New Insights on One-Lung Ventilation

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Learning Objectives: 1) To update practitioners on recent advances in our understanding of the physiology of one-lung ventilation. 2) To develop a plan to manage hypoxemia during one-lung anesthesia in a variety of clinical settings.

Case Synopsis: A 67 year old male with mild emphysema (FEV1 = 62% predicted) and no other significant co-morbidity is scheduled for a video-assisted (VATS) left upper lobectomy . After induction of general anesthesia and placement of a left-sided double-lumen tube (DLT) the patient is placed in the right lateral decubitus position and right one-lung ventilation (OLV) is commenced with a tidal volume of 6 ml/kg, respiratory rate 10/min and FiO2 of 0.5 in air with sevoflurane 1MAC. After insertion of the VATS telescope the surgeon complains the lung is not well collapsed. What should the anesthesiologist do?

Improving lung collapse during OLV. The initial diagnostic maneuver is to re-assess the position of the DLT with a fiberoptic bronchoscope to ensure that the non-ventilated lung is adequately isolated from the ventilated lung and also that there is no lobar obstruction impeding collapse of the non-ventilated lung. Application of suction (e.g. –20cmH2O) to the lumen of the DLT to the non-ventilated lung will aid the elastic recoil of the lung and improve the rate of lung collapse to its closing capacity. Once the non-ventilated lung reaches its closing capacity, which is abnormally large in emphysema patients, further collapse depends on absorption of the trapped gas in the lung. If the non-ventilated lung contains poorly soluble nitrogen it will collapse slowly. To facilitate lung collapse, the non-dependent lung should be de-nitrogenated by ventilation with a FiO2 of 1.0 for several minutes prior to the initiation of OLV.[1] After the initiation of OLV, if the oxygenation remains stable, then air can be re-introduced to the gas mixture to decrease atelectasis in the ventilated (dependent) lung.

Case Synopsis continued: Eventually the left lung collapses sufficiently to allow surgery to proceed. During the first 30 min. of OLV the airway pressures (17/2 cmH20), hemodynamic parameters (BP 120/70, HR 80) and end-tidal CO2 (36 mmHg) remain stable. However there is a slow and persistent fall in the SpO2 from an initial value of 99% to 90% in spite of increasing the FiO2 to 1.0. What diagnostic and/or therapeutic maneuvers should be used?

After a recruitment maneuver of the ventilated lung and application of 5 cmH_2O positive end-expiratory pressure (PEEP) to the ventilated lung the saturation continues to decline and is now 88%. The anesthesiologist suggests the use of continuous positive airway pressure (CPAP) to the non-ventilated lung. The surgeon says that it may not be possible to continue with a VATS technique if CPAP is used and the operation will have to be converted to an open thoracotomy. Is there any other useful therapy for hypoxemia that can be used in this situation?

MANAGEMENT OF HYPOXEMIA DURING OLV:

The incidence of hypoxemia (SpO2 < 90%) during one-lung ventilation (OLV) with an inspired oxygen concentration (FiO_2) of 1.0 has declined from levels of 20-25% in the 1970's to <10% today.[2] Several advances in thoracic anesthesia have aided this improved oxygenation. First, the routine use of fiberoptic bronchoscopy to position double-lumen tubes and bronchial blockers. Second, improved anesthetic techniques with lower doses of volatile agents.[3] And third, a better understanding of the pathophysiology of OLV.

Etiology: The major cause of hypoxemia is the shunt of de-oxygenated blood through the non-ventilated lung. Factors which influence this shunt are hypoxic pulmonary vasoconstriction (HPV), gravity, the pressure differential between the thoraces and physical lung collapse. HPV is inhibited by essentially all volatile anesthetics. Isoflurane seems to be less inhibitory than enflurane or halothane[4] and equivalent to sevoflurane or desflurane. Intravenous anesthetic techniques have not been shown to provide better oxygenation than the newer volatile anesthetics in <1MAC concentrations.

