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: 무작위대조시험

**Effect of recruitment maneuver
combined with protective ventilation
on the inflammatory response
in video-assisted thoracoscopic lobectomy
: A randomized controlled trial**

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A Thesis of the Degree of Doctor of Philosophy

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August 2016

Anesthesiology and Pain Medicine, Medicine
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**Effect of recruitment maneuver
combined with protective ventilation
on the inflammatory response
in video-assisted thoracoscopic lobectomy
: A randomized controlled trial**

by

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A Thesis Submitted

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Abstract

Effect of recruitment maneuver combined with protective ventilation on the inflammatory response in video-assisted thoracoscopic lobectomy : A randomized controlled trial

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Background: The role of the recruitment maneuver, combined with protective ventilation, on the inflammation for patients undergoing lung surgery remains uncertain. We hypothesized that the addition of recruitment maneuver on the protective ventilation would decrease pulmonary and systemic inflammatory response compared to the traditional ventilation or the protective ventilation in patients undergoing lung surgery.

Patients and methods: Sixty patients scheduled video-assisted thoracoscopic lobectomy were randomized into three groups: traditional ventilation, protective ventilation, and protective ventilation with recruitment maneuver. Two lung ventilation were performed with tidal volume (TV) of 10 ml/kg (traditional

ventilation) or 8 ml/kg with PEEP 5 cmH₂O (protective ventilation and protective ventilation with recruitment maneuver). One lung ventilation (OLV) was performed with TV of 10 ml/kg (traditional ventilation) or 6 ml/kg with PEEP 5 cmH₂O (protective ventilation and protective ventilation with recruitment maneuver). Recruitment maneuver was performed at 10 minutes after the start of OLV in protective ventilation with recruitment maneuver group. Fiberoptic bronchoalveolar lavage (BAL) was performed in dependent and non-dependent lungs two times: before the start of OLV and immediately after the end of OLV. At the same time points, blood samples were obtained. The cytokine, including TNF- α , IL-1 β , IL-6, IL-8, and IL-10, were measured in BAL fluid and serum.

Results: The TNF- α , after OLV in BAL fluid of dependent lung, was higher significantly in the protective ventilation group compared to the protective ventilation with recruitment maneuver group ($P = 0.049$), whereas the IL-1 β , IL-6, IL-8, and IL-10 were not different significantly among groups. In BAL fluid of non-dependent lung, all cytokines were not significantly different among groups. In serum, IL-10 was higher significantly after OLV in the traditional ventilation group compared to the protective ventilation with recruitment maneuver group ($P = 0.027$).

Conclusion: The recruitment maneuver, combined with protective ventilation, may limit pulmonary inflammatory response of the ventilated lung and systemic inflammatory response in patients undergoing video-assisted thoracoscopic

lobectomy, compared to the protective ventilation and traditional ventilation, respectively.

ClinicalTrials.gov ID: NCT01630395

Keywords: inflammation, one-lung ventilation, pulmonary surgical procedures, ventilation

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List of Abbreviations

ALI	acute lung injury
ARDS	acute respiratory distress syndrome
BAL	bronchoalveolar lavage
DLCO	carbon monoxide pulmonary diffusing capacity
ELISA	enzyme-linked immunosorbent assay
FEV ₁	forced expiratory volume in the first second
FVC	forced vital capacity
IQR	inter-quartile range
LMM	linear mixed model
OLV	one lung ventilation
PEEP	peak end-expiratory pressure
SD	standard deviation
TV	tidal volume

1. Introduction

Mechanical ventilation is one of the major causes inducing pulmonary and systemic inflammatory response along with the surgical manipulations in patients undergoing lung surgery (1, 2). The inflammatory response aggravates especially during one lung ventilation (OLV), which is the essential period for the lung resection, requiring the elevated airway pressure (1, 3). The possible underlying mechanisms are the pulmonary damage which is provoked by not only mechanical ventilation itself, consisting of alveolar overdistension and shear forces due to cyclic opening and closing of alveoli, but also pulmonary oxidative stress and ischemia-reperfusion (4). The magnitude of these pulmonary and systemic inflammations is associated with the occurrence of multi-organ dysfunction and mortality (2, 5, 6). Therefore, there have been several efforts to reduce the inflammatory response in patients undergoing lung surgery and one of the methods is adjusting the ventilatory strategy during OLV (7).

