EFFECTS OF PROPOFOL TOTAL INTRAVENOUS ANESTHESIA VERSUS SEVOFLURANE INHALATIONAL ANESTHESIA ON PULMONARY SHUNT FRACTION AND HAEMODYNAMICS DURING ONE LUNG VENTILATION IN THORACIC SURGERY.

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ABSTRACT
One-lung ventilation (OLV) is required for several thoracic operations. The effects of anesthesia and one-lung ventilation on arterial oxygenation are complex and not fully understood; adequate arterial oxygen is not achieved in some patients despite an accurately placed endobronchial tube and high inspired oxygen. The present study compared the effect of both total intravenous anesthesia using propofol infusion and inhalational anesthesia using sevoflurane on pulmonary shunt fraction and haemodynamic variables in patients requiring OLV during thoracic surgery. Twenty patients were studied, aged 20-65 years ASA physical status, II, and III; they were scheduled for lobectomy with OLV, and were randomly assigned into two groups. Group I: Patients were anesthetized using I.V. anesthesia using propofol. Group II: Patients were anesthetized with inhalation anesthesia using sevoflurane. Blood gas values (PaO2, PaCO2, SpaO2), mixed venous blood samples and haemodynamic data (HR, MAP, CVP, PAP and PCWP) were obtained. Four sets of measurements were taken: after 15 min. of stable total lung ventilation(TLV), after 15 min. of stable OLV in supine position (OLVsf), after 15 min. of stable OLV in lateral position(OLVl) and after opening thorax(OLVo). Shunt fraction (Qs/Qt) was calculated using standard formula Qs/Qt = (CcO2-CaO2)/(CcO2-CvO2). Also, CI, SVRI, and PVRI were calculated. There was significant increase in PaCO2 values in both groups but no significant difference was found between the two groups. Both groups showed significant decrease in PvO2 throughout the entire four-step sequences and no significance was found between the two groups. Average shunt during OLVs increased by 11.02% and 5.6% from TLV and during OLVl by 14% and 10.29% for propofol and sevoflurane respectively; this increase showed statistically significant value in both groups. Institution of OLVs was associated with a significant decrease in CI in each group and there was no significance between groups; turning the patient to lateral position together with opening chest was associated with significant increase in CI and these results did not show any significance between groups. Significant reduction in SVRI was observed after initiation of OLVs in propofol group while SVRI increased in sevoflurane group; these changes were statistically insignificant between the two groups. Turning the patient to lateral position together with opening the chest were associated with significant increase in SVRI in each group, but it was significantly higher in the propofol group. PVRI decreased after turning the patient to lateral position in propofol group while PVRI increased in sevoflurane group. In conclusion: both sevoflurane and propofol administration causes an increase in shunt fraction. PvO2, PaO2, PaCO2, CI did not differ between both groups. Both sevoflurane and propofol are considered as good drugs for thoracic operations but sevoflurane appears to give more cardiovascular stability.

Key words: One lung ventilation, pulmonary shunt fraction, anesthetics

INTRODUCTION
One lung ventilation (OLV) is required for several thoracic operations; adequate arterial oxygenation is not achieved in some patients despite an accurately placed endobronchial tube, due to, in part, to inhibition of hypoxic pulmonary vasoconstriction (HPV). HPV is an important mechanism by which blood is diverted from the hypoxic regions of the lung to a better-ventilated region, thereby reducing the venous admixture and minimizing the decrease in arterial oxygenation.

Intravenous anesthetic agents including propofol have not been shown to inhibit hypoxic pulmonary vasoconstriction. This might encourage the use of propofol in thoracic surgery where one lung ventilation is required.

Sevoflurane has useful effects during thoracic surgery. It is a potent bronchodilator and its low blood-gas partition coefficient allows rapid adjustment of the depth of anesthesia. Rapid emergence from anesthesia allows rapid return of spontaneous respiration and avoids the risk of postoperative mechanical ventilation.
This study was designed to compare the effects of both total intravenous anesthesia using propofol infusion and inhalational anesthesia using sevoflurane on pulmonary shunt fraction and haemodynamic variables in patients requiring one lung ventilation during thoracic surgery.