Manipulating the ventilating pressures and tidal volumes during one-lung anesthesia can improve the oxygenation for certain patients.[5] Some patients, particularly those with COPD, showed better oxygenation during OLV with pressure-controlled vs. volume-controlled ventilation.[6]

A third of the 25-35% shunt during OLV is due to ventilation-perfusion mismatch in the ventilated dependent-lung. Several factors under the control of the anesthesiologist can influence this dependent-lung shunt. An excess of intravenous crystalloids can rapidly cause desaturation of the pulmonary venous blood draining the dependent lungvii.Also, the use of nitrous oxide will lead to increased dependent-lung atelectasis since it causes greater instability of poorly ventilated lung regions than oxygen.

Monitoring: The risk of intraoperative hypoxemia is increased during OLV. Pulse oximetry is prone to malfunctions and does not give an early warning of the rapidly falling PaO2 that occurs during OLV

before any change in saturationviii. Patients whose PaO2 declines rapidly after initiating OLV are most likely to become hypoxemic. Side-stream spirometry permits on-line monitoring of pulmonary mechanics. This technology can provide an early warning of loss of lung isolation or accidental lobar obstruction. It may be possible to use this information to select the optimal ventilatory parameters for an individual patient during OLV.

Prediction of Hypoxemia: Several factors allow prediction of the risk of hypoxemia developing during OLV[9] (see Table 1). First, the side of lung collapse during OLV. The mean PaO_2 level is 70 mmHg higher for left vs. right thoracotomies. Second, patients with good preoperative spirometric pulmonary function tests tend to have lower PaO_2 values during OLV than patients with poor spirometry.[10] This may be related to auto-PEEP in patients with poor spirometry.[11] Other predictive factors include the A-aO_2 gradient during two-lung ventilation which correlates inversely with the PaO_2 during OLV. Also, hypoxemia occurs more frequently during OLV in the supine position[12] than the lateral position because there is not the increase of blood flow (approximately 10% increase) to the dependent (ventilated) lung due to gravity which is seen in the lateral position .

TABLE 1: FACTORS INCREASING THE RISK OF HYPOXEMIA DURING ONE-LUNG VENTILATION:

- High percentage of ventilation or perfusion to the operative lung on preoperative V/Q scan
- Poor PaO_2 during two-lung ventilation
- Right-sided surgery.
- Good preoperative spirometry (FEV1 or FVC)
- Supine (vs. lateral) patient position

Treatment of Hypoxemia during OLV: First, causes of hypoxemia such as malposition of an endobronchial tube or inadequate oxygen delivery should be ruled out. The use of the highest possible FiO_2 during OLV improves oxygenation. However, drugs such as Bleomycin, Mitomycin and Amiodarone, have been associated with pulmonary oxygen toxicity when a FiO_2 >0.4 was used intra-operatively for thoracic surgery.

Decreases in cardiac output during OLV decrease PaO2 via a fall in mixed venous oxygen content since these patients have a large shunt.[13] Therefore, assure that the cardiac output is maintained. However, raising cardiac output beyond baseline tends to decrease PaO2 as HPV is opposed by the passive increase in pulmonary arterial pressures (see Fig. 1).

CPAP to the non-ventilated lung has been used traditionally as the first-line of treatment for hypoxemia during OLV.[14] Useful increases in oxygenation can be achieved with 1-2 cm H_2O levels of CPAP.[15] For maximal clinical efficiency CPAP must be applied to the inflated lung.[11] Even short periods of lung collapse impair the efficiency of CPAP since the opening pressure of atelectatic lung units exceeds 20 cmH_2O. Because of the problems which re-inflation may cause at an in-opportune surgical moment, it is useful to predict which patients are most at risk of hypoxemia and to apply CPAP prophylactically at the onset of OLV. However CPAP to the non-ventilated lung can impede surgery during VATS procedures.[16]

