British Journal of Anaesthesia **98 (4):** 539–44 (2007)
doi:10.1093/bja/aem039 Advance Access publication March 8, 2007

BJA

Effects of propofol *vs* sevoflurane on arterial oxygenation during one-lung ventilation

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Background. The inhibitory effect of anaesthetic agents on hypoxic pulmonary vasoconstriction may depend upon their dose, especially when using a volatile agent. The aim of this randomized open study was to compare the effects of sevoflurane and propofol, as primary anaesthetic agents, on oxygenation during one-lung ventilation (OLV), with their administration being adjusted to maintain bispectral index (BIS) values between 40 and 60.

Methods. Eighty patients scheduled for a lobectomy, receiving an epidural mixture of ropivacaine and sufentanil, were randomly assigned to Group S (maintenance with sevoflurane) or Group P (maintenance with propofol). After placement of a double-lumen tube, the lungs were ventilated at an inspiratory fraction of oxygen of 1.0, a tidal volume of 6 ml kg^{-1}, and 12 bpm. Arterial blood gas samples were taken as follows: during two-lung ventilation before OLV, and during the first 40 min of OLV.

Results. Fifteen patients were excluded (incorrect placement of the tube or BIS outside the desired range). The two groups were comparable in terms of demographic variables, haemodynamic, and BIS levels during the operation. Four patients in each group had a $Sp_{O_2}<90\%$. Mean of the lowest Pa_{O_2} was 16.3 (7.5) kPa in Group S and 17.7 (9.3) kPa in Group P (ns).

Conclusions. Sevoflurane and propofol had similar effect on Pa_{O_2} during OLV when their administration is titrated to maintain BIS between 40 and 60.

Br J Anaesth 2007; **98**: 539–44

Keywords: anaesthetics i.v., propofol; anaesthetics volatile, sevoflurane; oxygenation; surgery, thoracic; ventilation, one-lung

Accepted for publication: January 22, 2007

The major cause of hypoxaemia during one-lung ventilation (OLV) is the pulmonary arterio-venous shunt of the de-oxygenated blood through the non-ventilated lung. Patients with good preoperative spirometric pulmonary function tests or a proportionately high perfusion of the operative lung tend to have lower Pa_{O_2} values during OLV. The side of lung collapse also affects the Pa_{O_2} during OLV with a higher incidence of hypoxaemia when the right lung is collapsed for surgery. Several intraoperative factors influence the shunt: hypoxic pulmonary vasoconstriction (HPV), gravity (lower decrease in oxygenation in the lateral position), changes in pulmonary vascular resistance (increase in the ipsilateral resistance to blood flow), cardiac output, and double-lumen tube malposition.

HPV is felt by most investigators to be the most important intraoperative variable.[1] A large number of factors (anaesthetic agents, acid/base imbalance, temperature changes, lung manipulation, vasodilators, etc.) can be involved in the magnitude of HPV in the non-ventilated lung. In animal studies, volatile anaesthetics have been shown to impair HPV, and increase intrapulmonary shunt fraction or reduce arterial oxygen tension in a dose–response manner[2–4] whereas propofol does not seem to affect HPV.[5]

Clinical investigations are contradictory regarding the effects of each anaesthetic agent on oxygenation. Two studies found no difference in arterial oxygenation and intrapulmonary shunt between patients receiving propofol or either isoflurane[6] or sevoflurane.[7] Conversely, Kellow and colleagues[8] showed a greater shunt fraction using isoflurane when compared with propofol, whereas Abe and colleagues[9] showed that oxygenation was greater and shunt fraction was lower using propofol than volatile anaesthetics during OLV.

In previous studies, comparison between inhaled and i.v. anaesthetic agents during OLV was made using predetermined doses.[6–10] This may have led to higher drug administration than clinically necessary. Assessment of the clinical relevance of potential inhibition of HPV by the inhaled volatile agent sevoflurane must be carried out using similar clinically effective dosage of i.v. anaesthetic agents. The use of a bispectral index monitor (BIS), or of another anaesthetic depth monitor, is therefore necessary to achieve such an assessment.

The aim of the present study was to compare the effect of sevoflurane, which permits quicker adjustment of depth of anaesthesia than isoflurane, and propofol, as primary anaesthetic agent, on oxygenation during OLV, their administration being adjusted to maintain BIS values between 40 and 60.