**PATIENTS AND METHODS**

This study was carried out in Menofiya University hospital between 2003-2005 after written informed consent one day before surgery. Twenty patients scheduled for one-lung ventilation (OLV) for thoracic surgery (lobectomy) were randomly enrolled in this study.

**Exclusion criteria:** Patients with fever, hypothermia, history of unusual response to anesthetics, History of chronic exposure to drugs known to affect anesthetic requirements. End-stage obstructive or restrictive pulmonary disease. Renal, hepatic, and endocrinal insufficiency. Ischemic or valvular heart disease.

**Preoperative criteria of pulmonary functions and blood gases values:**

- arterial O₂ tension (PaO₂) > 50 mmHg on room air,
- arterial carbon dioxide tension (PaCO₂) < 45 mmHg on room air,
- forced expiratory volume in one second (FEV₁) > 2L or 40% of the predicted value,
- forced expiratory volume in one second / forced vital capacity (FEV₁/FVC) > 50% of the predicted value,
- maximum breathing capacity > 50% of the predicted value.

Patients were randomly allocated into 2 groups; group I were assigned to receive total intravenous anesthesia (TIVA) with propofol and group II assigned to receive inhalation anesthesia with sevoflurane; patients were premedicated with midazolam 0.03 mg/kg IM in the ward half an hour before surgery. Routine monitors were connected and baseline measurements were recorded and repeated every 10 min till phase I recovery.

**Routine monitors** included ECG, EtCO₂, SpO₂, NIBP, nasopharyngeal temperature, neuromuscular monitoring in the form of train of four stimulation (TOF), urine output and peak airway pressure.

After pre oxygenation, fentanyl 0.2 mg I.V was given and anesthesia was induced with propofol (2-3mg/kg) I.V and maintained with continuous infusion (6-8 mg/kg/h.) in group I. This was supplemented with sevo-flurane 1.5-2% in 100% O₂ in group II.

After induction in both groups, patients were manually ventilated via tight fitted face mask with 100% oxygen. Neuromuscular block was achieved with vecuronium 0.1mg/kg intravenously. Once a complete muscle paralysis was achieved, a proper sized right or left double lumen tube was inserted. The correct position of the tube was initially confirmed by auscultation and then by direct visualization of atelectatic, non ventilated lung after thoracotomy. Tidal volume (Tv) and respiratory rate (RR) were adjusted to maintain acid base status and PaCO₂ within physiological limits. During two-lung ventilation (TLV) and one-lung ventilation (OLV), Tv of 10 ml/kg was used, RR was adjusted to maintain the EtCO₂ of 35-40 mmHg. During OLV, the lumen of non-ventilated lung remained open to atmosphere. Tv was reduced if peak airway pressure exceeded 35 cm of water. Increments of vecuronium were given to maintain suppression of the second twitch using train of four stimulation.

After induction of anesthesia, pulmonary artery floatation catheter (Arrow International, USA) was introduced via ipsilateral internal jugular vein using seldinger's technique and was zeroed at the level of the left atrium. Arterial and mixed venous blood were analyzed by using automated blood gas analyzer. Cardiac output (CO) was measured using thermodilution technique by forcibly injecting 10 ml of 5% dextrose at room temperature through the proximal port of the pulmonary catheter that was connected to cardiac output monitor. Arterial hypoxemia (SaO₂ < 90%) during OLV was initially treated using CPAP ventilation to the nondependent lung, followed by either PEEP to the dependent lung or restoration of TLV.

**Sampling and measurements:**

Four steps of measurements were taken. Step 1: (TLV) 15 min after induction and stable two-lung ventilation (base line). Step 2: (OLV₁) After 15 min of stable OLV in supine position. Step 3: (OLV₂) After 15 min of stable OLV in lateral position. Step 4: (OLV₀) After opening thorax. In the previous four steps, the following parameters were
taken: Heart rate (HR), Mean arterial pressure (MAP), Central venous pressure (CVP), Pulmonary capillary wedge pressure (PCWP), Partial pressure of oxygen in arterial and mixed venous blood (PaO₂, PvO₂), Arterial partial pressure of carbon dioxide (PaCO₂).

