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ORIGINAL ARTICLE

Effects of tidal volume and PEEP on arterial blood gases and pulmonary mechanics during one-lung ventilation

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Abstract
Purpose The main problem of one-lung ventilation (OLV) is hypoxemia. The use of a high tidal volume for preventing hypoxemia during OLV is controversial. We compared the effects of a high tidal volume versus a low tidal volume with or without PEEP on arterial oxygen tension (PaO_2) and pulmonary mechanics during OLV.
Methods Sixty patients (age range, 16–65 years; ASA I, II) who underwent wedge resection with video-assisted thoracostomy during OLV were assigned to three groups: group I received a high tidal volume (10 ml/kg) ($n = 20$), group II received a low tidal volume (6 ml/kg) ($n = 20$), and group III received a low tidal volume (6 ml/kg) with PEEP (5 cmH_2O) ($n = 20$). Patient hemodynamics, pulmonary mechanics, and arterial blood gases were measured before (T_0) OLV and 5 (T_1), 15 (T_2), 30 (T_3), and 45 min (T_4) after OLV.
Results The PaO_2/FiO_2 ratios of group II and III were significantly decreased and the incidence of hypoxemia was significantly higher in groups II and III than in group I ($P < 0.05$).
Conclusion During OLV, mechanical ventilation with a low tidal volume with or without PEEP increased hypoxemia as compared to that when performing OLV with a high tidal volume.

Keywords High tidal volume · Hypoxemia · Low tidal volume · One-lung ventilation · PEEP

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Introduction

One-lung ventilation (OLV) has been widely used to provide an ideal surgical condition with the isolation of the lung during thoracic surgery. However, it has been reported that OLV induces a right-to-left shunt and a ventilation/perfusion mismatch (V/Q mismatch), resulting in hypoxia and hypoxemia [1]. Hypoxemia is the biggest problem of OLV, and numerous methods have been introduced to prevent hypoxemia during OLV. The incidence of hypoxemia has currently been reduced to less than 1% by the introduction of prophylactic methods [2].

An appropriate inspired O_2 concentration and tidal volume are important factors to maintain oxygenation during OLV [3]. There has particularly been debate on which tidal volume might prevent hypoxemia during OLV. A high tidal volume of 10–12 ml/kg has traditionally been suggested because atelectasis occurs when tidal volume is less than 8 ml/kg [3]. However, recent studies have suggested that the use of a low tidal volume (5–6 ml/kg) and positive end-expiratory pressure (PEEP) was better than a high tidal volume because application of high tidal volume during OLV can more often lead to acute lung injury (ALI) than low tidal volume [4, 5]. Therefore, we examined the changes of arterial blood gases and pulmonary mechanics according to tidal volume and PEEP during OLV.

Materials and methods

The study was approved by our Institutional Review Board (IRB), and informed consent was obtained from all the patients or their guardians. Sixty patients who were ASA class I or II and aged 16–65 years were enrolled. They underwent right wedge resection with video-assisted

thoracostomy under general anesthesia. Patients who had deterioration of pulmonary function (FEV_1 <60%), asthma, or chronic obstructive pulmonary diseases were excluded.

As premedication, midazolam 0.05 mg/kg was injected intramuscularly 1 h before the induction of anesthesia. When the patients arrived in the operating room, noninvasive blood pressure, heart rate, and peripheral oxygen saturation were monitored. A modified Allen's test was performed; if the result was negative, a catheter was installed in the radial artery for continuous monitoring of blood pressure and arterial blood gas analysis. Anesthesia was induced by intravenously injecting propofol 2 mg/kg and rocuronium 0.6 mg/kg. A double-lumen endobronchial tube (Broncho-Cath R; Mallinckrodt Medical, Athlone, Ireland) was inserted, and the location was assessed by the use of a fiberoptic bronchoscope. O_2–air–desflurane was used for the maintenance of anesthesia. Tidal volume was regulated to 10 ml/kg by calculating predicted body weight (PBW) [3]. End-tidal carbon dioxide tension ($ETCO_2$) was continuously monitored and the respiratory rate was adjusted to maintain a normal $ETCO_2$ (35–45 mmHg).

