# Effects of Desflurane and Sevoflurane on Systemic and Pulmonary Hemodynamic Status and Pulmonary Shunt Fraction in Patients Undergoing One-Lung Ventilation for Thoracic Surgery

## Torasik Cerrahide Tek Akciğer Ventilasyonu Uygulanan Hastalarda Desfluran ve Sevofluranın Sistemik ve Pulmoner Hemodinamik Durum ve Pulmoner Şant Fraksiyonu Üzerine Etkileri

Hanife KARAKAYA KABUKCU[1], Nursel SAHİN[2], Bulent Naci KANEVETCİ[3], Abdullah ERDOGAN[4], Tulin AYDOGDU TİTİZ[5]

[1]MD, Assistant Professor, Akdeniz University Departments of Anesthesiology and Reanimation and Thoracic Surgery Antalya-TURKEY

[2]MD, Assistant Professor Akdeniz University Departments of Anesthesiology and Reanimation and Thoracic Surgery Antalya-TURKEY

[3]MD, Fellow Research Akdeniz University Departments of Anesthesiology and Reanimation and Thoracic Surgery Antalya-TURKEY

[4]MD, Assistant Professor Akdeniz University Departments of Anesthesiology and Reanimation and Thoracic Surgery Antalya-TURKEY

[5]MD, Professor Akdeniz University Departments of Anesthesiology and Reanimation and Thoracic Surgery Antalya-TURKEY

**İletişim Adresi**:
Dr. Hanife KARAKAYA KABUKCU
Akdeniz Üniversitesi Tıp Fakültesi
Dumlupınar Kampüsü
Anesteziyoloji ve Reanimasyon
Ana Bilim Dalı
07070-Antalya-TÜRKİYE
**Tel:** +90 242 249 62 35
**Fax:** +90 242 227 88 36
**E-mail:** hanifekabukcu@akdeniz.edu.tr

**SUMMARY**

**Objective:** The effects of desflurane and sevoflurane on pulmonary shunt fraction and also systemic and pulmonary hemodynamic status in patients requiring OLV for thoracic surgery were evaluated.

**Material and Methods:** Thirty adult patients, ASA I or II, requiring one-lung ventilation (OLV) for thoracic surgery. Anesthesia was induced with iv propofol (1-2 mg/kg), fentanyl (2 μg/kg), and vecuronium (0.1 mg/kg), and patients were ventilated via mask with in 100% $O_2$. Desflurane (Group 1) was administered to maintain an inspiratory concentration of 6-10%. Sevoflurane (Group 2) was administrated in order to maintain an inspiratory concentration of 1-1.5%. The patients were ventilated 50% $O_2$ and air. After induction of anesthesia, right internal jugular vein was cannulated and a flow-directed thermodilution catheter was placed in the pulmonary artery. After tracheal intubation in the supine position, patients were turned to lateral decubitus position. Systemic and pulmonary hemodynamic data were recorded and arterial and mixed venous blood samples were obtained during one-lung ventilation (OLV) in the lateral decubitis position at the beginning and $10^{th}$ and $20^{th}$ minutes of OLV.

**Results:** Patient demographics were similar between the desflurane and sevoflurane groups. Clinically important tachycardia, hypotension or hypertension with the transition to OLV were not detected and a stable cardiovascular condition was easily obtained by the use of both anesthetic agent. Statistically meaningful increases of cardiac output in sevoflurane group were found, but in desflurane group, increaments did not reach to statistically important points. With transition to OLV, 21% and 22% increaments in shunt fractions were observed in desflurane and sevoflurane respectively.

**Conclusion:** Desflurane and sevoflurane produce similar cardiovascular and pulmonary hemodynamic effects and shunt ratio before and during one-lung ventilation in patients undergoing thoracic surgery. Stable cardiovascular condition was obtained by the use of both anesthetic agents. Both agents did not cause clinically significant hypertension and tachycardia.

***Key Words: Anesthesia, one lung ventilation, thoracic surgery, inhalation anesthetic agent, shunt fraction***

## ÖZET

**Amaç:** Torasik cerrahide tek akciğer ventilasyonu (TAV) uygulanan hastalarda desfluran ve sevofluranın sistemik ve pulmoner hemodinamik durum ve pulmoner şant fraksiyonu üzerine etkilerinin değerlendirilmesidir.