Traditionally, the use of large tidal volume (TV) from 8 to 10 ml/kg combined with no peak end-expiratory pressure (PEEP) has been advocated to prevent atelectasis during OLV (8). However, a large TV has

been reported to be associated with the postoperative complications such as acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) and mortality after lung surgery (7, 9). This is because of over-distension of the alveoli and hyperinflation of the lung accompanied with the high peak and plateau airway pressures resulting in volutrauma, barotrauma, and lung inflammation (10). Therefore, the protective ventilation, which consists of small TV of 5-6 ml/kg with adequate PEEP, has been suggested to attenuate inflammatory response in experiment and clinical study (11, 12). The use of small TV prevents the excessive alveolar stretch compared to the large TV. The application of the adequate level of PEEP maintains the end-expiratory lung volume and further reduces the potential pulmonary injuries from the repeated opening and closing of atelectatic regions which is promoted by the use of small TV (13). Therefore, the use of small TV (protective ventilation) has been proven to decrease mortality and increase the number of ventilator-free days in patients with ALI and the ARDS (14). On the other hand, it was demonstrated that, in normal non-injured lung, protective ventilation had contradictory results in terms of inflammatory response and mortality compared to large TV without PEEP (15-18). It is thought that the optimal level of PEEP is difficult to determine and some alveolar areas become atelectatic or overdistended in

the insufficient low or high PEEP, respectively (10, 19).

Recently, recruitment maneuver is suggested as a resolution to overcome the limitation of lung collapse of the protective ventilation (20). The recruitment maneuver combined with the following the adequate level of PEEP reverses the anesthesia-induced atelectatic areas and stabilizes the newly opened pulmonary units increasing the lung homogeneity (21). In previous studies, a recruitment maneuver, which is performed with protective ventilation, improved oxygenation, dead space, and respiratory compliance in patients undergoing abdominal surgery and open thoracic surgery including heart surgery (22-24). In addition, in multicenter clinical trial and observational cohort study, ventilatory strategy, consisting of small TV, PEEP, and recruitment maneuver, decreased the incidence of ALI, atelectasis, admission to the intensive care unit, and hospital stay (25, 26). Whereas, in ALI and ARDS patients, the hospital mortality and the incidence of barotrauma were not different significantly depending on whether the recruitment maneuver was performed or not (27). It is likely that high inspiratory pressure, which is required during recruitment maneuver, may precipitate the overdistension of the lung, redistributing pulmonary blood flow toward atelectatic regions, worsening of gas exchange, and induce inflammatory response (28-30). Therefore, there is

an uncertainty about the role of recruitment maneuver (10). Furthermore, there have been no studies about the effect of recruitment maneuver on the inflammatory response in lung surgery.

We hypothesized that the addition of recruitment maneuver on the protective ventilation would influence the pulmonary and systemic inflammatory response compared to the protective ventilation and the traditional ventilation in patients undergoing lung surgery. Therefore, we compared the pulmonary and systemic inflammatory response among the three-different ventilatory strategy, including large TV without PEEP (traditional ventilation), small TV with PEEP (protective ventilation), and small TV with PEEP and recruitment maneuver (protective ventilation with recruitment maneuver), in patients undergoing lung surgery.

2. Patients and Methods

This prospective randomized study was approved by the Institutional Review Board of Seoul National University Hospital (ref: 1203-101-403) in Seoul, Republic of Korea, and was registered at the ClinicalTrials.gov web site (ref: NCT01630395, June 21, 2012). Written informed consent was obtained from all of the patients. The participants included adults who were aged between 20 and 80 years old, had an American Society of Anesthesiologists physical status of 1 or 2, and were scheduled for elective lobectomy using video-assisted thoracoscope under general anesthesia. Patients with a heart failure of New York Heart Association Functional Class more than 2, reduced preoperative pulmonary function test of forced vital capacity (FVC) less than 50% or forced expiratory volume in the first second (FEV1) less than 50% of predictive values, pulmonary hypertension of mean pulmonary arterial pressure more than 25 mmHg, coagulation disorders, acute pneumonia, extrapulmonary infection, previous treatment with corticosteroids within 3 months before surgery, or a history of recurrent pneumothorax or previous lung resection surgery were excluded.

