Laboratory Investigations

Sevoflurane anaesthesia for one-lung ventilation with PEEP to the dependent lung in sheep: effects on right ventricular function and oxygenation

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This study was undertaken to examine the effect of sevoflurane on right ventricular function, the safety of sevoflurane for one-lung ventilation and the effects of PEEP (positive end-expiratory pressure) to the dependent lung in this model using 12 open-chest sheep. Haemodynamic variables, including cardiac output, mean arterial blood pressure, right ventricular pressure and pulmonary arterial pressure, and right ventricular segment shortening (sonomicrometry) were measured. First, animals received 2.0, 3.0 or 4.0% sevoflurane for 20 min each, respectively, during two-lung ventilation to measure the dose-dependent haemodynamic effects of sevoflurane. Then one-lung ventilation was performed with a randomized sequence of 0 (ZEEP), 5 and 10 cm H_2O PEEP to the dependent lung under 2.0% sevoflurane anaesthesia after one-hour stabilization. A decrease in systolic segment shortening along with increases in both the end-diastolic and end-systolic lengths of the right ventricle were observed at 3.0 and 4.0% sevoflurane, while global right ventricular function remained substantially unchanged during two-lung ventilation. During one-lung ventilation the PaO_2 was greater with 5 cm H_2O PEEP 198 mmHg (±25 SEM) than with ZEEP 138 mmHg (±22) or with 10 cm H_2O PEEP 153 mmHg (±23) ($P < 0.05$). No differences in haemodynamic variables or segment shortening between ZEEP and PEEPs during one-lung ventilation were observed. We conclude that although sevoflurane causes a dose-dependent depression of right ventricular function, sevoflurane anaesthesia can be safely applied to one-lung ventilation, and that 5 cm H_2O PEEP to the dependent lung can improve arterial oxygenation without causing changes in right ventricular function.

Ce travail vise à évaluer les effets du sévoflurane sur la fonction ventriculaire droite, sa sécurité en ventilation monopulmonaire et les effets de la pression positive télé-expiratoire (PEEP) sur le poumon inférieur à thorax ouvert chez 12 moutons. On mesure les variables hémodynamiques habituelles: débit cardiaque, pression artérielle moyenne, pression ventriculaire droite, pression artérielle pulmonaire; et par sonomicrométrie, le raccourcissement segmentaire ventriculaire droit. D'abord, les animaux sont anesthésiés au sévoflurane à 2,0, 3,0, or 4,0% pour 20 minutes pendant la ventilation bipulmonaire pour mesurer les effets hémodynamiques du sévoflurane. Ensuite, la ventilation monopulmonaire est initiée avec une séquence randomisée de 0 (ZEEP), 5 et 10 cm H_2O PEEP sur le poumon inférieur et maintenue à une concentration de sévoflurane 2% pour une

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période de stabilisation d'une heure. Une diminution du raccourcissement systolique segmentaire droit et un allongement télédiastolique et télésystolique ventriculaire droit simultanés sont observés sous sévoflurane 3,0 et 4,0%. La fonction ventriculaire droite globale demeure inchangée pendant la ventilation bipulmonaire. Pendant la ventilation monopulmonaire, la PaO_2 est plus élevée sous PEEP 5 cm H_2O (198 mmHg ± 25 SEM) que sous ZEEP (138 mmHg ± 22) ou sous PEEP 10 cm H_2O (153 mmHg ± 23) ($P < 0,05$). On n'observe pas de différences entre les diverses variables hémodynamiques et au regard du raccourcissement segmentaire entre le ZEEP et le PEEP pendant la ventilation monopulmonaire. Nous concluons que bien que le sévoflurane puisse produire une dépression de la fonction ventriculaire droite proportionnelle à la dose, le sévoflurane peut être utilisé avec sécurité pour la ventilation monopulmonaire et qu'une PEEP de 5 cm H_2O appliquée au poumon inférieur peut améliorer l'oxygénation artérielle sans modifier la fonction ventriculaire droite.