**Calculated parameters:**
- Cardiac index (Cl) L/min/m² = CO/Body surface area (BSA)
- Systemic vascular resistance index (SVRI) in dynes. sec/cm⁵m²
  \[ SVRI = \frac{MAP - CVP \times 80}{Cl} \]
- Pulmonary vascular resistance index (PVRI) in dynes. sec/cm⁵m²
  \[ PVRI = \frac{MPAP - PCWP \times 80}{Cl} \]

Where: MAP = mean arterial pressure. CVP = central venous pressure.

MPAP = mean pulmonary artery pressure
PCWP = pulmonary capillary wedge pressure.

Calculation of shunt fraction: The shunt fraction was calculated using a standard formula proposed by Riely and his colleagues[7]:
\[ \frac{Q_s}{Q_t} = \frac{(CcO_2 - CaO_2)}{(CcO_2 - CvO_2)} \]

Where:- Qs = shunt flow, Qt = cardiac output and CcO₂, CaO₂ and CvO₂ represent the oxygen content of pulmonary end capillary, arterial and mixed venous blood respectively. Arterial and mixed venous oxygen content were calculated according to the following formula.
\[ CcO_2 = PO_2 \times 0.0031 + (Hb \times 1.34 \times SPO_2/100) \]

Where: PO₂ and SPO₂ represent the partial pressure (mmHg) and oxyhemoglobin saturation in arterial (CaO₂) or mixed venous (CvO₂) blood. PaO₂ and PvO₂ were directly measured.

In case of CcO₂, the relevant partial pressure and saturation have to be derived from alveolar oxygen tension (PAO₂) equation[7].
\[ PAO_2 = F_iO_2 \times (PB - PH_2O) - (PaCO_2/R) \]

Where: FiO₂ is the fractional inspired oxygen concentration, PB is barometric pressure (760 mmHg), PH₂O is the saturated vapor pressure of water (47 mmHg) at body temperature, PaCO₂ is arterial carbon dioxide partial pressure and R is the respiratory quotient (assumed to be 0.8).

\[ 0.003 = \text{oxygen solubility coefficient (O}_2\text{ml/mmHg) } \]
\[ 1.34 = \text{oxygen carrying capacity of Hb (ml O}_2\text{/gm Hb).} \]

So, CcO₂ = PaO₂ × 0.0031 + (Hb X 1.34 SPO₂/100)

PA CO₂ tension was assumed to be equal to PaCO₂.

Data were collected and statistically analyzed using SPSS version 1.00. Descriptive statistics (eg. Number, percent, Mean, standard deviation) were used. Analytic statistics (eg, student's test, paired t test and chi square) were applied. P < 0.05 was used to determine significance.

**DISCUSSION**

One-lung ventilation (OLV) is required for several thoracic operations. The effects of anesthesia and OLV on arterial oxygenation are complex and not fully understood; adequate arterial oxygen is not achieved in some patients despite an accurately placed endobronchial tube and high inspired oxygen[8]. Whether propofol and sevoflurane impair arterial oxygenation during OLV is controversial. The purpose of the present study was to determine whether there was a difference between sevoflurane and propofol as regards to oxygenation, shunt fraction and haemodynamics during OLV. There was no difference between groups in patient’s characteristics or preoperative data.

After induction of general anesthesia and endobronchial intubation, OLV was instituted and was associated with a decrease in HR in propofol group; this

<table>
<thead>
<tr>
<th>Table (1): Comparison between both groups as regards age, demographic data and preoperative investigations.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients</td>
</tr>
<tr>
<td>----------</td>
</tr>
<tr>
<td>Group I</td>
</tr>
<tr>
<td>Group II</td>
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<tr>
<td>P value</td>
</tr>
</tbody>
</table>

Data presented as mean and SD
Table (2): Comparison between the two groups regarding blood gases.

