Effects of head-up tilt on intrapulmonary shunt fraction and oxygenation during 1-lung ventilation in the lateral decubitus position

Sung Yong Park, MD,^a Dae Hee Kim, MD,^a Jin Soo Kim, MD, PhD,^a Sung Soo Lee, MD,^b and Yong Woo Hong, MD, PhD^a

Objective: Hypoxemia is a common problem of 1-lung ventilation. Arterial oxygenation progressively decreases after 1-lung ventilation. The surgical position influences the shunt and arterial oxygenation. Therefore we evaluated the effect of head-up tilt on intrapulmonary shunt and oxygenation during 1-lung ventilation in the lateral decubitus position.

Methods: Twenty patients requiring 1-lung ventilation were included in this study. During 1-lung ventilation, hemodynamic and respiratory variables were measured 15 minutes after horizontal positioning in the lateral decubitus position (baseline), 5 and 10 minutes after a 10-degree head-up tilt (T5 and T10, respectively), and 10 minutes after the patient was returned to a horizontal position (T20). Arterial and mixed venous blood analyses were performed at the same time points.

Results: Arterial oxygenation was increased, and shunt was decreased significantly during head-up tilt position in 1-lung ventilation. These changes were accompanied by decreases in the mean arterial pressure and cardiac filling pressures without significant changes in cardiac index.

Conclusions: Head-up tilt during 1-lung ventilation in the lateral decubitus position caused a significant decrease in shunt and an increase in arterial oxygenation that persisted after the patient was returned to the horizontal lateral decubitus position. (J Thorac Cardiovasc Surg 2010;139:1436-40)

During thoracic surgery requiring 1-lung ventilation (OLV), deterioration of oxygenation is a major problem.^{1,2} The lateral decubitus position causes compression of the dependent ventilated lung by the abdominal contents against the paralyzed diaphragm, which results in increased intrapulmonary shunt and dangerous hypoxemia.³⁻⁶

A previous study demonstrated that 10° head-down tilt for 10 minutes during OLV in the lateral decubitus position caused a significant increase in shunt and a decrease in Pao₂ values.⁷ In clinical situations, especially during robotic or endoscopic thoracic surgery, it is necessary to place the patients in the head-up tilt position for surgical exposure. How this position affects hemodynamic and respiratory variables and therefore influences cardiopulmonary function during surgical intervention has not been clarified. Accordingly, we evaluated the effect of head-up tilt on intrapulmonary shunt, oxygenation, and hemodynamic variables during OLV in the lateral decubitus position.

MATERIALS AND METHODS

Twenty unpremedicated adult patients scheduled for elective thoracic surgery requiring OLV were included in this study. All patients provided informed consent, and approval was obtained from the institutional review board before conducting this study. Patients with coronary artery occlusive disease, cerebrovascular disease, and preoperative forced expiratory volume in 1 second and forced vital capacity of less than 80% of the predicted value were excluded from this study.

Patients' demographics and clinical findings are presented in Table 1. All surgical interventions that were performed were lobectomies (left/right, 9/11; thoracotomy/thoracoscopy, 15/5). The lobes removed were the left upper lobe (5 patients), left lower lobe (4 patients), right upper lobe (3 patients), right middle lobe (4 patients), and right lower lobe (4 patients). None of the patients demonstrated dangerous levels of hypoxemia or hypotension requiring vasoactive drugs, and the study was successfully performed in all patients. The mean duration of pulmonary function testing to the day of thoracic surgery was 11.5 days (range, 2-40 days).

On arrival at the operating room, patients received routine monitoring consisting of 3-lead electrocardiographic analysis, pulse oximetry, and non-invasive blood pressure measurement. Anesthesia was induced with 5 mg/kg thiopental and 2 µg/kg fentanyl. Endobronchial intubation with a left-sided, double-lumen tube was facilitated with 0.1 mg/kg vecuronium, and the position of the double-lumen tube was verified with a fiberoptic bronchoscope. After the induction of anesthesia, a radial artery catheter was placed and a pulmonary artery catheter (Swan-Ganz CCOmbo CCo/Svo₂; Edwards Lifesciences LLC, Irvine, Calif) was inserted through the right internal jugular vein. The patients' lungs were ventilated with a tidal volume of 8 to 10 mL/kg, an inspiratory/expiratory ratio of 1:1.9, and an inspiratory pause of 10% of the total inspiration time at a rate of 8 to 12 breaths/min in 50% oxygen. The accordance of Paco₂ with end-tidal CO₂ was confirmed, and the respiratory rate was adjusted to maintain the Paco₂ within 33 to 38 mm Hg and the pH at approximately 7.4. Anesthesia was maintained with sevoflurane (0.8 vol% to 1 vol%), continuous infusion of remifentanyl (0.05-0.2 µg · kg⁻¹ · min⁻¹), and vecuronium (1-2 µg · kg⁻¹ · min⁻¹).

