

Update on thoracic anesthesia

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A variety of thoracic surgical procedures, such as lobectomy, pneumonectomy, esophago-gastrectomy, pleural decortication, bullectomy and bronchopulmonary lavage are commonly performed. Customarily they are classified either as absolute or as relative. *The absolute indications* include life-threatening complications, such as massive bleeding, sepsis and pus, where the non diseased contralateral lung, must be protected from contamination. Broncho-pleural and broncho-cutaneous fistulae are absolute indications since they offer a low resistance pathway for the delivered tidal volume during positive pressure ventilation. Giant unilateral bullae may rupture under positive pressure and ventilatory exclusion is mandatory. Finally, during bronchopulmonary lavage for alveolar proteinosis or cystic fibrosis, prevention of the contralateral lung from drowning is necessary. Video assisted thoracoscopy (VAT) brought to the practice of surgery for diagnostic and therapeutic procedures required a well collapse lung and should be included in the absolute indication for OLV category. *Relative indications* which includes lobectomies (particularly right upper), pneumonectomy, and thoracic aortic aneurysm repair, are primarily for surgical exposure. Lower or middle lobectomy and esophageal resection are of lower priority. In practice, the majority of the procedures where DLT is used, are in essence relative indication, and only a small fraction are absolute. The use of OLV for relative indications rely on the surgeon-anesthesiologist practice and preference.

Clinical approach to management of OLV: Once the patient has been placed in the lateral decubitus position, proper DLT position should be re-checked, since dislocation during position change is not uncommon. Two-lung ventilation should be maintained for as long as possible. When OLV is required, a FiO_2 of 1.0 provides a high margin of safety to protect against possible hypoxemia. With a FiO_2 of 1.0, assuming an intact hypoxic pulmonary vasoconstriction (HPV) response, PaO_2 during OLV should be between 150-210 mmHg. The patient should be ventilated with a

tidal volume of 10-12 ml/kg at a ventilatory rate to maintain a PaCO_2 of 35 ± 3 mmHg that can be estimated from the end-tidal CO_2 value. Low tidal volume might produce atelectasis in the ventilated lung (reduced FRC) and increases in the degree of shunt. High tidal volume might shift blood flow into the non-dependent lung (similar to the application of PEEP) to increase the transpulmonary shunt. Following the initiation of OLV, PaO_2 can continue to decrease for up to 45 minutes, hence, close monitoring of arterial blood gases and/or the use of a pulse oximeter are indispensable. Should hypoxia occur, proper positioning of the DLT should be re-conform by fiberoptic bronchoscopy. Several techniques can be employed to improve oxygenation. The most effective maneuver for improving PaO_2 is the application of a continuous positive airway pressure of 10 cm H_2O (CPAP_{10}) to the non-dependent lung. It consists of insufflation of oxygen under positive pressure to keep a "quiet" lung, while preventing it from collapsing completely. The beneficial effect of CPAP_{10} is not secondary to the positive pressure effect, potentially causing blood flow diversion to the dependent perfused lung, but from distending the alveoli with oxygen to allow gas exchange. Hyperinflation of nitrogen under positive pressure into the non-dependent lung failed to improve PaO_2 . Most studies confirmed that dramatic improvement in PaO_2 values with the application of CPAP_{10} . PEEP may be added to the dependent lung, or in combination with CPAP_{10} to the non-dependent lung. PEEP_{10} alone added to the dependent lung, resulted in either no change or in a decrease in PaO_2 values. Most probably, the beneficial effects of PEEP to the dependent lung, assumed to be subsequent to expansion of atelectatic alveoli, are offset by the increased blood flow diverted to the non-ventilated lung from the continuous positive pressure. In summary, the preferred method to manage hypoxemia during OLV is by application of CPAP to the non-dependent lung with a search for an optimal combination of PEEP and CPAP (between 5-10 cm of H_2O). In exceptional cases,

despite all of these maneuvers, PaO_2 will fail to improve and intermittent ventilation of the non-dependent lung should be reinstituted with the surgeon's collaboration. Depending on the stage of the surgery, if a pneumonectomy is planned, ligation of the pulmonary artery will eliminate the shunt through that lung. Should doubt arise as to the stability of the patient, (patient becomes hypotensive, dusky, or tachycardic), two-lung ventilation should be resumed until the problem is settled.

