One-lung anaesthesia-Pathophysiology, Conduct and Techniques

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One lung ventilation (OLV) or one lung anaesthesia means separation of the two lungs and each lung functioning independently by preparation of the airway. It continues to remain as one of the challenging and skilled techniques of anesthesia practice. The need for isolating one lung from the other for different surgical procedures on the lungs, mediastinum or great vessels is well established. A variety of techniques have been developed to isolate one lung and considerable clinical experience has been gained over last two decades. Knowledge about the physiology of ventilation and perfusion at different positions and during one lung ventilation has made one lung anaesthesia a safe technique in the majority of patients. However in some, severe hypoxemia may occur. This review focuses on the different physiological and anatomical variables causing hypoxia and measures to counteract it.

Selective lung ventilation for thoracic surgery was first reported in 1931[1]. OLV was originally performed for basically two reasons, to prevent spillage of infectious material from one lung to the other and for bronchospirometry[2, 3]. Early on one lung anesthesia was performed using modifications of single lumen tubes and bronchial blockers. Carlens introduced double lumen tube[2] in 1949, which is the precursor of our current polyvinyl chloride double lumen endobronchial tube (DLT). Arndt endobronchial blockers are used in pediatric patients and Univent tubes (single lumen tube with built in bronchial blocker) are meant for adults. DLT continues to remain as the preferred technique for most anesthesiologists. Many of our patients undergo OLV without any derangements in oxygenation and ventilation. However significant hypoxemia can occur with a reported incidence as high as 40%-50% in some series[4].

Objectives of this review
- Indications for OLV
- Common techniques of lung isolation
- Physiological changes (ventilation & perfusion) in lateral decubitus position and OLV
- Factors affecting ventilation and perfusion during OLV
- Anesthetic drugs and ventilatory strategies and their effects on OLV
- Management of hypoxia associated with OLV

How it is useful
1. Protection of healthy lung from infected or bleeding one
2. Diversion of ventilation from damaged airway or lung
3. Improved exposure of surgical field

How it is detrimental
1. More manipulation of airway so more damage
2. Significant physiological change and possible development of hypoxia

INDICATIONS
- ❑ **Absolute**
  - Isolation of one lung from the other to avoid spillage or contamination
    - o Infection
    - o Massive hemorrhage
  - Control of distribution of ventilation
    - o Bronchopleural fistula
    - o Bronchopleural cutaneous fistula
    - o Giant unilateral bullae or cyst
    - o Tracheobronchial disruption
    - o Surgical opening of a major conducting airway
    - o Life threatening hypoxemia due to unilateral lung disease
  - Unilateral broncho pulmonary lavage
- ❑ **Relative**
  - Surgical exposure (high priority)
    - o Thoracic aortic aneurysm
    - o Mediastinal exposure
    - o Thoracoscopy
  - Surgical exposure (low priority)
    - o Esophageal surgery
    - o Thoracic spine procedure
    - o Minimally invasive cardiac surgery

COMMON TECHNIQUES OF LUNG ISOLATION
OLV can be provided by various airway techniques. These are

- Double lumen tubes
- Univent tubes
- Bronchial blockers
- Single lumen tubes

Double lumen tubes

Common size DLTs for adults include 32, 35, 37, 39, and 41 French and they are available from Portex, Mallinckrodt, Rusch and Sheridan. For pediatric age group, sizes 26 and 28 F are now available from some manufacturers. The 26 F size from Rusch is appropriate for children as young as 8 years. DLTs are in essence 2 single tubes bonded together allowing each tube to ventilate a specified lung.