From the Departments of Anesthesiology and Pain Medicine^a and Thoracic and Cardiovascular Surgery,^b Ajou University College of Medicine, Suwon, Korea. Disclosures: None.

Received for publication March 2, 2009; revisions received June 16, 2009; accepted for publication July 16, 2009; available ahead of print Aug 26, 2009.

Address for reprints: Yong Woo Hong, MD, PhD, Department of Anesthesiology and Pain Medicine, Ajou University College of Medicine, San 5, Wonchon-dong, Yeongtong-gu, Suwon 443-721, Korea (E-mail: ywhong7292@gmail.com).

0022-5223/\$36.00

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doi:10.1016/j.jtcvs.2009.07.041

Abbreviations and Acronyms

- A-aO₂ = alveolar–arterial O₂ gradient
- CI = cardiac index
- HPV = hypoxic pulmonary vasoconstriction
- MAP = mean arterial pressure
- OLV = 1-lung ventilation
- PCWP = pulmonary capillary wedge pressure
- PVRI = pulmonary vascular resistance index
- SVRI = systemic vascular resistance index
- V/Q = ventilation–perfusion

Patients were turned to the lateral decubitus position, after which OLV was initiated with 100% oxygen, and the tidal volume and respiratory rate were adjusted within the predefined range to maintain a peak airway pressure of less than 40 cm H₂O and normocarbica. After the lung resection was completed but before the chest wall closure and inflation of the residual lung were started, the patients were maintained in the horizontal decubitus position. Fifteen minutes later, the patients were positioned in a 10° head-up tilt for 10 minutes and then returned to the horizontal decubitus position. Pressure transducers were located at the level of the right atrium during all phases of the study and were recalibrated after each position change. Arterial blood samples and mixed venous blood samples were measured with an automated blood gas analyzer (CCX Analyzer; Nova Biomedical Corporation, Waltham, Mass).

Hemodynamic variables, arterial and mixed venous blood gas analyses, peak airway pressure, and dynamic pulmonary compliance were recorded at the following points: 15 minutes after the lung resection was completed (baseline), 5 and 10 minutes after 10° head-up tilt (T5 and T10, respectively), and 10 minutes after the patient was returned to the horizontal decubitus position (T20). Hemodynamic measurements included heart rate, mean arterial pressure (MAP), central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), and cardiac index (CI). The systemic vascular resistance index (SVRI), pulmonary vascular resistance index (PVRI), oxygen content in arterial and mixed venous blood, alveolar–arterial O₂ gradient (A-aO₂), and intrapulmonary shunt were calculated at the same time points.

The following calculations were performed⁸:

1. Artery oxygen content ($Cao_2[mL/dL]$) = $(1.36 \times \text{Hemoglobin} \times Sao_2) + (0.0031 \times Pao_2)$
2. Mixed venous oxygen content ($Cvo_2[mL/dL]$) = $(1.36 \times \text{Hemoglobin} \times Svo_2) + (0.0031 \times Pvo_2)$
3. Pulmonary capillary blood oxygen content ($Cco_2[mL/dL]$) = $(1.36 \times \text{Hemoglobin} \times 100\%) + (0.0031 \times \text{alveolar oxygen pressure}[PAo_2])$
4. PAo_2 (mm Hg) = Fraction of inspired oxygen (FIo_2) $\times$ ($P_{atm} - PH_2O$) $- PaCO_2 / 0.8$,

where P_{atm} = 760 mm Hg and PH_2O = 47 mm Hg

5. Shunt fraction ($Qs / Qt[\%]$) = $(Cco_2 - Cao_2) / (Cco_2 - Cvo_2) \times 100$
6. A-aO₂ (mm Hg) = ($PAo_2 - Pao_2$)
7. CI ($L \cdot \text{min}^{-1} \cdot m^{-2}$) = Cardiac output/Body surface area
8. SVRI ($\text{dynes} \cdot s \cdot \text{cm}^{-5} \cdot m^2$) = $(MAP - CVP) \times 80 / CI$
9. PVRI ($\text{dynes} \cdot s \cdot \text{cm}^{-5} \cdot m^2$) = $(\text{Mean pulmonary artery pressure} [MPAP] - PCWP \times 80 / CI)$