One-lung ventilation (OLV) has gained widespread acceptance because of good surgical exposure for thoracic surgery in the lateral decubitus position. It is, however, often associated with a wide range of variability in PaO_2 despite a high inspired oxygen fraction.[1] Maintenance of adequate arterial oxygenation is thus still the most important factor for successful OLV.[2] Except for improper positioning of a double lumen endobronchial tube, perfusion of the collapsed non-dependent lung and inefficient oxygenation in the dependent lung are the two main causes for impairment of arterial oxygenation during OLV.[2] Accordingly, CPAP (continuous positive airway pressure) to the nondependent lung[3,4] and PEEP (positive end-expiratory pressure) to the dependent lung during OLV have been reported to improve arterial oxygenation. The former technique may, however, interfere with surgical manipulation, but with PEEP, on the other hand, surgical procedures would be better facilitated due to the collapsed lung.[5] While an adequate level of PEEP to the dependent lung may reverse tortuosity of perialveolar vessels caused by gravity compression, a high level of PEEP may increase right ventricular afterload due to increased tissue pressure in the dependent lung. This may result in right ventricular dysfunction if it cannot be compensated for by right ventricular function. This possibility remains to be investigated.

Sevoflurane is a new potent inhaled anaesthetic with a blood/gas partition coefficient of 0.6–0.7.[6,7] It is known to have a relatively small left ventricular depressant effect with a vasodilatory function similar to that of isoflurane,[8,9] although its effect on right ventricular function has not been determined. Furthermore, a recent study showed that 1.0 MAC sevoflurane does not inhibit hypoxic pulmonary vasoconstriction in dogs.[10] These characteristics suggest that sevoflurane could also play a potent role for OLV.

We investigated the effects of various concentrations of sevoflurane on right ventricular function and evaluated the effects of PEEP application to the dependent lung during one-lung ventilation under sevoflurane anaesthesia.

Methods

The experimental protocol was approved by the Medical Research Center Committee of Kawasaki Medical School. The experiment was performed using 12 sheep of either sex (weight 30.2 ± 1.6 kg, mean ± SEM). The animals were anaesthetized with intravenous pentobarbital (30 $mg \cdot kg^{-1}$) after overnight fasting. The trachea was intubated and the lungs were ventilated initially at a rate of 15 breaths per minute with a tidal volume of 10 $ml \cdot kg^{-1}$ (Model R10, Aika, Tokyo). The tidal volume was adjusted to obtain normocapnoea ($PaCO_2$ 35–45 mmHg). Vecuronium (8 mg), pentazocine (30 mg) and diazepam (10 mg) were administered *iv*. During the surgical preparation 2% sevoflurane in a mixture of oxygen and nitrous oxide (1:1) was administered from a vaporizer. Ringer's solution was infused at a rate of 5 $ml \cdot kg^{-1} \cdot hr^{-1}$ throughout the experiment. Body temperature was maintained between 37.0–38.0°C with a warming blanket. End-tidal carbon dioxide and sevoflurane concentrations (Anesthetic Agent Monitor, Datex Instrumentarium, Finland) were monitored continuously through a small-bore tube connected to an endotracheal tube.

Surgical preparation

A 7 Fr. polyethylene catheter was inserted into the right femoral artery for mean arterial blood pressure (mAP) measurement and blood sampling. After a tracheostomy was performed, a 37 Fr. left-sided double-lumen endotracheal tube (Broncho-Cath®, Mallinckrodt, NY) was inserted into the *right* bronchus to avoid obstruction of the left upper lobe orifice during left-sided one-lung ventilation for Study 1. The endobronchial cuff was not inflated during surgical preparation and Study 1. Its proper position was confirmed initially by fluoroscopic monitoring and later by visual observation of lung collapse after thoracotomy. A thermodilution catheter with a tip transducer (7.5 Fr. PT-157J, Goodtec, Huntington Beach, CA) was inserted via a branch of the left external jugular vein into the pulmonary artery for determination of cardiac output (CO), and pulmonary arterial pressure (PAP). Under fluoroscopic monitoring, a 6 Fr. double micromanometer catheter (Gaeltec, U.K.) mounted at the catheter tip and at 5 cm from the tip was advanced via a branch of the right external jugular vein into the right ventricle to measure right ventricular pressure (RVP) and mean right atrial pressure (mRAP). The time derivative

of RVP (RV dP/dt) was obtained with an electronic differentiator (EG-601G, Nihon Kohden, Tokyo, Japan).

