Hypoxia during Thoracic Surgery

Javier H. Campos, MD
Division of Cardiothoracic Anesthesia
Department of Anesthesia
University of Iowa Carver College of Medicine
Iowa City, Iowa

Learning Objectives:

As a result of completing this activity, the participant will be able to

- List the factors that predict hypoxia during one-lung ventilation
- Describe the different ventilation maneuvers to restore or improve arterial oxygenation
- Describe the effects of anesthetics on hypoxia and inflammation, as well as their protective effects on the lung

Double-lumen tubes or bronchial blockers are used to provide one-lung ventilation (OLV) in patients undergoing lung, esophageal, thoracic vascular, minimally invasive cardiac, and occasionally mediastinal surgery.[1,2] During OLV, an intrapulmonary shunt may result in hypoxemia related in part to collapse of the nondependent lung and increased atelectatic areas in the dependent lung.[3] Hypoxemia by definition is a decrease in oxygen saturation (SpO_2) to less than 90%[4] or an arterial oxygen tension (PaO_2) <60 mm Hg when the patient is being ventilated at an inspired oxygen fraction (FiO_2) of 1.0.[5] The incidence of hypoxemia during OLV has been reported to be 1–10%. This relative infrequency is related in part to advances with routine use of a fiberoptic bronchoscope for optimal placement of lung isolation devices. It is also attributable to the introduction of newer volatile anesthetics that cause less inhibition of hypoxic pulmonary vasoconstriction (HPV) in a dose-dependent fashion and less venous admixture during OLV.[6–9]

This review focuses on the predictors of hypoxia during OLV, the pathophysiology of HPV, protective ventilation maneuvers to restore or improve arterial oxygenation, the effects of anesthetics on hypoxia and inflammation.

PATIENTS AT RISK OF DEVELOPING HYPOXIA DURING OLV

Slinger et al,[10] using a regression analysis model in 80 patients undergoing OLV, showed that the three most significant predictors for PaO_2 were (1) side of operation (because the right lung is approximately 10% larger than the left lung, there is better oxygenation during left than right thoracotomy), (2) the percentage of forced expiratory volume in one second (FEV_1), and (3) reduced intraoperative PaO_2 during two-lung ventilation when patients were breathing spontaneously in a lateral decubitus position. Others[11,12] have shown better oxygenation during left-sided thoracic procedures as compared to right-sided surgeries when an FiO_2 of 1.0 is used.

Schwarzkopf et al.[11] reported that patients undergoing lobectomy and pneumonectomy had better oxygenation during OLV than patients undergoing video thoracoscopic metastasectomy. Lung perfusion studies in these patients showed that perfusion of the nonventilated lung was more impaired in patients presenting for pneumonectomy.

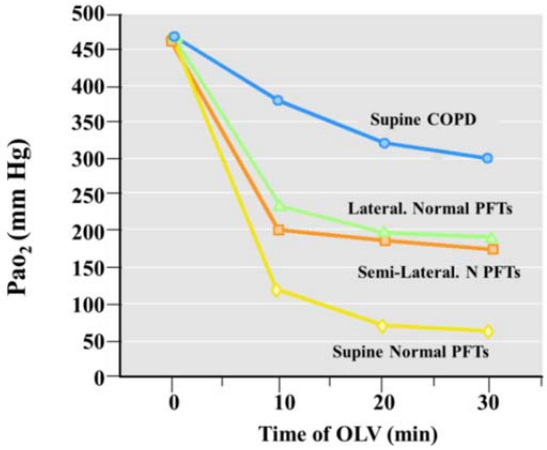
Another group of patients identified to be at risk of developing hypoxia are the morbidly obese. One study reported that patients with a body mass index >30 $kg \cdot m^2$ undergoing thoracic surgical procedures with OLV developed more intraoperative hypoxemia and increased alveolar oxygen difference than nonobese patients.[13] Also, patients with previous lobectomy requiring a second procedure in the contralateral lung may be at risk of developing hypoxemia during total lung collapse because 25% of the lung function was compromised in the previous lobectomy.[14]

The effects of the supine or lateral decubitus position on arterial oxygen tension will vary depending on whether or not the lung is exposed to OLV. In general, surgeons who perform thoracic surgery with OLV most commonly operate with the patient in a lateral decubitus position. Therefore, gravity is a major determinant of shunt fraction and perfusion.[15] Recent studies[16,17] have examined the changes in PaO_2 during procedures requiring OLV. In a study by Watanabe et al,[16] patients undergoing OLV were ventilated with an FiO_2 of 1.0 and divided into three groups. One group was placed in the supine position, another group in a left semilateral position, and the third group in left full-lateral position. In the supine position, 9 out of 11 patients had arterial oxyhemoglobin saturations <90% while receiving OLV. Only one patient each in the other two groups developed hypoxemia.

A study by Bardoczky et al[17] compared the positional effects and the inspired fraction of oxygen during OLV. Patients were randomly assigned and received different concentrations of oxygen (FiO_2 of 0.4, 0.6, or 1.0) during two-lung ventilation and thereafter OLV in the supine and lateral positions. PaO_2 decreased more during OLV compared to two-lung ventilation regardless of the position. However, in all three groups, PaO_2 was significantly higher during OLV in patients in the lateral compared to the supine position.