Finally, there are several important issues to keep in mind during management of OLV. First, when using a right-sided DLT, adequate ventilation to the right upper lobe should always be confirmed with the help of fiberoptic bronchoscopy. Second, when using a left-sided DLT for right thoracotomy, where the patient is dependent on the left lung, the tip of the left-sided tube may obstruct the left upper lobe orifice. Thus, should hypoxemia result during right thoracotomy with left-sided tube, correct position of the tube should be readjusted with fiberoptic bronchoscopy. Withdrawal of the tube by 1 cm and re-expansion of the left upper lobe typically resolve the hypoxemia. Manipulation of DLT can involve the surgeon as well. Palpation and manual occlusion of the main bronchial lumens can guide the tip of the DLT to the correct position. Finally, peak airway pressure, delivered tidal volume (as measured by spirometer) and the capnogram shape, should be inspected continuously to identify an obstructive or a low end-tidal value from inadequate gas exchange subsequent to DLT malposition. A peak airway pressure of up to 40 cm of H_2O on OLV is acceptable. A sudden increase in the peak airway pressure may be from tube dislocation due to surgical manipulation.

VIDEO ASSISTED THORACOSCOPY

The improvements in video endoscopic surgical equipment and a growing enthusiasm for minimally invasive surgical approaches brought VAT to the practice of surgery for diagnostic and therapeutic procedures. Most of these procedures required a well collapsed lung and should be included in the absolute indication for OLV category. The indications for VAT are summarized in the Table. The patient population tends to be either very healthy, undergoing diagnostic procedures, or high-risk patients who are undergoing VAT to avoid the stress of a thoracotomy. The patients with advanced cardiopulmonary disease should have an extensive pre-operative evaluation and intraoperative monitoring the same as for thoracotomy. Small incision in the chest wall permit the insertion of the video camera and the surgical instruments. In most cases general anesthesia with one lung ventilation is required. The lung should be well collapsed to allow the surgeon an optimal view of the surgical field and to palpate the lesion in the lung parenchyma. Because

it may take 30 minute for the lung to collapse it is advisable to initiate OLV immediately following endobronchial intubation. In some cases CO_2 is insufflated to facilitate visualization, at pressure < 10 mm H_2O with a flow of less than 2 l/min. Higher pressure may cause mediastinal shift with cardiovascular compromise. In a very small selected group of patients to avoid the need for endobronchial intubation, VAT may be performed under regional anesthesia using an intercostal block or epidural. The patients for regional VAT should be carefully selected and the risk benefit should be considered. The procedure should be simple and of a short duration, the patient's airway should be easy to establish in case of an emergency. Pain stimuli from the apex of the lung and the pleura are poorly blocked by the intercostal nerve block, therefore procedure such as pleural abrasion should be done under general anesthesia. Finally, the awake patient breathing spontaneously with an open chest, may develop hypoxemia and hypercarbia from paradoxical breathing and hypotension from mediastinal shift. These complications can be treated by applying a slight positive pressure via face mask.

NITRIC OXIDE AND ONE LUNG VENTILATION

Nitric oxide (NO) is an important endothelium-derived relaxing factor. It is produced by the endothelium from L-arginin via metabolic pathway that requires NO synthase, and diffuses into subjacent vascular smooth muscle to cause relaxation and vasodilatation. NO combine intracellularly with the heme present in guanylate cyclase. This activation of the guanylate cyclase lead to smooth muscle relaxation through the synthesis of guanosine 3,5-cyclic monophosphate (cGMP). Clinically used nitrovasodilators such as nitroprusside and nitroglycerin exert their effects by releasing NO intracellularly. In addition to regulating vascular tone endogenously produced NO is important in regulating several other physiologic functions, including platelet aggregation, neurotransmission, and antitumor and antimicrobial activity. Inhaled NO (5-80 ppm) has been shown to decrease pulmonary vascular resistance. NO have selective dilating effects on the pulmonary circulation without effect on the systemic circulation. Because inhaled NO is quickly inactivated by the hemoglobin in the vascular lumen, it has direct systemic effects. The half life of NO is between 110-130 ms, thus exogenous inhaled NO may diffuse from the alveoli to pulmonary vascular smooth muscle and produce pulmonary vasodilatation, but any NO that diffuses into blood will be inactivated before it can produce systemic vasodilatation. Since NO distributes directly into the alveoli, it is a selective microvasodilator only of those capillaries adjacent to ventilated alveoli, and therefore improve V/Q matching. This effect is in contrast to NTP or NTG, that

when given IV, cause a non-selective capillary dilatation of poorly ventilated alveoli, which result in deterioration of oxygenation. These properties of NO on the pulmonary circulation are used in a variety of clinical applications. Frost-ell et al, described the effect of inhaled NO during hypoxia in human volunteers, inhaled NO selectively reversed HPV without causing systemic vasodilatation. A major application of inhaled NO is in congenital heart disease associated with pulmonary hypertension with large left to right shunt and pulmonary hypertension.