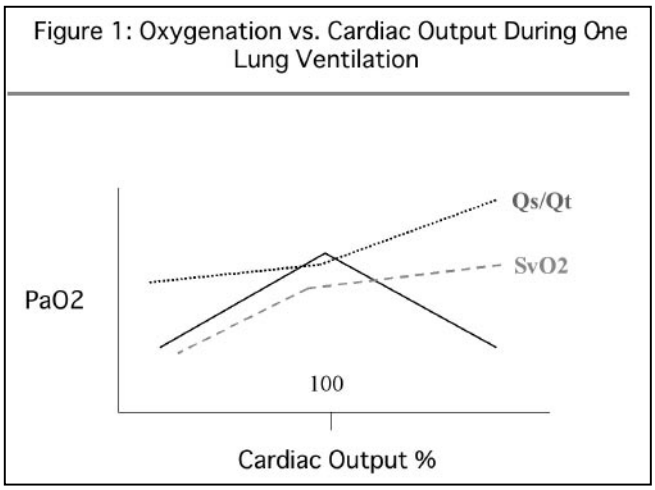

Figure 1: The effects of changes in cardiac output (C.O.) on arterial oxygenation (PaO_2) during OLV. A decrease in cardiac output will lead to a decrease in shunt (Qs/Qt) as hypoxic pulmonary vasoconstriction (HPV) becomes more effective as pulmonary artery pressures fall passively. However the attendant decrease in mixed venous oxygen saturation (SvO_2) as C.O. falls leads to a net decrease in PaO_2. Conversely raising the C.O. above baseline (100%) with an inotrope leads to an increase in SvO2 but also an increase in Qs/Qt and a net fall in PaO_2. (Diagram based on data from Refs. # 4 &13). During OLV it is very important to maintain C.O.

Added PEEP to the ventilated lung decreases PaO_2 in some groups of patients during OLV, particularly COPD patients who usually develop auto-PEEP during OLV. Some patients, often those with the poorest PaO_2 values, benefit from added dependent-lung PEEP. The beneficial effects of PEEP during OLV are related to changes in the end-expiratory dependent-lung volume and its static compliance curve.[17] The goal of ventilation during OLV is to maintain the volume of ventilated lung as closely as possible to its functional residual capacity (FRC), which is the lung volume at which the pulmonary vascular resistance is minimal (see Figs. 2 and 3).

Those patients most likely to benefit from PEEP to the dependent lung are patients with an increased A-aO_2 gradient (PaO_2/FiO_2 ratio < 300) in the lateral position during two-lung ventilation and a low level of auto-PEEP during OLV. Groups who can be expected to improve with PEEP are patients with healthy lungs on the side of thoracotomy (e.g. younger patients or esophageal surgery)i or patients with low functional residual capacities (obese or pulmonary fibrosis). It is important to recruit the ventilated lung to eliminate atelectasis at the start of OLV and when PEEP is appliedii. The concept of individualizing ventilation parameters during OLV to accommodate individual patient differences in lung mechanical function and to maintain the ventilated lung as close as possible to its FRC is possibly the most important lesson that we have learned in the

past 20 years in thoracic anesthesia. Unfortunately the ventilators on modern anesthesia machines do not permit easy measurement of auto-PEEP or true total-PEEP so the application of PEEP during OLV is mainly on the basis of the clinician's best guess. As a personal guideline, I usually apply 5cmH_2O PEEP to most patients during OLV with the exception of patients with moderate or severe COPD (FEV1/FVC ratio <70%) when I avoid added PEEP.