These studies clearly demonstrate that during OLV in a lateral decubitus position, gravity augments the redistribution of perfusion to the ventilated (dependent) lung, improving V/Q matching. Therefore, supine patients requiring OLV will likely experience more transient episodes of hypoxemia. Figure 1 displays PaO_2 values during OLV in the supine or lateral decubitus position in patients with normal lung function[16] or with chronic obstructive pulmonary disease.[17] Table 1 lists risk factors that will play a role in desaturation and hypoxemia.[18]

Figure 1:



This figure displays the PaO_2 values over time during one-lung ventilation (OLV) in supine-lateral and semi-lateral position. **(**Modified with permission from reference #18**)**

- Normal pulmonary function test (PFT) patients (Modified from reference #16)
- Chronic obstructive pulmonary disease (COPD) patients (modified from reference #17)

Table 1: Factors That Will Increase the Risk of Desaturation during OLV

- High percentage of ventilation or perfusion to the operative lung on preoperative V/Q scan
- Poor PaO_2 during two-lung ventilation in the lateral decubitus position
- Right-sided thoracotomy
- Normal preoperative spirometry (FEV_1 or FVC)
- Supine position during OLV
- Morbidly obese patient during OLV
- Previous lobectomy and contralateral lung collapse surgery

(Modified with permission from reference #18)

PATHOPHYSIOLOGY OF HYPOXIA DURING OLV

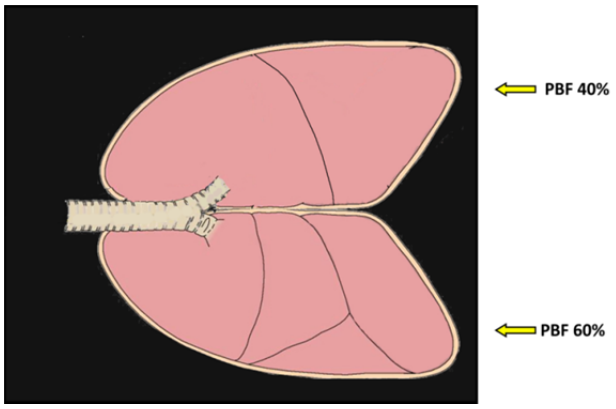
Hypoxemia during OLV is caused by venous admixture through shunts and areas of low V/Q gas exchanging units. During OLV, the collapsed, nondependent lung is an obligate shunt while the dependent lung also causes a venous admixture through the shunt and areas of low V/Q. V/Q mismatch is a consequence of atelectasis and is perhaps increased with the patient in the lateral decubitus position by the weight of the mediastinum, abdominal organs, retraction, and low compliance of the chest wall.[19]

The determinants of arterial oxygen content include hemoglobin concentration, hemoglobin dissociation curve (P_{50}), oxygen consumption, total cardiac output, FiO_2, arterial carbon dioxide level ($PaCO_2$), blood flow through the nonventilated (nondependent) lung, and unventilated or low V/Q areas of the ventilated lung (dependent lung). The last two factors are often associated together as shunt (Q) or shunt fraction (Qs/Qt).[20]

In a lateral decubitus position when both lungs are being ventilated, the proportion of the pulmonary blood flow is distributed as follows: the dependent (or down) lung receives approximately 60% of the pulmonary blood flow (more perfusion) while the nondependent lung receives 40% of the total pulmonary blood flow. When OLV is instituted, the nondependent lung becomes atelectatic. Because the alveolar arterial oxygen tension decreases, there is a response to hypoxia, HPV, in which increased pulmonary vascular resistance diverts blood flow toward the dependent lung. Figure 2A shows the redistribution of pulmonary blood flow in the lateral decubitus position while both lungs are being ventilated. Figure 2B displays the atelectatic nonventilated lung along with the percentage of HPV response.

Figure 2:

A



B

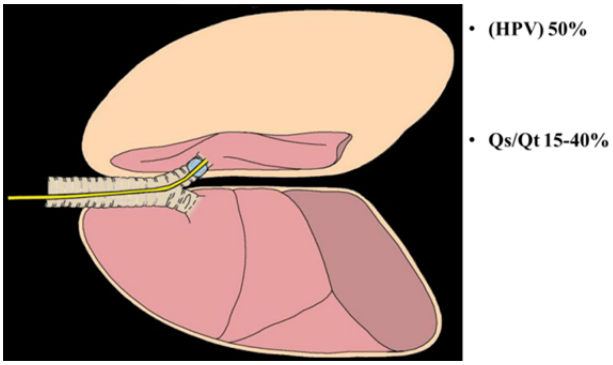


Figure A). The lungs of a patient positioned in the lateral decubitus position. The dependent, or down, lung receives approximately 60% of the total pulmonary blood flow (PBF). The non-dependent but ventilated lung receives approximately 40% of the total PBF. **B**) A non-dependent (collapsed) lung. This leads to a 50% response of HPV as blood flow is being diverted to the dependent lung. In general, the Qs/Qt fraction seen during general anesthesia and OLV ranges from 20 to 40% (this is the amount of blood not being oxygenated).