**Gereç ve Yöntemler:** Çalışmaya, torasik cerrahide tek akciğer ventilasyonu (TAV) yapılan 30 erişkin hasta dâhil edildi. Anestezi indüksiyonunda, intravenöz (IV) propofol (1-2 mg/kg), fentanil (2 μg/kg) ve vekuronyum (0.1 mg/kg) uygulanan hastalar %100 $O_2$ maske ile ventile edildi. Anestezi idamesinde desfluran %6-10 (Grup 1) sevofluran (Grup 2) %1-1.5 inspiratuar konsantrasyonlarda uygulandı. %50 $O_2$ ve kuru hava ile mekanik ventilasyon yapıldı. Trakeal entübasyondan sonra sağ internal juguler ven kanülü uygulandı ve pulmoner arter kateteri yerleştirildi. Lateral dekübitis pozisyonunda tek akciğer ventilasyonu başlangıcında, 10. ve 20. dakikalarda sistemik ve pulmoner hemodinami kaydedilerek, arteriyel ve miks venöz kan örnekleri alındı.

**Bulgular:** Desfluran ve sevofluran gruplarındaki hastaların demografik özellikleri benzerdir. TAV'a geçişte klinik olarak önemli taşikardi, hipotansiyon veya hipertansiyon bulunmamıştır. Her iki anestezik ajanın kullanımında kardiyovasküler stabilite kolay elde edilmiştir. Sevofluran grubunda istatistiksel olarak anlamlı kardiyak "output" artışı bulunmuştur, desfluran grubunda istatistiksel olarak anlamlı bir artış gözlenmemiştir. TAV'a geçişte şant fraksiyonunda artış desfluranda %21, sevofluranda %22 olarak gözlenmiştir.

**Sonuç:** Desfluran ve sevofluran torasik cerrahi hastalarında tek akciğer ventilasyonu öncesinde ve sonrasında kardiyovasküler ve pulmoner hemodinamik etkileri ve şant oranları benzerdir. Her iki anestezik ajanın kullanımında kardiyovasküler stabilite kolay elde edilmiştir. Her iki anestezik ajan klinik olarak anlamlı hipertansiyon ve taşikardiye neden olmamıştır. Anahtar Kelimeler: Anestezi, tek akciğer ventilasyonu, torasik cerrahi, inhalasyon anestezik ajanı, şant fraksiyonu


## INTRODUCTION

Inhalation anaesthesia is generally favored over iv anaesthesia for surgery requiring one-lung ventilation (OLV) because of its several advantages. These include ease of drug delivery, offset independent of hepatic or renal function, end-tidal concentration monitoring, bronchodilatation and the option of gaseous induction (1,2). In contrast, inhalation anaesthetics inhibit the hypoxic pulmonary vasoconstriction (HPV) which is a homeostatic mechanism of pulmonary circulation. HPV maintains the optimal oxygenation of the arterial blood with a mechanism that diverts pulmonary blood flow away from lung regions with low alveolar oxygen tensions towards better ventilated areas of the lung, thus reducing venous admixture (2,3).

Desflurane and sevoflurane are very commonly used volatile anesthetic agents. Their pharmacokinetic properties make them highly attractive for use in thoracic surgery (4). All inhalated anesthetics inhibit HPV to varying degrees and therefore affect intrapulmonary shunting (2). In this study, we compared the effects of desflurane and sevoflurane on pulmonary shunt fraction and on systemic and pulmonary hemodynamic status in patients requiring OLV for thoracic surgery.

## MATERIAL AND METHODS

After acquiring the approval from the hospital's ethics committee and informed consent, 30 adult patients, ASA I or II, requiring OLV for thoracic surgery were randomized to receive inhalational anesthesia with desflurane (n= 15, patients 1-15) or sevoflurane (n= 15, patients 16-30). None had a history of obstructive airways disease. Patients demonstrating hemodynamic instability, renal hepatic insufficiency or neurological disease were excluded. Heart disease was evaluated by means of personal medical history, physical status, electrocardiography, and echocardiography.

No preanesthetic medication was administered. After arrival in the operating room, an IV cannula was placed for infusion of 0.9% saline 10 $mL.kg^{-1}$ and a radial arterial catheter was placed for continuous monitorisation of arterial blood pressure.

