

Hypoxaemia during One-Lung Ventilation

Dr S Kumar, Cardiac Anesthesiologist,

Meenakshi Mission Hospital and Research Centre, Madurai.

Introduction

One-lung ventilation is required when providing anaesthesia for operative procedures in the thoracic cavity. During this process, hypoxaemia is reported to occur with an incidence of approximately 5–10%. Hypoxaemia is an adverse but inevitable consequence of one-lung ventilation (OLV). Unfortunately, hypoxaemia affects postoperative outcome as there is an increased risk of complications such as cognitive dysfunction, atrial fibrillation, renal failure, and pulmonary hypertension. Thus, prevention and treatment of hypoxaemia associated with one-lung ventilation is a priority for anaesthetists. This article covers the indications for lung isolation, elucidates why hypoxaemia occurs, and explains the relationship between developments in methods of lung isolation and attenuation of hypoxaemia.

Indications

Lung isolation is necessary for:

- facilitating surgery, for example, lobectomy, pneumonectomy, pleurectomy, decortication, bullectomy, diaphragmatic hernia repair, pericardial window, and oesophagectomy;
- preventing cross-contamination of the contralateral lung, for example, endobronchial haemorrhage, abscess with empyema, bronchiectasis, and lavage;
- controlling distribution of ventilation, for example, bronchopleural fistula.

Methods used for lung isolation

There are two main methods for lung isolation: use of a double-lumen endobronchial tube or insertion of an endobronchial blocker.

Double-lumen endobronchial tubes : Features

Modern-day double-lumen endobronchial tubes have the following features. They allow: collapse of either the left or right lung to assist surgery; airway toilet; left or right lung isolation to prevent cross-contamination; ventilation of the left lung; ventilation of the right lung, including its upper lobe; surgical access for any lung resection, including sleeve resection and pneumonectomy.

Modern-day double-lumen endobronchial tubes are based on the Robertshaw design. In the current practice, there are two main types of double-lumen endobronchial tubes:

- Plastic type, for example, the Broncho-Cath (Mallinckrodt, Tyco Healthcare). For adults, the standard sizes are 35, 37, 39, and 41 Fr. Other companies, for example, Rusch and Sheridan, manufacture this type of tube as well.
- Disposable Robertshaw (Phoenix Medical). The Robertshaw tube (Fig. 1A) comes in small, medium, or large sizes.

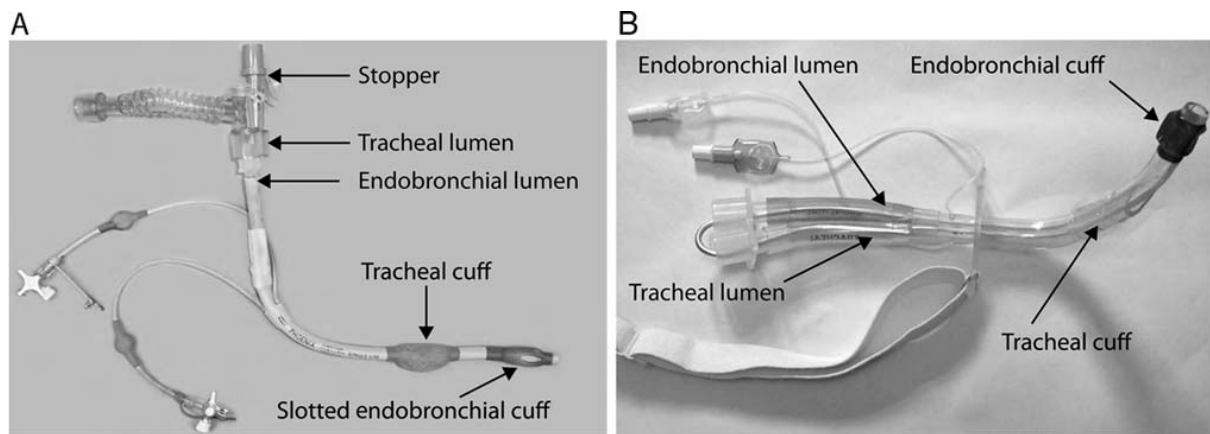


Fig 1 Types of double-lumen tubes: (A) right-sided Robertshaw double-lumen tube and (B) left-sided Rusch double-lumen tracheostomy tube.