During OLV, the nondependent (operated) lung remains atelectatic and hypoperfused because of HPV. Thus, the HPV in the nondependent lung ameliorates the pulmonary ventilation/perfusion relationship, preserving systemic oxygenation by constricting pulmonary vessels in poorly ventilated or atelectatic hypoxic lung regions to divert the pulmonary blood flow to better aerated areas.[21] Although HPV decreases the shunt fraction and attempts to resolve hypoxemia,[22] there are associated factors to consider when ventilation is restored because pulmonary reexpansion to correct hypoxemic episodes promotes reentry of oxygen through the airways, causing the release of excessive oxidative radicals.[23]

MANAGEMENT OF INTRAOPERATIVE HYPOXEMIA DURING OLV

During the management of OLV, if the patient's oxygen saturation as measured by pulse oximetry falls below 90% or the PaO_2 is <60 mm Hg, any nonurgent surgical procedure must be stopped. FiO_2 should be increased to 1.0% and two-lung ventilation restored. One should reassess the lung isolation device to ensure that the double-lumen tube or bronchial blocker is in the correct position.[24] During a pneumonectomy, hypoxemia can often be eliminated by surgical clamping of the pulmonary artery feeding the operated lung (Table 2).

Table 2. Treatment of Hypoxia during OLV

- Increase the fraction of inspired oxygen to 100%
- Convert to two-lung ventilation
- Re-expand the collapsed lung
- Confirm optimal position of lung isolation device with a flexible fiberoptic bronchoscope. After SpO_2 has been improved (i.e., SpO_2 >98%), convert to OLV and apply CPAP 5 cm H_2O to the non-dependent lung.
- Unless counterproductive (i.e., auto PEEP >10 cm H_2O), use PEEP 5 cm H_2O to the dependent lung
- Adjust ventilation according to the patient's needs to maintain a $PaCO_2$ 35–40 mm Hg and peek pressure of <35 cm H_2O
- Use pressure-controlled ventilation in patients with severe emphysema or who are morbid obese
- Perform ventilatory maneuvers prior to or during OLV if the patient is at risk for hypoxia during OLV
- If a DLT is being used in video thoracoscopic surgery, consider selective O_2 insufflation to the non-ventilated lung with a fiberoptic bronchoscope
- If a bronchial blocker is used, consider selective lobar blockade in patients with previous contra-lateral lobectomy
- Use intermittent O_2 insufflation with 2 L/min for short intervals to the non-dependent lung
- To reduce the shunt fraction, clamping pulmonary vessel by the surgeon during pneumonectomy cases will improve oxygenation

Malposition of lung isolation devices is a common cause of hypoxemia. A study by Inoue et al[5] showed that patients who experience malposition of the double-lumen tube after being turned into the lateral decubitus position had more malpositions during OLV and more hypoxemic episodes that required intervention to treat the hypoxemia. A study by Campos et al[25] showed that anesthesiologists with limited thoracic surgery experience reported a frequent incidence of malposition (38%) of lung isolation devices. If hypoxemia occurs during OLV, the first step is to ventilate the patient's lung with FiO_2 1.0%. After the device is correctly positioned, fiberoptic bronchoscopy can determine whether all lobar bronchi and segmental branches are patent and free of secretions. In addition, it is important that the patient under OLV be maintained normocapnic, normotensive, and normothermic. Any extreme alteration of these factors will also contribute to the development of hypoxemia by modifying HPV. Hemodynamic parameters must be checked to ensure that the desaturation episodes are not related to profound hypotension caused by compression of the vena cava or direct compression of the aorta or pulmonary vessels.

VENTILATORY MANEUVERS TO IMPROVE ARTERIAL OXYGENATION

Alveolar recruitment maneuvers, continuous positive airway pressure (CPAP), positive end-expiratory pressure (PEEP), fiberoptic oxygen insufflation, and selective lobar ventilation during hypoxia and OLV may be used to improve arterial oxygenation.

Alveolar Recruitment Maneuvers

Lung recruitment is a ventilator maneuver aimed to reverse atelectasis by means of a brief, controlled increase in the airway with expansion of the lungs. Clinical studies involving thoracic surgical patients undergoing OLV have shown that employing lung recruitment prior to OLV or in the minute thereafter reduces atelectasis, improves arterial oxygenation, and decreases pulmonary shunt and dead space with an adequate level of PEEP.[26-28] One study[28] showed that recruitment of both lungs before instituting OLV—by giving 10 consecutive breaths at a plateau pressure of 40 and incremental levels of 5–10 up to 20 cm H_2O of PEEP—improved arterial oxygenation. Mean PaO_2 values for the control group (no recruitment maneuvers) during OLV were 182 ± 79 mm Hg; in contrast, the group that received alveolar recruitment maneuvers had a mean PaO_2 value of 251 ± 69 mm Hg during OLV. In some cases, increasing the PaO_2 value to a safer level during OLV might eliminate the need for additional therapeutic intervention such as CPAP. Although alveolar recruitment maneuvers improve oxygenation, they may cause transient hypotension because the excessive intrathoracic pressure interferes with blood venous return.