Methods

With Ethics Committee approval and written informed consent, 80 consecutive patients undergoing lobectomy requiring OLV were included in a randomized open study. The study population was selected if they met the following criteria: age between 18 and 70 yr, ASA physical status I–III, and indication for thoracic epidural anaesthesia. Patients taking a vasoactive drug at the time of selection (angiotensin converting enzyme inhibitor, angiotensin II-receptor antagonist, calcium channel antagonist, nitrate, sildenafil) were excluded. Other medications, including beta-blockers, were continued as usual. Preoperative cardiac or pulmonary function and persistent tobacco abuse were not taken into account for selection of patients.

Arterial blood gas analysis without oxygen and routine spirometry were performed preoperatively.

The patients received hydroxyzine 100 mg orally 1 h before surgery. In the operating room, a 16 or 18-gauge peripheral i.v. canula was inserted into a large forearm vein and standard monitors were applied (GE Datex-Ohmeda S/5™ Anaesthesia Monitor, Helsinki, Finland). A 20-gauge radial artery canula was inserted for invasive monitoring of blood pressure. A thoracic epidural catheter was inserted under local anaesthesia at the T5–6 or T6–7 interspace; to exclude intravascular or intrathecal positioning of the catheter tip, a test dose of 3 ml of 2% lidocaine with epinephrine was given. A BIS® self-adhesive EEG electrode strip (ZipPrep; Aspect Medical Systems) was positioned on the forehead as recommended by the manufacturer after the skin of the forehead had been carefully wiped with an alcohol swab and allowed to dry. The BIS (BIS version 4.0, XP) was calculated with a smoothing rate of 30 s by the BIS plug-in module of a Datex-Ohmeda S/5 monitor. Electrode impedance was considered acceptable if below 10 kΩ (manufacturers' recommendations).

After preoxygenation, anaesthesia was induced in all patients with i.v. etomidate 0.25 mg kg^{-1} (actual body weight) and sufentanil 0.2 μg kg^{-1}. Atracurium 0.5 mg kg^{-1} was given initially and thereafter adjusted according to train of four monitoring. According to a computer-generated randomization list, patients were assigned to maintenance of anaesthesia with sevoflurane (Group S) or i.v. propofol (Asena® PK pump, Alaris Medical Systems, Hampshire, UK) (Group P) in order to maintain a BIS between 40 and 60. The Asena PK pump permits target controlled delivery of propofol using Schnider's pharmacokinetic model. The patient was excluded from the study if two consecutive BIS values were outside the target range (40–60).

A left-sided double-lumen tube (Broncho-Cath; Mallinckrodt Medical Ltd, Athlone, Ireland) was placed. Its position was checked using a fibreoptic bronchoscope. Ventilatory settings (Julian ventilator, Dräger, Lübeck, Germany) were identical during two-lung ventilation (TLV) and OLV: 6 ml kg^{-1} tidal volume, 12 min^{-1} ventilatory rate, constant flow, inspiratory to expiratory ratio 1:2, and $F_{I_{O_2}}$ 1.

After positioning the patient in lateral decubitus, the correct position of the double-lumen tube was checked again and a 5 ml h^{-1} epidural infusion of a mixture of 0.2% ropivacaine plus 0.5 μg ml^{-1} of sufentanil was initiated.

Fluid administration was limited to 1500 ml Ringer Lactate (500 ml before epidural insertion and 1000 ml during surgery). Additional boluses of i.v. sufentanil were administered as necessary. Intraoperative haemodynamic disturbances were managed as deemed to be clinically necessary by the anaesthetist in charge. Hypotension and bradycardia were treated with i.v. ephedrine and atropine, respectively.

Arterial blood gas analysis (ABL 700 Radiometer®, Copenhagen, Denmark) was first performed 15 min after positioning in the lateral decubitus position during TLV. Then OLV began and the surgeon was allowed to open the chest. Arterial blood gas analysis was performed every 5 min for 40 min during OLV. Sp_{O_2} was continuously monitored. If Sp_{O_2} reached, or decreased below 90%, arterial blood was immediately drawn for gas analysis and TLV was reinstituted. This blood gas sample was used as the patient's lowest Pa_{O_2} for analysis, and no further blood sample was drawn for the study.

Incomplete lung deflation on visual inspection of the surgical field was considered as a criterion for exclusion from the study.

The following variables were also recorded at the same intervals as arterial blood gas analysis: end-tidal carbon dioxide concentration, heart rate, mean arterial pressure, and BIS value. End-tidal concentration of sevoflurane (ET_{sevo}) was noted in Group S and propofol calculated target blood concentration, reported on the Asena PK pump screen, in Group P.