Animals were placed in the lateral decubitus position. A thoracotomy was performed in the fifth right intercostal space and the pericardium was incised. Two pairs of piezoelectric crystals (CSL 2100, Sonotek, San Diego, CA), 1.5–2.0 mm in diameter, were implanted in the subendocardium of the outflow and inflow areas of the right ventricular free wall, parallel to the short axis of the heart, and connected to ultrasonic amplifiers (AP-601G, Nihon Kohden), which were synchronized with the pressure amplifiers of the double micromanometer to avoid interference with each other. The segmental lengths of the outflow and inflow regions were measured continuously by the sonomicrometry technique. The sonomicrometry signals, RVP, RV, dP/dt and arterial blood pressure signals were stored in a multichannel magnetic cassette tape recorder (MR-30, TEAC, Tokyo, Japan). The data were digitized at a sampling rate of 200 Hz by a 12-bit, simultaneous eight-channel analogue to digital converter (AZI-273, Interface, Hiroshima, Japan) over a period of five seconds and stored for subsequent analysis by a personal computer (PC-286V-STD, Epson, Tokyo, Japan). Arterial and mixed venous blood samples were analyzed with a blood gas analyzer (IL 1304 pH and Blood Gas Analyzer, Instrumentation Laboratory Lexington, MA) for pH, PCO_2 and PO_2, and with a CO-oximeter (IL 382, Instrumentation Laboratories) for haemoglobin concentrations and oxygen saturations.

Protocol

After the completion of surgical preparations, nitrous oxide was discontinued and at least 60 min were allowed with an end-tidal anaesthetic concentration of 2.0% sevoflurane in oxygen before starting measurements. Each animal was studied for the dose-response haemodynamic effects of sevoflurane under two-lung ventilation, and the effects of PEEP to the dependent lung under OLV.

STUDY 1

The dose-response effects of sevoflurane were studied with increasing concentrations of 2.0, 3.0 and 4.0% end-tidal sevoflurane in oxygen. Two-lung ventilation was performed with the endobronchial cuff deflated. At each sevoflurane concentration, 20 min at a steady state was allowed before measurements. Equilibration at 2.0% sevoflurane was allowed for 60 min to ensure haemodynamic stability prior to Study 2.

STUDY 2

This study was performed using 2% sevoflurane anaesthesia. After carrying out haemodynamic measurements during two-lung ventilation, OLV was begun. VEntilatory separation of the two lungs was performed by inflation of the endobronchial cuff located in the right lung. Three levels of PEEP, 0, 5 and 10 cm H_2O (ZEEP, $PEEP_5$, $PEEP_{10}$, respectively) were applied for 30 min in a randomized sequence to the dependent left lung by submerging the expiratory tube to an appropriate depth below water. The right lung was allowed to collapse with its bronchial lumen open to the atmosphere. Tidal volume was reduced to two-thirds of that during two-lung ventilation, and $PaCO_2$ was maintained between 35–45 mmHg by adjusting the respiratory rate. Haemodynamic measurements, except for the determination of CO, were obtained while ventilation was stopped at end-expiration.

Calculation

Systolic segment shortening (SS) was defined as:

$$SS = \{(\text{end-diastolic length}) - (\text{end-systolic length})\}/(\text{end-diastolic length}) \times 100,$$

where end-diastolic length and end-systolic length were measured at the peak positive RV dP/dt and at the peak negative RV dP/dt, respectively.[11] The SSs of the inflow (iSS) and outflow (oSS) areas of the right ventricular free walls were calculated.

Physiological shunt ($\dot{Q}s/\dot{Q}t$) was calculated as:

$$\dot{Q}s/\dot{Q}t = (Cc'O_2 - CaO_2)/(Cc'O_2 - CvO_2) \times 100,$$

where $Cc'O_2$, CaO_2, CvO_2 are oxygen contents in pulmonary capillary, systemic arterial and mixed venous blood, respectively. These were calculated from the haemoglobin, oxygen saturation and PaO_2 in the animals' blood.[1]

Statistical analysis

The data are presented as the mean ± the standard error of the mean (SEM). Statistical analysis was performed by one-way analysis of variance for repeated measures (ANOVA). When the ANOVA showed a significant difference in treatment, comparisons were made by Fisher's LSD test. A P value of <0.05 was considered statistically significant. In Study 2, statistical comparisons were made among the three levels of PEEP.