Anaesthesia was induced by IV propofol (1-2 mg/kg), fentanyl (2 μg/kg), and vecuronium (0.1 mg/kg), and patients were ventilated via a mask with in 100% $O_2$. After induction of anaesthesia, a left-sided double-lumen endobronchial tube (Broncho-Cath, Mallinckrodt Medical, Athlone, Ireland) was placed in all patients and initially positioned by auscultation. The endobronchial tube position was confirmed and adjusted with

fiberoptic bronchoscopy. Tracheal and bronchial cuff pressures were measured and kept between 20-40 cm $H_2O$ with intermittant measurements and manuel pressure release. Desflurane (Group 1) was administered to maintain an inspiratory concentration of 6-10%. Sevoflurane (Group 2) was administered to maintain an inspiratory concentration of 1-1.5%. The patients were ventilated with 50% $O_2$ and air. During two-lung ventilation, tidal volumes were adjusted to be 10 mL/kg. As one-lung ventilation began, tidal volumes were readjusted to be 4-5 mL/kg so as to keep the arterial carbondioxide concentrations between 35 and 45 mmHg.

After tracheal intubation, right internal jugular vein was cannulated and a flow-directed thermodilution catheter (Edwards Lifesciences LLC, Irvine, CA, USA) was placed in the pulmonary artery. In all patients, other monitoring parameters including ECG, pulse oximeter, nasopharyngeal temperature, neuromuscular block, ventilation pressures and volumes, end-tidal carbondioxide concentration and urine output were performed. Peak airway pressure, tidal volume, ventilatory rate, both tracheal and bronchial cuff pressures were followed up.

After intubation in the supine position, patients were turned to lateral decubitus position. Systemic and pulmonary hemodynamic datas were recorded and during OLV in the lateral decubitis position, arterial and mixed venous blood samples were obtained at the beginning

(0 minutes) and $10^{th}$ and $20^{th}$ minutes during OLV.

In patients with PA catheters; thermodilution cardiac output (CO), stroke volume (SV), systemic and pulmonary vascular resistances (SVR and PVR, respectively), mixed venous blood gases were measured. At the same time, haemodynamic variables were recorded, including heart rate (HR), mean arterial pressure, mean pulmonary arterial pressures (MPAP), pulmonary arterial occlusion pressures (PCWP) and central venous pressures (CVP). Oxygen consumption ($VO_2$) was determined as the product of cardiac output and the difference between arterial and venous oxygen content. The shunt fraction (Qs/Qt) was computed using a standard formula $Qs/Qt= (Cc_{02}-Ca_{02})/(Cc_{02}-Cv_{02})$ where Qs= shunt flow, Qt= cardiac output and $Cc_{02}$= oxygen content of pulmonary end-capillary, $Ca_{02}$= arterial oxygen content, $Cv_{02}$= mixed venous oxygen content (5).

The two-tailed Student's unpaired t-test compared groups for normally distributed data, which are reported as means ± SD. Comparisons of treatment within each group were made by Paired t-test significance required a value of $p< 0.05$.

## RESULTS

### 1) Demographic Data

Patient demographics were similar between the desflurane and sevoflurane groups (Table 1).

Table 1. Demographics and preoperative data

| | **Desflurane (n= 15)** | **Sevoflurane (n= 15)** |
|---|---|---|
| Age (year) | 47 ± 12 | 54 ± 13 |
| Weight (kg) | 67.8 ± 12 | 65.5 ± 13 |
| BSA ($m^2$) | 1.76 ± 0.15 | 1.70 ± 0.19 |
| HR (bpm) | 76.6 ± 10 | 82.3 ± 16 |
| MAP (mmHg) | 81.9 ± 8.5 | 86 ± 18 |
| $PaO_2$ (mmHg) | 93 ± 12 | 94 ± 11 |
| $PaCO_2$ (mmHg) | 35.5 ± 3.6 | 36.6 ± 3.9 |
| Oxygen saturation (%) | 97.4 ± 1.2 | 97.3 ± 0.9 |
| Duration of operation (min) | 120 ± 59 | 143 ± 62 |
| Duration of anaesthesia (min) | 171 ± 47 | 185 ± 66 |

BSA= body surface area, HR= heart rate, MAP= mean arterial pressure.