Continuous Positive Airway Pressure

CPAP has traditionally been used to treat hypoxemia because of the obligatory shunt developed by the nondependent (collapsed) lung. Application of CPAP has been suggested in the deflation phase of tidal volume (V_T) breath. CPAP is thought to improve oxygenation by a passive mechanism (uptake of oxygen by the alveoli with

continuous oxygen administration). It is recommended to start with 5 cm H_2O of CPAP and progressively increase to no more than 10 cm H_2O. For CPAP to work, it must be applied to an at least partially reexpanded lung before it is adjusted to the desired value. Of the new CPAP devices, the easiest to use is the Mallinckrodt™ (Covidien, Mansfield, MA) that can be attached to a lumen of the double-lumen tube or the center channel of a bronchial blocker. Figure 3 shows the CPAP circuit attached to the center channel of a bronchial blocker.

Figure 3:

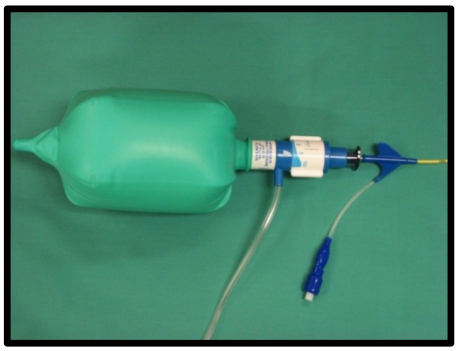


Figure 3 shows the Mallinckrodt® continuous positive airway pressure (CPAP) circuit attached to the center channel of a bronchial blocker.

Oxygen insufflation by CPAP of up to 10 cm H_2O into the nondependent lung, along with no PEEP to the dependent lung during OLV, has improved oxygenation to an average mean PaO_2 value of 286 ± 49 mm Hg and a Qs/Qt fraction of 28 ± 2.5% in thoracic surgical patients.[29] The beneficial effects of CPAP are primarily due to oxygen uptake from the nonventilated lung, not to blood flow diversion to the ventilated lung.

CPAP, even when properly administered, is not completely reliable for improving oxygenation during OLV when the bronchus of the nonventilated lung is obstructed or open to the atmosphere, as in a patient with a bronchopleural fistula or during endobronchial surgery. Also, in some situations such as thoracoscopic surgery, CPAP may prevent adequate surgical exposure because of the partial expansion of the lung.

Positive End-Expiratory Pressure

Should PEEP be used routinely in all patients undergoing OLV? Resistance to blood flow through the lung is related to lung volume in a biphasic pattern and is lowest when the lung is at its functional residual capacity (FRC). Keeping the ventilated lung as close as possible to its normal FRC encourages pulmonary blood flow to the lung. In select patients, the application of PEEP to the dependent (ventilated) lung is beneficial because it restores FRC to close to normal values.[30] This ventilatory maneuver will prevent atelectasis when its value is titrated along the static compliance curve of the flow/volume curves.[31] Many patients do not reach their end-expiratory equilibrium FRC lung volume as they try to exhale a relatively large V_T through one lumen of the double-lumen tube during OLV. These patients develop dynamic hyperinflation and a positive end-expiratory pressure (auto-PEEP).[32]

Auto-PEEP occurs more often in patients with decreased lung elastic recoil, such as in the severely emphysematous patient, and the estimated auto-PEEP averages 4–6 cm H_2O in most lung cancer patients with chronic obstructive pulmonary disease (COPD). The effects of applying external PEEP through the ventilator circuit to the lung in the presence of auto-PEEP are complex. Slinger et al[31] have shown that patients with a very low auto-PEEP (i.e., <2 cm H_2O) will have a greater increase in total PEEP from moderate (5 cm H_2O) external PEEP than those with a high level of the already present auto-PEEP (>10 cm H_2O). The same study[31] showed that if the application of PEEP shifts the expiratory equilibrium position on the compliance curve toward the lower inflection point of the curve, then the external PEEP will be beneficial.

A study by Yokota et al[33] showed that auto-PEEP becomes more evident during OLV and is worst in patients who have poor FEV_1/forced vital capacity ratios. Also, this study showed no correlation with the intraoperative PaO_2 and auto-PEEP. Auto-PEEP is difficult to detect and measure intraoperatively with our anesthesia machines. The application of PEEP during OLV to improve oxygenation depends on the individual patient's lung mechanics. Patients with normal lung parenchyma or those with restrictive lung disease tend to fall below their FRC at the end-expiration during OLV and may benefit from the application of PEEP to the dependent lung.[34]

When should PEEP be applied during OLV? A study by Ren et al[35] in patients requiring OLV examined the best time to apply PEEP and the ideal level of PEEP application. In their study, 30 patients undergoing thoracic

surgery were divided into three groups. No PEEP application or 5–10 cm H_2O PEEP was applied at different intervals. In one group, 5 cm H_2O of PEEP was maintained during the entire time of OLV. The authors reported that PEEP applied at the initial time of OLV improves oxygenation. Also, they reported that 5 cm H_2O of PEEP may produce a beneficial effect without the increase in airway pressure associated with 10 cm H_2O of PEEP.

Another study, this one by Hoftman et al,[36] reported that a V_T of 6.6 mL·kg plus 5–10 cm H_2O of PEEP in patients undergoing OLV improved oxygenation >20% from baseline in only 29% (12 of 41 patients); in contrast, 71% (29 patients) had no improvement of oxygenation. This study clearly confirms that the response to PEEP is related to the lung mechanics of each individual patient. Another alternative to treat hypoxia during OLV is by intermittent positive airway pressure to the nonventilated lung. Russell[37] managed 10 patients whose desaturated SpO_2 was <90% during OLV. They received slow inflation of 2L/min O_2 in the nonventilated lung from 2 to 10 seconds for 5 min. In all 10 patients, the SpO_2 rose to an average of 96% and PaO_2 improved from a mean value of 67 ± 13 to 99 ± 20 mm Hg.