Finally, all patients were visited on the first postoperative day and interviewed about intraoperative awareness using the specific Sebel's score.[11]

The primary outcome of the study was defined as the lowest Pa_{O_2} during the studied period (lowest Pa_{O_2}).

A power analysis (two-tailed α error of 5% and β error of 20%) was performed before the study. The calculation of the required sample size was based on the lowest Pa_{O_2} measured during OLV under propofol anaesthesia in a previous study [16.5 (7.3) kPa][12] and on the experimental HPV inhibition by sevoflurane leading to an increase of 9% of shunt fraction[1 2] which corresponds theoretically to a Pa_{O_2} decrease of 5.3 kPa during FI_{O_2} 1 ventilation.[13] Thirty-one patients per group were required in order to detect a difference of 5.3 kPa in the lowest Pa_{O_2} between groups and we decided to enrol 40 patients per group.

Continuous variables are presented as mean (SD) and extreme values. The Fisher exact test was used to compare qualitative variables, and Student's *t*-test was used to compare continuous variables. From TLV in the lateral decubitus position until the end of the observation period (OLV-40), consecutive measurements were compared using the repeated-measures analysis of variance with Bonferroni's adjustments for multiple comparisons. *Post hoc* analyses were performed with a paired Student's *t*-test (intragroup comparison) or unpaired Student's *t*-test (intergroup comparison). Changes were considered significant at the 5% level ($P<0.05$). Statistical tests were performed using SPSS® version 11.0.5 (SPSS Inc., Chicago, IL, USA).

Results

Eighty patients were enrolled in the study. Fifteen patients were excluded from analysis (seven in Group S and eight in Group P): 12 because of difficulties of lung exclusion (five in Group S and seven in Group P) and three because of two consecutive BIS values outside fixed limits (two in Group S and one in Group P). Statistical analysis was therefore conducted with 65 patients: 33 in Group S and 32 in Group P.

Table 1 Patient characteristics. Values are mean (range), mean (SD) or absolute numbers. Group S: group sevoflurane; Group P: group propofol; FEV_1: forced expired volume in 1 s; FVC: forced vital capacity

	Group S ($n=33$)	Group P ($n=32$)
Demographic data		
Age (yr)	62 (37–68)	57 (18–70)
Sex (M/F)	26/7	25/7
Weight (kg)	74 (13)	69 (12)
Height (cm)	173 (7)	170 (7)
BMI (kg m^{-2})	24.5 (4.0)	24.1 (3.6)
ASA I/II/III (*n*)	3/24/6	2/18/12
Preoperative arterial blood gases		
pH	7.42 (0.04)	7.41 (0.03)
Pa_{O_2} (kPa)	10.4 (1.5)	10.7 (1.7)
Pa_{CO_2} (kPa)	5.1 (0.7)	5.3 (0.4)
HCO_3^- (mEq $litre^{-1}$)	24.3 (2.0)	25.5 (1.7)
Preoperative spirometric tests		
FEV_1 (% of predicted value)	83 (18)	86 (17)
FVC (% of predicted value)	88 (18)	93 (19)
FEV_1/FVC (%)	83 (14)	82 (15)
FEV_1/FVC<70% (*n*)	6	5

The two groups were comparable in terms of age, male/female ratio, weight, height, BMI, preoperative arterial blood gas, and spirometry results (Table 1).

The right lung was ventilated in 21 of the 33 patients in Group S and in 19 of the 32 patients in Group P. Four patients in each group had a Sp_{O_2} below 90%, thus requiring a return to TLV which corrected the Sp_{O_2} levels to above 98%. The lowest Pa_{O_2} were similar: 16.3 (7.5) [range between 8.9 and 29.7] kPa in Group S and 17.7 (9.3) [8.3–37.7] kPa in Group P (Fig. 1B). Pa_{O_2} decreased from TLV to OLV in both groups ($P<0.05$ for each measurement during OLV); Pa_{O_2} was similar in both groups at any time (Table 2, Fig. 1A). Pa_{CO_2} and pH did not change significantly (Table 2); maximal Pa_{CO_2} values were 6.9 kPa in Group S and 7.1 kPa in Group P, minimal pH values were 7.26 in Group S and 7.27 in Group P.