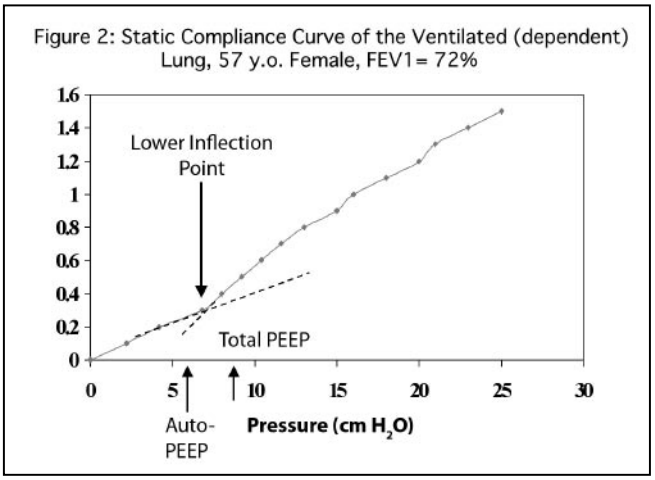

Figure 2: The static compliance (volume vs. pressure) curve of the dependent lung during OLV. During routine OLV this patient with mild COPD developed 6 cm H_2O of auto-PEEP. The FRC estimated from the lower inflection point of the compliance curve was 7 cm H_2O. The addition of 5 cm H_2O PEEP via the ventilator raised the total PEEP to 9 cm H_2O (the additive interaction of PEEP and auto-PEEP is not predictable, see ref. # 11). This increase of end-expiratory lung volume above FRC with PEEP raises the pulmonary vascular resistance of the ventilated lung and resulted in a decrease of PaO_2 during OLV. This is the common pattern seen when patients with COPD have PEEP added to the ventilated lung during OLV (Based on data from Ref. # 17).

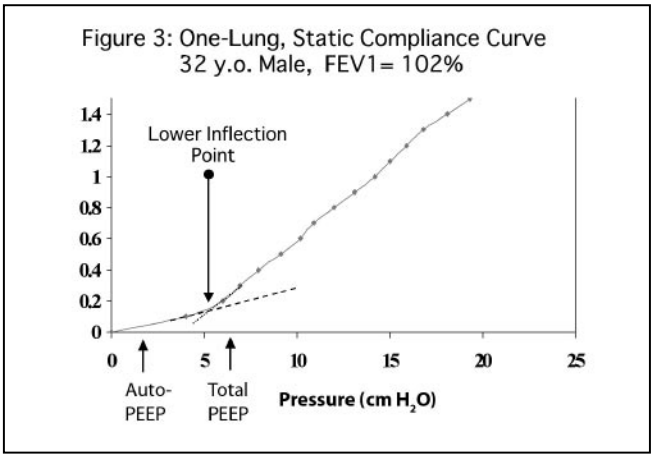

Figure 3: The compliance curve of the ventilated lung during OLV in a patient with normal pulmonary function. This patient developed 2 cmH2O auto-PEEP during standard OLV. The addition of 5 cmH2O PEEP via the ventilator raised the total PEEP to 6 cmH2O and by raising the end-expiratory lung volume closer to the FRC resulted in an increase of PaO2 during OLV for this patient. This is the typical pattern of response with the addition of PEEP during OLV in patients with normal pulmonary function or in patients with restrictive (vs. obstructive lung disease). (Based on data from Ref. # 17).

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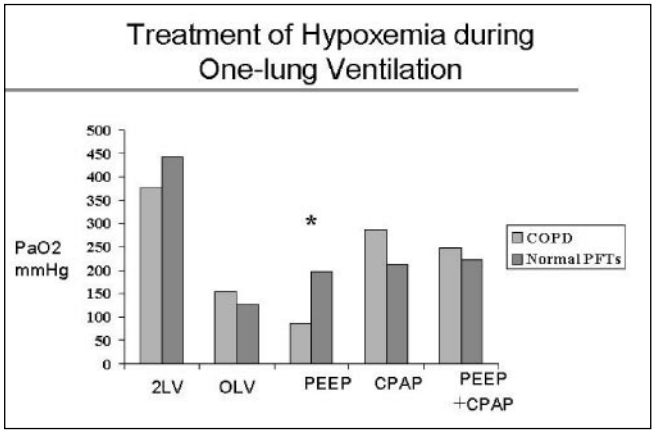

Figure 4: A comparison of the effects on PaO_2 of strategies to improve oxygenation during OLV in patients with COPD vs. patients with normal preoperative pulmonary function tests (PFTs) during thoracic surgery. 2LV= two-lung ventilation. PEEP= application of PEEP to the ventilated lung. CPAP = application of CPAP to the non-ventilated lung. PEEP + CPAP= the simultaneous combination of ventilated-lung PEEP and non-ventilated lung CPAP. * Note that in patients with normal PFTs, PEEP improves PaO_2 during OLV while it decreases PaO_2 in COPD patients (Based on data from Refs. # 14 &18).