Selective Lobar Ventilation

Campos et al[38] reported improved arterial oxygenation in thoracic surgical patients undergoing OLV when they used selective lobar blockade with a bronchial blocker to block only the surgical lobe while ventilating the rest of the lobes in the ipsilateral side, and with 5 cm H_2O of CPAP delivered to the nondependent lung. Therefore, for patients who have a history of previous contralateral lobectomy and years later require a surgical procedure on the opposite lung, a selective lobar blockade should be considered in order to ventilate the remaining lobe on the surgical side and to preserve or improve arterial oxygenation.[14,38]

Fiberoptic Oxygen Insufflation

An alternative method to treat hypoxemia during OLV for thoracoscopic surgery is by selective insufflation of oxygen using the fiberoptic bronchoscope (with the suction channel connected to the O_2 source), where the tip of the bronchoscope is selectively positioned into the basilar segments of the nondependent lung and positioned in the distal (nonsurgical) lobe. Apneic oxygenation with 5 L/min has been shown to improve oxygenation with this technique.[39] When applying CPAP or oxygen insufflation to the nondependent lung, it is always advisable to observe the direct distention of the collapsed lung and make necessary adjustments to avoid overdistention of the nonventilated lung.

EFFECTS OF ANESTHETIC TECHNIQUE ON HYPOXIA AND INFLAMMATORY RESPONSE DURING ONE-LUNG VENTILATION

In the 1980's, special attention was given to the effects of anesthetics and HPV during OLV.[40] One study[40] reported that, in the canine model, the effect of isoflurane on HPV was dose-dependent inhibition. Others,[9] using the pig model, compared the effects of different minimal alveolar concentration (MAC) concentrations of desflurane and isoflurane on lung perfusion, Qs/Qt fraction, and oxygenation during OLV. This study reported that pulmonary venous oxygen, mixed venous oxygen saturation, and Qs/Qt fraction decreased in a dose-dependent fashion (i.e., MAC of 0.5, 1.0, and 1.5, respectively) without affecting arterial oxygenation. In addition, the influence of thoracic epidural analgesia plus 0.5% MAC of isoflurane has been studied in patients undergoing OLV and compared with total intravenous anesthesia with propofol.[41] This study showed that, based on PaO_2, arterial oxygenation was better in the group who received general anesthesia with volatile agents plus thoracic epidural analgesia. Thus, the effects of inhaled anesthetics on HPV are dose-dependent and thoracic epidural analgesia has no influence on HPV as long as arterial blood pressure is maintained.

OLV increases a proinflammatory response, including cytokine release and leukocyte recruitment in the ventilated lung. In the past 10 years, special interest has been focused on the effects of anesthetics and ventilation on the inflammatory response by measurement of interleukins—interleukin (IL), interleukin Iβ (Iβ), tumor necrosis factor (TNF∝), and $IL_{1\text{-}6\text{-}8}$—from bronchoalveolar lavage or systemic circulation, as well as the potential effect/complications after OLV, including the development of acute lung injury, which has a reported incidence of 4.2% after thoracic surgery.[42] Some have recommended the use of low levels of V_T (i.e., 5 mL·kg) during OLV to reduce the risk of lung injury. One study[43] showed that 5 mL·kg of V_T during OLV was associated with lower levels of interleukin ($IL_{6\text{-}8\text{-}10}$, IL18, and TNF∝) when compared to a high V_T of 10 mL·kg. However, the use of small V_T might predispose patients to atelectasis during general anesthesia.[44] As a result, other investigators have used different ventilatory strategies to maintain or improve oxygenation during OLV. Michelet et al[45] reported that protective ventilation with V_T of 5 mL·kg plus 5 cm H_2O of PEEP to the dependent lung in esophagectomy patients has lower plasmatic levels of interleukins (IL, Iβ, $IL_{6\text{-}8}$, and TNF∝) when compared to conventional ventilation of 9

mL·kg during OLV. These same studies reported a lower extravascular lung water index and intrathoracic blood volume ratio. The patients in the Michelet study had improved lung function, their tracheas were extubated earlier, and they had a decreased proinflammatory systemic response compared with the group who received conventional ventilation of a large V_T.

The anesthetic technique plays an important role in the proinflammatory response during OLV. Schilling et al[46] reported an immunomodulatory effect with the use of a volatile anesthetic, desflurane, in the dependent ventilated lung when compared to intravenous propofol anesthesia. This study reported an induced proinflammatory response in the dependent lung during OLV while it was ventilated with a decidedly nonphysiological V_T of 10 mL·kg. The study also showed that the immune response was attenuated by desflurane, and the administration of propofol resulted in a relative increase of alveolar granulocyte fraction. DeConno et al[47] showed potential lung protection with the use of sevoflurane (vs. propofol) in thoracic surgical patients undergoing OLV. Bronchoalveolar lavage was performed before and after OLV on the nondependent lung, showing a marked reduction of inflammatory mediators and a significantly better outcome in the group that received sevoflurane anesthesia. In contrast, a similar study[48] evaluated the effects of propofol, desflurane, and sevoflurane and the relationship between pulmonary and systemic inflammation in patients undergoing thoracic surgery who required OLV. That study showed that desflurane and sevoflurane suppressed the local alveolar response (lower cytokines) when compared to propofol; however, the systemic inflammatory response was not evident with the three anesthetics studied. All the studies discussed in this paragraph appear to support the beneficial effects of using volatile anesthetics to suppress pulmonary cytokines as well as their potential protective effects against inflammation.