The patients were randomly assigned in a 1:1:1 ratio to one of three groups using a computer-generated randomization table: the “traditional ventilation” group, the “protective ventilation” group, or the “protective ventilation with recruitment maneuver” group. Random sequence of size 3 blocks that included A, B or C were generated. The intraoperative mechanical ventilation was performed differently according to the group assignments.

A bispectral index sensor (BISTM sensor; Covidien, Boulder, CO, USA) was attached to the patient’s forehead. Anesthesia was induced with propofol 4-5 $\mu\text{g/ml}$ and remifentanyl 4 ng/ml via target controlled intravenous infusion (Orchestra® Base Primea, Fresenius Vial, Brezins, France). Rocuronium of 0.6 mg/kg was administered intravenously to achieve neuromuscular blockade. Tracheal intubation was performed with a double-lumen tube of 35 Fr to 39 Fr (Broncho-Cath®, Mallinckrodt Medical Ltd, Athlone, Ireland). Correct placement of the double-lumen tube was evaluated using fiberoptic bronchoscope (BF-MP60, Olympus, Tokyo, Japan). The 20G catheter was placed in the radial artery to monitor arterial pressure and cardiac index continuously (FloTrac/Vigileo system, Edwards Lifesciences, Irvine, CA, USA). The central venous catheter was

placed in the internal jugular vein. Anesthesia was maintained with propofol 3.5-5 $\mu\text{g/ml}$ and remifentanyl 2-4 ng/ml using target controlled infusion. Vecuronium of 1 mg was administered intermittently for the maintenance of the neuromuscular relaxation. Lactated Ringer's solution was infused at 6 ml/kg/h during surgery and 6% hydroxylethyl starch 130/0.4 (Voluven®, Fresenius Kabi Korea, Seoul, Korea) of 3-5 ml/kg was infused if the cardiac index was less than 2.5 L/min/m^2 .

Two lung ventilation was performed using pressure-controlled ventilation with FiO_2 of 0.5 using the anesthesia machine (GE Datex-Ohmeda S/5 Avance, Madison, WI, USA). In traditional ventilation group, inspiratory pressure was adjusted to achieve TV of 10 ml/kg and PEEP was not applied. In protective ventilation and protective ventilation with recruitment maneuver groups, inspiratory pressure was adjusted to achieve TV of 8 ml/kg and PEEP of 5 cmH_2O was applied. Predicted body weight was used to calculate the TV: $50 + 0.91(\text{centimeters of height} - 152.4)$ in male and $45.5 + 0.91(\text{centimeters of height} - 152.4)$ in female (14, 24, 31). Respiratory rate was adjusted to achieve end-tidal CO_2 of 35-45 mmHg and inspiration to expiration ratio was maintained as 1:2.

OLV was performed using pressure-controlled ventilation and

FiO₂ was applied at 1.0 initially. In traditional ventilation group, inspiratory pressure was adjusted to achieve TV of 10 ml/kg and PEEP was not applied likewise two lung ventilation. In protective ventilation and protective ventilation with recruitment maneuver groups, inspiratory pressure was adjusted to achieve TV of 6 ml/kg and PEEP of 5 cmH₂O was applied. In all three groups, TV was reduced by 1 ml/kg if a peak airway pressure was more than 30 cmH₂O or plateau pressure was more than 25 cmH₂O. FiO₂ was decreased by 0.2 until 0.5 if oxygen saturation was more than 95% and increased by 0.2 until 1.0 if oxygen saturation was less than 95% or PaO₂ was less than 80 mmHg.