<table>
<thead>
<tr>
<th></th>
<th>TLV</th>
<th>OLV₆</th>
<th>OLV₄</th>
<th>OLV₀</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PaO₂ (mmHg)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Propofol</td>
<td>414.6 ± 49.22</td>
<td>394.8 ± 60.72 *</td>
<td>257.86±51.1 *</td>
<td>231.9±41.38*#</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>415.2 ±50.13</td>
<td>376.4 ±51.34 *</td>
<td>328.8 ±53.93 *</td>
<td>291.7±59.11* #</td>
</tr>
<tr>
<td>P</td>
<td>0.97</td>
<td>0.06</td>
<td>0.07</td>
<td>0.07</td>
</tr>
<tr>
<td><strong>PaCo₂ (mmHg)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Propofol</td>
<td>39.5 ± 4.21</td>
<td>42.27 ±4.5   *</td>
<td>46.7 ± 4.79 *</td>
<td>51.97±4.8 *#</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>37.63 ±4.49</td>
<td>40.76 ±6.77 *</td>
<td>43.42 ±5.27 *</td>
<td>47.74±5.48 *#</td>
</tr>
<tr>
<td>P</td>
<td>0.35</td>
<td>0.19</td>
<td>0.16</td>
<td>0.08</td>
</tr>
<tr>
<td><strong>PvO₂ (mmHg)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Propofol</td>
<td>43.98 ± 2.26</td>
<td>40.6 ± 1.39 *</td>
<td>38.6 ± 1.65 *</td>
<td>37.2 ±1.49 *#</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>43.58 ±1.37</td>
<td>41.4 ±2.06 *</td>
<td>38.7 ±1.34 *</td>
<td>37.9 ±1.1 *#</td>
</tr>
<tr>
<td>P</td>
<td>0.63</td>
<td>0.32</td>
<td>0.9</td>
<td>0.81</td>
</tr>
<tr>
<td><strong>Qs/Qt</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Propofol</td>
<td>16.28 ± 4.0</td>
<td>27.3 ± 3.86 *</td>
<td>30.28 ± 3.6 *</td>
<td>31.85 ±3.89 *</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>15.98 ±3.97</td>
<td>21.58 ±4.22 *</td>
<td>26.27 ±4.21 *</td>
<td>27.57±3.62 *#</td>
</tr>
<tr>
<td>P</td>
<td>0.86</td>
<td>0.00</td>
<td>$</td>
<td>0.02</td>
</tr>
</tbody>
</table>

*significant in relation to TLV  # significant in relation to OLV₄
$significant difference between the two groups.
PaO₂=Arterial oxygen tension. PaCo₂= Arterial carbon dioxide tension. PvO₂= Venous oxygen tension.
Qs/Qt=Shunt fraction.

decrease was insignificant but after turning the patient to the lateral position more decrease was reported and this decrease was statistically significant and these results were in agreement with that of Abe et al. (9) and Beck et al., (10) who found the same observation. It has been suggested that propofol either resets or inhibits the baroreflex thus reducing the tachycardia response to hypotension (11).

On the other hand, our results were different from those obtained by Spies et al., (12) who compared the use of propofol/fentanyl technique with enflurane before and after OLV. In their study, HR increased with much more increase in enflurane group.

Stephan et al., (13) found that HR might increase, decrease or remain unchanged when anesthesia was maintained with propofol. In the present study, HR in the sevoflurane group has significantly increased throughout the four steps study sequence and this was in agreement with Wang et al., (8) who studied the effects of sevoflurane and isoflurane on arterial oxygenation during OLV and found that, there was no difference in HR or MAP between sevoflurane and isoflurane and both the studied drugs increased HR. Abe et al., (14) Beck et al., (10) and Chow et al., (15) obtained the same results.

Patients in the propofol group had a decrease in MAP during OLV₆ and that decrease was statistically significant compared to TLV and this was possibly due to vasodilatation of the peripheral vasculature, decrease in myocardial contractility, and/or resetting of the baroreflex activity (16). And this also could explain the decrease in SVRI after initiation of OLV in our study.