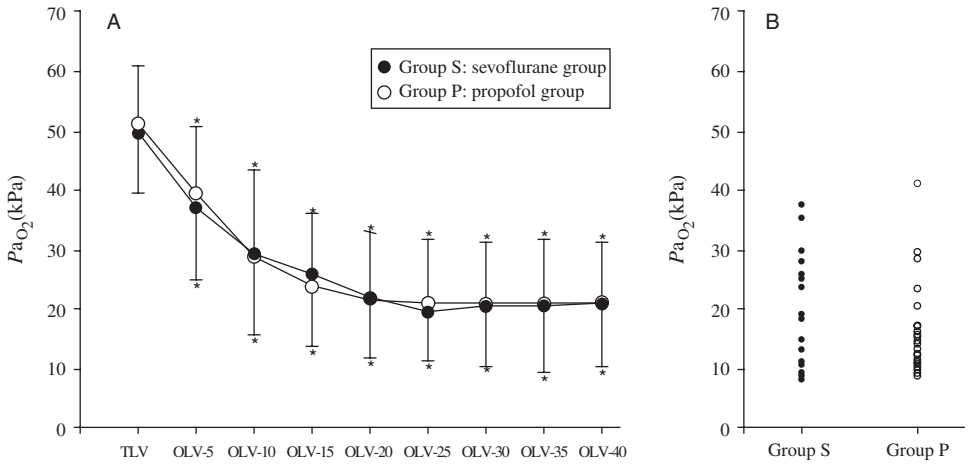

Fig 1 Time-course changes of Pa_{O_2} and individual lowest Pa_{O_2}. (A) Time-course changes of Pa_{O_2} and (B) individual lowest Pa_{O_2} in each group. TLV, two-lung ventilation; OLV-5, 5 min after the initiation of OLV; OLV-10, 10 min after the initiation of OLV; OLV-15, 15 min after the initiation of OLV; OLV-20, 20 min after the initiation of one-lung ventilation; OLV-25, 25 min after the initiation of OLV; OLV-30, 30 min after the initiation of OLV; OLV-35, 35 min after the initiation of OLV; OLV-40, 40 min after the initiation of OLV. *$P<0.05$ within group comparison *vs* TLV.

Table 2 Changes in blood gas analysis, end-tidal carbon dioxide concentration, heart rate, and mean arterial pressure. Values are means (SD). Group S, group sevoflurane; Group P, group propofol; E'_{CO_2}, end-tidal carbon dioxide concentration (kPa); TLV, two-lung ventilation; OLV-5, 5 min after the initiation of OLV; OLV-10, 10 min after the initiation of OLV; OLV-15, 15 min after the initiation of OLV; OLV-20, 20 min after the initiation of OLV; OLV-25, 25 min after the initiation of OLV; OLV-30, 30 min after the initiation of OLV; OLV-35, 35 min after the initiation of OLV; OLV-40, 40 min after the OLV. *$P<0.05$ intragroup comparison *vs* TLV

	TLV	OLV-5	OLV-10	OLV-15	OLV-20	OLV-25	OLV-30	OLV-35	OLV-40
Group S (n=33)									
pH	7.37 (0.04)	7.40 (0.05)	7.40 (0.05)	7.39 (0.05)	7.39 (0.05)	7.39 (0.05)	7.40 (0.05)	7.39 (0.05)	7.40 (0.06)
Pa_{O_2} (kPa)	49.9 (10.4)	37.2 (12)*	29.5 (13.9)*	25.9 (12.1)*	22 (10.1)*	19.7 (8.4)*	20.7 (10.1)*	20.8 (11.3)*	21.1 (10.7)*
Pa_{CO_2} (kPa)	5.6 (0.5)	5.1 (0.5)	5.2 (0.8)	5.3 (0.7)	5.3 (0.8)	5.3 (0.7)	5.3 (0.7)	5.3 (0.5)	5.3 (0.7)
E'_{CO_2} (kPa)	4.3 (0.5)	4.1 (0.7)	4 (0.5)	4 (0.5)	4.1 (0.5)	4.1 (0.5)	4.3 (0.5)	4.3 (0.5)	4.3 (0.4)
HR (beats min^{-1})	70 (17)	72 (16)	71 (17)	70 (17)	74 (16)	79 (16)	78 (16)	78 (16)	78 (16)
MAP (mm Hg)	83 (18)	85 (16)	87 (22)	81 (23)	84 (19)	77 (15)	81 (21)	83 (21)	87 (18)
Group P (n=32)									
pH	7.38 (0.05)	7.40 (0.05)	7.40 (0.05)	7.39 (0.05	7.40 (0.05)	7.39 (0.07)	7.40 (0.05)	7.41 (0.05)	7.40 (0.04)
Pa_{O_2} (kPa)	51.3 (9.7)	39.7 (11.1)*	29.1 (14.5)*	24 (12)*	21.9 (11.3)*	21.2 (10.9)*	21.1 (10.4)*	21.1 (10.8)*	21.3 (10.3)*
Pa_{CO_2} (kPa)	5.6 (0.6)	5.3 (0.7)	5.5 (0.7)	5.5 (0.7)	5.3 (0.8)	5.3 (0.7)	5.3 (0.7)	5.5 (0.7)	5.3 (0.7)
E'_{CO_2} (kPa)	4.1 (0.5)	3.9 (0.5)	4.1 (0.5)	4.1 (0.5)	4.1 (0.5)	4 (0.5)	4 (0.4)	4 (0.4)	4 (0.4)
HR (beats min^{-1})	70 (14)	69 (17)	71 (16)	72 (16)	74 (16)	72 (13)	72 (12)	73 (11)	74 (7)
MAP (mm Hg)	76 (13)	84 (17)	91 (14)	86 (16)	80 (13)	80 (24)	88 (16)	85 (15)	87 (19)