Recruitment maneuver was performed only in protective ventilation with recruitment maneuver group. Recruitment maneuver was applied at 10 minutes after the start of OLV and the method as follows (23, 24, 32, 33): Inspiration time was increased by 50% and respiratory rate was set at 12/minutes. Peak inspiratory pressure and PEEP was set at 30 cmH₂O and 10 cmH₂O, respectively during first 3 breaths. Peak inspiratory pressure and PEEP was set at 35 cmH₂O and 15 cmH₂O, respectively during second 3 breaths. Peak inspiratory pressure and PEEP was set at 40 cmH₂O and 20 cmH₂O, respectively during third 6 breaths. After performing recruitment maneuver, the inspiratory pressure and PEEP were

changed the same as the value immediately before starting recruitment maneuver.

At the end of surgery, lung inflation was performed using the inspiratory pressure of 30 cmH₂O for 7 seconds both on the dependent and non-dependent lungs in all three groups. Tracheal extubation was performed after confirmation of neuromuscular recovery. Chest X-ray was performed for 3 days after the surgery.

Bronchoalveolar lavage (BAL) was performed via a fiberoptic bronchoscope in both dependent and non-dependent lungs (12, 34, 35). The end of fiberoptic bronchoscope was positioned at the segmental bronchioles and the sterile saline of 50 ml was administered and aspirated. The BAL was performed twice: during two lung ventilation before the start of OLV and immediately after the end of OLV. Blood sample of 10 ml was also obtained at the same time points as the BAL sample. BAL sample was centrifuged at 200 g and 4 °C for 10 minutes. Blood sample was centrifuged at 1000 g and 4 °C for 15 minutes. The supernatant of BAL and the separated plasma were stored at -70°C. Cytokines such as TNF- α , IL-1 β , IL-6, IL-8, and IL-10 were analysed via enzyme-linked immunosorbent assay (ELISA) using Procarta Cytokine Assay Kit

(Affymetrix, Santa Clara, California, USA).

Anesthetic time, operation time, and the duration of OLV were recorded. The amount of propofol, remifentanyl, and infused fluid during surgery were recorded. Hemodynamic data (including heart rate, mean arterial pressure, and cardiac index) and respiratory data (including TV, respiratory rate, peak inspiratory pressure, plateau pressure, end-tidal CO₂, PaO₂/FiO₂ ratio, PaCO₂, and SaO₂) were recorded at several time points as follows: during two lung ventilation and before the start of OLV (TLV), at 30 minutes after the start of OLV (OLV 30), at 50 minutes after the start of OLV (OLV 50), at 70 minutes after the start of OLV (OLV 70), and at 10 minutes after the end of OLV (OLV end). The physiological dead space to VT ratio was calculated using the following formula: $V_D/V_T = (PaCO_2 - EtCO_2)/PaCO_2$, where EtCO₂ is the end-tidal CO₂. The occurrence of intraoperative events such as PaO₂ less than 80 mmHg, SpO₂ less than 95%, peak inspiratory pressure more than 30 cmH₂O, and plateau pressure more than 25 cmH₂O was recorded. The occurrence of postoperative pulmonary and cardiovascular complications such as pneumonia and atrial fibrillation was recorded. The abnormal findings of postoperative chest X-ray such as atelectasis, pulmonary edema, consolidation, and subcutaneous emphysema were recorded.

Statistical analysis

The primary outcome was the TNF- α in BAL fluid of the dependent lung. TNF- α is a well-known pro-inflammatory mediator produced by macrophages and monocytes and detected in BAL fluid after the mechanical ventilation referring to the previous studies (12, 35, 36). Secondary outcomes were IL-1 β , IL-6, IL-8, and IL-10 in BAL fluid of dependent lung and TNF- α , IL-1 β , IL-6, IL-8, and IL-10 in serum and BAL fluid of non-dependent lung. A minimum of 60 patients was estimated to be required to detect a difference of more than 40% in TNF- α based on a type 1 error of 0.05, a power of 0.8, and a drop-out rate of 20% based on the results of previous study (12, 34, 35). Gender, American Society of Anesthesiologists physical status, preoperative pulmonary function test, lung pathology, operative region, and intraoperative and postoperative adverse events were compared using the Chi-square test or the Fisher's exact test. Weight, height, age, operation time, anesthetic time, the duration of OLV, the amount of propofol and remifentanil, the volume of infused fluid and preoperative respiratory data such as PaO₂, PaCO₂, FEV₁, FVC, FEV₁/FVC ratio, and carbon monoxide pulmonary diffusing capacity