After performing mechanical ventilation for 15 min with the patient in the supine position, mean arterial pressure (MAP), heart rate (HR), arterial blood oxygen tension (PaO_2), and PaO_2/FiO_2 (inspired O_2 concentration) ratio were recorded as the basal values (T_0). A spirometer (S/5 Compact Airway Module, E-CaioV; GE Healthcare Finland Oy, Helsinki, Finland) was installed, and peak airway pressure (P_{aw} peak), plateau airway pressure (P_{aw} plat), mean airway pressure (P_{aw} mean), and compliance were measured and recorded as the basal values (T_0). After changing the patient's position to the lateral position, the location of the double-lumen endobronchial tube was again assessed by a fiberoptic bronchoscope. The patients were randomly assigned to three groups using a random number table. These groups were classified according to the tidal volume with the volume-controlled mode (VCV mode) and the application of PEEP. A tidal volume of 10 ml/kg was applied to group I and a tidal volume of 6 ml/kg was applied to group II. For group III, a tidal volume of 6 ml/kg was applied with PEEP 5 cmH_2O. OLV was initiated from the time of the performing the first incision. The respiration rates were regulated according to the equal minute ventilation during two-lung ventilation.

Pulmonary mechanics and arterial blood gas values were recorded 5 min after OLV and every 15 min until the end of surgery. Hypoxemia was defined as arterial oxygen tension (PaO_2) <80 mmHg after OLV, which was the highest limitation of another study [6], and according to our hospital hypoxemia treatment protocol and the IRB approval that hypoxemia (<80 mmHg) must be treated for the patient's safety. When hypoxemia developed, the inspired O_2 concentration (FiO_2) was raised to 1.0, and the patients were excluded in analyzing the PaO_2/FiO_2 ratio and spirometer parameters results. If hypoxemia was not improved, then a procedure was performed according to the established course to raise arterial oxygen saturation (SpO_2) (Table 1) [7].

All the measured values are presented as means ± standard deviations. In each group, the homodynamic parameters (MAP and HR), the spirometer parameters (P_{aw} peak, P_{aw} plat, P_{aw} mean, compliance), and the PaO_2/FiO_2 ratio were compared by repeated-measures analysis of variance (ANOVA). Post hoc testing was performed using the Scheffe test. One-way ANOVA was applied for comparison of the groups at each time point. Chi-square tests were applied for comparison of the incidence of arterial hypoxemia during OLV. For all analyses, a *P* value lower than 0.05 was considered to be significant.

Table 1 Therapies for desaturation during one-lung ventilation [7]

Severe or precipitous desaturation: resume two-lung ventilation (if possible)
Gradual desaturation:
1. Ensure that delivered FiO_2 is 1.0
2. Check position of double-lumen tube or blocker with fiberoptic bronchoscopy
3. Ensure that cardiac output is optimal; decrease volatile anesthetics to <1 MAC (minimal anesthetic concentration)
4. Apply a recruitment maneuver to the ventilated lung (this will transiently make the hypoxemia worse)
5. Apply positive end-expiratory pressure (PEEP) 5 cmH_2O to the ventilated lung (except in patients with emphysema)
6. Apply continuous positive airway pressure (CPAP) 1–2 cmH_2O to the nonventilated lung (apply a recruitment maneuver to this lung immediately before CPAP)
7. Intermittent reinflation of the nonventilated lung
8. Partial ventilation techniques of the nonventilated lung:
a. Oxygen insufflation
b. High-frequency ventilation
c. Lobar collapse (using a bronchial blocker)
9. Mechanical restriction of the blood flow to the nonventilated lung

Results

Sixty patients were enrolled in the study. However, 5 patients were excluded upon analyzing PaO_2/FiO_2 ratio and spirometer parameter results because they showed hypoxemia; we treated the hypoxemia by increased FiO_2, which may change the PaO_2/FiO_2 ratio.

Gender, age, height, weight, and operation time of the three groups were not significantly different (Table 2).

The incidence of hypoxemia during surgery was 1 case (5%) in group I, 14 cases (70%) in group II, and 13 cases (65%) in group III: the incidence was significantly higher in group II and group III as compared with that of group I ($P < 0.05$, Table 2). MAP and HR did not show significant changes among the three groups (Table 3).