Two patients in Group S and one in Group P received atropine 0.5 mg at the time of epidural puncture for treatment of vasovagal bradycardia. Twenty-nine patients in Group S and 25 patients in Group P required at least one bolus of ephedrine (ns) with similar doses during the study period, 15 (10) and 12 (11) mg, respectively (ns). End-tidal carbon dioxide concentration and haemodynamic parameters did not change significantly from TLV to OLV and were similar between groups (Table 2).

Mean E'_{sevo} was 1.3 (0.3) [0.6–1.7]%. E'_{sevo} was similar throughout the study (Table 3). Mean propofol plasma target concentration was 2.6 (0.9) [1.4–6.1] $\mu g\ ml^{-1}$. Propofol plasma target concentrations were similar throughout the study (Table 3). Nineteen patients in Group S and 18 patients in Group P received one additional sufentanil bolus (ns). Nine patients in Group S and seven patients in Group P received two additional sufentanil boluses (ns). Patients received 11.1 (5.0) and 11.4 (4.2) μg sufentanil in Groups S and P, respectively (ns).

Mean BIS values were 51 (4) in Group S and 46 (8) in Group P (ns). BIS values were similar throughout the studied period in each group and between the two groups (Table 3).

All patients were questioned about intraoperative awareness the day after surgery. Every Sebel's score was below 3, excluding patient awareness.

Discussion

Our study shows that there is no difference in arterial oxygen tension during OLV when sevoflurane or propofol is administered in a BIS-controlled manner.

Volatile anaesthetics, which cause bronchodilatation, are widely used for lung surgery patients but the inhibition of HPV, demonstrated in animal studies,[2–4] can be a major drawback. Previous reports of the effects of inhalation and i.v. anaesthetics on oxygenation during OLV could have been confounded since anaesthetic agents were administered at a predetermined dosage. This is particularly important for halogenated agents due to their concomitant direct and indirect effects on oxygenation. Direct effect is demonstrated in some studies: a large inhaled agent concentration caused increased inhibition of HPV.[8 9] Indirect effect is observed when cardiac output is decreased, improving the effect of HPV on oxygenation.[14] This relation is not valid when cardiac output is very low as arterial oxygenation is impaired by the desaturation of mixed venous blood. Consequently, the administered concentration of a halogenated agent must be adjusted to a clinical parameter. The index of the hypnotic state seems a good candidate since the study aimed to compare an inhalation anaesthetic to an i.v. anaesthetic agent.

Table 3 Time-course changes of end-tidal sevoflurane, propofol target blood concentration, and bispectral index. Values are means (SD). Group S, group sevoflurane; Group P, group propofol; E'_{sevo}, end-tidal concentration of sevoflurane; BIS, bispectral index; TLV, two-lung ventilation; OLV-5, 5 min after the initiation of OLV; OLV-10, 10 min after the initiation of OLV; OLV-15, 15 min after the initiation of OLV; OLV-20, 20 min after the initiation of OLV; OLV-25, 25 min after the initiation of OLV; OLV-30, 30 min after the initiation of OLV; OLV-35, 35 min after the initiation of OLV; OLV-40, 40 min after the initiation of OLV