During pneumonectomy, lung transplantation or in life threatening situations, the ipsilateral pulmonary artery can be compressed or clamped by the surgeon to transiently improve PaO_2. Pulmonary artery balloon-tipped floatation catheters can be placed under fluoroscopic control prior to OLV and inflated to decrease regional pulmonary blood flow. High frequency jet ventilation (HFJV) to the operative lung provides superior oxygenation.[20] However, HFJV tends to increase the diameter of central airways and can impede surgery during pulmonary resections. HFJV is useful for non-pulmonary intrathoracic surgery.

Various pharmacological methods of modulating the unilateral pulmonary vascular tone such as prostaglandin E1 and nitric oxide (NO) are available. The combination of NO (20 ppm) to the ventilated lung and an intravenous infusion of almitrene (a pulmonary vasoconstrictor) can restore PaO2 during OLV to levels close to these during two-lung

ventilation.[21] NO alone does not improve PaO_2 in the majority of patients during OLViii. However, there is a small minority of hypoxemic patients who benefit from NO. It is not yet clear how to identify these patients prospectively.[23]

Contradictory studies have been published on the use of combined thoracic epidural with general anesthesia and PaO2 during OLV.[24,25] It is unlikely that thoracic epidural blockade per se has any significant effect on HPV or PaO2 during OLV. However falls in cardiac output due to the sympathectomy of local anesthetic epidural blockade will decrease PaO2 if not corrected.

The traditional use of large tidal volume (10 ml/kg ideal body weight) ventilation during OLV may not be the optimal anesthetic management. High airway pressures and volumes during OLV may be associated with injury to the non-operated lung.[26] This is particularly a concern in pneumonectomy patients who are prone to develop increased permeability in the residual lung.[27] Post-pneumonectomy pulmonary edema is related to the use of large tidal volumes during one-lung ventilation[28] and has a high mortality rate.[29] There is a trend to use lower tidal volumes and/or pressure-controlled ventilation during OLV particularly in COPD patients who tend to develop auto-PEEP[30] (see Table 2).

Table 2: Individualizing One-Lung Ventilation

		Exceptions:
Tidal Volume	5-6 ml/kg	Peak Pa/w <35 cmH2O Plateau Pa/w P<25
PEEP	Total 5 cm.	Not added if COPD
FiO2	1.0	Add Air as tolerated
Resp. Rate	12	Mild hypercapnia accepted
Mode	Volume or Pressure Control Ventilation	Press Cont Vent COPD, Transplant, Pneumonectomy

Table 2: Pa/w= airway pressure. The peak and plateau airway pressures during OLV should be maintained at < 35 and 25 cmH_2O respectively. With the use of smaller tidal volumes (5-6 ml/kg ideal body weight), most patients will required added PEEP during OLV. After a period (20-30 min.) of stable OLV with FiO_2 1.0, air can be added to the gas mixture to decrease atelectasis in the ventilated lung. Mild hypercapnia = $PaCO_2$ < 60mmHg. Pressure control ventilation is recommended in patients at increased risk for lung injury such as COPD patients, lung transplantation and pneumonectomies.

Summary: Recent advances in anesthetic agents, equipment and monitoring for lung isolation and in techniques of one-lung ventilation have improved the safety and reliability of one-lung anesthesia for thoracic surgery. Also our understanding of the respiratory physiology of OLV has evolved. It is possible to identify in advance patients who will benefit from PEEP to the ventilated lung Ventilation parameters should be individualized for each patient depending on the patho-physiology of the underlying lung disease. A strategy to avoid and treat hypoxemia during OLV is presented in Table 3. Steps 1-5 under "Gradual Desaturation" should be considered for all patients.

Table 3: Management of Hypoxemia During One-Lung Ventilation:

Severe or Acute desaturation: Resume two-lung ventilation, deflate the bronchial cuff of the double-lumen tube or blocker.