It has been found that the orifice within the slotted cuff ('Murphy's eye') for the right-sided Robertshaw double-lumen endobronchial tube enlarges with cuff inflation whereas that for right-sided plastic double-lumen tubes does not do so. In theory, therefore, the risk of hypoxaemia during right-lung ventilation may be minimized by the right-sided Robertshaw design. A double-lumen endobronchial tube of appropriate size should be selected for patient height, gender, and size of main stem bronchi. In general, small tube sizes are appropriate for females since the diameter of their cricoid cartilage is smaller than that of males. Depending on the type of surgery, either a left sized or right sized double-lumen endobronchial tube should be used.

Endobronchial blockers

In addition to double-lumen endobronchial tubes, endobronchial blockers may be utilized for OLV. Interest in their design and use has increased recently and a number of new models have become available. Endobronchial blockers consist of a balloon and a central lumen for application of suction and lung deflation or administration of oxygen. There are two main designs for endobronchial blockers:

(i) Individual endobronchial blocker with:

(a) an adjustable monofilament loop at the tip, called the wire-guided Arndt endobronchial blocker (Cook Critical Care,) Sizes available are 5 and 9 F3 (Fig. 2A).

(b) fixed flexion at the tip (Smiths Medical) (Fig. 2B). One size, equivalent to 9 F, is available.

(c) adjustable flexion at the tip called the Cohenw Tip Deflecting Endobronchial Blocker (Cook Critical Care). There is one size of 9 F which is suitable for a tracheal tube of at least 7.5 mm in internal diameter.⁴

(ii) Endobronchial blocker combined with a tracheal tube such as:

(a) Univent Torque Control Blocker, consisting of a blocker with spherical balloon incorporated into a single-lumen tracheal tube whose internal diameter size range is from 3.5 to 9.0mm. Except for size 3.5 mm, all tracheal tubes have a tracheal cuff (Fig. 2C)

(b) an endobronchial blocker accompanying the Papworth Bivent tube which is a single cuffed, double-lumen tracheal tube with a central flange. The flange enables blind tube positioning on the carina and the side-by-side arrangement of each lumen in situ allows selective blind placement of a bronchial blocker to each lung as required.

When lung resection is not required, or if there are no issues with cross-contamination (e.g. pleural surgery, lung biopsy, and oesophagectomy), either a double-lumen tube or an endobronchial blocker may be utilized for OLV. In this situation, endobronchial blockers have a natural tendency to dislodge and cause airway obstruction. Hypoxaemia and cardiac arrest associated with malpositioning and airway obstruction have been reported.⁹

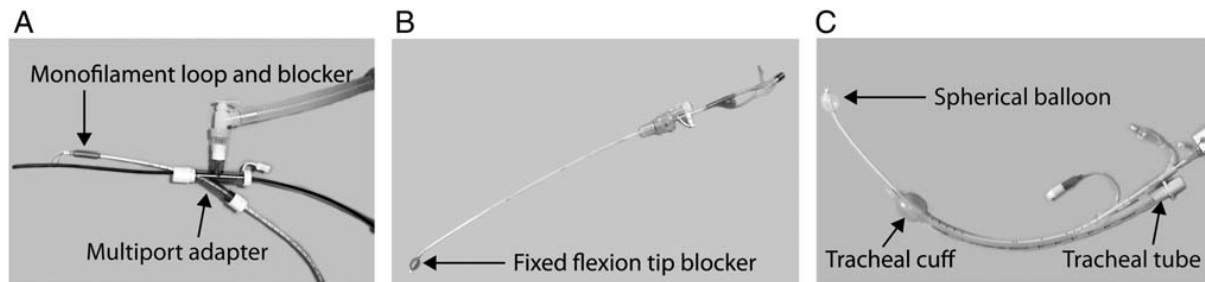


Fig 2 Endobronchial blockers: (A) Arndt endobronchial blocker, (B) Coopdech endobronchial blocker, and (C) Univentw Torque Control blocker

Pathophysiology of hypoxaemia

Patients who require OLV for thoracic surgery are placed in the lateral decubitus position. The lower, dependent lung is ventilated, whereas the upper, non-dependent lung is allowed to collapse when opening the chest. Factors predictive of hypoxaemia during OLV include: ventilation of the left rather than the right lung, low oxygen partial pressure on two lungs, absence of reduction of perfusion to areas of lung pathology, and supine position rather than the lateral decubitus position.¹

Development of hypoxaemia, caused by OLV may be explained by considering oxygen storage, dissociation of oxygen from haemoglobin, the relationship between ventilation and perfusion, and factors that reduce the effect of hypoxic pulmonary vasoconstriction.