Results

Study 1 (Dose-response haemodynamic effects of sevoflurane under two-lung ventilation)

The effects of sevoflurane on haemodynamic variables and a right ventricular function are summarized in Table I. Sevoflurane decreased mAP in a dose-dependent manner, while CO remained unchanged. There was a slight decrease in the HR at 4.0% sevoflurane compared with that at 2.0% sevoflurane. Although there was no difference in PAPs, RVPs was slightly less at 4.0% sevoflurane than at 2.0% sevoflurane. Both iSS and oSS

TABLE I Haemodynamic effects of sevoflurane during two-lung ventilation

	Sevoflurane		
Variables	*2.0%*	*3.0%*	*4.0%*
HR (beats · min^{-1})	108 ± 4‡	104 ± 4	102 ± 4*
CO (L · min^{-1})	2.9 ± 0.2	2.7 ± 0.3	2.7 ± 0.3
mAP (mmHg)	80.5 ± 3.9‡	77.7 ± 4.1‡	68.2 ± 3*†
PAPs (mmHg)	21.4 ± 1.2	21.8 ± 1.0	21.8 ± 1.5
PAPd (mmHg)	15.4 ± 4.0	14.8 ± 1.2	15.6 ± 0.9
RVPs (mmHg)	21.9 ± 1.2‡	21.2 ± 0.8	20.3 ± 0.7*
RVEDP (mmHg)	1.7 ± 0.4	1.9 ± 0.4	1.8 ± 0.4
mRAP (mmHg)	1.6 ± 0.4†‡	2.1 ± 0.4*	2.5 ± 0.5*
iSS (%)	18.5 ± 2.2‡	16.6 ± 2.2	14.6 ± 2*
oSS (%)	15.4 ± 0.8†‡	12.9 ± 1.0*	12.2 ± 1.0*

Mean ± SEM (n = 12).
Abbreviations. mAP: mean aortic pressure, CO: cardiac output, HR: heart rate, PAPs: systolic pulmonary arterial pressure, PAPd: diastolic pulmonary pressure, RVPs: systolic right ventricular pressure, RVEDP: right ventricular end-diastolic pressure, mRAP: mean right atrial pressure, iSS: systolic segment shortening of the right ventricular inflow region, oSS: systolic segment shorting of the right ventricular outflow region.
*Significantly ($P < 0.05$) different from 2% sevoflurane.
†Significantly ($P < 0.05$) different from 3% sevoflurane.
‡Significantly ($P < 0.05$) different from 4% sevoflurane.

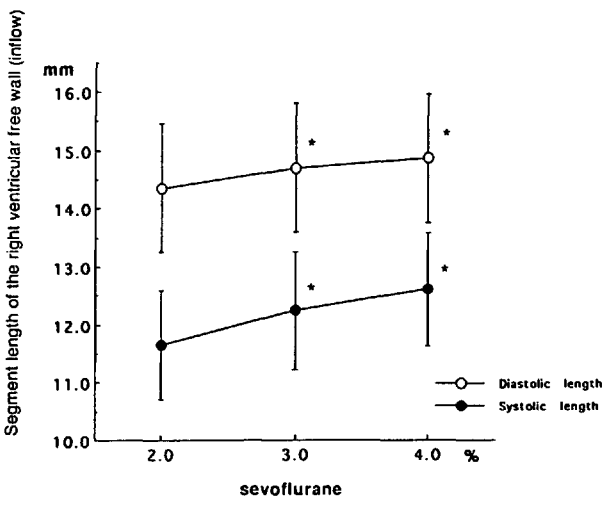

FIGURE Effects of sevoflurane on the segment length of the right ventricular free wall (inflow region) (n = 12). Sevoflurane increased both the systolic and diastolic segment lengths of the inflow area of the right ventricular free wall in a dose-dependent manner. Systolic segment shortening decreased with increasing sevoflurane concentrations (Table I). The systolic and diastolic segment length of the outflow region showed a similar change. Values are presented as mean ± SEM. *Significantly ($P < 0.05$) different from 2% sevoflurane.

decreased with increasing concentrations of sevoflurane. Both end-diastolic length and end-systolic length were also greater at 3.0 and 4.0% sevoflurane than at 2.0% sevoflurane (Figure).

Study 2 (One-lung ventilation with ZEEP, $PEEP_5$ and $PEEP_{10}$)

Haemodynamic values returned to near base-line values after one-hour stabilization at 2% sevoflurane, as seen in the Tables I and II. There were no differences in HR, CO and mAP among the three levels of PEEP. The PAPs and RVPs were greater at $PEEP_{10}$ than at ZEEP or $PEEP_5$. But, PEEP had no effect on iSS or oSS (Table II).