Advantages of DLT (Over single lumen tubes)

- Independent isolation and ventilation of selected lung
- Independent lung suctioning is possible
- Allows CPAP for the operated lung

- Allows rapid conversion from two lung to OLV and vice versa

Univent tubes

The Univent tube, an alternative device for providing OLV was first introduced in 1982, and underwent design modifications in 2001. It is silastic single lumen tube with a built-in internal channel housing a 2-mm diameter movable integrated blocker. The blocker has a small lumen along the entire length to facilitate lung deflation and limited suctioning. Univent tubes are available for both adults and pediatric patients (size 3.5 to 9)

Advantages of Univent tube over DLT and other blockers

- Less traumatic than DLT
- Easier to insert and properly position
- Can be repositioned during continuous ventilation and in the lateral decubitus position
- Selective capability to collapse either a single lobe or entire lung
- Useful in the presence of difficult airway
- Can be used in tracheostomized patients for OLV
- Useful rapid lung isolation in the presence of hemoptysis
- No need to change the tube if post-operative ventilation is planned

Bronchial blockers

The blockers that are independent of the single lumen tube most often used are the Fogarty occlusion catheters and the Arndt blocker. The Fogarty catheters come with a rigid wire stylet in place. This allows the creation of a hockey stick curve at the end of the catheter, which allows better directional control of the blocker. Once the blocker is positioned under FOB guidance, the stylet is removed. The main disadvantages of Fogarty catheter are inability to suction or insufflate distal to the occluded bronchial lumen and utilization of high pressure low-volume cuff.

The Arndt endobronchial blocker or wire-guided endobronchial blocker is a fairly recent addition and this mimimizes some of the difficulties associated with Fogarty catheters. The balloon of the blocker is less likely to dislodge than that of a Fogarty because of its elliptical shape and high-volume, low-pressure cuff and its 1.4 mm lumen can be used to insufflate oxygen or suction gas from the blocked lung. It can be used in pediatric patients also (upto a size of 4.5 mm ETT)

Single lumen tubes

Deliberate endobronchial intubation with a SLT may be useful in a bleeding lung. Intubation of the right main bronchus is easier than the left. FOB may be used to intubate the left main bronchus.

PHYSIOLOGY OF LATERAL DECUBITUS POSITION & OLV

Ventilation

When we change the position of an anaesthetised patient from supine to lateral decubitus position significant physiological changes occur in the matching of ventilation and perfusion. In the lateral

position in an anaesthetised and paralysed patient, approximately 55% of tidal volume is delivered to the upper (non dependent) lung unlike spontaneous ventilation in the awake[5].

Figure 1. *Distribution of ventilation in lateral decubitus position*
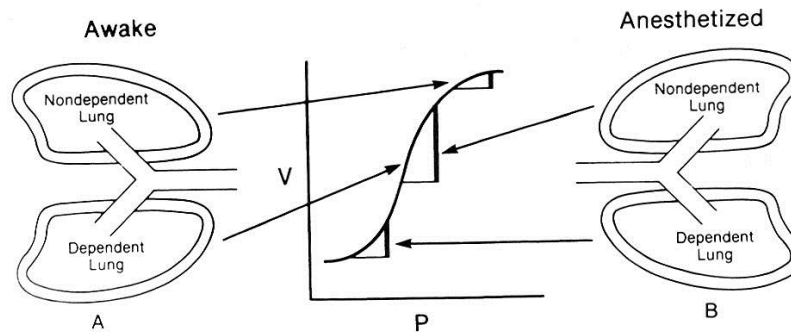
During spontaneous ventilation the dependent lung is better ventilated than the non-dependent one. On induction of general anaesthesia with neuromuscular paralysis both lungs move down on the pressure volume curve (fig 1) [6]. It means that the dependent lung moves from an initial, favourable position on the steep portion of the curve (more compliance) to a position on the lower flat portion of the curve (less compliance). There is reduction in the functional residual capacity and compliance of the dependent lung. The reasons for these phenomena are compression (with loss of FRC) of the dependent lung and restriction of its excursion (decrease in compliance) by the mediastinum, the cephalic movement of the abdominal organs via the flaccid diaphragm, and the exaggerated flexed position with chest rolls to free the axillary contents. The net result is that the non-dependent lung is better ventilated and its FRC may be approximately 1.5 times that of the dependent lung[5]. This leads to ventilation perfusion (V/Q) mismatching, as the dependent lung is better perfused and under ventilated and the non-dependent lung is under perfused and better ventilated. Gravity has no significant effect on distribution of ventilation during IPPV in the lateral decubitus position[7].