(DLCO) were compared using ANOVA. Intraoperative hemodynamic and respiratory data including heart rate, mean arterial pressure, cardiac index, TV, respiratory rate, peak inspiratory pressure, plateau pressure, end-tidal CO₂, PaO₂/FiO₂ ratio, PaCO₂, SaO₂, and V_D/V_T were compared using linear mixed model (LMM). The cytokine level, including TNF- α , IL-1 β , IL-6, IL-8, and IL-10 in serum and BAL fluids of dependent and non-dependent lung, were logarithmically transformed to achieve homogenous variances of data sets. If the mediator concentration was below the detection limits of the assays, a value of 0.01 was entered (12). LMM was used to analyze the cytokine data. Post hoc analysis was performed with Bonferroni's correction. SAS software (version 9.2, SAS Institute, Inc., Cary, NC, USA) was used for the statistical analysis. The results were considered to be statistically significant if the *P* value was less than 0.05. Data are presented as the number of patients, mean values with standard deviation (SD) or median values with inter-quartile range (IQR), as appropriate.

3. Results

Sixty patients were included and successfully completed the study (Fig.1). Patient characteristics are presented in Table 1. There were no significant differences in preoperative pulmonary function test, operation time, anesthetic time, OLV duration, infused fluid volume, and dose of propofol or remifentanil among the three groups.

Respiratory data is shown in Table 2. TV decreased significantly during OLV compared to the baseline value in all groups. The decrease of TV from the baseline value was significantly lesser in traditional ventilation group compared to the protective ventilation group and protective ventilation with recruitment maneuver group. The decrease of respiratory rate from the baseline value was significantly greater in traditional ventilation group compared to the protective ventilation group and protective ventilation with recruitment maneuver group. Peak inspiratory pressure and plateau pressure increased significantly during OLV compared to the baseline value in all groups. The increase of the peak inspiratory pressure and plateau pressure from the baseline value were significantly greater in traditional ventilation group compared to the

protective ventilation and protective ventilation with recruitment maneuver groups. End-tidal CO₂ was not significantly different among three groups. PaO₂/FiO₂ ratio decreased significantly during OLV in all groups. At 10 minutes after the end of OLV, the decrease of PaO₂/FiO₂ ratio from the baseline value was greater significantly in traditional ventilation group compared to the protective ventilation group. There was no significant difference in PaCO₂ among three groups although PaCO₂ increased significantly from the baseline value at certain time points in all groups. There was no significant difference in SaO₂ among three groups although SaO₂ decreased significantly from the baseline value at certain time points during OLV in protective ventilation group. There was no significant difference in V_D/V_T among three groups although V_D/V_T increased significantly from the baseline value at certain time points during OLV in protective ventilation group.

Hemodynamic data including heart rate, mean arterial pressure, and cardiac index is shown in Table 3. There was no significant difference among three groups. Intraoperative and postoperative complications are shown in Table 4. There were no significant differences among the three groups except that the incidence of intraoperative peak inspiratory pressure more than 30 cmH₂O was higher in traditional ventilation group compared

to the protective ventilation with recruitment maneuver group ($P = 0.024$). Intraoperative hypoxia (PaO_2 less than 80 mmHg) occurred in 12% of patients and the lowest PaO_2 level was 72 ± 5 mmHg.

Cytokine level is shown in Tables 5, 6 and 7. In BAL fluid of dependent lung, $\text{TNF-}\alpha$ was higher significantly after OLV in the protective ventilation group compared to the protective ventilation with recruitment maneuver group ($P = 0.049$), whereas the $\text{IL-1}\beta$, IL-6 , IL-8 , and IL-10 were not significantly different among three groups (Table 5). In BAL fluid of non-dependent lung, all cytokines were not significantly different among groups (Table 6). In serum, IL-10 after OLV was higher significantly in the traditional ventilation group compared to the protective ventilation with recruitment maneuver group ($P = 0.027$), whereas $\text{IL-1}\beta$, IL-6 , IL-8 , and $\text{TNF-}\alpha$ were not significantly different among three groups (Table 7).