	TLV	OLV-5	OLV-10	OLV-15	OLV-20	OLV-25	OLV-30	OLV-35	OLV-40
Group S (n=33)									
E'_{sevo} (%)	1.1 (0.3)	1.2 (0.4)	1.4 (0.4)	1.4 (0.4)	1.3 (0.4)	1.3 (0.4)	1.3 (0.3)	1.3 (0.4)	1.3 (0.3)
BIS	50 (5)	48 (4)	47 (5)	46 (5)	47 (4)	48 (5)	48 (4)	47 (5)	49 (4)
Group P (n=32)									
Propofol target blood concentration ($\mu g\ ml^{-1}$)	2.7 (0.8)	2.8 (1.0)	2.7 (1.0)	2.7 (1.1)	2.7 (1.0)	2.6 (1.1)	2.5 (1.1)	2.7 (1.1)	2.6 (0.8)
BIS	49 (6)	47 (4)	46 (6)	44 (5)	45 (6)	47 (7)	47 (5)	48 (7)	47 (6)

For lack of a well-defined clinical endpoint for depth of anaesthesia, we used the BIS to produce seemingly comparable levels of anaesthesia with propofol and sevoflurane. An upper limit at 60 has been validated by studies in which maintaining BIS below 60 decreased the incidence of intraoperative awareness.[15] A lower limit at 40 has been disputed recently as it has been reported that cumulative deep hypnotic time with a BIS lower than 45 is an independent predictor of mortality in the first year after major non-cardiac surgery.[16] However, the range 40–60 for BIS remains widely used and reported by a large number of publications despite large variability and overlap in BIS scores at distinct depths of anaesthesia, which would make differentiation of anaesthetic depth difficult.[17]

Maintaining BIS between 40 and 60 was obtained in our patients with an E'_{sevo} lower than the minimal alveolar concentration (2.05% in pure oxygen). This can be explained by the fact that all patients received an epidural solution of ropivacaine and i.v. sufentanil. The mean E'_{sevo} administered in our patients [1.3 (0.3)%] is lower than that used in all the human studies on the effect of sevoflurane on HPV. Sevoflurane was administered at an *a priori* concentration or mainly at a concentration derived from the MAC, and not adapted to a hypnotic index in the previous studies (Table 4).[7 18–21] As the effect of halogenated agents on oxygenation during OLV depends on their concentration, this difference could explain our results.

We did not find any modification of Pa_{O_2} with time during OLV whereas the time course of HPV has been described in patients.[22] Our study was performed during lung surgery with an active surgeon. Certain surgical manoeuvres can be compared with lung compression which can improve oxygenation.[23] Our interpretation is that many factors are involved during the study period and that one can hide another (e.g. time).

We chose lowest Pa_{O_2} as primary outcome including data from patients with Sp_{O_2} equal to or below 90%. Study was discontinued in these patients but their lowest Pa_{O_2} was used for statistical analysis. This attitude differs from several studies that excluded data of some patients who had a Sp_{O_2} lower than 90% or 91% during OLV.[15 16] The choice of a lower cut-off of Sp_{O_2} for changing from OLV to TLV would probably not have modified our results and we considered it unethical to expose patients to a risk of severe hypoxaemia.

Table 4 Mean end-tidal sevoflurane concentration as reported in the literature

	E'_{sevo} (%)
Wang *et al.*[20]	2.1
Beck *et al.*[7]	1.8
Saito *et al.*[21]	1.7
Shimizu *et al.*[18]	1.7
Abe *et al.*[19]	1.5–2
Present study	1.3

One limitation of our study is the fact that we measured Pa_{O_2} and not arterial oxygen transport. This is of major importance as cardiac output can be modified in such patients by the anaesthetics, the position, and the association of general anaesthesia and thoracic epidural ropivacaine administration. We did not consider the use of invasive monitoring (Swan-Ganz catheter or PICCO®) in patients submitted for lobectomy as this is not our routine practice. Transoesophageal Doppler is not appropriate as a characteristic aortic blood flow signal cannot be obtained in half of the patients in similar conditions.[24] Consequently, we could not have any access to cardiac output evaluation. However, requirements for ephedrine, heart rate, and mean arterial pressure were similar between the two groups. The relatively high rate of ephedrine administration (29/33 in Group S and 25/32 in Group P) is easily comprehensible since epidural infusion of ropivacaine was initiated at the beginning of surgery and since fluid loading was deliberately limited to prevent postoperative pulmonary oedema.