Open chest

When the non-dependent hemithorax is opened, there is further increase in FRC and compliance of the non-dependent lung and a decrease of these parameters of the dependent lung with two-lung ventilation[6]. This causes further deterioration of ventilation perfusion mismatch. When the non-dependent lung is collapsed, the blood flow to that lung is not oxygenated leading to increased P (A-a) O2 gradient and impaired oxygenation.

Pulmonary blood flow

The principal determinant of pulmonary blood flow in lateral position is gravity, which means that the dependent lung is better perfused than the non-dependent one. Thoracic operations are usually performed in the lateral position with selective ventilation of the dependent lung. The collapsed non-dependent lung continues to be perfused. This is wasted perfusion or shunt and this maldistribution of

pulmonary blood flow is the most common cause of impaired oxygenation of arterial blood. There are three major determinants of distribution of pulmonary blood flow. They are

- *Hypoxic pulmonary vasoconstriction (HPV)*
- *Gravity*
- *Non gravitational factors*

Hypoxic pulmonary vasoconstriction

Regional hypoxia in the lung causes arteriolar constriction, with diversion of the blood flow away from hypoxic segments (e.g.: atelectatic lung) to normal areas of the lung. The matching of perfusion to the ventilation is important for adequate oxygenation and removal of CO_2. This intrinsic mechanism of lung to maintain V/Q matching was first described in 1946 and has been termed hypoxic pulmonary vasoconstriction (HPV)[8].

It is the small arterioles (less than 500 μm), which undergoes constriction during HPV. Capillaries and venules contribute to a lesser extent. This response is unique to the pulmonary system. The stimulus for HPV is a combination of both the alveolar partial pressure of oxygen and the mixed venous PO_2 (PvO_2)[9]. Between the two, alveolar PO_2 is the predominant one. HPV results in the diversion of blood flow away from hypoxic or atelectatic alveoli to those that are better ventilated thus V/Q is maximised.

Figure 2. *Effect of HPV on PaO_2*

The effectiveness of the HPV response depends on the size of the atelectatic lung. It is most effective when the size (atelectatic lung) is between 20% to 80% of the lung (fig 2)[10]. During one lung ventilation the percentage of lung that is atelectatic is usually between 30% to 70% and this falls within the range of effective HPV[10]. The expected shunt comes down by 50% during OLV because of effective HPV[11]. Many factors modify HPV response. These are pulmonary disease, pharmacological agents, acid base status (respiratory or metabolic alkalosis blunts HPV; metabolic acidosis enhances it), surgical manipulation, hemodynamic changes and sepsis.
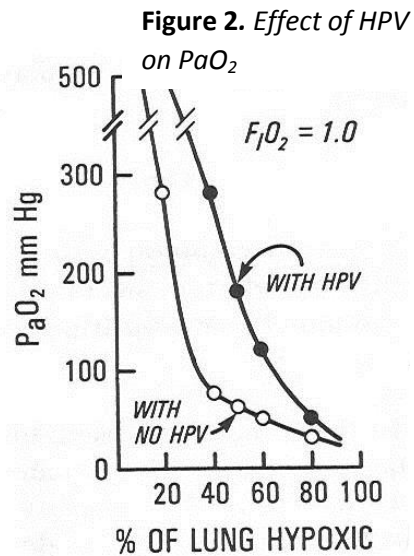
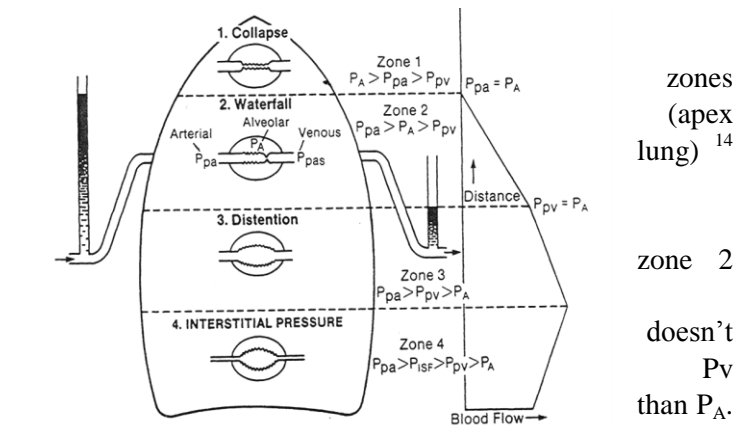**Figure 3.** *Relationship between cardiac output &PaO_2 for different shunts*
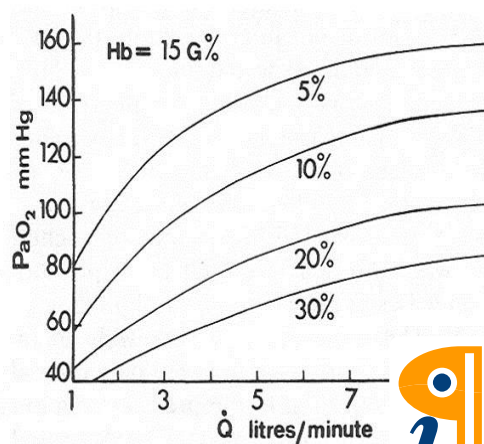
Changes in cardiac output (both decrease and increase) attenuate HPV. Increase in cardiac output results in increase in pulmonary artery pressure and mixed venous PO_2 that reduces the effectiveness of HPV. In the low output states (hemorrhage), the alveolar vessels in the ventilated lung have high pulmonary vascular resistance and are