This study was designed to terminate if the MAP decreased by more than 20% of the postinduction value, if vasoactive drug administration was

TABLE 1. Patient characteristics (n = 20)

Age (y)	58.5 ± 9.6
Sex (M/F)	16/4
BSA (m ²)	1.7 ± 0.1
FEV ₁ (% predicted)/FVC (% predicted)	93.3 ± 10.2
Operative time (min)	160.7 ± 81.5

Values are shown as mean ± standard deviations or the number of patients. BSA, Body surface area; FEV₁, forced expiratory volume in 1 second; FVC, forced vital capacity.

required, if the oxygen saturation as measured with pulse oximetry decreased to less than 90%, or if the Pao₂ values decreased to less than 60 mm Hg. In a previous study evaluating the effect of head-down tilt during OLV in the lateral decubitus position in patients undergoing thoracic surgery, the number of patients required to detect a 10% difference in percentage change of Pao₂ from the baseline value with an α value of .05 and a β value of .2 was 17.⁷

Statistical analyses were performed with SPSS software 12.0 (SPSS Inc, Chicago, Ill). All data are expressed as means ± standard deviations or the number of patients. We examined subjects' responses to head-up tilt by using repeated-measures analysis of variance with contrast. Contrast comparisons (baseline to after the position change) were performed at each time point and used Greenhouse–Geisser Epsilon as a correction factor for within-subject tests. The rejection level for the pairwise comparison was .05.

RESULTS

Comparisons of hemodynamic variables with baseline values revealed that the MAP and PCWP values at T5 (−6% and −36%, $P = .041$ and $<.001$) and T10 (−8% and −34%, $P = .002$ and $<.001$) were significantly decreased compared with baseline values and returned to the baseline value at T20. CVP values at T5 (−66%, $P < .001$), T10 (−67%, $P < .001$), and T20 (−21%, $P = .02$) were significantly decreased compared with baseline values. Heart rate, CI, SVRI, and PVRI values showed no significant changes during the study period (Table 2).

When the respiratory variables were compared with the baseline values, the Pao₂ value was significantly increased at T10 (+9%, $P = .022$) and T20 (+15%, $P = .009$). Shunt was decreased at T5 (−17%, $P = .038$), T10 (−24%, $P = .01$), and T20 (−19%, $P = .017$) significantly. Peak airway pressure was significantly decreased at T10 (−3%, $P = .006$). A-aO₂ and dynamic pulmonary compliance did not show any significant changes from baseline values (Table 3). The power to detect a change in Pao₂ and intrapulmonary shunt values from baseline is greater than 80% (0.802 in shunt fraction and 0.888 in arterial oxygenation).

There were no complications associated with the placement of the pulmonary artery catheter. All pulmonary artery catheters were used for postoperative management.

DISCUSSION

In this study assessing the effect of 10° head-up tilt for 10 minutes during OLV in the lateral decubitus position, we observed a significant decrease in shunt and peak airway pressure and an increase in arterial oxygenation. These changes were accompanied by decreases in MAP and cardiac filling

TABLE 2. Changes in hemodynamic variables (n = 20)

Variable	Baseline	T5	T10	T20
HR (beats min ⁻¹)	74.7 ± 9.5	76.7 ± 8.5	78.0 ± 8.6	76.0 ± 8.1
MAP (mm Hg)	82.6 ± 9.1	77.3 ± 11.8*	76.0 ± 10.6*	81.1 ± 9.7
CVP (mm Hg)	8.2 ± 3.3	2.8 ± 2.7*	2.7 ± 2.9*	6.5 ± 2.9*
PCWP (mm Hg)	14.6 ± 3.3	9.3 ± 4.2*	9.6 ± 4.5*	13.3 ± 3.7
CI (L · min ⁻¹ · m ⁻²)	3.1 ± 0.9	3.0 ± 0.9	3.0 ± 1.0	2.9 ± 0.9
SVRI (dynes sec cm ⁻⁵ m ²)	2159.8 ± 722.7	2158.1 ± 620.5	2144.5 ± 662.2	2270.6 ± 708.4
PVRI (dynes sec cm ⁻⁵ m ²)	185.6 ± 69.1	178.0 ± 55.1	171.9 ± 46.2	200.8 ± 69.2

Values are shown as means ± standard deviations. *Baseline*, Fifteen minutes after horizontal position; *T5*, 5 minutes after head-up tilt; *T10*, 10 minutes after head-up tilt; *T20*, 20 minutes after horizontal position after head-up tilt; *HR*, Heart rate; *MAP*, mean arterial pressure; *CVP*, central venous pressure; *PCWP*, pulmonary capillary wedge pressure; *CI*, cardiac index; *SVRI*, systemic vascular resistance index; *PVRI*, pulmonary vascular resistance index. **P* < .05 versus baseline.

pressures without any significant changes in CI. This result might have been due to decreased tension on the dependent paralyzed diaphragm, resulting in decreased atelectasis in the dependent lung, and intrapulmonary blood flow redistribution, leading to improved ventilation-perfusion (V/Q) mismatch.