The PaO_2 was greater at $PEEP_5$ (198 ± 25 mmHg) than at ZEEP (138 ± 22 mmHg). Corresponding to the difference in PaO_2, the $\dot{Q}s/\dot{Q}t$ was less at $PEEP_5$ than that at ZEEP. There were no differences in PaO_2 between ZEEP and $PEEP_{10}$, although $\dot{Q}s/\dot{Q}t$ was less during $PEEP_{10}$ than during ZEEP.

Discussion

We selected sheep as the animal model for OLV. In the present study, the left endobronchial tube was inserted into the right bronchus in order to avoid left upper lung atelectasis during left OLV, since the distance between the carina and the upper lobe orifice is close even in sheep. This animal model thus permitted ventilatory separation of the two lungs with a commercially available double lumen endobronchial tube, which is very difficult in dogs.[12]

In Study 1, sevoflurane was administered with increasing concentrations, and not in a randomized sequence. Hence, this design is accompanied by time-effects. However, we believe that statistical comparison of the three concentrations of sevoflurane is still valid, because the absence of any time effect on contractility for up to two hours has been established with other volatile anaesthetics such as halothane and enflurane.[13] Animals were exposed to sevoflurane for a short time (20 min) at each concentration.

The minimum alveolar concentration (MAC) of sevoflurane in sheep has not been determined. Assuming the ratio of isoflurane and sevoflurane is constant across species as proposed by Drummond[14] and Scheller *et al.*[15] the MAC in sheep for sevoflurane should be 2.85% based on the MAC values for isoflurane in sheep (1.58%)[16] and in rabbits (2.05%),[14] and for sevoflurane in rabbits (3.70%).[15] The concentrations of sevoflurane, 2.0%, 3.0% and 4.0%, tested in the present study correspond therefore to 0.70, 1.05 and 1.40 MAC, respectively.

The results of the present study showed that sevoflurane in concentrations of 2.0–4.0% was associated with dose-dependent arterial hypotension, while CO remained

TABLE II Effects of sevoflurane on haemodynamic variables and an oxygenation during two-lung ventilation and one-lung ventilation with PEEP.

Variables	Two-lung ventilation	One-lung ventilation		
		ZEEP	$PEEP_5$	$PEEP_{10}$
HR (beats · min^{-1})	106 ± 6	108 ± 4	101 ± 4	98 ± 8
CO (L · min^{-1}	2.9 ± 0.3	3.2 ± 0.3	3.2 ± 0.4	2.9 ± 0.4
mAP (mmHg)	75.6 ± 3.4	73.9 ± 3.7	73.2 ± 4.4	77.6 ± 4.6
PAPs (mmHg)	23.01 ± 1.7	22.3 ± 1.4	21.9 ± 1.7‡	24.5 ± 1.6†
PAPd (mmHg)	13.6 ± 1.4	14.9 ± 1.1	14.5 ± 1.4	15.6 ± 1.0
RVPs (mmHg)	23.1 ± 1.4	21.8 ± 1.0‡	22.6 ± 1.1‡	24.9 ± 1.5*†
RVEDP (mmHg)	1.4 ± 0.6	2.0 ± 0.6	2.5 ± 0.6	2.2 ± 0.6
mRAP (mmHg)	2.0 ± 0.5	1.9 ± 0.4	1.8 ± 0.4	2.3 ± 0.4
iSS (%)	17.9 ± 2.3	17.8 ± 2.5	17.4 ± 1.9	17.2 ± 1.9
oSS (%)	14.3 ± 1.4	14.7 ± 1.2	13.9 ± 1.2	13.2 ± 1.4
PaO_2 (mmHg)	297 ± 29	138 ± 22†	198 ± 25*	153 ± 23
$PaCO_2$ (mmHg)	41 ± 1	40 ± 1	39 ± 1	41 ± 2
$\dot{Q}s/\dot{Q}t$(%) $n = 8$	32.0 ± 2.3	36.8 ± 4.1†	33.8 ± 4.8*	34.0 ± 3.7*