compressed leading to attenuation of HPV[12]. Low cardiac output states also can lead to low PO_2 in the presence of physiological shunts greater than 5%[13] (as in lung collapse, or atelectasis etc) (fig 3). Over distension of ventilated lung as occurs in excessive tidal volumes and high PEEP, can divert blood flow to hypoxic alveoli (non dependent lung) attenuating HPV resulting in worsening of V/Q mismatch.

Figure 4. *Distribution of blood flow in upright lung*

Gravity

The principal determinant of pulmonary blood flow is gravity dependent and this results in the distinct zones of West with least blood flow in zone 1 (apex of lung)[14] and best flow in zone 3 (base of lung) (Fig 4). Airway pressure (P_A) exceeds pulmonary arterial pressure (Pa) in zone 1 resulting in no blood flow, whereas in zone 2 blood flow is determined by the difference between Pa and P_A. Airway pressure doesn't affect blood flow in zone 3 as both Pa and Pv (pulmonary venous pressure) is higher than P_A. There exists a zone 4 in the most dependent portion of the lung where blood flow is less than zone 3. The cause of decreased flow in this zone is because of increased interstitial pressure and increased resistance of the extra-alveolar vessels. There is a progressive increase in airway pressure in the non dependent atelectatic lung resulting in decreasing perfusion in that lung. The gravity dependent increase in blood flow is seen in all positions (supine, sitting, lateral) and it is not affected by anesthesia and paralysis but the vertical gradient in the lateral decubitus position is less than in upright position[15]. In the lateral position the non-dependent lung has less zone 1 and the rest comes under zone 2 whereas the dependent lung has zone 2 & 3 areas.

Non gravitational factors

Some studies had shown that there is a central to peripheral gradient of pulmonary blood flow in supine, awake human volunteers[16]. It means that pulmonary blood flow follows a concentric pattern with maximum flow in the center and least in the periphery even though there is some tendency of greater blood flow in the lower half of the lung compared to the upper half. This study was done using SPECT technology and questioned the conventional gravity dependent pulmonary blood flow pattern. The relative importance of gravity and non-gravity factors in humans is difficult to assess. Overall there is a greater amount of blood flow to the dependent lung in the lateral decubitus position during two lung mechanical ventilation.

Lung volume and cardiac output changes can change the distribution of pulmonary blood flow significantly. The mechanism of action is either direct or through the determinants of pulmonary blood flow described above. The classical teaching is that increase in cardiac output attenuate HPV and distributes pulmonary blood more evenly. However studies using SPECT techniques had demonstrated that an increase in cardiac output results in the maintenance of flow in a central to peripheral gradient, without an increase in attenuation of the gradient[17].