The concept that the pulmonary circulation can be modulated by the administration of NO to selective areas of the lung may be important during one lung ventilation. In theory, the administration of NO during OLV will cause vasodilatation of that lung, that will enhance the effect of HPV in the non-dependent lung to increase blood flow to the dependent lung and reduce the degree of shunt. Preliminary studies during OLV presents mixed results. Booth et al, administered NO at 40 ppm to 9 patients during OLV reported improvement in oxygenation during OLV compared to a control group of 6 patients (26.8 Pka vs 12.6 Pka). More recently Wilson et al, found no improvement in 6 patients with the administration of NO at 40 ppm. Os/Qt did not change in these patients with a normal PVR. The beneficial effect of NO is limited in the absence of hypoxemia and pulmonary vasoconstriction. Most likely those patients with hypoxemia and elevated pulmonary pressure during OLV will benefit from the application of NO. One of the most interesting future concepts to keep adequate oxygenation during OLV, is the ability to modulate the lung circulation. Almitrine bimesylate a peripheral chemoreceptor agonist, increases pulmonary hypoxic vasoconstriction in low doses. In fact inhaled NO and intravenous Almitrine have been combined with additive effects on gas exchange. In case of OLV using that combination will maximize the HPV of the non-dependent lung while dilate the dependent lung to practically eliminate the transpulmonary shunt (Figure 1). That theory was tested by Moutafis et al, in 20 patients undergoing thoroscopic lung resection. The group of patients that received the combination of Almitrine and NO has almost no decrease in PaO_2 during OLV. That is important for those patients with a marginal respiratory reserve who are unable

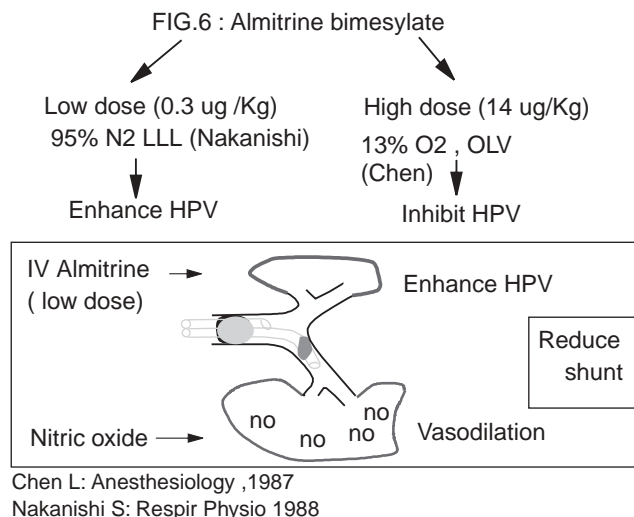


Figure 1. Almitrine bimesylate.

to maintain oxygenation on OLV. Finally, it would be useful during VAT where the application of CPAP to the non-dependent lung interferes with the surgeon's ability to view the surgical field.

CLINICAL APPROACH TO OLV MANAGEMENT

1. Use FiO_2 of 1.0.
2. Ventilate with a TV of 10-12 ml/kg.
3. Respiratory rate to maintain $PaCO_2$ between 33-35 mmHg.
4. Check the DLT position subsequent to the lateral decubitus positioning.
5. If peak airway pressure exceeds 40 mmHg during OLV DLT malposition should be excluded.
6. For hypoxemia, apply CPAP10 cm H_2O to the non-dependent lung.
7. If additional correction of hypoxemia is necessary add PEEP 5-10 cm H_2O to the ventilated lung.
8. Or intermittently inflate and deflate the operated lung.
10. Keep in mind that arterial oxygenation can decrease up to 45 minutes following initiation of OLV.

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