MAP increased after turning the patient to the lateral position (OLV₄) and after opening the chest and this increase was statistically significant, SVRI significantly increased also in both steps and this was in agreement with Beck et al., (10). Also cardiac indices had increased significantly and were associated with an increase in pulmonary perfusion which could increase Qs/Qt. Similar findings were reported in the study done by Domino et al., (17). This increase in MAP might be due to position and hypercarbia as well; this increase was accompanied by an increase in CVP together with SVRI.

In the sevoflurane group, there was statistically significant increase in MAP throughout the four sampling times and this was in agreement with the study done by Beck et al., (10) who found that sevoflurane during one-lung ventilation (OLV₆-OLV₄-OLV₀) caused a significant increase in MAP and this might be attributed to
Table (3): Comparison between the two groups regarding haemodynamic data.

<table>
<thead>
<tr>
<th></th>
<th>TLV</th>
<th>OLVs</th>
<th>OLVS</th>
<th>OLVO</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR</td>
<td>76.2 ± 19</td>
<td>75.1 ± 17.27</td>
<td>73 ± 16.06 *</td>
<td>72.5 ± 15.84 *</td>
</tr>
<tr>
<td></td>
<td>0.46</td>
<td>0.82</td>
<td>0.71</td>
<td>0.43</td>
</tr>
<tr>
<td>MAP</td>
<td>87.4 ± 8.83</td>
<td>80.1 ± 8.94*</td>
<td>102.7 ± 10.88*</td>
<td>104 ± 8.24 *</td>
</tr>
<tr>
<td></td>
<td>0.06</td>
<td>0.8</td>
<td>0.01 $</td>
<td>0.02 $</td>
</tr>
<tr>
<td>CI</td>
<td>2.64 ± 0.32</td>
<td>2.44 ± 0.28 *</td>
<td>2.9 ± 0.26 *</td>
<td>2.68 ± 0.27 *</td>
</tr>
<tr>
<td></td>
<td>0.76</td>
<td>0.22</td>
<td>0.55</td>
<td>0.36</td>
</tr>
<tr>
<td>CVP</td>
<td>11 ± 1.94</td>
<td>11 ± 1.94</td>
<td>13 ± 1.41 *</td>
<td>12 ± 1.41</td>
</tr>
<tr>
<td></td>
<td>0.20</td>
<td>0.16</td>
<td>.012</td>
<td>0.13</td>
</tr>
<tr>
<td>PCWP</td>
<td>12 ± 1.41</td>
<td>13 ± 1.41 *</td>
<td>14 ± 1.33*</td>
<td>15 ± 1.05*#</td>
</tr>
<tr>
<td></td>
<td>0.7</td>
<td>0.14</td>
<td>0.01 $</td>
<td>0.85</td>
</tr>
<tr>
<td>PAP</td>
<td>22.4 ± 3.97</td>
<td>23.2 ± 3.76*</td>
<td>26 ± 3.29*</td>
<td>26.7 ± 2.88*#</td>
</tr>
<tr>
<td></td>
<td>0.16</td>
<td>0.18</td>
<td>0.6</td>
<td>0.82</td>
</tr>
<tr>
<td>SVRI</td>
<td>2335.8 ± 331.8</td>
<td>2281.6 ± 327*</td>
<td>2728.4 ± 403.7*</td>
<td>2802.1 ± 371.44* #</td>
</tr>
<tr>
<td></td>
<td>0.1</td>
<td>0.25</td>
<td>.01 $</td>
<td>0.03 $</td>
</tr>
<tr>
<td>PVRI</td>
<td>317.9 ± 165.7</td>
<td>341 ± 163.2 *</td>
<td>334.4 ± 117.4</td>
<td>322.5 ± 103.11</td>
</tr>
<tr>
<td></td>
<td>0.02 $</td>
<td>0.2</td>
<td>0.3</td>
<td>0.6</td>
</tr>
</tbody>
</table>

*significant in relation to TLV           # significant in relation to OLV L.
$ significant difference between the two groups.

