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Hypoxemia During One-Lung Ventilation

KEYWORDS: One-lung ventilation, Hypoxemia, Thoracotomy

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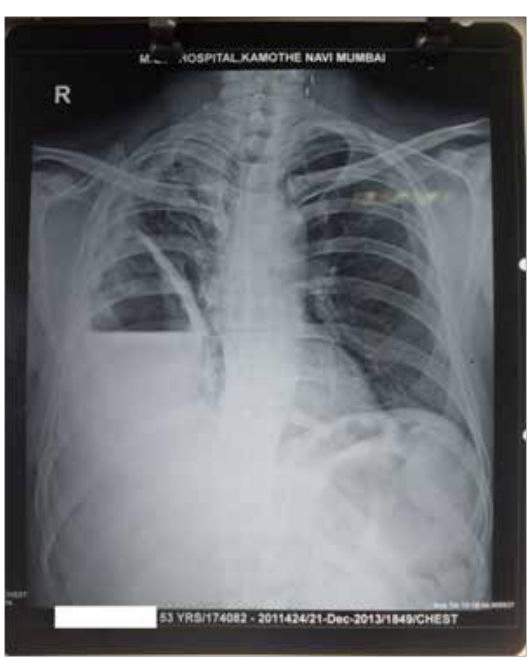
ABSTRACT *Modern techniques to isolate the lungs, coupled with accurate continuous non-invasive monitoring, have made one-lung ventilation (OLV) during surgical procedures safe and easy to perform. Hypoxemia i.e. Arterial haemoglobin oxygen saturation of less than 90%, occurs in 5-10% of cases during OLV1. The pathophysiology of hypoxemia is complex, and the management of hypoxemia during OLV remains a challenge for anaesthesiologists. Here is a case report of hypoxemic event during OLV for thoracotomy, wherein efforts were directed towards optimizing perfusion and ventilation to the ventilated lung and increasing the oxygen content of blood returning from the collapsed lung.*

CASE REPORT

A 53-year-old, 68-kg male having a right lung empyema was scheduled for decortication of the right pleura under general anaesthesia. Preanaesthetic examination revealed diminished breath sounds and coarse crackles over the right middle and lower lung zones on chest auscultation. Chest radiograph showed obliteration of the right costophrenic angle and a thickened, calcified pleura with an air-fluid level. The adjacent middle and lower lobes of lung were collapsed and fibrosed with deviation of the trachea towards the right. Computed tomography (CT) scan showed fluid density collection in the right pleural space with collapse and fibrosis of the adjacent middle and lower lung lobes. Pulmonary function testing showed FEV1, FVC, FEV1/FVC, FEF25-75, and MVV of 107%, 9%, 114%, 128%, and 72% of the age predicted values respectively. He had good exercise tolerance. All the other clinical and biochemical investigations were normal. The patient was accepted for surgery under American Society of Anaesthesiologists grade II.