OLV is frequently used in thoracic operations, and in some patients severe hypoxemia occurs, mandating the implementation of other therapies to provide adequate oxygenation. The cause of hypoxemia during OLV is mainly an increase in intrapulmonary shunt through the nonventilated lung. In addition to total collapse of the nonventilated lung, increased V/Q mismatch in the ventilated lung contributes to shunting and affects the deterioration of arterial oxygenation.¹⁻³ The principle mechanism used to achieve better oxygenation is reduction of the V/Q mismatch in the nonventilated and ventilated lung.

It has been shown that the lateral decubitus position prevents life-threatening hypoxemia (oxygen saturation as measured with pulse oximetry, <90%) in 92% of patients, which developed approximately 10 minutes after the start of OLV in 82% of patients in the supine position among anesthetized human subjects.⁹ In case of dangerous hypoxemia, conventional treatment for hypoxemia always should be initiated: manual ventilation to assess dynamic compliance of the pulmonary system, optimization of ventilator settings, suction of blood and secretion, and confirmation of the correct endotracheal tube position. Nonocclusive insufflations of oxygen through the open endotracheal double-lumen tube, continuous positive airway pressure to the nonventi-

lated lung, and positive end-expiratory pressure to the ventilated lung are other therapies. Intermittent 2-lung ventilation occasionally might be necessary.³

Surgical positions considerably influence the deterioration of oxygenation after the OLV.^{7,9} In the lateral decubitus position during OLV, the nondependent lung is responsible for two thirds of the total shunt, whereas the dependent lung contributes an additional one third of the total shunt.¹⁰ Both hypoxic pulmonary vasoconstriction (HPV) and gravity would cooperate to reduce pulmonary blood flow in the nondependent lung from 40% to 20% of total blood flow in the lateral decubitus position.⁹ Gravity is considered to be an important factor involved in the redistribution of pulmonary perfusion, with proportionally more blood flow in the dependent areas of a lung in the lateral decubitus position.¹¹ However, in the supine position the shift of pulmonary blood flow caused by gravity does not occur because there is no vertical distance between the ventilated and nonventilated lungs.⁹

Although the lateral decubitus position is safer than the supine position during OLV,⁹ placing anesthetized patients in this position during OLV also results in significant V/Q mismatch. With mechanical ventilation, the expansion of the dependent lung is restricted by abdominal contents impinging on the diaphragm, resulting in underventilation of the dependent lung.^{3,7} Various maneuvers have been applied to improve V/Q mismatch in the ventilated dependent lung.^{10,12,13} However, few studies have been conducted to evaluate the clinical effects of surgical positions on arterial oxygenation during OLV.^{7,9}

TABLE 3. Changes in respiratory variables (n = 20)

Variable	Baseline	T5	T10	T20
PaO ₂ (mm Hg)	266.3 ± 103.0	267.5 ± 110.9	289.6 ± 110.3*	306.0 ± 107.9*
A-aO ₂ (mm Hg)	378.4 ± 133.4	397.2 ± 109.9	375.7 ± 108.7	359.8 ± 107.3
Qs/Qt (%)	29.5 ± 11	24.4 ± 10.3*	22.3 ± 9.4*	23.7 ± 10.7*
Paw (cm H ₂ O)	24.2 ± 4.1	23.7 ± 3.4	23.4 ± 3.8*	24.0 ± 3.6
Cdyn (mL · cm H ₂ O ⁻¹)	23.9 ± 6.4	23.1 ± 4.3	23.6 ± 4.7	23.7 ± 5.0

Values are shown as means ± standard deviations. *Baseline*, Fifteen minutes after horizontal position; *T5*, 5 minutes after head-up tilt; *T10*, 10 minutes after head-up tilt; *T20*, 20 minutes after horizontal position after head-up tilt; A-aO₂, alveolar-arterial O₂ gradient; *Qs/Qt*, pulmonary shunt fraction; *Paw*, peak airway pressure; *Cdyn*, dynamic compliance.