4. Discussion

TNF- α was higher after OLV, in BAL fluid of dependent lung, in the protective ventilation group compared to the protective ventilation with recruitment maneuver group, whereas other cytokines were not significantly different among three groups. In BAL fluid of non-dependent lung, all cytokines were not significantly different among groups. In serum, IL-10 was higher after OLV in the traditional ventilation group compared to the protective ventilation with recruitment maneuver group, whereas other cytokines were not significantly different among three groups.

Intraoperative mechanical ventilation precipitates local pulmonary and systemic inflammatory response in patients undergoing elective surgery (10). Previous studies demonstrated that, in patients undergoing lung surgery and the other types of surgery such as abdominal surgery, esophageal surgery, and gynaecologic surgery, the cytokines increased after surgery in BAL fluid and in serum (5, 12, 34, 37-39). Furthermore, the cytokines such as IL-6 in serum have been reported to increase after OLV (35, 38) and this response was reduced by the protective ventilatory strategy during OLV in patients undergoing esophagectomy (17). Therefore,

it is certain that the release of local pulmonary inflammatory mediators occurs during mechanical ventilation, which aggravates especially during OLV, and these cytokines are translocated into the bloodstream due to the increased alveolar-capillary permeability, resulting in systemic inflammation (10, 29). This corresponds with the result of our study showing that the release of cytokines after OLV compared to the value before OLV in BAL fluid and in serum. The main underlying mechanism of the inflammatory response is different according to the position where the inflammation takes place. In dependent ventilated lung, the cause of the inflammation is the oxidative stress developed by hyperoxia and cycling opening and closing of the airways throughout mechanical ventilation (4). In non-dependent operative lung, the reexpansion of the completely atelectatic and hypoperfused lung after lung resection, provokes ischemia-reperfusion (4, 40). Surgical manipulation also induces inflammatory response of the non-dependent lung during OLV (1, 41). Therefore, the inflammatory response occurs in both lungs as reported in patients undergoing lobectomy, segmentectomy, or esophagectomy (3, 37, 42) and experiment of pigs which observed diffuse alveolar damage in both lungs (1).

In previous study, protective ventilation improved oxygenation,

compliance and dead space compared to the traditional ventilation in patients undergoing elective surgeries under general anesthesia such as lung surgery, esophagectomy, and cardiac surgery (17, 43, 44). It corresponds with our results showing that the decrease of $\text{PaO}_2/\text{FiO}_2$ was less in protective ventilation group compared to the traditional ventilation group. However, this positive effect did not extend to the decrease of inflammatory response in the lungs and serum in our study. This may be due to the absence of the use of the recruitment maneuver. Recruitment maneuver, which was performed before OLV, was shown to enhance aeration and reduce cyclic recruitment during OLV in experiment of pigs (45). Previous retrospective study demonstrated that small TV with PEEP without recruitment maneuver was linked to the increased risk of 30-day mortality in patients receiving general anesthesia (18). Therefore, we speculate that the recruitment maneuver may be the critical component of the lung-protective ventilatory strategy and to the best of our knowledge this is the first study investigating and comparing the inflammatory response among three different ventilation methods.