### 2) Evaluation of Hemodynamical Changes with Transition to One Lung Ventilation According to Baseline (Two-Lung Ventilation) (Table 2)

**Heart rate:** There were no significant differences in desflurane group. In sevoflurane group, statistically meaningful increases were found at the beginning and the $10^{th}$ minute of OLV. They returned back to base-line levels at $20^{th}$ minute.

**Mean arterial pressure:** Significant increases were measured at the beginning and during OLV in desflurane group. In group sevoflurane, at the beginning and at the $10^{th}$ minute of OLV, statistically significant increases of mean arterial pressures were observed and at the $20^{th}$ minute of OLV, they returned to base-line levels.

**Central venous pressure:** CVP did not change at the beginning or during OLV in both groups.

**Mean pulmonary artery pressure:** MPAP increased at the beginning and during OLV in both two groups.

**Pulmonary capillary wedge pressure:** It increased at the beginning and during OLV in desflurane group. In sevoflurane group, it increased after 10th minute of OLV.

**Cardiac output:** In sevoflourane group, statistically meaningful increases were measured. In desflurane group, increases did not reach to statistically important point.

**Stroke volume:** SV did not change at the beginning or during OLV in desflurane group. In sevoflurane group, it did not change at the beginning and the 10th minute of OLV but increased at the 20th minute of OLV.

**Systemic vascular resistance:** It did not change at the beginning and during OLV in desflurane group. In sevoflurane group, it decreased at the 10th and 20th minutes of OLV.

**Pulmonary vascular resistance:** In desflurane group, it increased at 10th and 20th minutes of OLV. It did not change at the beginning or during OLV in sevoflurane group.

**Shunt fraction:** It increased at the beginning and during OLV in both group.

**3) Evaluation of Changing in the Blood Gases with Transition to One Lung Ventilation According to Baseline (Two-Lung Ventilation) (Table 3-4)**

**$PaO_2$ and $SaO_2$:** reductions in $PaO_2$ and $SaO_2$ occurred during OLV in desflurane and sevoflurane groups.

**$PaO_2$:** increases in $PaCO_2$ occurred during OLV in desflurane and sevoflurane groups.

**$PvO_2$ and $SvO_2$:** Statistically meaningful changes were not observed in both groups at the beginning and during OLV.

**Oxygen consumption:** It remained unchanged during OLV in both groups.

**Tidal volume:** It reduced in both groups during OLV

**Peak airway pressure:** It increased in both groups during OLV. Tracheal and bronchial cuff pressures did not change with transition and during OLV.

## DISCUSSION

In this study, the effects of desflurane and sevoflurane on systemic and pulmonary hemodynamic status and pulmonary shunt fraction in patients requiring OLV for thoracic surgery were investigated.

### Evaluation of the Heart Rate and Arterial Pressure

In desflurane group, while heart rate did not change with transition to OLV, an increase in blood pressure within normal range, however, was determined. In sevoflurane group, while heart rate increased at beginning and the 10th minute, blood pressure was seen to be increased within normal range. Clinically significant tachycardia or hypertension was not determined. It has been reported that a rapid ascent to desflurane concentration over 1 MAC had caused CNS activation via tracheopulmonary receptors, and resulted in tachycardia and hypertension (6-8). That's why concentration over 1 MAC was never reached in our study. It was also reported that desflurane concentration below 1 MAC did not cause any significant alteration in heart rate (6-8). In our study, although a slight increase in heart rate was seen, it was not clinically significant. We think that, the restrictive effect of fentanyl on sympathetic nervous system, used in induction and maintenance of anaesthesia, may have prevented significant tachycardia and hypertension.

### Evaluation of Cardiac Output and its Determinants

Consecutive physiopathological interactions exist among cardiac output, mixed venous $O_2$ saturation, and hypoxic pulmonary vasoconstriction (9). A decrease in cardiac output reduces mixed venous $O_2$ saturation and results in increases in pulmonary vasoconstriction. An increase in cardiac output, however, increases mixed $O_2$ saturation and reduces hypoxic pulmonary vasoconstriction (9). In our study; increase in cardiac output was statistically significant in sevoflurane group. In desflurane group, a slight but insignificant increase was determined.