Ventilatory settings used in the study were low tidal volume, zero end-expiratory pressure, and FI_{O_2} 1. A major goal of ventilation is to minimize atelectasis and this explains the approach with large tidal volumes; common teaching is to use similar tidal volumes (10–12 ml kg^{-1}) for both TLV and OLV. But focus has been made recently on the link between high intraoperative ventilation pressure and the risk of postoperative acute lung injury after pulmonary resection. A study of either large (12–15 ml kg^{-1}) or small (6 ml kg^{-1} plus added PEEP 10 cm H_2O) tidal volume ventilation during OLV could not demonstrate differences in inflammatory cytokines in serum or tracheal aspirates,[25] but this study was not powered to evaluate more important outcomes. Quite the opposite result has been reported recently in patients undergoing oesophagectomy.[26] The debate is not closed.[27 28] A second question is the use of PEEP which has been advocated by many authors. Valenza and colleagues[29] reported that PEEP improved oxygenation only in patients with high FEV_1 (above 72% of the predicted value). This report should be compared with Slinger's.[30] Thus, it is now appreciated that the effects of applied PEEP during OLV depend on the lung mechanics of the individual patient. Most patients with chronic obstructive pulmonary disease develop auto-PEEP during OLV and external PEEP can lead to hyperinflation and increased shunt. Conversely, patients with normal lung parenchyma, or those with restrictive lung diseases, tend to fall below their functional residual capacity at end-expiration during OLV and may benefit from applied external PEEP. PEEP application must be discussed in each case and an unconsidered application of PEEP during OLV could lead to a deterioration of arterial oxygenation.[31] As we did not take into account lung function for selection of patients and did not measure auto-PEEP during OLV, we chose not to use PEEP during OLV. This could have increased dependent lung vascular

resistance and consequently increased non-dependent lung perfusion and shunt. Finally, the use of 100% oxygen ventilation during OLV in this study could have caused vasodilatation of the vessels in the dependent lung, increasing their capability to increase dependent lung blood flow due to HPV without increasing pulmonary artery pressure.[1] On the other hand, 100% oxygen could cause absorption atelectasis and therefore increase the shunt.[32] We used pure oxygen because it would limit the number of patients becoming severely hypoxaemic during OLV and thus prevent exclusion of many patients from the study due to requirement of an increase in $F_{I_{O_2}}$.[7]

In conclusion, sevoflurane and propofol had a similar effect on Pa_{O_2} during OLV when their administration was titrated to maintain BIS between 40 and 60. Choice between these anaesthetic agents can be independent of their effects on oxygenation.

References

1 Cohen E. Management of one-lung ventilation. *Anesthesiol Clin North America* 2001; **19**: 475–95

2 Domino KB, Borowec L, Alexander CM, *et al*. Influence of isoflurane on hypoxic pulmonary vasoconstriction in dogs. *Anesthesiology* 1986; **64**: 423–9

3 Ishibe Y, Gui X, Uno H, Shiokawa Y, Umeda T, Suekane K. Effect of sevoflurane on hypoxic pulmonary vasoconstriction in the perfused rabbit lung. *Anesthesiology* 1993; **79**: 1348–53

4 Loer SA, Scheeren TW, Tarnow J. Desflurane inhibits hypoxic pulmonary vasoconstriction in isolated rabbit lungs. *Anesthesiology* 1995; **83**: 552–6

5 Schwarzkopf K, Schreiber T, Preussler NP, *et al*. Lung perfusion, shunt fraction, and oxygenation during one-lung ventilation in pigs: the effects of desflurane, isoflurane, and propofol. *J Cardiothorac Vasc Anesth* 2003; **17**: 73–5

6 Reid CW, Slinger PD, Lenis S. A comparison of the effects of propofol-alfentanil versus isoflurane anesthesia on arterial oxygenation during one-lung ventilation. *J Cardiothorac Vasc Anesth* 1996; **10**: 860–3

7 Beck DH, Doepfmer UR, Sinemus C, *et al*. Effects of sevoflurane and propofol on pulmonary shunt fraction during one-lung ventilation for thoracic surgery. *Br J Anaesth* 2001; **86**: 38–43

8 Kellow NH, Scott AD, White SA, Feneck RO. Comparison of the effects of propofol and isoflurane anaesthesia on right ventricular function and shunt fraction during thoracic surgery. *Br J Anaesth* 1995; **75**: 578–82

9 Abe K, Shimizu T, Takashina M, Shiozaki H, Yoshiya I. The effects of propofol, isoflurane, and sevoflurane on oxygenation and shunt fraction during one-lung ventilation. *Anesth Analg* 1998; **87**: 1164–9

10 Funahara T, Konishi A, Izumi K, Matsuyama H. Effect of low sevoflurane concentration on systemic oxygenation during one-lung ventilation. American Society of Anesthesiology Annual Meeting, 2004, A-1579