Distribution of blood flow is also affected by changes in lung volume. The pulmonary vascular resistance (PVR) is lowest at FRC. When lung volume is above FRC, the PVR is raised due to the stretch of the capillaries. At lung volumes less than FRC, PVR is again high due to reduced size of the extra alveolar vessels. The mechanism may be a loss of radial traction supporting these vessels at low lung volumes.

ONE LUNG VENTILATION (summary of physiology)

Thoracic operations are usually performed with the patient in the lateral position with selectively ventilating the dependent lung. The principle physiological change in OLV is the redistribution of pulmonary perfusion between the ventilated lung and the collapsed lung. The collapsed non-dependent lung continues to be perfused. This is wasted perfusion or shunt (responsible for arterial desaturation), which is about 20-25% of cardiac output under the best of circumstances. The factors determining it were already reviewed above; the main factors are HPV and gravity. The effect of gravity increases blood flow to the dependent lung during OLV in lateral position. Apart from gravity, the other passive mechanical mechanisms that decrease blood flow to the non-dependent lung include surgical manipulation and severity of preexisting disease in the non-dependent lung.

OLV has much less of an effect on $PaCO_2$ than on the PaO_2 because of the linear shape of the CO_2 dissociation curve. Blood flowing through poorly ventilated alveoli will retain more than normal amount of CO_2 and not take up normal amount of O_2. Blood flowing through well-ventilated alveoli gives up a greater amount of CO_2, but unable to take up higher amount of O_2. The top flat portion of the oxygen-hemoglobin dissociation curve explains this. Thus there is only a small $PACO_2$ to $PaCO_2$ gradient during OLV and main defect is inability to take up enough oxygen.

The mechanism of poor ventilation of the dependent lung is already reviewed earlier. *In order to maximize oxygenation efforts are directed towards either optimizing matching of ventilation with perfusion in the dependent lung or increasing the oxygen content of blood returning from the collapsed non-dependent lung.*

Strategies to maximize oxygenation during single lung ventilation
Operated lung
Oxygenate shunt blood

- o Continuous oxygen insufflation
- o Intermittent single breath with oxygen
- o CPAP
- o High frequency ventilation

Decrease shunt

- o Vasoconstriction (phenylephrine, noradrenaline)
- o Pulmonary artery obstruction
- o Lateral or semi lateral position

Non operated lung
Optimize ventilation

- o High FIO_2
- o Optimal tidal volume

o   Pressure controlled ventilation
o   Optimal PEEP

Improve perfusion

o   Selective vasodilatation (NO, prostacyclin)

Effect of positioning

Recently two studies had evaluated the effect of positioning on arterial PO_2. In the first study, patients were divided into three groups-supine, left semi lateral decubitus position and the left lateral position[18]. Arterial blood gas analysis was sequentially done with left OLV on an FIO_2 of 1. It was observed that life threatening hypoxemia frequently occurred in the supine group approximately 10mts after starting OLV. The left semi lateral decubitus position was as effective as the left lateral decubitus position in avoiding life threatening hypoxia during OLV.

In the second study, the authors evaluated the effects of position and FIO_2 on arterial PO_2 in COPD patients undergoing OLV[19]. Patients were randomly assigned to receive FIO_2 of 0.4, 0.6, and 1 in both supine and lateral position during two-lung ventilation and OLV. In all 3 groups PaO_2 was significantly higher during OLV in lateral compared to supine position.

These studies have shown that during OLV, it is necessary to keep the patient in the lateral position for augmenting redistribution of pulmonary blood flow to the dependent (ventilated) lung by way of gravity. This will improve V/Q matching in the ventilated lung resulting in higher PO_2.

Choice of anaesthetic agents (effect on HPV)

Many studies have shown that intravenous anesthetics (thiopentone, benzodiazepines, opioids, ketamine etc) do not alter the HPV response[10]. Recent studies have reconfirmed these findings and also have shown that propofol may actually potentiate HPV response[20]. In contrast inhalational anesthetics inhibit HPV in a dose dependent manner[21].