During OLV, it is not uncommon to switch from volume-controlled ventilation to pressure-controlled ventilation in order to improve oxygenation. One study involving thoracic surgical patients demonstrated that better oxygenation was achieved when pressure-controlled ventilation was used during OLV.[49] Another study[50] used a crossover design involving thoracic surgical patients requiring OLV who had relatively normal lung function. Arterial oxygenation was similar in the groups who received volume-controlled ventilation (PaO_2 mean value 206 ± 62 mm Hg) when compared to pressure-controlled ventilation (PaO_2 202 ± 56 mm Hg). In addition, this study reported a decrease in peak pressure in the group who received pressure-controlled vs. volume-controlled ventilation (24 ± 3 cm H_2O vs 34 ± 5 cm H_2O). Pressure-controlled ventilation appears more beneficial for the morbidly obese patient or the severely emphysematous patient undergoing OLV.

Pharmacological interventions have been used to control pulmonary blood flow, prevent decreases of PaO_2 during OLV, or improve oxygenation.[51–53] Some studies have shown that the use of a respiratory stimulant such as almitrine intravenously improved oxygenation during OLV, probably due to the molecular mechanism of action on the pulmonary vessels combined with direct stimulation of chemoreceptors and direct pulmonary vasoconstrictor action.[51] Another study showed no effect on oxygenation with inhaled nitric oxide (NO) in patients undergoing OLV.[52] However, the combination of NO and almitrine showed an improvement in PaO_2 values during OLV in two studies.[53,54] Almitrine is not FDA approved and is unavailable in the United States.

Others have reported improved arterial oxygenation with inhaled epoprostenol plus intravenous phenylephrine infusion during OLV in a patient with interstitial lung disease undergoing video-assisted thoracoscopic surgery.[55] Doering et al[56] reported improved arterial oxygenation with phenylephrine and NO in patients with adult respiratory distress syndrome. The mechanism by which vasoconstrictors such as phenylephrine may improve oxygenation is unclear, but could result from enhancing HPV.

RESTORATION OF TWO-LUNG VENTILATION

During OLV, the collapsed lung remains atelectatic and hypoperfused. After restoration of two-lung ventilation, reentry of oxygen through the airways and alveoli produces a reactive pulmonary vascular dilation due in part to the phenomenon of reperfusion as subsequent free oxygen radicals are released. The reoxygenation injury is the structural damage caused by the excessive production of free radicals. The free radicals interact with cellular structural molecules, producing dysfunction to the endothelial cells. One study[23] has shown that switching from OLV to two-lung ventilation during video-assisted thoracoscopic surgery induces a massive production of reactive oxygen species, but with minimal lung injuries. In this study, extravascular lung water index, intrathoracic blood volume, and permeability index were insignificantly changed after resuming two-lung ventilation. Also, this study showed that the amount of total antioxidants was adequate to counteract the reactive oxygen species. However, these values may be different in patients with extensive lung tumors or pulmonary trauma. Another study[57] showed that OLV lasting longer than1 hour can cause cardiovascular complications (i.e., cardiac arrhythmias) through the generation of severe oxidative stress during reexpansion and conversion to two-lung ventilation. Further studies are needed to validate these results as a potential cause/effect.

SUMMARY

Hypoxemia usually does not occur during OLV. When it does, one should use fiberoptic bronchoscopy to rule out a malposition of the isolation device, then institute ventilatory maneuvers (as necessary) to optimize and improve oxygenation. Alveolar recruitment maneuvers during two-lung ventilation prior to conversion to OLV may serve to prevent the problem. Compared with propofol anesthesia, desflurane and sevoflurane appear to attenuate the inflammatory response to OLV. Smaller V_T plus 5 cm H_2O of PEEP to the dependent lung along with CPAP in the nondependent lung should be considered while treating hypoxemic episodes during OLV.[58]