HR=Heart rate. MAP=Mean arterial pressure. CI=Cardiac index. CVP=Central venous pressure.
PCWP=Pulmonary capillary wedge pressure. PAP=Pulmonary artery pressure. SVRI=Systemic vascular
resistance index.
PVRI=Pulmonary vascular resistance index.

increase in HR and SVRI. In contrast to these findings, Abe et al.,(14)and Ebert et al.,(18) in their study found that sevoflurane decreased MAP and they postulated that this decrease in MAP might be due to decrease in SVRI.

These changes in MAP after institution of OLV were associated with significant decrease in CI, SVRI and a significant increase in PVRI in propofol group during OLVs, and these results are in agreement with the study done by Kellow et al.,(19)and Reid et al.,(20)and this might be attributed to slight decrease in myocardial contractility leading to increase in end-diastolic pressure and more pulmonary congestion leading to increase in PVRI and PCWP. On turning the patient to lateral position (OLV L), there was increase in CI, SVRI together with increase in PVRI in propofol group. In contrast to this result, Beck et al.,(10) reported increases in CI after propofol during OLV.

CI in sevoflurane group during OLV L was preserved or slightly increased; also SVRI increased together with PVRI and these results were in agreement with a study done by Wang et al.,(6) who compared
the effects of sevoflurane and isoflurane on arterial oxygenation during OLV and they concluded that there was no significant difference in PvO₂ or CI. This was in contrast to study done by Beck et al.⁴ who reported increases in CI together with increase in PVRI after propofol and sevo- flurane during OLV. Ebert et al.⁸ stated that CI was stable during sevoflurane anesthesia due to preserved myocardial contractility.

In the present study, there was a statistically significant increase in shunt fraction together with decrease in PaO₂ in both groups in the four sampling times and this was in agreement with the studies done by Abe et al.,⁹ Wang et al.,⁸ and Beck et al.,¹⁰. The reported changes in shunt and PaO₂ during OLV might be explained by the fact that, during OLV, the non-dependent lung is non-ventilated so, any blood flow to the nonventilated lung becomes shunt flow; in addition to whatever shunt flow might exist in the dependent lung, consequently, it was not surprising to find that, given the same FiO₂, haemodynamics and metabolic status as TLV; OLV resulted in a much larger alveolar to arterial oxygen tension difference P(A-a)O₂ and lower PaO₂ than does TLV¹.

Turning the patient from supine to lateral position was associated with significant increase in shunt fraction; this was in agreement with the studies done by Abe et al.,⁹ and Beck et al.,¹⁰. Turning the patient from supine to the lateral position would decrease the blood flow of the nondependent lung due to gravity; this decrease has been assumed to be approximately 10% of the total pulmonary blood flow²¹,²². Intrapulmonary shunt would usually increase from approximately 5% in supine to 10-15% in lateral position²³,²⁴. This might explain why shunt fraction increased after turning the patient to lateral position together with loss of the dependent lung volume with general anesthesia, compression by mediastinum, abdominal contents and/or suboptimal positioning effects. In addition, poor mucocilliary clearance may cause further dependent lung volume loss.

The increase in shunt and decrease in PaO₂ on opening chest (OLV₀) might be due to the fact that upon opening the nondependent hemithorax, there is an increase in compliance and FRC in the nondependent lung and nondependent lung vascular resistance decreases significantly associated with a decrease in compliance and FRC of the dependent lung; this resulted in a further increase in the non-dependent lung blood flow²⁵.

Another explanation is that, when the nondependent lung falls away from open chest, the vertical distance between the heart and the nondependent lung may decrease, which might result in increased perfusion in the nondependent lung²¹. This was in agreement with Chow et al.,¹⁵ as regards to sevoflurane results and with Von Dossow et al.,²⁶ as regards to propofol results.