All inhalational anaesthetic agents produce a modest reduction of PaO_2. This is by increasing the shunt by partial inhibition of HPV[22]. This effect is mainly seen in in-vitro experiments, but the result in-vivo is variable. In a dog model of OLV, the median effective dose for inhibition of HPV was 2 times MAC for isoflurane[23]. In humans one study had shown that 1.2 MAC of isoflurane did not affect HPV in a model of one lung hypoxia[24].

An inhalational anaesthetic agent that lowers cardiac output more than it decreases oxygen consumption will also lower mixed venous PO_2. A reduction in cardiac output will lead to decreased blood flow to the collapsed lung because of high PVR already present in the lung. These actions counter the direct depression of HPV by the anaesthetic agents.

The above-mentioned action was demonstrated in pigs anesthetized with propofol and then with desflurane[25]. In this study TIVA with propofol was used to anesthetize the pigs and then it was discontinued. These pigs were then ventilated with increasing concentrations of desflurane. Mean arterial pressure, cardiac output, PvO_2 and shunt fraction all decreased in a dose dependent manner during OLV, but PaO_2 remained unchanged. The authors concluded that increasing concentrations of desflurane had hemodynamic effects that negated its direct inhibitory effect on HPV. In humans, when desflurane was compared with isoflurane in patients undergoing OLV, there was no difference in arterial PO_2[26, 27]

TIVA or Inhalational agents

Inhalational agents may depress HPV and intravenous anesthetics maintain it, so some anesthesiologists recommend TIVA for OLV[28]. In a study, fifty patients undergoing OLV were divided into two groups[29]. First group received TIVA and the second group received inhalational anesthetic agent. Blood pressure, heart rate, and PCO_2 were similar in both groups. Patients in both groups maintained adequate oxygenation but, the TIVA patients had a higher PO_2 as a group compared to those receiving inhalational anesthetic agent during OLV.

The clinical effects of intravenous or inhalational anesthetics on oxygenation through HPV are more theoretical than real for most patients. Concerns about inhalational anesthetic effects on HPV are even less important during surgery because surgical manipulation of lung releases vasoactive substances like thromboxane and prostacyclin. These agents cause local vasodilatation that blunts HPV[30]. These evidences prove that both intravenous and inhalational anesthetics can be used during OLV without much physiological consequence.

Vasodilators and inotropes

Most vasodilators inhibit HPV. These are nitroglycerine, sodium nitroprusside, calcium channel blockers, prostacyclin etc. Beta agonists like isoprenaline, salbutamol also have similar effects. Aminophylline and hydralazine have minimal effect on HPV. Inotropes like adrenaline, nor-adrenaline and dopamine at low doses have negligible effect on HPV, but will impair it at higher doses. Propranolol may augment HPV.

VENTILATION

Traditionally, the ventilatory parameters used during OLV were the same as those used during two-lung ventilation, which is about 10-12 ml/kg. The PCO_2 is kept in the normal range by adjusting the ventilatory rate. This tidal volume will recruit alveoli and prevent atelectasis of the dependent lung. Optimizing the dependent lung FRC is important and tidal volumes less than 8 ml/kg will result in decrease in FRC. Use of large tidal volume (14ml/kg) is effective in improving PaO_2 to some extent[31]. Tidal volumes in excess of 15ml/kg will over-distend the alveoli and increase PVR in the ventilated lung leading to an increase in shunt to the non-ventilated lung.

There is some concern about large tidal volume during OLV causing barotrauma to the lungs. Pressure controlled ventilation instead of volume controlled appeared to be better alternative for OLV with lower peak and plateau pressure and decreased pulmonary shunt with higher PaO_2[32].

High frequency ventilation (HFV) is suggested as an alternative to volume controlled ventilation (VCV) [33]. The study concluded that there is no difference in SaO_2 or end tidal CO_2 between HFV group and VCV group during OLV.

Apnea during OLV can have disastrous consequences, as desaturation is rapid compared to two-lung ventilation. During OLV the mean time for SaO_2 to fall to 95% from 100% is 3.2 minutes compared to 6.3 minutes with two-lung ventilation[34].