All the parameters of airway pressure (P_{aw} peak, P_{aw} plat, P_{aw} mean) were lowest in group II ($P < 0.01$, Fig. 1).

Compliance showed no statistical significance among the groups ($P < 0.01$, Fig. 2).

The PaO_2/FiO_2 ratio was significantly reduced during OLV, and the PaO_2/FiO_2 ratios of group II and group III were significantly lower than that of group I (Fig. 3).

Discussion

In this study, we examined the effect of tidal volume and PEEP on arterial blood gases and pulmonary dynamics during OLV. OLV with high tidal volume showed a higher PaO_2/FiO_2 ratio and less hypoxemia than did OLV with low tidal volume.

Hypoxemia is one of the major complications during OLV. Although the incidence of severe hypoxemia during anesthesia has decreased by virtue of the many efforts to develop a method to prevent and treat hypoxemia in clinics, hypoxemia during OLV is still problematic and sometimes life threatening [2]. The definition of hypoxemia during OLV is controversial, and it has been suggested that the optimal lower limit of arterial oxygen tension during OLV is between 60 and 80 mmHg [6, 8]. In this study, we defined hypoxemia as arterial oxygen tension below 80 mmHg.

The proper tidal volume to maintain appropriate oxygenation during OLV is controversial. Katz et al. [9] have reported that the highest PaO_2 was shown when ventilation was performed by high tidal volumes during OLV. They

Table 2 Demographic variables and incidence of hypoxemia

	Group I ($n = 20$)	Group II ($n = 20$)	Group III ($n = 20$)
Age (year)	34 ± 18.1	28.4 ± 13.9	35.3 ± 17.1
Height (cm)	172.0 ± 7.3	172.4 ± 8.2	173.1 ± 7.4
Weight (kg)	60.1 ± 7.5	60.7 ± 4.7	62.6 ± 7.8
Sex (male/female)	18/2	17/3	18/2
Operation time (min)	69.8 ± 9.8	61.6 ± 19.0	64.2 ± 21.4
Incidence of hypoxemia (%)	5 (1/20)	70 (14/20)*	65 (13/20)*

Values are expressed as mean ± SD or a number of patients (%). There are no significant differences among groups in demographic variables. The incidence of hypoxemia was significantly higher in group II and III. Hypoxemia is less than 80 mmHg arterial oxygen tension

Group I tidal volume 10 ml/kg, *group II* tidal volume 6 ml/kg, *group III* tidal volume 6 ml/kg with positive end-expiratory pressure (PEEP) 5 cmH_2O

* $P < 0.05$ compared with group I

Table 3 Mean arterial pressure and heart rate (HR) during one-lung ventilation (OLV)

	T_0	T_1	T_2	T_3	T_4
Mean arterial pressure (mmHg)					
Group I	83.05 ± 11.89	92.75 ± 19.68	86.85 ± 14.2	81.59 ± 13.76	82.17 ± 23.64
Group II	83.82 ± 10.02	93.53 ± 16.49	93.06 ± 14.45	85.82 ± 10.48	86.76 ± 7.64
Group III	91.11 ± 12.04	96.28 ± 13.08	93.78 ± 8.73	87.33 ± 11.32	85.06 ± 7.13
Heart rate (bpm)					
Group I	85.35 ± 16.31	86.45 ± 15.93	83.25 ± 12.92	79.83 ± 10.31	75 ± 11.15
Group II	86.00 ± 17.08	86.71 ± 11.58	89.24 ± 9.76	89.12 ± 12.20	84.47 ± 8.87
Group III	91.83 ± 14.38	85.11 ± 13.29	86.61 ± 11.02	84.39 ± 15.00	87.17 ± 11.69

Values are expressed as mean ± SD

Group I tidal volume 10 ml/kg, *group II* tidal volume 6 ml/kg, *group III* tidal volume 6 ml/kg with PEEP 5 cmH_2O, T_0 before OLV, T_1 5 min after OLV, T_2 15 min after OLV, T_3 30 min after OLV, T_4 45 min after OLV (group I, $n = 20$; group II, $n = 17$; group III, $n = 18$)