REFERENCES

1. Campos JH. Progress in lung separation. Thorac Surg Clin 2005; 15:71-83
2. Campos J: Lung Isolation. Chapter 16 in Principles and Practice of Anesthesia for Thoracic Surgery, 2011 pp 227-246. Ed Slinger P. Springer Publisher.
3. Rozé H, Lafargue M, Ouattara A. Case scenario: Management of intraoperative hypoxemia during one-lung ventilation. Anesthesiology 2011; 114:167-74
4. Karzai W, Schwarzkopf K. Hypoxemia during one-lung ventilation: prediction, prevention, and treatment. Anesthesiology 2009; 110:1402-11.
5. Inoue S, Nishimine N, Kitaguchi K, et al. Double lumen tube location predicts tube malposition and hypoxaemia during one lung ventilation. Br J Anaesth 2004; 92:195-201.
6. Slinger P. Pro: low tidal volume is indicated during one-lung ventilation. Anesth Analg 2006; 103:268-70.
7. Klein U, Karzai W, Bloos F, et al. Role of fiberoptic bronchoscopy in conjunction with the use of double-lumen tubes for thoracic anesthesia: a prospective study. Anesthesiology 1998; 88:346-50.
8. Campos JH. Current techniques for perioperative lung isolation in adults. Anesthesiology 2002; 97:1295-1301
9. Schwarzkopf K, Schreiber T, Bauer R, et al. The effects of increasing concentrations of isoflurane and desflurane on pulmonary perfusion and systemic oxygenation during one-lung ventilation in pigs. Anesth Analg 2001; 93:1434-1438
10. Slinger P, Suissa S, Triolet W. Predicting arterial oxygenation during one-lung anaesthesia. Can J Anaesth 1992; 39:1030-35.
11. Schwarzkopf K, Klein U, Schreiber T, et al. Oxygenation during one-lung ventilation: the effects of inhaled nitric oxide and increasing levels of inspired fraction of oxygen. Anesth Analg 2001; 92:842-7.
12. Lewis JW Jr, Serwin JP, Gabriel FS, et al. The utility of a double-lumen tube for one-lung ventilation in a variety of noncardiac thoracic surgical procedures. J Cardiothorac Vasc Anesth 1992; 6:705-10
13. Suemitsu R, Sakoguchi T, Morikawa K, et al. Effect of body mass index on perioperative complications in thoracic surgery. Asian Cardiovasc Thorac Ann 2008; 16:463-7
14. Campos JH, Ledet C, Moyers JR. Improvement of arterial oxygen saturation with selective lobar bronchial block during hemorrhage in a patient with previous contralateral lobectomy. Anesth Analg 1995; 81:1095-6.
15. Brodsky JB. Approaches to hypoxemia during single-lung ventilation. Curr Opin Anaesthesiol 2001; 14:71-6.
16. Watanabe S, Noguchi E, Yamada S, et al. Sequential changes of arterial oxygen tension in the supine position during one-lung ventilation. Anesth Analg 2000; 90:28-34.
17. Bardoczky GI, Szegedi LL, d'Hollander AA, et al. Two-lung and one-lung ventilation in patients with chronic obstructive pulmonary disease: the effects of position and FiO_2. Anesth Analg 2000; 90:35-41.
18. Slinger P, Campos J. Anesthesia for Thoracic Surgery. Chapter 59 in Miller's Anesthesia, 7th edition, 2009, pp 1219-1287
19. Larsson A, Malmkvist G, Werner O. Variations in lung volume and compliance during pulmonary surgery. Br J Anaesth 1987; 59:585-91.
20. Ward DS. Intra-operative ventilation strategies for thoracic surgery. Chapter 21 in Principles and Practice of Anesthesia for Thoracic Surgery, 2011 pp 297-305. Ed Slinger P Springer Publisher.
21. Leite CF, Calixto MC, Toro IF, et al. Characterization of Pulmonary and Systemic Inflammatory Responses Produced by Lung Re-expansion After One-Lung Ventilation. Cardiothorac Vasc Anesth 2012; 26:427-32
22. Lohser J. Evidence-based management of one-lung ventilation. Anesthesiol Clin 2008; 26:241-72
23. Cheng YJ, Chan KC, Chien CT, et al. Oxidative stress during 1-lung ventilation. J Thorac Cardiovasc Surg 2006; 132:513-8.
24. Campos JH, Update on Tracheobronchial Anatomy and Flexible Fiberoptic Bronchoscopy in Thoracic Anesthesia, Curr Opin Anaesthesiol 2009; 22: 4-10