OPTIMIZING OXYGENATION

The major reason for arterial desaturation during OLV is because of the large alveolar to arterial oxygen tension gradient (due to continued perfusion of the non dependent lung). Still *other miscellaneous factors* contribute to hypoxemia during OLV. They are

1. Low FIO_2
2. Gross hypoventilation of dependent lung
3. Malposition of DLT
4. Malfunction of the dependent lung airway lumen (blockage by secretions, bronchial cuff herniation)

Accurate placement of DLT is paramount in OLV to prevent hypoxia. *If there is increased peak inspiratory pressure or decreased ventilation of hemithorax on initiation of OLV, the cause may be in the lung (obstructive or restrictive lung disease), DLT (malposition or obstructed lumen) or the double connector (which connects anesthesia circuit to DLT)* [35]. We have developed *a simple and practical algorithm* for rapid identification of the cause of high airway pressures / inadequate ventilation with a DLT (fig 5)[36]

Figure 5. A *simple and practical algorithm for rapid identification of high airway pressure with DLT.* ▲ = *diagnosis; DLT = double lumen tube; FOB = fiberoptic bronchoscopy; PIP = peak inspiratory pressure*
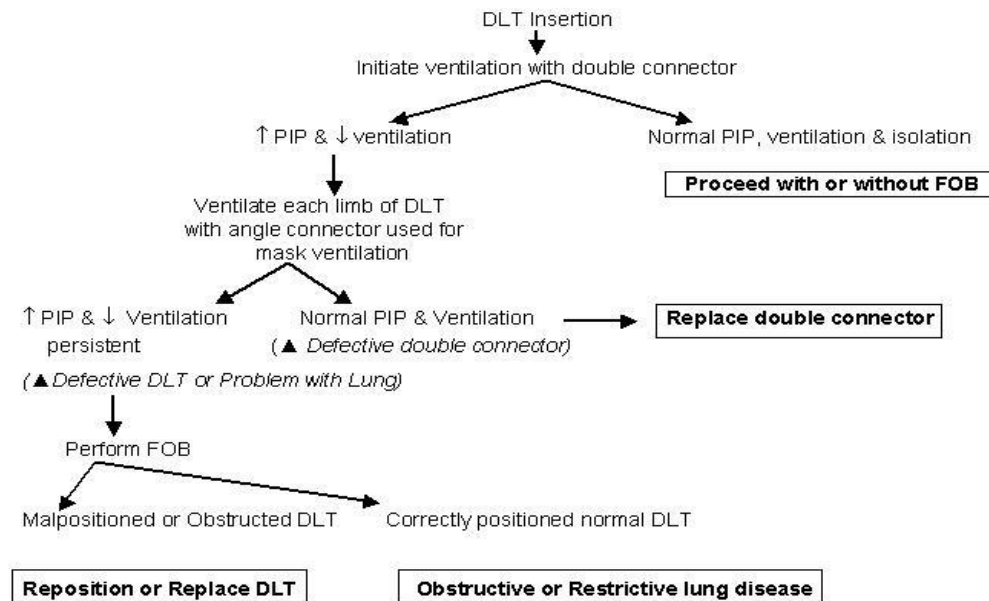
When hypoxemia occurs during surgery the placement of DLT should be reassessed preferably with a fiberoptic bronchoscope. Mechanical problems like tube malposition, blockage or bronchospasm should be ruled out again. It can also result from decreased perfusion of ventilated lung from causes like hypotension, hemorrhage or arrhythmias.

PEEP

There is reduction of FRC in the dependent lung in lateral position as mentioned earlier. The application of PEEP to the dependent lung is intended to restore FRC, recruit collapsed alveoli, and

improve oxygenation. All patients do not show improvement with PEEP. Patients' preexisting lung condition determines their response[37]. Dynamic pulmonary hyperinflation occurs when expiratory flow does not cease and the lung does not expire to FRC before the next inspiration. This leads to the development of positive pressure at the end of expiration. This is called auto-PEEP or intrinsic PEEP. This is commonly seen during mechanical ventilation of COPD patients. Recent studies have demonstrated that auto PEEP may be present in the majority of patients undergoing OLV[37]. In patients who have low dependent lung volumes, the application of PEEP to the ventilated lung has been shown to be beneficial[38].