(Image 1) Chest radiograph showing right sided empyema

In the operating room, peripheral venous, central venous, and arterial lines were taken and monitors attached. A 16G catheter was introduced and fixed in thoracic epidural space for continuous epidural analgesia. After routine pre-medication, general anaesthesia was induced with 120mg of Propofol and intubation was facilitated with 6mg of Vecuronium once adequacy of bag and mask ventilation was confirmed. After laryngoscopy, Robertshaw left-sided double lumen tube (DLT) number 37 was introduced, tracheal cuff inflated, and fixed at 30 cm mark after confirmation by fibreoptic bronchoscopy and chest radiography by C-arm. Ability to isolate right lung was confirmed by clamping the tracheal lumen with the bronchial cuff inflated. Both lungs were kept ventilated at 12cycles/min rate, 500ml tidal volume, 40% FiO_2, and 5cm H_2O positive end-expiratory pressure (PEEP). Baseline arterial blood gas (ABG) analysis was done. End-tidal carbon dioxide ($EtCO_2$) was 32mmHg and plateau pressure was 20cm H_2O. Patient was given left lateral position. DLT position was reconfirmed. Once the right pleura was adequately exposed, the right lung was isolated. FiO_2 was increased to 100% and respiratory rate adjusted to 16 cycles/minute to maintain arterial carbon dioxide tension between 36-40mmHg. Within 15 minutes, the patient exhibited profound hypoxemia with a decrease in pulse oximetry from 97% to 86%. ABG values were pH 7.39, PaO_2 54mmHg (100% FiO_2), $PaCO_2$ 40mmHg, HCO_3 24.8mM, and 87% SaO_2. Haemodynamics and electrocardiogram remained stable. Expiratory flow and expiratory tidal volume were unchanged, and no circuit leak was noticed. The correct position of the DLT was immediately confirmed by fibreoptic 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 suspected. Once the surgeon was informed, the non-ventilated lung was expanded manually (checked visually) by administration of 100% oxygen at airway pressure of 20cmH_2O for 10s every 5 minutes for a total of 15 minutes. This produced rapid improvement in the patient's oxygenation. Haemodynamics remained stable during the maneuver. Thereafter, a separate breathing circuit was attached to the tracheal lumen of the DLT and this was used to maintain an airway pressure of 5-10cm H_2O by adjustment at the pressure limiting valve of resevoir bag. The pulse oximetry value remained above 95% for remaining 2 hour duration of surgery. The postoperative course of this patient was uneventful. He did not exhibit any hypoxemia post extubation.

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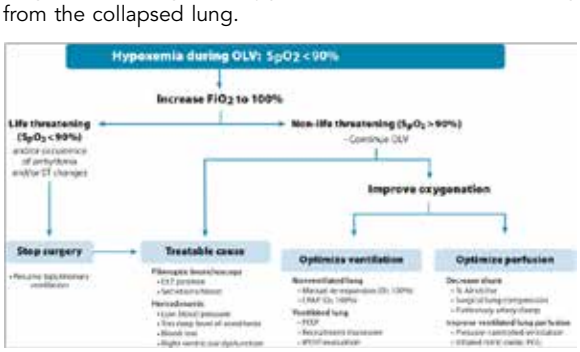
(Image 2) Head-end view showing lines and tubes in-situ-- A: 16G thoracic epidural catheter; B: 6Fr Triple-lumen central venous catheter in right-sided internal jugular vein; C: 16FG Nasogastric tube; D: No.37 DLT with clamp (open) over tracheal (right-sided) lumen; E: 20G arterial cannula in right radial artery.

(Image 3) Chest radiograph showing correct placement of left-sided DLT

DISCUSSION

Thoracic operations are usually performed in the lateral position with selective ventilation of the dependent lung. The intentionally collapsed non-dependent lung continues to be perfused with around 20-25% of cardiac output (Shunt)[2]. Therefore, in order to maximize oxygenation, efforts are directed towards either optimizing the matching of ventilation with perfusion in the dependent ventilated lung or increasing the oxygen content of blood returning from the collapsed lung.



(Figure 1) Proposed algorithm for management of hypoxemia during one-lung ventilation (From Hadrien R, Mathieu L,Alexandre O.Hypoxemia and One-lung ventilation.Anesthesiology 2011; 114: 168.)

DIAGNOSIS OF A TREATABLE CAUSE OF HYPOXEMIA

In case of hypoxemia, correct positioning of the DLT must be systematically confirmed by fibreoptic[1,3] and the respiratory tract must be cleared of any blood or secretions. As OLV induces increases in right ventricular overload, intraoperative assessment of ventricular function may be useful.[4] Right ventricular dysfunction may also be related to volume overload or intrinsic myocardial depression.[4] Despite some limitations in the analysis of right ventricular function, transesophageal echocardiography is probably the best intraoperative technique to use during OLV> The two main signs that must be sought are the right ventricular dilatation as well as paradoxical septal motion.[5] Volume depletion and too deep a plane af anaesthesia must be looked into.