Gradual Desaturation:
1. Assure FiO_2= 1.0
2. Check double-lumen tube or bronchial blocker placement with fiberoptic bronchoscopy
3. Optimize cardiac output
4. Recruit the ventilated lung
5. Apply PEEP 5cm H_2O to ventilated lung (except COPD patients)
6. Apply CPAP 1-2 cm to the non-ventilated lung (after recruitment)
7. Partial ventilation of the non-ventilated lung:
   i) Lobar/segmental re-inflation with fiberoptic bronchoscopy (see Fig. 5)
   ii) Lobar collapse*
   iii) Whole lung oxygen insufflation
8. Intermittent re-inflation of the non-ventilated lung
9. Mechanical restriction of non-ventilated lung pulmonary blood flow

(* Lobar collapse requires the placement of a bronchial blocker in the lobar bronchus of the lobe to be collapsed while ventilating the remaining lung. This can be a useful technique in patients having repeat pulmonary resections but the blocker needs to be placed during the initial intubation)

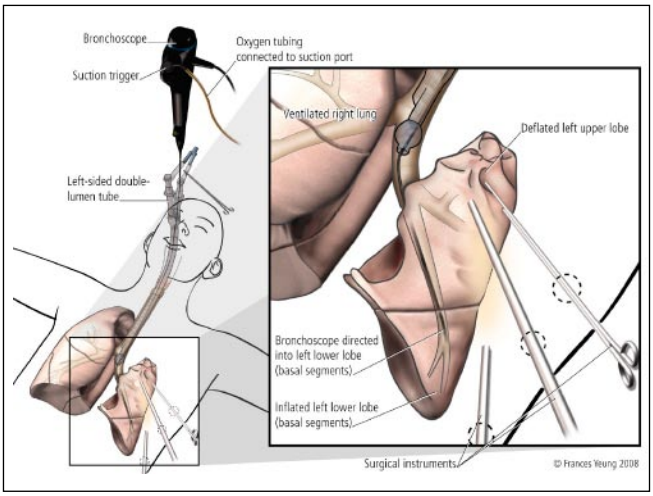

Figure 5: Treatment of hypoxemia during VATS. Oxygen 5L/min. from an auxiliary source is attached to the suction channel of a FOB and insufflated to recruit a segment of the non-ventilated lung distant from the surgical site with direct thoracoscopic observation. In this Figure, basal segments of the left lower lobe are recruited during a left upper lobe VATS surgery. (Reference: Ku M, et al. J Cardiothorac Vasc Anesth 2009, in press). This treatment can improve oxygenation during VATS and does not interfere with the exposure at the site of surgery. This would be a useful strategy in the case presented.

QUESTIONS:

Question 1: What is the incidence of hypoxemia during OLV for thoracic surgery?

a) < 10%
b) 10-20%
c) 20-40%
d) >40%

Question 2: Which of the following factors is associated with an increased risk of hypoxemia during OLV?

a) Left-sided surgery
b) COPD
c) Increased A-aO2 gradient during two-lung ventilation
d)Lateral (vs. supine) position

Question 3: The initial treatment of acute severe desaturation during OLV should be?

a) Fiberoptic bronchoscopy to verify tube/ blocker position
b) Resumption of two-lung ventilation
c) Change to an intravenous anesthetic technique
d)Apply PEEP to the ventilated lung

Question 4: Which of the following has not contributed to the decrease in the incidence of hypoxemia during OLV?

a) Better understanding of the physiology of OLV
b) The routine use of FOB to position double-lumen tubes and blockers
c) Decreased inhibition of HPV by newer volatile anesthetics
d) The use of thoracic epidural analgesia

Question 5: The addition of PEEP via the ventilator is most likely to cause a decrease in PaO2 during OLV in which patient?

a) A morbidly obese patient
b) A child
c) A patient with COPD
d) A patient with pulmonary fibrosis

Answers: 1:a; 2:c; 3:b; 4:d; 5:c

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