**a- Hypoxic pulmonary vasoconstriction:** Hypoxic pulmonary vasoconstriction (HPV) which has been mainly detected by alveolar $O_2$ concentration and mixed venous $O_2$ saturation is inversely related to the cardiac output (10). In our study, when we had turned to OLV, no significant difference was observed in respect to mixed venous $O_2$ saturation and pressure (partial pressure of $O_2$) in both groups. Addition-

**Table 2.** Systemic and pulmonary haemodynamia

| | TLV | OLV-1 | OLV-2 | OLV-3 |
|---|---|---|---|---|
| HR (bpm) | | | | |
| Des | 73 ± 14 | 73 ± 16 | 73 ± 10 | 81 ± 7.0 |
| Sevo | 69 ± 11 | 77 ± 11* | 79 ± 11* | 71 ± 22 |
| MAP (mmHg) | | | | |
| Des | 77 ± 3.9 | 91 ± 16* | 92 ± 13* | 87 ± 11* |
| Sevo | 72 ± 8.5 | 93 ± 12* | 81 ± 12* | 78 ± 15 |
| CVP (mmHg) | | | | |
| Des | 12.8 ± 3.9 | 13.5 ± 4.0 | 13.5 ± 3.1 | 12.0 ± 2.7 |
| Sevo | 10.3 ± 2.2 | 11.4 ± 3.4 | 12.2 ± 4.2 | 9.7 ± 3.3 |
| MPAP (mmHg) | | | | |
| Des | 20.5 ± 3.6 | 30.0 ± 6.5* | 27.9 ± 5.4* | 26.2 ± 5.8* |
| Sevo | 18.6 ± 3.7 | 25.5 ± 6.6* | 23.9 ± 6.5* | 24.6 ± 5.8* |
| PCWP (mmHg) | | | | |
| Des | 10.4 ± 1.3 | 14.5 ± 2.3* | 12.5 ± 2.5* | 13.4 ± 3.2* |
| Sevo | 9.2 ± 2.1 | 11.0 ± 4.0 | 13.0 ± 3.4* | 12.7 ± 4.2* |
| CO (L/min) | | | | |
| Des | 5.5 ± 1.5 | 5.7 ± 1.1 | 6.0 ± 1.6 | 6.4 ± 1.0 |
| Sevo | 5.3 ± 1.0 | 6.7 ± 1.4* | 6.6 ± 1.2* | 7.4 ± 1.0* |
| SV (mL) | | | | |
| Des | 82 ± 24 | 89 ± 27 | 82 ± 26 | 90 ± 31 |
| Sevo | 85 ± 24 | 90 ± 25 | 92 ± 22 | 108 ± 30* |
| SVR (dynes.s.cm$^5$) | | | | |
| Des | 946 ± 175 | 986 ± 231 | 1091 ± 280 | 899 ± 239 |
| Sevo | 960 ± 182 | 1005 ± 212 | 726 ± 182* | 710 ± 213* |
| PVR (dynes.s.cm$^5$) | | | | |
| Des | 87 ± 29 | 92 ± 25 | 131 ± 39* | 135 ± 60* |
| Sevo | 95 ± 18 | 89 ± 30 | 100 ± 43 | 88 ± 31 |

Data are mean ± SD.

TLV= two-lung ventilation, OLV= one-lung ventilation, OLV-1= data at the beginning of OLV, OLV-2= data at the 10th min OLV, OLV-3= data at the 20th min OLV, Des= desflurane, Sevo= sevoflurane, HR= heart rate, MAP= mean arterial pressure, CVP= central venous pressure, MPAP= mean pulmonary artery pressures, PCWP= pulmonary capillary wedge pressure, CO= cardiac output, SVR= systemic vascular resistance, PVR= pulmonary vascular resistance, SV= stroke volume.

* Significantly ($p< 0.05$) different from TLV value.