In our study, $\text{TNF-}\alpha$, in BAL of dependent lung, was significantly higher after OLV in the protective ventilation group compared to the protective ventilation with recruitment maneuver group, although the other cytokines

were not significantly different among the three groups. TNF- α is the well-known pro-inflammatory cytokine produced by the alveolar macrophages and monocytes, which are recruited into the alveolar space after the injury of the alveolo-capillary units, and detected in BAL fluid after mechanical ventilation (34, 35). Therefore, the low level of TNF- α , in the ventilated dependent lung, means that the addition of the recruitment maneuver on the protective ventilation may improve the pulmonary inflammation, which is induced by the alveolar damage after one lung ventilation in video-assisted thoracoscopic lobectomy. In our study, the inflammatory responses were not significantly different in ventilated dependent lung between the traditional ventilation group and protective ventilation group. This is consistent with the finding of the previous report which observed no significant difference in TNF- α , IL-1 β , IL-6, IL-8, L-10, and L-12 in BAL between the large TV of 12-15 ml/kg with no PEEP and the small TV of 6 ml/kg with PEEP of 10 cmH₂O in thoracotomy or laparotomy (15). Whereas, in other previous report, IL-8 and TNF- α increased significantly after OLV in BAL of dependent lung and the increase of TNF- α was significantly lower when small TV of 5 ml/kg with no PEEP was used compared to the large TV of 10 ml/kg with no PEEP in the open thoracic surgery (12). Moreover, Zupancich et al. observed that small TV of 8 ml/kg

with PEEP 10 cmH₂O attenuated the release of IL-6 and IL-8 in BAL, compared to the large TV of 10-12 ml/kg with PEEP 2-3 cmH₂O, in the cardiac surgery (46). This variation in cytokine concentrations of BAL may be due to the difference of the type and extent of surgical trauma and the components of the ventilatory strategy (the level of TV and PEEP). In our study, the increases of cytokine levels were not significantly different in BAL of non-dependent operated lung among three groups. The predominant cause of the inflammation in non-dependent lung is not the OLV but the direct surgical injury, therefore, the different application of the ventilatory strategy may not improve the inflammatory response of the non-dependent lung.

In our study, serum cytokine IL-10 was higher after OLV in traditional ventilation group compared to the protective ventilation with recruitment maneuver group, whereas there were no significant differences in other serum cytokines among groups. IL-10 is an immunoregulatory cytokine, functioning to reduce the severity of lung injury by counteracting the expression of proinflammatory cytokines, which has been reported to increase after OLV in serum in the clinical study and experiments in animals (37, 40). Our finding is consistent with the result of previous report in which the recruitment maneuver, combined with protective

ventilation, decreased the release of IL-8 and IL-10 in serum significantly after the cardiopulmonary bypass in patients undergoing the cardiac surgery, compared to the traditional ventilation (47). Therefore, based on our study result, recruitment maneuver combined with the protective ventilation may improve systemic inflammatory response compared to the traditional ventilation. However, we did not observe the significant difference in plasmatic IL-6 among the groups, although serum IL-6 has been reported to increase less with protective ventilation compared to the traditional ventilation in patients undergoing esophagectomy (17). Moreover, in our study, there was no significant difference in serum cytokines between the protective ventilation group and the protective ventilation with recruitment maneuver group, even though the pulmonary cytokine levels were different significantly between these two groups. As can be seen from these various different reports, serum cytokines may not differentiate the specific influence of the ventilation method on the systemic inflammation from the surgical stimulation (3). This is because the surgical injury precipitates the severe degree of the inflammation concurrently combined with the mechanical ventilation. This is consistent with the previously reported results that the increase of the serum IL-8 was not significantly different after surgery between the traditional ventilation

and the protective ventilation with recruitment maneuver, although the pulmonary level of IL-8 was attenuated with protective ventilation in abdominal surgery (39). Therefore, we find no evidence that the addition of recruitment maneuver can affect the systemic inflammation compared to the protective ventilation in patients undergoing video-assisted thoracoscopic lobectomy, despite the improvement of the local inflammatory response of the ventilated lung. Whereas, the recruitment maneuver combined with the protective ventilation may improve systemic inflammatory response compared to the traditional ventilation, although the mechanism is not clear.