Oxygen storage, oxygenation, and ventilation

Patients are susceptible to hypoxaemia owing to a reduction in the functional residual capacity and oxygen storage during OLV, in addition to the effects of anaesthesia and the lateral decubitus position. Atelectasis in the ventilated dependent lung occurs as a result of:

- compression caused by the weight of the mediastinum;
- compression by abdominal contents after diaphragmatic muscle relaxation;
- increased closure of small airways with old age, reduced elastic recoil, and the lateral decubitus rather than the erect position.

Dissociation of oxygen from haemoglobin

During OLV, there is a reduction in arterial oxygen partial pressure and also permissive hypercarbia and respiratory acidosis. These physiological changes lead to rapid dissociation of oxygen from haemoglobin (Bohr effect), as shown by the steep slope of the oxygen haemoglobin dissociation curve.

Ventilation–perfusion relationship

Abnormalities of ventilation and perfusion of the respiratory system may be considered in three situations:

Spontaneous ventilation.

When the patient is breathing spontaneously, ventilation, under negative pressure, is greater in the dependent lung than in the non-dependent lung (except at the very bottom of the dependent lung). Similarly, perfusion in the dependent lung is greater than that in the non-dependent lung. Thus, the ventilation–perfusion relationship is normal and is similar to that observed if the patient is sitting in the standard upright position (Fig. 3A).

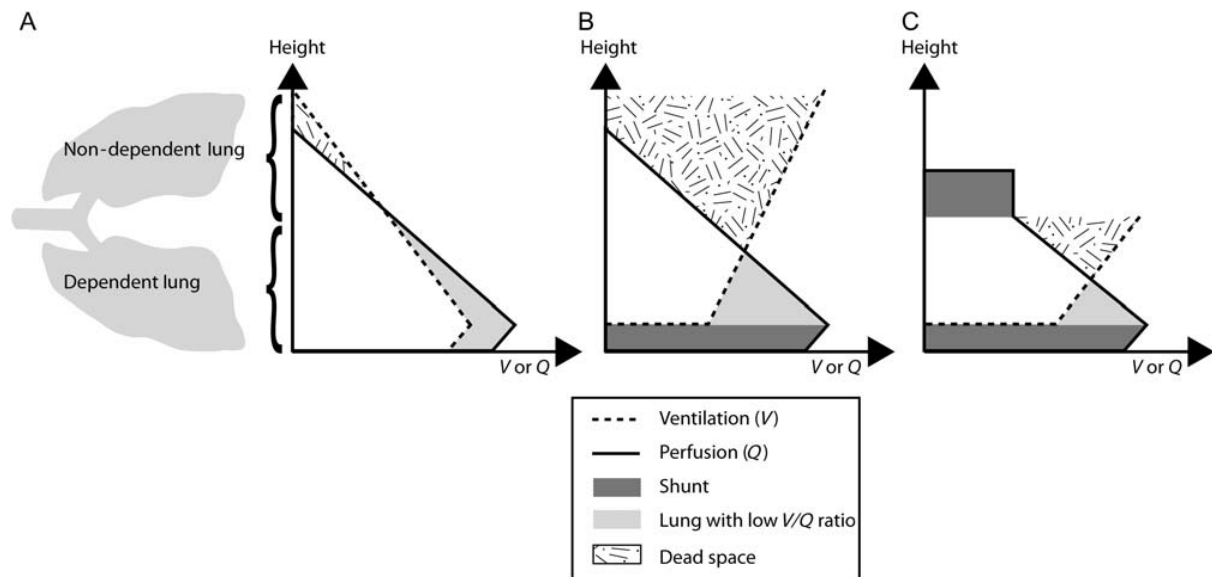


Fig 3 Schematic diagrams showing ventilation–perfusion relationship in the lateral decubitus position during: (A) spontaneous ventilation, with two lungs, (B) positive pressure ventilation to both lungs, and (C) positive pressure ventilation to the dependent lung.

Positive pressure ventilation with two lungs.

The relationship of ventilation and perfusion changes when the patient is paralysed. In this situation, positive pressure ventilation is directed preferentially to the non-dependent lung, whereas perfusion remains greater in the dependent lung than in the non-dependent lung (Fig. 3B). Thus, compared with spontaneous ventilation, positive pressure ventilation leads to an increase in areas of lung with low ventilation–perfusion ratios. The volume of dead space, that is to say, ventilated lung with no perfusion, also increases during positive pressure ventilation; this effect contributes to hypercarbia.

Positive pressure ventilation to one lung.

During OLV, when the non-dependent lung is not ventilated, some perfusion still remains in this lung, despite hypoxic pulmonary vasoconstriction (Fig. 3C). Thus, there may be a substantial increase in shunt and hence hypoxaemia.

