in patients with chronic obstructive pulmonary disease or cirrhosis.^{44,45} In addition, HPV failure may be related to iatrogenic and anesthetic factors. Inhaled anesthetics, calcium channel blockers and other vasodilators, alkalosis, high pulmonary vascular pressure, and hypothermia decrease HPV and worsen oxygenation during OLV.⁴⁶ Although it is not possible to classify anesthetic management as a predicting factor for hypoxemia before surgery, airway management, fluid strategy, and ventilation remain a challenge in OLV. The importance of the preoperative workup must be emphasized, and a clear protocol for each patient, involving both the anesthesiologist and the thoracic surgeon as a team, should be decided before surgery. As mentioned above, oxygenation during OLV will be higher during left thoracotomy and in the lateral decubitus position. Surgical manipulations may impede expansion of the dependent lung, and mediastinal pressure can result in indirect compression or modification of cardiac output resulting in hypoxemia.

Knowledge Gap and Research Perspectives

Hypoxemia is a constant threat during OLV and the ventilation strategy is crucial to decrease its incidence. However,

as with the management of acute respiratory distress syndrome 10–20 yr ago, new concepts arise. To avoid intraoperative hypoxemia as the primary endpoint of OLV, the same V_T during TLV and OLV has been recommended as in the past. However, it is important to remember that ventilation can be harmful, and acute lung injury is now known to be a problem after thoracic surgery.

Is OLV Similar to Acute Respiratory Distress Syndrome?

Large volume ventilation might not be sufficient *per se* to induce acute lung injury and volume-induced lung injury in healthy lungs during short periods of anesthesia, but conditions are totally different during OLV and thoracic surgery. Use of high F_{IO_2} , frequent underlying pulmonary diseases, surgical manipulations, and resections provide a pathophysiologic basis for multiple hit aggression and subsequent susceptibility to acute lung injury and volume-induced lung injury. In both acute respiratory distress syndrome and OLV, clinicians will have to deal with a reduced lung volume and reduced functional residual capacity resulting in a heterogeneous lung and an increase in shunt and ventilation/perfusion mismatch. In these conditions, it is understandable that usual ventilatory setting in the past (high V_T and no positive end-expiratory pressure) might not be adequate. Gama de Abreu *et al.* demonstrated that OLV with high V_T (10 ml/kg) caused secondary lung injury in the dependent lung.⁴⁷ Licker *et al.*⁴⁸ found intraoperative ventilatory hyperpressure to be an independent risk factor for acute lung injury after lung resection. Likewise, a retrospective study found that postoperative pneumonectomy respiratory failure was associated with the use of higher intraoperative tidal volume during OLV.⁴⁹

Should Low V_T Be Used during OLV?

The ideal setting during OLV remains unclear. Michelet *et al.*¹² reported a decrease inflammatory response and improved lung function after OLV with protective low volume ventilation using a reduced V_T of 5 ml/kg and 5 cm H_2O PEEP. Schilling *et al.*⁵⁰ found similar results, including a decrease in alveolar inflammation. However, Shultz *et al.*,⁵¹ in a recent review of the literature, could not find clear data supporting the use of decrease V_T . The choice depends on whether the focus is on intraoperative oxygenation or on postoperative complications and clinical outcome. Focusing on intraoperative gas exchange, Katz *et al.*⁵² showed that large tidal volume ventilation produced the highest Pa_{O_2} . Although it is multifactorial, excessive V_T and inadequate settings may create an insult in susceptible patients, and acute respiratory distress syndrome network results should clearly alert the thoracic anesthesiologist to the potential repercussions of overinflation. It is noteworthy that the normal tidal volume in mammals is 6.3 ml/kg, and protective ventilation seems to be the right approach.

The consequence of a reduction in tidal volume is consistent alveolar derecruitment with the occurrence of atelecta-

sis. In acute respiratory distress syndrome patients, reducing end-expiratory lung volume or pressure can be injurious, even when tidal volume or peak pressures are controlled; conversely, increasing PEEP can be protective.⁵³ In fact, the coexistence of atelectatic and open alveoli may result in shear forces that exceed transpulmonary pressure.⁵⁴ Mechanical ventilation of areas of lung that are atelectatic is associated with repetitive collapse and reexpansion with each breath; this cyclic recruitment is also a source of pulmonary inflammation. Therefore, PEEP may play a key role in reducing pulmonary inflammation and maintaining oxygenation once V_T is reduced. A really important concept is the interaction between V_T and PEEP during bipulmonary ventilation and especially during OLV. Depending on the lung mechanics and perfusion status for any individual patient, there is a need to adjust ventilation strategies to keep the lung open without impeding perfusion and minimizing alveolar damage. As Karzai *et al.*¹ stated in their recent review on hypoxemia and OLV: “Clinically, there are no viable means of determining this best ventilation strategy in an individual patient. Therefore, patient condition, physiologic reasoning, and best available clinical evidence must be used to tailor a ventilatory strategy that best fits the needs of the individual patient.” The clinical postoperative benefits of lung protective strategies using decrease V_T combined with a certain concentration of PEEP during OLV are still to be demonstrated. In the meantime, it appears logical that decrease V_T with limited plateau pressure must be applied with the initiation of OLV. In addition to a reduction of V_T , PEEP is also required and should be adjusted to reduce alveolar derecruitment and maintain oxygenation with values probably between 5 and 15 cm H_2O . The interaction between PEEP and iPEEP during volume-controlled ventilation in chronic obstructive pulmonary disease patients is complex. In fact, Caramez *et al.* observed three specific responses during stepwise PEEP application in these patients during TLV: (1) paradoxical response with an increased isovolume expiratory flow and a decrease in functional residual capacity, plateau pressures, and total PEEP while external PEEP was applied; (2) biphasic response (no deleterious effect on expiratory-flow, functional residual capacity, plateau pressures, or total PEEP until external PEEP exceeded a specific concentration); and (3) classic overinflation (any application of external PEEP resulted in evident lung overinflation).⁵⁵ These three specific responses in chronic obstructive pulmonary disease patients during TLV will also have different effect on the dependent lung perfusion during OLV. Therefore, any modification of the concentration of PEEP during OLV in chronic obstructive pulmonary disease patients has to be controlled because the effects on thoracopulmonary compliance and oxygenation are unpredictable.¹¹

In conclusion, hypoxemia may occur in 5–10% of patients during OLV. The prevention and treatment of hypoxemia during OLV involves control of the correct position of the DLT by fiberoptic bronchoscopy and the use of an ap-

appropriate ventilation strategy.¹ High inspired oxygen fraction associated with CPAP can be applied to the nondependent lung, and this strategy is both effective and safe. Alveolar recruitment, higher concentrations of PEEP, and almitrine can also be used, but pure oxygen flow with CPAP with a constant acceptable inflation to the nondependent lung remains the safer strategy.

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