VENTILATION STRATEGY DURING ONE-LUNG VENTILATION

In Non-ventilated lung:

100% oxygen may be administered followed by application of CPAP of 5-10cm H_20. If no CPAP valve is available, as in this reported case, a separate breathing circuit may be used to ventilate non-dependent lung, with airway pressure maintained at 5-10cm H_2O using pressure limiting valve of reservoir bag.

In Ventilated lung:

Volume Controlled Ventilation (VCV)

Even with a shunt as high as 25%, a FiO_2 of 1.0 and large tidal ventilation (10-12ml/kg) usually results in a PaO_2 greater than 150mmHg during OLV.[6] At this PaO2, arterial haemoglobin is 100% saturated. A high FiO_2 causes vasodilation of the vessels in the dependent lung that increases perfusion of that lung and further decreases shunt. Tidal volumes less than 8ml/kg decrease FRC further and tidal volumes greater than 15ml/kg over-distend the alveoli and increase PVR in the dependent lung, resulting in an increase in shunt to non-dependent lung.

Application of PEEP

Conflicting results have been reported concerning the application of PEEP to the dependent lung during OLV.[7,8] In some patients, the application of PEEP to the dependent lung might be beneficial by restoring functional residual capacity to normal values,[9] thus preventing atelectasis when its value is titrated along the static compliance curve.[10] In obstructive patients, the use of PEEP may sometimes interefere with iPEEP and decrease oxygenation.[7,10,11]

Pressure Controlled Ventilation (PCV)

The use of PCV during OLV does not lead to improved oxygenation during OLV compared with VCV for patients with good preoperative pulmonary function, but PCV did lead to lower peak airway pressures.[12]

High frequency jet ventilation(HFJV) to the nondependent lung provides not only satisfactory oxygenation but also good cardiac output, thereby maintaining better oxygen transport than CPAP or deflation to atmospheric pressure, while the dependent lung is ventilated with Intermittent positive pressure ventilation(IPPV) during one-lung ventilation for thoracotomy.[13]

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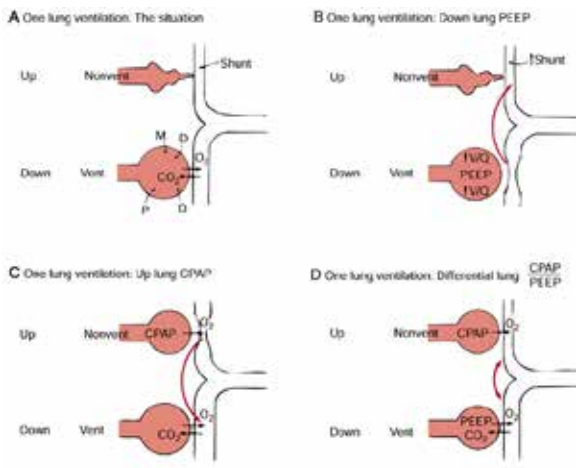


(Figure 2) Four-part schematic diagram showing the effects of various differential lung management approaches. (A) The one-lung ventilation situation. The DOWN (dependent) lung is ventilated (VENT) but is compressed by the weight of the mediastinum (M) from above, the pressure of the abdominal contents against the diaphragm (D), and by positioning effects of rolls, packs, and shoulder supports (P). The UP (nondependent) lung is nonventilated (NONVENT), and blood flow through this lung is shunt flow. (B) The dependent lung has been selectively treated with PEEP. (C) Selective application of CPAP to the nondependent lung. (D) Differential lung CPAP (nondependent lung)/PEEP (dependent lung).