When extrinsic PEEP of 5 is applied in the presence of intrinsic PEEP, most often the total PEEP increases by 1 to 2 cm of H_2O. In patients with significant auto-PEEP, high extrinsic PEEP may not improve oxygenation or may even decrease oxygenation. In these patients application of PEEP increases the alveolar pressure-raising lung PVR, which in turn diverts blood flow to the non-ventilated lung increasing hypoxia. Although intrinsic PEEP may improve oxygenation, some studies have failed to show a correlation between intrinsic PEEP and PaO_2 during OLV[39]. High levels of intrinsic PEEP may reduce cardiac output by over distension of lung. Therapies that may reduce it include altering the ventilator settings (decrease tidal volume, respiratory rate, and I: E ratio), treating bronchospasm, clearing secretions or applying extrinsic PEEP in some cases.

Other maneuvers

If hypoxemia persists, the collapsed lung can be partly or fully re-expanded after informing the surgeon. This maneuver can be repeated every 5 to 10 minutes. Oxygen insufflations to the operated lung are usually unsuccessful as it fails to reach and recruit collapsed alveoli. Early clamping of pulmonary artery during pneumonectomy completely eliminates shunt. This cannot be recommended for lobectomy as reperfusion after interruption can produce lung injury.

CPAP

Less severe cases of hypoxemia can be managed by selective CPAP with 100% oxygen to the operated lung. CPAP maintains the patency of the alveoli with oxygen, so the blood to the operated lung becomes oxygenated. Another useful effect is that it increases the airway pressure in that lung, increasing the PVR that will divert shunt blood to the ventilated lung[10]. Applying CPAP of 10 cm H_2O to non-ventilated lung with zero PEEP to the ventilated lung, was the most effective way of improving oxygenation and decreasing shunt flow through collapsed lung during OLV according to one study[40].

The combination of PEEP to the dependent lung and CPAP to the non-dependent lung has been described[41]. This is effective in improving oxygenation and decreasing shunt, but it reduces cardiac output mildly.

Pharmacological management

Pulmonary vasoconstrictors

Pulmonary vasoconstrictors have been tried to decrease the shunt to the non-ventilated lung. Prostaglandin F2α a potent pulmonary vasoconstrictor was directly infused into the pulmonary artery of non-ventilated lungs of dogs[42]. It leads to significant decrease in the shunt and increase in PaO_2. Phenylephrine was also used as non-specific vasoconstrictor to improve oxygenation[43]. To date these drugs have not been very useful in human beings.

Nitric oxide

There is considerable interest in using NO to improve blood flow to the dependent lung. Inhaled NO at 20 and 40 ppm did not improve oxygenation in patients with normal PVR in two studies[44, 45] For patients with raised PVR, inhaled NO in combination with other vasoactive agents like aerosolized prostacyclin can improve oxygenation and decrease shunt[46]. Currently routine use of NO is not recommended for OLV.

SUMMARY

The following strategy summarizes actions for achieving satisfactory oxygenation during OLV.

1. Confirm proper positioning of DLT in the supine and then in the lateral position and its patency preferably with fiberoptic bronchoscope.
2. Maintain two-lung ventilation till the pleura is opened.
3. Initial setting of OLV (volume controlled ventilation)
   - $FIO_2 = 1$
   - TV = 8-10 ml/kg and adjust according to airway pressure
   - RR to initially target $PaCO_2$ of 40 mm Hg, then adjust according to presence of dynamic hyperinflation.
   - Consider pressure controlled ventilation in cases with high peak airway pressures
4. If hypoxemia develops
   - Reconfirm DLT position and patency with fiberoptic bronchoscope.
   - Non-dependent lung CPAP of 5-10 cm or H_2O
   - Dependent lung PEEP of 5-10 cm or H_2O if CPAP fails
   - Consider intermittent inflation of non-dependent lung
   - Consider high-frequency ventilation of non-dependent lung
   - Consider NO if hypoxemia co-exists with pulmonary hypertension.
   - Consider early clamping of pulmonary artery of operative lung during pneumonectomy

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