Mean ± SEM ($n = 12$ except for $\dot{Q}s/\dot{Q}t$). PEEP (positive end-expiratory pressure) was applied to the dependent lung, while the non-dependent lung was allowed to collapse.
Abbreviations. mAP: mean aortic pressure, CO: cardiac output, HR: heart rate, PAPs: systolic pulmonary arterial pressure, PAPd: diastolic pulmonary pressure, RVPs: systolic right ventricular pressure, RVEDP: right ventricular end-diastolic pressure, mRAP: mean right atrial pressure, iSS: systolic shortening of the right ventricular inflow region, oSS: systolic shortening of the right ventricular outflow region. $\dot{Q}s/\dot{Q}t$ was calculated from the data of eight animals due to technical failure to collect the mixed venous blood. Statistical comparisons were performed among the data of one-lung ventilations.
*Significantly ($P < 0.05$) different from ZEEP.
†Significantly ($P < 0.05$) different from $PEEP_5$.
‡Significantly ($P < 0.05$) different from $PEEP_{10}$.

unchanged. These findings indicate that the decrease in arterial blood pressure was mainly an effect of vasodilatation rather than of myocardial depression. The systemic haemodynamic effects are thus similar to those of isoflurane, as reported by Bernard J-M *et al.*[9] in chronically instrumented dogs.

To the best of our knowledge, the effects of sevoflurane on right ventricular contractility have not been determined. We found no changes in the global right ventricular variables except for a slight but significant decrease in RVPs and an increase in mRA at 4.0% sevoflurane. On the other hand, decreases in iSS and oSS along with increases in both end-systolic length and end-diastolic length occurred at 3.0 and 4.0% sevoflurane in comparison with those values at 2.0% sevoflurane. However, both afterload; i.e., PAPs and PAPd, and preload; i.e., RVEDP remained unaltered. These data indicate that the contractility of the right ventricle begins to be depressed by sevoflurane at a concentration of 3.0%, while no substantial changes in the global variables of the right ventricular haemodynamic variables occur. The effects of other halogenated volatile anaesthetics on right ventricular systolic segment shortening are currently unknown.

Although most volatile anaesthetics inhibit hypoxic pulmonary vasoconstriction to some extent, it has been demonstrated that isoflurane and halothane can be used safely for OLV along with an efficacy of CPAP to the nondependent lung.[17] The safety of sevoflurane for OLV has not, however, been examined. The results of the present study show that oxygenation is relatively well preserved during OLV under sevoflurane anaesthesia, and that OLV with $PEEP_5$ achieves an improvement in arterial oxygenation without causing haemodynamic changes, including systolic segment shortening of the right ventricle.

In the lateral decubitus position, the dependent lung receives an increased amount of blood flow due to the effect of gravity, while its lung volume is decreased by compression of the mediastinal weight and cephalad displacement of the diaphragm. This results in inefficient oxygenation in the dependent lung due to low $\dot{V}/\dot{Q}$ and shunt flow.[18] Application of PEEP to the dependent lung should theoretically improve oxygenation by restoring the lung volume and dimishing the low $\dot{V}/\dot{Q}$ area, so long as the PEEP does not interfere with pulmonary perfusion in the dependent lung by increasing the pulmonary tissue pressure.

In their clinical study Capan *et al.*[19] reported that $PEEP_{10}$ applied to the dependent lung did not improve arterial oxygenation, but arterial oxygenation was optimized during OLV by 10 cmH_2O CPAP. Likewise, Cohen *et al.*[20] showed that $PEEP_{10}$ to the dependent lung did not cause any increases in PaO_2, whereas 10 cmH_2O CPAP to the nondependent lung alone or their combination was asssociated with increased PaO_2. In the present study, $PEEP_5$ caused an improvement in arterial oxygenation, as indicated by increased PaO_2 and decreased $\dot{Q}s/\dot{Q}t$, but $PEEP_{10}$ failed to do so. Besides the improvement in arterial oxygenation, no differences in haemodynamic variables between ZEEP and $PEEP_5$ were observed. Therefore, $PEEP_5$ is considered to be optimal in terms of pulmonary oxygen exchange and right ventricular haemodynamics in this model. The ineffectiveness of $PEEP_{10}$ to improve oxygenation in our study and theirs may be explained by reduction in pulmonary perfusion to the dependent lung due to the increased pulmonary tissue pressure.[21] The increase in PAPs during $PEEP_{10}$ may support this explanation.