**P* < .05 versus baseline.

Head-up tilt shifts abdominal viscera away from the diaphragm and improves ventilation of the dependent lung bases. It has been demonstrated in a previous study that head-down tilt in the lateral decubitus position results in increased atelectasis formation through impingement of abdominal contents against the paralyzed diaphragm, which causes an increase in shunt and a decrease in arterial oxygenation.⁷

There are no comprehensive data describing the effect of head-up tilt on intrapulmonary shunt and oxygenation during OLV in the lateral decubitus position. We observed that a 10° head-up tilt for 10 minutes during OLV in the lateral decubitus position resulted in a significant decrease in shunt and an increase in arterial oxygenation. Theoretically, after the head-up tilt position, gravity change might influence the V/Q mismatch in the dependent lung. In the lateral decubitus position the dependent lung behaves as the inferior portion of the upright lung, with a high blood flow area compared with ventilation.¹¹ In addition, change in gravity after the head-up tilt might increase the perfusion in the dependent lung. Therefore head-up tilt redistributes intrapulmonary blood flow to the base of the dependent lung, which is ventilated more by the release of compression of the diaphragm, and increases blood flow to the dependent lung, which results in improved V/Q mismatch and decreased shunt. The hypothesis of improvement in intrapulmonary shunt and oxygenation is consistent with our results. Therefore head-up tilt can be another mode of treatment for severe hypoxemia during OLV in the lateral decubitus position.

Heneghan and colleagues¹⁴ showed that there was no improvement in oxygenation when lung volumes were increased significantly by using the reverse Trendelenburg position (head-up 30°) in normal anesthetized subjects. However, the head-up tilt during 2-lung ventilation in the supine position is different than that seen during OLV in the lateral decubitus position. The head-up tilt during 2-lung ventilation in the supine position can cause an increase in perfusion of the lower part of both lungs but an increase in ventilation of the upper part to a greater degree. Although ventilation in the lower portion of both lungs increases because of placing abdominal viscera away from the diaphragm, there was no improvement in V/Q mismatch and oxygenation.

For hemodynamic variables, head-up tilt reduced venous return and decreased MAP without a change in the CI. Intuitively, a decrease in MAP might cause a decrease in the systemic oxygenation, but the cardiac output, which could alter Qs/Qt,¹⁵ was maintained at a constant level in our study. Thus we can speculate that the hemodynamics did not influence the systemic oxygenation.

This study design had some limitations. First, our study was not a randomized controlled study. The best evidence from our study would come from such a study, but anesthetic agents and the base–acid status were controlled throughout

the study period to minimize the factors affecting HPV response. In addition, vessel manipulation and vasoactive agents were also withheld. Thus we could suggest that position was the only factor affecting the hemodynamic and respiratory variables during the study period.

Second, the duration of this study might have been insufficient to investigate changes in the respiratory variables. Maximal HPV response in anesthetized human subjects in the supine position occurs within 15 minutes,¹⁶ and for ethical reasons associated with increasing the duration of anesthetic time, we decided the duration of head-up tilt to be 10 minutes. Considering that the time of maximal HPV response during OLV in the lateral decubitus position has not been demonstrated clearly and that respiratory variable changes (eg, shunt and arterial oxygenation) do not return to baseline value 10 minutes after the patient is returned to the horizontal lateral decubitus position, more prolonged duration of the head-up tilt position might have led to better results.

Third, only patients with normal pulmonary function were included in our study, and the mean duration of pulmonary function testing to the day of thoracic surgery was relatively long. To generalize our results to all patients undergoing thoracic surgery, further study, especially in patients with impaired lung function, is needed.

Fourth, mean Pao₂ values at T20 were higher than at T10, but considering that the standard deviations were more than 100, it is reasonable that the value of T10 and T20 showed no significant change. Actually, the *P* value of the difference between T10 and T20 arterial oxygenation was .064. Thus it could be considered that the values were in plateau status.

CONCLUSIONS

Head-up tilt during OLV in the lateral decubitus position during robotic or endoscopic surgery caused a significant decrease in shunt and increase in arterial oxygenation that persisted after a return to the horizontal lateral decubitus position. This result might have been due to decreased tension on the dependent paralyzed diaphragm. Therefore during OLV in the lateral decubitus position, head-up tilt can be another treatment for hypoxemia during OLV anesthesia.

The effect of head-up tilt on arterial oxygenation should be further evaluated for the treatment of serious hypoxemia during OLV in the lateral decubitus position.

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