EFFECT OF GRAVITY ON SHUNT

Studies conducted by Watanabe S et al demonstrated that during OLV with a patient in the lateral position, gravity augments the redistribution of perfusion to the ventilated lung, resulting in a better V/Q match and a higher PaO2, as compared to supine or semi-lateral positions.[14]

HYPOXIC PULMONARY VASOCONSTRICTION AND ANAESTHETIC AGENTS

Regional hypoxia in the lung causes arteriolar constriction, with diversion of the blood flow away from the hypoxic segment to normal areas of lung (Hypoxic pulmonary vasoconstriction; HPV). By redistributing cardiac output from poorly perfused areas to better-ventilated regions, V/Q is maximised. Under experimental conditions, HPV is an important regulator of blood flow to atelectatic lung. The onset of HPV is within 7s in isolated lungs and resolves within minutes of normoxic ventilation.[15]

From in-vitro animal studies, all intravenous anaesthetic and sedative agents do not alter the HPV response.[16] Propofol may actually potentiate HPV.[17,18]

In contrast, in-vitro studies[19] have demonstrated that inhaled halogenated anaesthetic agents all inhibit HPV in a dose-dependent manner. However, an anaesthetic agent that lowers cardiac output more than it decreases oxygen consumption will also lower PvO2 producing a potent stimulus for HPV. A decrease in cardiac output will result in less blood flow to the collapsed lung because of the higher pulmonary vascular resistance (PVR) already present in that lung. These actions counter the direct depression of HPV by the anaesthetic agent.

CHOICE OF ANAESTHETIC AGENT FOR THORACIC SURGERY

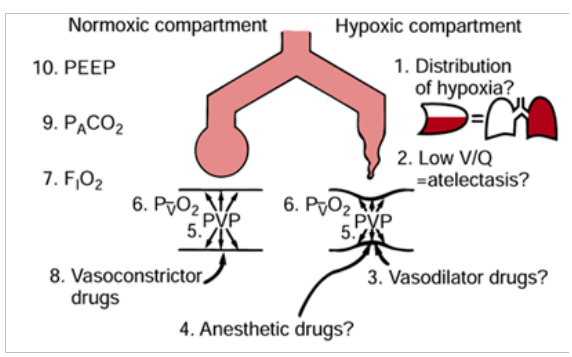
Pilotti et al[20] conducted a study on 50 patients undergoing OLV for pulmonary resection. One group received total intravenous anaesthesia (TIVA) and the second, an inhalational anaesthetic. Blood pressure, heart rate and arterial carbon dioxide tension levels were similar in both groups. As a group, the TIVA group patients had higher PaO_2 levels during OLV.

SURGEON'S ROLE

The surgeon has access to the perfusion of the non-dependent lung. Consequently, he/ she is able to reduce pulmonary blood flow, and therefore transpulmonary shunt.

PHARMACOLOGICAL MANAGEMENT OF ONE-LUNG VENTILATION

In animal studies[21] direct infusion of prostaglandin F_{2a}, a potent pulmonary vasoconstrictor, into the pulmonary artery of the non-ventilated lung causes a significant decrease in shunt and an increase in PaO_2. Phenylephrine has also been used as a non-specific pulmonary vasoconstriction to improve oxygenation.[22] Aerosolized prostacyclin[23] and inhaled nitric oxide (iNO) improve gas exchange and pulmonary shunt by redistributing pulmonary blood flow from non-ventilated to aerosol-accessible ventilated lung regions. Administration of almitrine besylate (a potent pulmonary vasoconstrictor) to decrease shunt to poorly ventilated lung increases oxygenation in animals.[24]



(Figure 3) Potential therapeutic target for improving oxygenation during one-lung ventilation

CONCLUSION

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),[15,25] preoperative function tests,[26] underlying disease, anaesthetic management, and surgical proecedure. During OLV ventilation with 100% oxygen and optimization of dependent lung FRC will produce a safe PaO_2 for most patients. If hypoxemia does occur, manipulation of ventilatory parameters and the application of PEEP to the dependent lung or CPAP to the collapsed lung will usually correct the problem. In the future, pharmacological manipulation may be helpful during OLV, but to date such interventions have not been very successful.

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