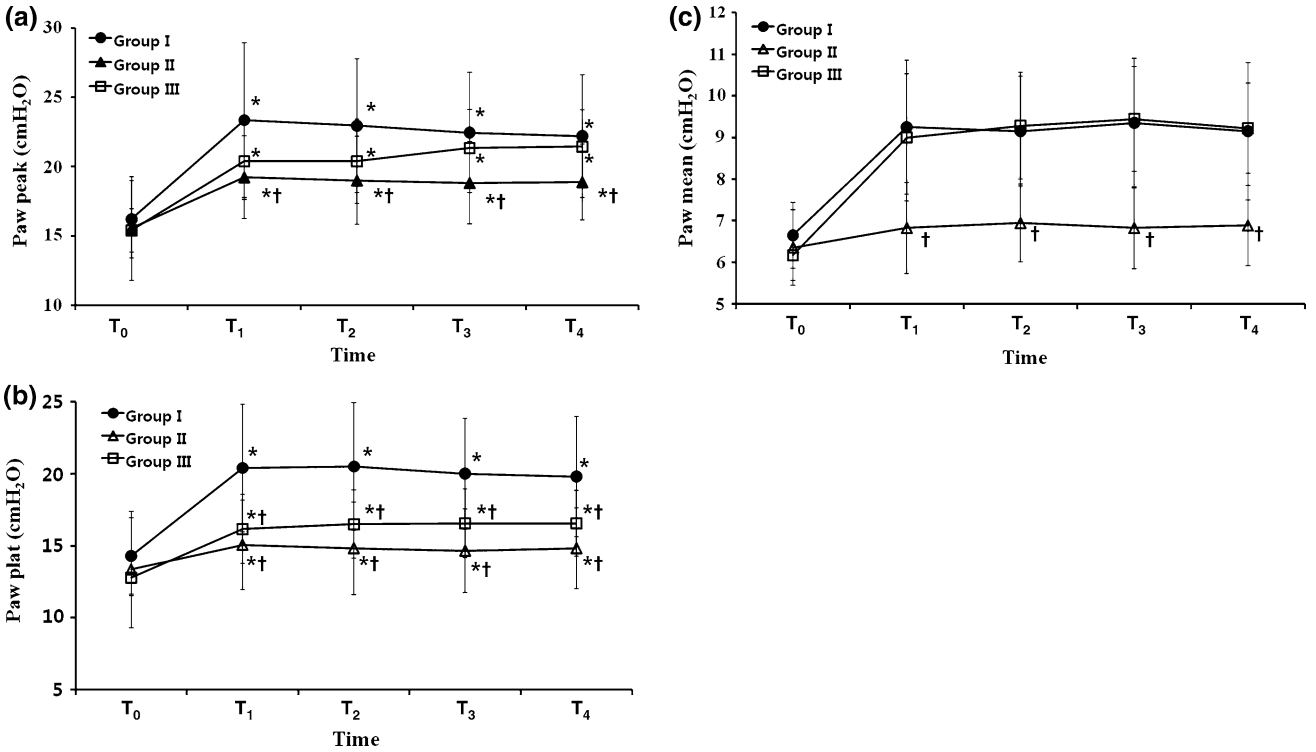

Fig. 1 Airway pressures during one-lung ventilation (OLV). Peak airway pressure (P_{aw} peak) (**a**), plateau airway pressure (P_{aw} plat) (**b**), and mean airway pressure (P_{aw} mean) (**c**) were increased significantly compared with T_0 in all groups. Pressures were lowest in group II. Values are expressed as mean ± SD. *Group I* tidal volume 10 ml/kg, *group II* tidal volume 6 ml/kg, *group III* tidal volume 6 ml/kg with positive end-expiratory pressure (PEEP) 5 cmH_2O, T_0 before OLV, T_1 5 min after OLV, T_2 15 min after OLV, T_3 30 min after OLV, T_4 45 min after OLV (group I, $n = 20$; group II, $n = 17$; group III, $n = 18$). *Compared with T_0; †compared with group I