Hypoxic pulmonary vasoconstriction

Hypoxic pulmonary vasoconstriction in the extra-alveolar pulmonary arterioles supplying the unventilated lung is an essential physiological response to minimize shunt and hence hypoxaemia during OLV. This effect occurs when there is a reduction in alveolar partial

pressure of oxygen to between 4 and 8 kPa. Factors that impair hypoxic pulmonary vasoconstriction in the non-ventilated lung and hence promote hypoxaemia during OLV include:

(i) Increase in pulmonary artery pressure which may occur when:

(a) there is an increase in pulmonary vascular resistance caused by atelectasis in the ventilated lung;

(b) excessive positive end-expiratory pressure is applied or develops intrinsically in the ventilated lung. There is a diversion of blood to the non-ventilated lung;

(c) vasoconstrictor drugs, for example, phenylephrine and epinephrine, are administered.

(ii) Supine position. In this position, the vertical height of the lungs is less than in the lateral decubitus or erect position.

This reduction of height allows blood flow to a greater proportion of unventilated collapsed lung, thus attenuating

hypoxic pulmonary vasoconstriction

iii) Failure of lung collapse. Provided there are no adhesions, the unventilated non-dependent lung should collapse when the

chest wall is open to the atmosphere. If this unventilated lung is held partially open, then intrapleural pressure becomes

increasingly negative during inspiration. This effect leads to an increase in transpulmonary pressure (i.e. the pressure

difference between alveoli and pleura) and hence distension of extra-alveolar pulmonary vessels that oppose hypoxic pulmonary vasoconstriction in the non-ventilated lung.

(iv) Vasodilators, for example, calcium antagonists, sodium nitroprusside, nitrates, α -antagonists, inhalation anaesthetic agents, and endogenous prostaglandins, released during lung handling.

Management of intraoperative hypoxaemia during OLV

Strategies to manage hypoxaemia may be divided into three main categories: delivery of oxygen to the patient, treatment of causes associated with high airway pressure, and management of physiological hypoxaemia.

Physiological hypoxaemia

In the absence of problems related to delivery of oxygen and high airway pressure, hypoxaemia may be due to shunt and inadequate ventilation caused by OLV. In this situation, a number of strategies may be utilized to prevent further desaturation, these include:

(i) insufflation of oxygen to the non-ventilated lung to improve oxygenation of shunted blood.

(ii) continuous positive airway pressure (with oxygen) to the nonventilated lung to attenuate hypoxaemia of shunted blood.

(iii) application of positive end-expiratory pressure to the dependent lung to improve functional residual capacity and hence oxygenation. However, excessive PEEP may divert blood to the non-ventilated lung and exacerbate shunt and hence hypoxaemia.

(iv) optimization of haemoglobin and cardiac output to improve oxygen delivery.

(v) utilization of high-frequency jet ventilation rather than conventional ventilation, provided complete lung collapse is not required.

(vi) pharmacological methods such as administration of nitric oxide and almitrine on an occasional basis.

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 and equivalent to sevoflurane or desflurane. Intravenous anesthetic techniques have not been shown to provide better oxygenation than the newer volatile anesthetics. Manipulating the ventilating pressures and tidal volumes during one-lung anesthesia can improve the oxygenation for certain patients. It is not yet possible to predict the optimal ventilatory settings for an individual patient. The use of a 10 ml/kg tidal volume while limiting plateau airway pressure to 25 cm H₂O at end inspiration are useful initial parameters for OLV in the majority of patients. A third of the 35-40% 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 lung. 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 saturation. Patients whose PaO₂ declines rapidly 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.

Predictors of Hypoxemia: Several factors allow prediction of the risk of hypoxemia developing during OLV. First, the A-aO₂ gradient during two-lung ventilation. Second, the side of lung collapse during OLV. The mean PaO₂ level is 70 mmHg higher for left vs right thoracotomies. Third, patients with good preoperative spirometric pulmonary function tests tend to have lower PaO₂ values during OLV than patients with poor spirometry. This may be related to auto-PEEP in patients with poor spirometry.