25. Campos JH, Hallam EA, Van Natta T and Kernstine KH: Devices for lung isolation used by anesthesiologists with limited thoracic experience: comparison of double-lumen endotracheal tube, Univent torque control blocker, and Arndt wire-guided endobronchial blocker. Anesthesiology 2006; 104:261-266
26. Tusman G, Böhm SH, Melkum F, et al: Alveolar recruitment strategy increases arterial oxygenation during one-lung ventilation Ann Thorac Surg 2002;73:1204-1209
27. Tusman G, Böhm SH, Sipmann FS, et al. Lung recruitment improves the efficiency of ventilation and gas exchange during one-lung ventilation anesthesia. Anesth Analg 2004; 98:1604-9
28. Unzueta C, Tusman G, Suarez-Sipmann F, et al: Alveolar recruitment improves ventilation during thoracic surgery: a randomized controlled trial. Br J Anaesth 2012;108:517-24.
29. Capan LM, Turndorf H, Patel C, et al. Optimization of arterial oxygenation during one-lung anesthesia. Anesth Analg 1980;59:847-51.
30. Cohen E, Eisenkraft JB. Positive end-expiratory pressure during one-lung ventilation improves oxygenation in patients with low arterial oxygen tensions. J Cardiothorac Vasc Anesth 1996; 10:578-582.
31. Slinger PD, Kruger M, McRae K, et al. Relation of the static compliance curve and positive end-expiratory pressure to oxygenation during one-lung ventilation. Anesthesiology 2001; 95:1096-102.
32. Ducros L, Moutafis M, Castelain MH, et al. Pulmonary air trapping during two-lung and one-lung ventilation. J Cardiothorac Vasc Anesth 1999;13:35-39.
33. Yokota K, Toriumi T, Sari A, et al. Auto-positive end-expiratory pressure during one-lung ventilation using a double-lumen endobronchial tube. Anesth Analg 1996;82:1007-1010.
34. Slinger PD, Hickey DR. The interaction between applied PEEP and auto-PEEP during one-lung ventilation. J Cardiothorac Vasc Anesth 1998; 12:133-136.
35. Ren Y, Peng ZL, Xue QS, et al. The effect of timing of application of positive end-expiratory pressure on oxygenation during one-lung ventilation. Anaesth Intensive Care 2008;36:544-548.
36. Hoftman N, Canales C, Leduc M, et al. Positive end expiratory pressure during one-lung ventilation: selecting ideal patients and ventilator settings with the aim of improving arterial oxygenation. Ann Card Anaesth 2011;14:183-187.
37. Russell WJ. Intermittent positive airway pressure to manage hypoxia during one-lung anaesthesia. Anaesth Intensive Care 2009;37:432-434.
38. Campos JH. Effects of oxygenation during selective lobar versus total lung collapse with or without continuous positive airway pressure. Anesth Analg 1997; 85:583-586.
39. Ku CM, Slinger P, Waddell TK. A novel method of treating hypoxemia during one-lung ventilation for thoracoscopic surgery. J Cardiothorac Vasc Anesth 2009; 23:850-852
40. Domino KB, Borowec L, Alexander CM, et al. Influence of isoflurane on hypoxic pulmonary vasoconstriction in dogs. Anesthesiology. 1986;64:423-429.
41. Von Dossow V, Welte M, Zaune U, et al. Thoracic epidural anesthesia combined with general anesthesia: the preferred anesthetic technique for thoracic surgery. Anesth Analg 2001;92:848-854.
42. Licker M, de Perrot M, Spiliopoulos A, et al. Risk factors for acute lung injury after thoracic surgery for lung cancer. Anesth Analg. 2003;97:1558-1565.
43. Schilling T, Kozian A, Huth C, et al. e pulmonary immune effects of mechanical ventilation in patients undergoing thoracic surgery. Anesth Analg. 2005;101:957-965
44. Duggan M, Kavanagh BP. Pulmonary atelectasis: a pathogenic perioperative entity. Anesthesiology 2005;102:838-854.
45. Michelet P, D'Journo XB, Roch A, et al. Protective ventilation influences systemic inflammation after esophagectomy: a randomized controlled study. Anesthesiology 2006;105:911-919.
46. Schilling T, Kozian A, Kretzschmar M, et al. Effects of propofol and desflurane anaesthesia on the alveolar inflammatory response to one-lung ventilation. Br J Anaesth 2007;99:368-375
47. De Conno E, Steurer MP, Wittlinger M, et al. Anesthetic-induced improvement of the inflammatory response to one-lung ventilation. Anesthesiology 2009;110:1316-1326.
48. Schilling T, Kozian A, Senturk M, et al. Effects of volatile and intravenous anesthesia on the alveolar and systemic inflammatory response in thoracic surgical patients. Anesthesiology 2011;115:65-74.
49. Tuğrul M, Camci E, Karadeniz H, et al. Comparison of volume controlled with pressure controlled ventilation during one-lung anaesthesia. J Anaesth 1997;79:306-310.
50. Unzueta MC, Casas JI, Moral MV. Pressure-controlled versus volume-controlled ventilation during one-lung ventilation for thoracic surgery. Anesth Analg 2007;104:1029-1033
51. Dalibon N, Moutafis M, Liu N, et al. Treatment of hypoxemia during one-lung ventilation using intravenous almitrine. Anesth Analg 2004;98:590-594

52. Rocca GD, Passariello M, Coccia C, et al. Inhaled nitric oxide administration during one-lung ventilation in patients undergoing thoracic surgery. J Cardiothorac Vasc Anesth 2001;15:218-223.
53. Moutafis M, Liu N, Dalibon N, et al. The effects of inhaled nitric oxide and its combination with intravenous almitrine on PaO_2 during one-lung ventilation in patients undergoing thoracoscopic procedures. Anesth Analg 1997;85:1130-1135.
54. Silva-Costa-Gomes T, Gallart L, Vallès J, et al. Low- vs high-dose almitrine combined with nitric oxide to prevent hypoxia during open-chest one-lung ventilation. Br J Anaesth 2005;95:410-416.
55. Raghunathan K, Connelly NR, Robbins LD, et al. Inhaled epoprostenol during one-lung ventilation. Ann Thorac Surg 2010;89:981-983.
56. Doering EB, Hanson CW 3rd, Reily DJ, et al. Improvement in oxygenation by phenylephrine and nitric oxide in patients with adult respiratory distress syndrome. Anesthesiology 1997;87:18-25.
57. Misthos P, Katsaragakis S, Theodorou D, et al. The degree of oxidative stress is associated with major adverse effects after lung resection: a prospective study. Eur J Cardiothorac Surg 2006;29:591-595
58. Campos JH. Hypoxia during Thoracic Surgery: Practical Advice for the Anesthesiologist. ASA Refresher Course. 2013; 41:38-46