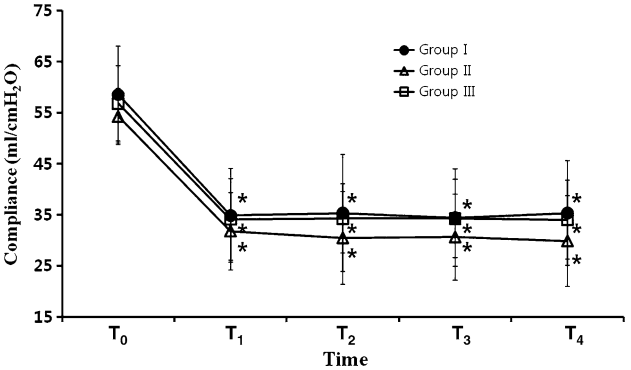

Fig. 2 Compliance during one-lung ventilation. Compliance was decreased significantly compared with T_0 in all groups. However, there were no differences among the groups. Values are expressed as mean ± SD. *Group I* tidal volume 10 ml/kg, *group II* tidal volume 6 ml/kg, *group III* tidal volume 6 ml/kg with PEEP 5 cmH_2O, T_0 before OLV, T_1 5 min after OLV, T_2 15 min after OLV, T_3 30 min after OLV, T_4 45 min after OLV (group I, $n = 20$; group II, $n = 17$; group III, $n = 18$). *Compared with T_0

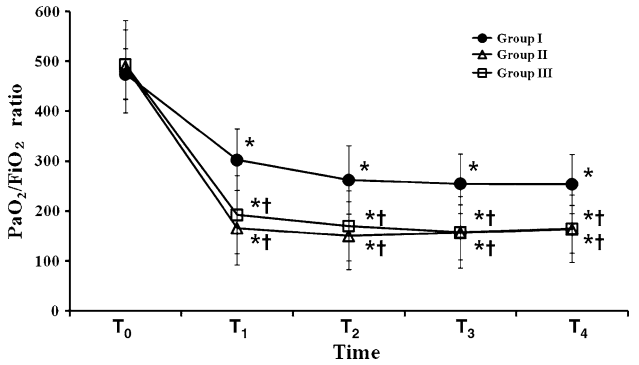

Fig. 3 The ratio of partial pressure of arterial oxygen and fraction of inspired oxygen (PaO_2/FiO_2 ratio) was decreased during one-lung ventilation. The ratio was significantly high in group I than groups II and III. Values are expressed as mean ± SD. *Group I* tidal volume 10 ml/kg, *group II* tidal volume 6 ml/kg, *group III* tidal volume 6 ml/kg with PEEP 5 cmH_2O, T_0 before OLV, T_1 5 min after OLV, T_2 15 min after OLV, T_3 30 min after OLV, T_4 45 min after OLV (group I, $n = 20$; group II, $n = 17$; group III, $n = 18$). *Compared with T_0; †compared with group I

suggested that a tidal volume of 10–12 ml/kg during OLV should be applied because a tidal volume lower than 8 ml/kg may cause atelectasis, which may induce hypoxemia [3, 10]. In our study, with high tidal volume (10 ml/kg), a noticeably higher arterial partial oxygen pressure and PaO_2/FiO_2 ratio were shown as compared with ventilation with a low tidal volume (6 ml/kg).

However, it has been reported that lung injury during OLV was induced by application of the same tidal volume as used for two-lung ventilation [9, 11]. Szegedi et al. [12]