Table 3. Arterial and mixed venous blood Gas values.

| | TLV | OLV-1 | OLV-2 | OLV-3 |
|---|---|---|---|---|
| $PaO_2$ (mmHg) | | | | |
| Des | 235 ± 69 | 143 ± 89* | 139 ± 91* | 128 ± 82* |
| Sevo | 260 ± 96 | 138 ± 45* | 120 ± 61* | 130 ± 58* |
| $PaCO_2$ (mmHg) | | | | |
| Des | 34 ± 5.4 | 38 ± 5.5* | 39 ± 5.0* | 45 ± 8.7* |
| Sevo | 33 ± 4.3 | 39 ± 7.7* | 38 ± 6.5* | 38 ± 6.7* |
| $SaO_2$ (%) | | | | |
| Des | 99.6 ± 0.4 | 96.7 ± 3.5* | 97.3 ± 2.8* | 94.7 ± 5.9* |
| Sevo | 99.5 ± 0.6 | 98.4 ±1.6 | 97.3 ± 1.9* | 97.9 ± 1.7* |
| $PvO_2$ (mmHg) | | | | |
| Des | 55 ± 16 | 47 ± 5.8* | 51 ± 7.4 | 52 ± 12 |
| Sevo | 45 ± 6.2 | 50 ± 7.3 | 53 ± 14 | 52 ± 11 |
| $SvO_2$ (%) | | | | |
| Des | 85 ± 7.3 | 80 ± 5.5 | 82 ± 5.4 | 80 ± 9.1 |
| Sevo | 83 ± 4.9 | 84 ± 4.9 | 85 ± 7.0 | 85 ± 6.2 |
| $VO_2$ (mL $0_2$) | | | | |
| Des | 200 ± 171 | 160 ± 83 | 180 ± 117 | 211 ± 156 |
| Sevo | 229 ± 99 | 231 ± 113 | 228 ± 101 | 305 ± 166 |
| Qs/Qt (%) | | | | |
| Des | 7.3 ± 3.5 | 16.3 ± 4.9* | 21.8 ± 7.2* | 20.6 ± 6.9* |
| Sevo | 5.2 ± 1.4 | 18.3 ± 4.8* | 20.8 ± 8.7* | 22.2 ± 6.8* |

Data are mean ± SD.

TLV= two-lung ventilation, OLV= one-lung ventilation, OLV-1= data at the beginning of OLV, OLV-2= data at the 10th min OLV, OLV-3= data at the 20th min OLV, $PvO_2$= venous partial pressure oxygen, $SaO_2$ and $SvO_2$= arterial and venous hemoglobin saturation, $VO_2$= oxygen consumption,

Qs/Qt= shunt fraction Des= desflurane, Sevo= sevoflurane.

* Significantly ($p< 0.05$) different from TLV value.

**Table 4.** Respiratory parameters and cuff pressures.

| | TLV | OLV-1 | OLV-2 | OLV-3 |
|---|---|---|---|---|
| Tidal volume (mL) | | | | |
| Des | 652 ± 104.5 | 339 ± 51.0* | 346 ± 106.9* | 359 ± 101.2* |
| Sevo | 605 ± 107.1 | 334.9 ± 53.4* | 362.5 ± 54.2* | 346 ± 63.3* |
| Peak airway pressure (cm $H_2O$) | | | | |
| Des | 22.4 ± 5.4 | 27.5 ± 7.6* | 28.0 ± 7.1* | 24.8 ± 7.3* |
| Sevo | 20.4 ± 3.5 | 29.5 ± 7.3* | 29.0 ± 6.3* | 27.9 ± 5.6* |
| Tracheal cuff pressure (cm $H_2O$) | | | | |
| Des | 30.1 ± 14.8 | 31.2 ± 15.5 | 29.1 ± 10.3 | 22.4 ± 5.9 |
| Sevo | 27.0 ± 17.6 | 21.4 ± 4.6 | 21.3 ± 4.1 | 20.4 ± 4.1 |
| Bronchial cuff pressure (cm $H_2O$) | | | | |
| Des | 24.1 ± 12.2 | 23.2 ± 13.7 | 21.7 ± 11.8 | 18.4 ± 9.5 |
| Sevo | 23.8 ± 11.6 | 22.2 ± 5.4 | 20.5 ± 4.9 | 18.7 ± 4.0 |
| Ventilatory rate | | | | |
| Des | 12 ± 0 | 20.5 ± 3.2* | 19.4 ± 2.1* | 19.5 ± 2.0* |
| Sevo | 12 ± 0 | 19.5 ± 1.5* | 19.3 ± 1.5* | 20.1 ± 1.5* |

TLV= two-lung ventilation, OLV= one-lung ventilation, OLV-1= data at the beginning of OLV,
OLV-2= data at the 10th min OLV, OLV-3= data at the 20th min OLV,
* Significantly ($p< 0.05$) different from TLV value.

ally, $O_2$ consumption which may affect mixed venous $O_2$ saturation was found to be similar in both groups. Alveolar $O_2$ concentration was regulated by $O_2$ fraction according to arterial $O_2$ saturation. Finally, basic determinants of the HPV were similar in two groups. Therefore, we think that sevoflurane and desflurane have similar effects on the HPV.