11 Sebel PS. Awareness during general anesthesia. *Can J Anaesth* 1997; **44**: R124–30

12 Moutafis M, Liu N, Dalibon N, *et al*. The effects of inhaled nitric oxide and its combination with intravenous almitrine on Pa_{O_2} during one-lung ventilation in patients undergoing thoracoscopic procedures. *Anesth Analg* 1997; **85**: 1130–5

13 Lawler PG, Nunn JF. A reassessment of the validity of the iso-shunt graph. *Br J Anaesth* 1984; **56**: 1325–35

14 Eisenkraft JB. Effects of anaesthetics on the pulmonary circulation. *Br J Anaesth* 1990; **65**: 63–78

15 Myles PS, Leslie K, McNeil J, Forbes A, Chan MT. Bispectral index monitoring to prevent awareness during anaesthesia: the B-Aware randomised controlled trial. *Lancet* 2004; **363**: 1757–63

16 Monk TG, Saini V, Weldon BC, Sigl JC. Anesthetic management and one-year mortality after noncardiac surgery. *Anesth Analg* 2005; **100**: 4–10

17 Anderson RE, Barr G, Assareh H, Jakobsson J. The AAI index, the BIS index and end-tidal concentration during wash in and wash out of sevoflurane. *Anaesthesia* 2003; **58**: 531–5

18 Shimizu T, Abe K, Kinouchi K, Yoshiya I. Arterial oxygenation during one lung ventilation. *Can J Anaesth* 1997; **44**: 1162–6

19 Abe K, Mashimo T, Yoshiya I. Arterial oxygenation and shunt fraction during one-lung ventilation: a comparison of isoflurane and sevoflurane. *Anesth Analg* 1998; **86**: 1266–70

20 Wang JY, Russell GN, Page RD, Jackson M, Pennefather SH. Comparison of the effects of sevoflurane and isoflurane on arterial oxygenation during one lung ventilation. *Br J Anaesth* 1998; **81**: 850–3

21 Saito M, Cho S, Morooka H, Hasuo H, Shibata O, Sumikawa K. Effects of sevoflurane compared with those of isoflurane on arterial oxygenation and hemodynamics during one-lung ventilation. *J Anesth* 2000; **14**: 1–5

22 Ishikawa S. Oxygenation may improve with time during one-lung ventilation. *Anesth Analg* 1999; **89**: 258–9

23 Ishikawa S, Nakazawa K, Makita K. Progressive changes in arterial oxygenation during one-lung anaesthesia are related to the response to compression of the non-dependent lung. *Br J Anaesth* 2003; **90**: 21–6

24 Dalibon N, Moutafis M, Liu N, Law-Koune JD, Monsel S, Fischler M. Treatment of hypoxemia during one-lung ventilation using intravenous almitrine. *Anesth Analg* 2004; **98**: 590–4

25 Wrigge H, Uhlig U, Zinserling J, *et al*. The effects of different ventilatory settings on pulmonary and systemic inflammatory responses during major surgery. *Anesth Analg* 2004; **98**: 775–81

26 Michelet P, D'Journo XB, Roch A, *et al*. Protective ventilation influences systemic inflammation after esophagectomy: a randomized controlled study. *Anesthesiology* 2006; **105**: 911–9

27 Gal TJ. Con: low tidal volumes are indicated during one-lung ventilation. *Anesth Analg* 2006; **103**: 271–3

28 Slinger P. Pro: low tidal volume is indicated during one-lung ventilation. *Anesth Analg* 2006; **103**: 268–70

29 Valenza F, Ronzoni G, Perrone L, *et al*. Positive end-expiratory pressure applied to the dependent lung during one-lung ventilation improves oxygenation and respiratory mechanics in patients with high FEV1. *Eur J Anaesthesiol* 2004; **21**: 938–43

30 Slinger PD, Kruger M, McRae K, Winton T. Relation of the static compliance curve and positive end-expiratory pressure to oxygenation during one-lung ventilation. *Anesthesiology* 2001; **95**: 1096–102

31 Capan LM, Turndorf H, Patel C, Ramanathan S, Acinapura A, Chalon J. Optimization of arterial oxygenation during one-lung anesthesia. *Anesth Analg* 1980; **59**: 847–51

32 Rothen HU, Sporre B, Engberg G, Wegenius G, Hogman M, Hedenstierna G. Influence of gas composition on recurrence of atelectasis after a reexpansion maneuver during general anesthesia. *Anesthesiology* 1995; **82**: 832–42