Prophylaxis and Treatment: Other potential 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 an $\text{FiO}_2 > 0.3$ was used intraoperatively for thoracic surgery. Continuous positive airway pressure (CPAP) to the non-ventilated lung is the other first-line of defence and treatment. Useful increases in oxygenation can be achieved with 1-2 cm H₂O CPAP⁸. CPAP must be applied to the fully inflated lung. Even short periods of lung collapse impair the efficiency of CPAP since the opening pressure of atelectatic lung units exceeds 20 cm H₂O. Because of the problems which re-inflation may cause at an inopportune surgical moment, it is useful to predict which patients are most at risk of hyperemia and to apply CPAP prophylactically at the onset of OLV. Increasing cardiac output during OLV increases PaO₂ via an increase in mixed venous oxygen content since these patients have such a large shunt. PEEP to the ventilated lung decreases PaO₂ in the majority of patients during OLV probably by exacerbating the pressure differential between the thoraces. A minority of patients, often those with the poorest PaO₂ values, benefit from 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. The patients most likely to benefit from PEEP are those patients with an increased A-aO₂ gradient in the lateral position during two-lung ventilation and a low level of auto-PEEP during OLV. During pneumonectomy, lung transplantation or in life threatening situations, the ipsilateral pulmonary artery can be compressed or clamped by the surgeon. Pulmonary artery balloon-tipped floatation catheters can be placed under fluoroscopic control and inflated to decrease regional pulmonary blood flow. High frequency jet ventilation (HFJV) to the operative lung provides superior oxygenation. 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 now 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.

Summary and Conclusion.

A systematic approach should be used to treat hypoxia. Oxygen supply and ventilator function should be checked. Blockage or disconnection of the breathing circuit must be ruled out. It is vital to ensure that the endotracheal tube, be it single or double lumen, is still correctly positioned. If available this should be done with a fibre optic bronchoscope.

Hypoxaemia may occur at any time and with rapidity during OLV. The use of a high FiO₂ and CPAP to the non-ventilated lung remain the first line of therapy for Desaturation during OLV. It is now possible to identify in advance the minority of patients who will benefit from PEEP to the ventilated lung.

Administer 100% Oxygen

- Check Ventilator, Circuit and catheter mount
- Clear secretions and debris by suctioning dependent lung
- Check tube position
- Apply CPAP or entrain Oxygen to non dependent lung
- Perform recruitment manoeuvre and apply PEEP to dependent lung
- Revert to two lung ventilation
- Clamp non-dependent pulmonary artery

If oxygenation remains problematic, definitive management of the shunt caused by one lung ventilation is to instruct the surgeon to clamp the pulmonary artery. If this does not work it may be necessary to revert back to double lung ventilation, which may be quicker and easier to perform if a double lumen tube has been used. If adequate oxygenation cannot be established with ventilation of both lungs it may be necessary to abandon the surgical procedure.

References

1. Karzai W, Schwarzkopf K. Hypoxaemia during one lung ventilation. *Anesthesiology* 2009; 110: 1402–11
2. Joen Y, Ryu HG, Bahk JH, Jung CW, Goo JM. A new technique to determine the size of double-lumen endobronchial tubes by the two perpendicularly measured bronchial diameters. *Anaesth Intensive Care* 2005; 33: 59–63
3. Narayanaswamy M, McRae K, Slinger P et al. Choosing a lung isolation device for thoracic surgery: a randomized trial of three bronchial blockers versus double-lumen tubes. *Anesth Analg* 2009; 108: 1097–101
4. Cohen E. The Cohen flexitip endobronchial blocker: an alternative to a double lumen tube. *Anesth Analg* 2005; 101: 1877–9
5. Campos JH, Kernstine KH. A comparison of a left-sided Broncho-Cath with the Torque Control Blocker Univent and the wire-guided blocker. *Anesth Analg* 2003; 96: 283–9
6. Ghosh S, Klein AA, Prabhu M, Falter F, Arrowsmith JE. The Papworth BiVent tube: a feasibility study of a novel double-lumen endotracheal tube and bronchial blocker in human cadavers. *Br J Anaesth* 2008; 101:424–8
7. Ho CYA, Chen CY, Yang MW, Liu HP. Use of the Arndt wire-guided endobronchial blocker via nasal for one-lung ventilation in patient with anticipated restricted mouth opening for oesophagectomy. *Eur J Cardiothorac Surg* 2005; 28: 174–5
8. Knoll HK, Ziegeler S, Schreiber JU et al. Airway injuries after one-lung ventilation: a comparison between double-lumen tube and endobronchial blocker. *Anesthesiology* 2006; 105: 471–7
9. Sandberg WS. Endobronchial blocker dislodgement leading to pulseless electrical activity. *Anesth Analg* 2005; 100: 1728–30
10. Campos JH, Hallam EA, Van Natta T, Kernstine KH. Devices for lung isolation used by anesthesiologists with limited thoracic experience. *Anesthesiology* 2006; 104: 261–6