The PaCO₂ also increased significantly in the two studied groups in all sampling times and this was matched with the studies done by Abe et al.,⁹ Wang et al.,⁸ and Beck et al.,¹⁰. This increase in PaCO₂ could be attributed to the fact that, during OLV, the ventilated lung can eliminate almost enough CO₂ to compensate for nonventilated lung, and PACO₂ and PETCO₂ gradient are small. However, the ventilated lung cannot take up enough O₂ to compensate for nonventilated, and PAO₂ to PaO₂ gradients are usually large with a constant minute ventilation. The retention of CO₂ by blood traversing the nonventilated lung usually slightly exceeds the increased elimination of CO₂ from blood traversing the ventilated lung, and the PaCO₂ will usually slowly increase over the time unless RR is increased².

PvO₂ decreased significantly in the two groups in all sampling times and there was no significant difference between the two groups and this was in agreement with Beck et al.,¹⁰ Chow et al.,¹⁵ and Domino et al.,¹⁷.

Shunt blood flow through the atelectatic lung during OLV depends principally on hypoxic pulmonary vasoconstriction (HPV), rather than mechanical effects of lung collapse². So, HPV is a function of both PaO₂ and PvO₂. General anesthetic agents can affect PaO₂ by two possible mechanisms: first, they can alter PvO₂ by altering
CO; second, they can alter shunt flow directly by inhibition of HPV and indirectly by altering HPV through change in PvO₂. Several studies have focused on inhaled versus intravenous anesthetic agents in humans undergoing OLV and it has often been difficult to show clinically significant difference between inhaled and intravenous anesthetics; this was in contrast to the results of the more easily controlled in-vitro studies done by Eisenkraft. These conflicting results of human studies are widely attributed to haemodynamic changes particularly their effect on CO, which may be more pronounced especially with the old volatile agents. The efficacy of HPV is inversely related to the CO, but interactions between several physiological variables can obscure the effect of CO on HPV and shunt fraction in clinical setting.

In the present study, both propofol and sevoflurane resulted in nearly similar increases in shunt fraction during OLV and this was possibly attributed to haemodynamic changes, particularly the reduction in CO which was more with propofol and these results agreed well with the reports of Beck et al. In contrast to this, propofol infusion in the range of 6-12 mg/kg/h did not inhibit HPV or decrease PaO₂ in the study done by Nakayama and Murray. Also, Pass and Dusing and Heller et al. stated that propofol has less effect on HPV than volatile anethetics during OLV. Reid et al. compared isoflurane with propofol anesthesia and they found a significant increase in shunt and CO during isoflurane anesthesia only. The increase in CO during isoflurane anesthesia could explain why there was no difference in PaO₂ despite the increase in shunt. This was demonstrated in study done in pigs by Karzai et al. who found that increasing concentrations of desflurane have haemodynamic effects that negate its direct inhibitory effect on HPV. The increase in shunt fraction by direct inhibition of HPV by inhalation anesthetics administered in concentrations of 1 MAC could be expected to be rather small. Despite maximal stimulation of the HPV response, there will be an obligatory shunt fraction of 25% during OLV. Attenuation of the maximal HPV response by 25% would increase shunt fraction from 25% to 30% in the presence of 1MAC sevoflurane provided other confounding variables remain unchanged. Thus direct inhibition of HPV could cause a 5% increase in shunt fraction. In our study, the increase in Qₐ/Qₜ that was affected with both groups occurred in conjunction with increases in PVRI and this might suggest that changes in Qₐ/Qₜ were caused by global pressure acting throughout the pulmonary vasculature and this was in agreement with the study done by Abe et al. who compared isoflurane and sevoflurane.
In conclusion: both sevoflurane and propofol administration causes an increase in shunt fraction. \(PvO_2, PaO_2, PaCO_2, CI\) did not differ between both groups. Both sevoflurane and propofol are considered as good drugs for thoracic operations but sevoflurane appear to be more cardio-vascular stable. Much of the overall shunt fraction during OLV may result from sources other than inhibition of HPV response; haemodynamic stability and appropriate ventilatory maneuvers are more important for achieving optimal arterial oxygenation during OLV than choice of anesthetic agent which has to be considered for achieving optimal arterial oxygenation.

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