In summary, the present study demonstrated that sevoflurane has a slight depressant effect on right ventricular function in concentrations of 2.0–4.0%, as evidenced by systolic segment shortening, while global right ven-

tricular variables were relatively well preserved. In addition, arterial oxygenation was maintained during one-lung ventilation and sevoflurane anaesthesia. Furthermore, application of 5 cmH_2O PEEP to the dependent lung is advocated to improve arterial oxygenation during OLV with little change in right ventricular function including segment shortening of the right ventricle.

References

1 *Katz JA, Laverne RG, Fairley HB, Thomas AN.* Pulmonary oxygen exchange during endobronchial anesthesia: effect of tidal volume and PEEP. Anesthesiology 1982; 56: 164–71.

2 *Benumof JL.* One-lung ventilation and hypoxic pulmonary vasoconstriction: implications for anesthetic management. Anesth Analg 1985; 64: 821–33.

3 *Hughes SA, Benumof JL.* Operative lung continuous positive airway pressure to minimize FIO_2 during one-lung ventilation. Anesth Analg 1990; 71: 92–5.

4 *Obara H, Tanaka O, Hoshimo Y, Kaetsu H, Maekawa N, Iwai S.* One lung ventilation: the effect of positive end expiratory pressure to the nondependent and dependent lung. Anaesthesia 1986; 41: 1007–10.

5 *Marshall BE.* Anesthesia for one-lung ventilation (Letter). Anesthesiology 1988; 69: 630–1.

6 *Wallin RF, Regan BM, Napoli MD, Stern IJ.* Sevoflurane: a new inhalational anesthetic agent. Anesth Analg 1975; 54: 758–66.

7 *Strum DP, Eger EI II.* Partition coefficients for sevoflurane in human blood, saline, and olive oil. Anesth Analg 1987; 66: 654–6.

8 *Kazama T, Ikeda K.* The comparative cardiovascular effects of sevoflurane with halothane and isoflurane. Masui 1988; 2: 63–8.

9 *Bernard J-M, Wouters PF, Doursout M-F, Florence B, Chelly JE, Merin RG.* Effects of sevoflurane and isoflurane on cardiac and coronary dynamics in chronically instrumented dogs. Anesthesiology 1990; 72: 659–62.

10 *Okutomi T, Ikeda K.* Sevoflurane has no inhibitory effect on hypoxic pulmonary vasoconstriction (HPV) in dogs. Masui 1990; 4: 123–30.

11 *Morris JJ, Pellom GL, Hamm DP, Everson CT, Wechsler AS.* Dynamic right ventricular dimension: relation to chamber volume during the cardiac cycle. J Thorac Cardiovasc Surg 1986; 91: 879–87.

12 *Muneyuki M, Konishi K, Horiguchi R, et al.* Effects of altering lung ventilation on cardiopulmonary functions in dogs. Anesthesiology 1983; 58: 353–6.

13 *Mote PS, Pruett JK, Gramling ZW.* Effects of halothane and enflurane on right ventricular performance in hearts of dogs anesthetized with pentobarbital sodium. Anesthesiology 1983; 58: 53–60.

14 *Drummond JC.* MAC for halothane, enflurane and isoflurane in the New Zealand white rabbit: and a test for the validity of MAC determinations. Anesthesiology 1985; 62: 336–8.

15 *Scheller MS, Saidman LJ, Partridge BL.* MAC of sevoflurane in humans and the New Zealand white rabbit. Can J Anaesth 1988; 35: 153–6.

16 *Palahniuk RJ, Shnider SM, Eger EI II.* Pregnancy decreases the requirement for inhaled anesthetic agents. Anesthesiology 1974; 41: 82–5.

17 *Benumof JL, Augustine SD, Gibbons JA.* Halothane and isoflurane only slightly impair arterial oxygenation during one-lung ventilation in patients undergoing thoracotomy. Anesthesiology 1987; 67: 910–5.

18 *Benumof JL.* Isoflurane anesthesia and arterial oxygenation during one-lung ventilation. Anesthesiology 1986; 64: 419–22.

19 *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.

20 *Cohen E, Eisenkraft JB, Thys DM, Kirschner PA, Kaplan JA.* Oxygenation and hemodynamic changes during one-lung ventilation: effects of $CPAP_{10}$, $PEEP_{10}$, and $CPAP_{10}$/$PEEP_{10}$. Journal of Cardiothoracic Anesthesia 1988; 2: 34–40.

21 *Benumof JL.* One-lung ventilation: which lung should be PEEPed? Anesthesiology 1982; 56: 161–3.