reported that in the initial period of OLV the P_{aw} peak and the P_{aw} plat could be increased immediately by approximately 49% and 51%, respectively, which agrees with our results. In this current study, the mean P_{aw} peak, mean P_{aw} plat, and mean P_{aw} were increased after OLV and particularly when a high tidal volume was used. Increase of pressure during OLV causes hyperinflation of the alveoli and stretch of the pulmonary parenchyma, which may induce acute lung injury by direct volume injury [4, 13, 14]. It has also been reported that tidal volume less than 9 ml/kg did not increase acute lung injury [15]. In addition, Gama de Abreu et al. [16] have reported that in animal experiments, when OLV was performed with a tidal volume of 8 ml/kg, ventilator-induced lung injury (VILI) may be induced in the dependent lung. VILI did not develop when two-lung ventilation was performed with the identical tidal volume or OLV was performed with a half-tidal volume. For these reasons, they suggested using a low tidal volume during OLV. Nevertheless, pulmonary ventilation with a low tidal volume also limits expansion of the alveoli, and it may induce pulmonary collapse [17]. In anesthetized patients, use of low tidal volume may cause atelectasis, and thus alveolar collapse may more readily occur [4, 17, 18]. Ferreira et al. [19] compared the volume fraction of collapsed and normal pulmonary areas of rat lung parenchyma after one-lung ventilation. The fractional area of alveolar collapse was higher in the group that was ventilated with tidal volume of 5 ml/kg and PEEP 2 cmH_2O than the group with tidal volume 10 ml/kg and PEEP 2 cmH_2O. These distal airway/airspace closures may lead to worsening of arterial oxygenation and pulmonary mechanics [19, 20]. Therefore, if sufficient PEEP to prevent alveolar collapse is not applied, then it is not only difficult to maintain the appropriate arterial oxygen saturation, but it can also induce the development or deterioration of lung injury [5, 21]. Application of a low tidal volume (5–7 ml/kg) and a moderate level of PEEP (5–6 cmH_2O) were recommended to reduce the development of acute lung injury [15, 22]; this could prevent the hyperinflation of the lung as well as provide the recruitment of pulmonary alveoli, and thus oxygenation could be maintained. Consequently, it has been suggested that, as a rule, a 10–12 ml/kg high tidal volume could induce acute lung injury and that application of a 5–6 ml/kg low tidal volume and PEEP is preferable [5]. However, application of PEEP with low tidal volume also increases the end-inspiratory volumes and final mean airway pressures as much as those when using a high tidal volume [3]. Mascotto et al. [23] have reported that the selective PEEP applied to the dependent lung during OLV did not improve oxygenation and did not increase pulmonary compliance. Further, Hoftman et al. [24] reported PEEP improved arterial oxygenation by >20% in only 29% of patients. Intrinsic PEEP is also the major concerning factor to determine optimal PEEP during OLV. OLV could produce intrinsic PEEP (2–6 mmHg) that varies from patient to patient, and applying external PEEP equal to intrinsic PEEP increased PaO_2 [25]. However, applying external PEEP greater than intrinsic PEEP could disturb the improving effect on gas exchange of PEEP [25]. In our study, hypoxemia was more commonly developed and the PaO_2/FiO_2 ratio was markedly lower in the group with a low tidal volume, regardless of the application of 5 cmH_2O PEEP, than that in the group with a high tidal volume. It is thought that a low tidal volume might develop atelectasis as a consequence of alveolar collapse [19], resulting in hypoxemia [20], and that application of PEEP 5 cmH_2O cannot improve the PaO_2. It is thought that the PEEP effect in improvement of arterial oxygenation was lost because of differences of intrinsic PEEP in patients of low tidal volume and external PEEP 5 cmH_2O.

A limitation of the present study is that additional tests for lung injury were not performed. However, Taskar et al. [26] reported that healthy lungs tolerated mechanical ventilation with physiological tidal volumes without apparent damage and did not seem to be damaged when terminal units were repeatedly opened and closed for short periods. Furthermore, the distending pressure of the normal relaxed human respiratory system (equivalent to the P_{aw} plat) at the total lung capacity is approximately 37 cmH_2O [27]. The mechanical forces associated with these distending pressures could be safe because normal humans can inspire voluntarily to their total lung capacities without apparent adverse effects [28]. It is thought that all the airway pressures in this study were lower than the pressure that could induce pulmonary barotrauma. Thus, we did not carry out additional tests for lung injury. In addition, surgery was finished early, within 1 h, which is not sufficient time to induce VILI. No patients needed admission to the ICU and mechanical ventilation. Complications such as VILI were not indentified in any patient after surgery. Postoperative chest X-ray did not show any specific changes or abnormalities in the dependent lung, and all patients were discharged without any complicated events. It seems that the patients with a higher tidal volume had higher arterial oxygen tension without complications because they had normal pulmonary function.

In conclusion, OLV with a high tidal volume, 10 ml/kg, in patients with normal pulmonary function does not induce any complications such as lung injury, and this method can prevent hypoxemia more effectively than a low tidal volume, 6 ml/kg, with or without PEEP 5 cmH_2O.

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