Only dramatic changes in the alveolar $O_2$ and mixed venous $O_2$ saturation may effect the pulmonary arterial vasoconstriction. In our study, pulmonary vascular resistance, which is proportioned to HPV, didn't change with the transition to OLV. Therefore we think that, effect of sevoflurane and desflurane on HPV is clinically unimportant.

**b- Systemic vascular resistance:** Systemic vascular resistance is inversely related with cardiac output (9). There was a reduction in SVR that resulted an increased cardiac output in sevoflorane group. In our study, it could not be possible to reveal the contribution of HPV inhibition. In desflurane group, a slight but insignificant increase in CO and a slight but insignificant decrease in SVR were determined.

In fact, changes in the cardiac output causing haemodynamically important situations occur only in the presence of dramatic changes in systemic vascular resistance and pulmonary vascular resistance (9,10). In our study, clinically important tachycardia, hypotension or hypertension with the transition to OLV were not detected and a stable cardiovascular condition was easily obtained by the use of both anesthetic agents. Therefore, we think that the effects of sevoflurane and desflurane on PVR and SVR are not clinically important.

**Affects on $PaO_2$, $PaCO_2$ and Shunt Fraction**

Despite the fact that OLV greatly facilitates the surgical manipulation on the non-ventilated lung, it creates serious ventilation-perfusion mismatch abnormalities. In OLV, two main factors cause the ventilation-perfusion mismatch. One of these factors is true shunt in the non-ventilated lung, where there is blood flow to the alveoli but no ventilation. The other factor is the shunt that gives rise to the alveoli with low ventilation-perfusion ratios. The blood in the upper lung can not take up sufficient oxygen, and therefore retains its poorly oxygenated mixed venous composition. A second factor causes approximately 10-15 percent venous admixture during two-lung ventilation. Venous admixture increases to a level of 30-40 per cent during one-lung ventilation (9). Handling of pulmonary vessel by surgeons during operation may increase shunt rate via HPV inhibition (9). In our study, to minimize this effect, blood gas analysis was performed before surgical handling of the lung.

During OLV, $PaO_2$ is maintained in a safe range with a high-inspired oxygen concentration at

level of 50% or over. Additionally, we reduced tidal volume and increased ventilation rate. In our study, with transition to OLV, a similar reduction of $PaO_2$ in both groups was observed. There were slight increases in $PaCO_2$ values but they were within normal ranges. Reduction in $O_2$ saturation of clinical importance was not determined. In patients in whom sufficient $PaO_2$ could not be obtained by $FiO_2$ level and tidal volume/ventilation rate regulations, PEEP has been proposed for restriction of blood flow at nonventilated lung (11,12). In our study, although there were not any patients requiring this application, during OLV, it is advised to be aware of all necessary maneuvers to increase $PaO_2$.

In our study with transition to OLV, 21% and 22% increases in shunt fraction were observed in desflurane and sevoflurane groups respectively. In present study, in both groups, adequate arterial oxygenation could be maintened with these shunt rates. Beck et al (13) reported 30% shunt fraction in OLV with sevoflurane, and Slinger et al (14) found 39% and 41% shunt fraction with isoflurane and enflurane respectively in thoracic surgery. Pagel and et al (15) tested the effects of desflurane and isoflurane on shunt fraction in patients undergoing one-lung ventilation. They found 40% shunt fraction in desflurane group and 34% shunt fraction in isoflurane group. In all of these studies, as in our study, increases in shunt fraction with the onset of OLV were well tolerated with implement of a high concentrated oxygen inspiration.

## CONCLUSION

In present study, it was concluded that, desflurane and sevoflurane produce similar cardiovascular and pulmonary haemodynamic effects and shunt fraction before and during one-lung ventilation in patients undergoing thoracic surgery. A stable cardiovascular condition was easily obtained by the use of both anesthetic agents. Both agents did not cause clinically significant hypertension and tachycardia.

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