There are several factors influencing the inflammatory response during OLV such as OLV duration, FiO_2 , the type and dose of anesthetic agents, fluid infusion volume, and surgical methods. The longer duration of OLV, which augments ischemic times of operative lung and mechanical ventilation times of dependent lung, was associated with the higher degree of inflammatory response proportionally (4, 49, 50). Moreover, the inflammatory response may become worse due to the production of radical oxygen species and development of absorption atelectasis when the high FiO_2 is applied (51, 52). In our study, the OLV duration was not significantly different among the three groups and the FiO_2 was adjusted

according to the oxygen saturation and PaO₂ regardless of the groups. Volatile anesthetic agents such as sevoflurane and desflurane have been shown an immunomodulatory effect and cell-protective effect against the pulmonary damage caused by ischemia-reperfusion, compared to the propofol (34, 35, 42, 50). In this study, we used propofol and remifentanyl for the maintenance of anesthesia and the dose of these agents were not significantly different. Fluid volume overload during lung surgery could affect the inflammatory response by increasing fluid translocation to the pulmonary interstitial space. Intraoperative total volume of administered fluid is a critical factor for the occurrence of postoperative ALI and ARDS (9, 53). Therefore, we administered same types of crystalloid with monitoring the cardiac index and there was no significant difference in the fluid amount regardless of the groups. We investigated the patients who received lobectomy using video-assisted thoracic surgical approach. Previous study demonstrated that video-assisted thoracic surgery provoked less cytokine response such as serum IL-6 levels with the less postoperative pain and better postoperative pulmonary function compared to conventional open surgery (41, 54, 55). This may be due to the reduced amount and extent of operation. Therefore, we applied the same surgical methods regardless of the group assignments to eliminate the surgical bias.

Therefore, we think that the significant difference in TNF- α in ventilated dependent lung, which was observed in our study, may be result from the different ventilatory strategy.

There are several limitations in our study. First, we performed BAL to measure the inflammatory mediators. Whereas, several previous reports suggested bronchoscopic microsampling method to obtain the epithelial lining fluid of small airways due to the concern about hypoxia which can occur during repeated examination of BAL (42, 49, 56). The values of the cytokine may be different if we chose to use this bronchoscopic microsampling method. However, in our study, the investigator, who was not aware of the group assignments, performed the BAL in each patient as the methods which was described in several previous studies investigating the pulmonary cytokines (3, 12, 34, 35, 37, 38). Therefore, we think that the results of our study have the clinical value. Second, several different recruitment maneuver methods such as maintaining high inspiratory pressure of 35-40 cmH₂O for 7 to 10 seconds were suggested previously (25, 57). Therefore, our result could be changed if a different recruitment method is chosen. However, we chose to use the recruitment method which is described in previous studies investigating the pulmonary inflammatory response during OLV (23, 24, 32, 33) and applied

into all groups in the same way. Third, the incidence of postoperative pulmonary and cardiovascular complications was not significantly different among the groups although there was significant difference in pulmonary inflammatory response. This is contradict to the previous report demonstrating that recruitment maneuver combined with protective ventilation decreased incidence of postoperative ALI and atelectasis (25). This may be due to the small sample size in our study. However, our findings are thought to be clinically relevant because previous report showing that the inflammatory cytokine levels such as pulmonary and plasmatic IL-6, IL-8, IL-10, and TNF- α can be an early marker of postoperative pulmonary complications or systemic inflammatory response syndrome in esophagectomy or thoracic surgery (5, 6, 50). Further large-scale study is warranted to find out the difference of the postoperative complications and mortality precisely.

In conclusion, recruitment maneuver combined with protective ventilation improves the local inflammation of the ventilated lung compared to the protective ventilation and systemic inflammation compared to the traditional ventilation. Therefore, we think that a recruitment maneuver may be needed to apply on the protective ventilation to reduce inflammatory response in ventilated lung effectively during

video-assisted thoracoscopic lobectomy whereas the protective ventilation without recruitment maneuver is not enough to induce such effects.

5. References

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Table 1. Patient characteristics

	Traditional ventilation (n = 20)	Protective ventilation (n = 20)	Protective ventilation with recruitment maneuver (n = 20)	<i>P</i> -value
Age (yrs)	57 ± 10	57 ± 10	57 ± 10	0.984
Sex, male/female	9/11	11/9	7/13	0.446
Height (cm)	162 ± 10	163 ± 8	160 ± 6	0.599
Weight (kg)	63 ± 9	64 ± 10	60 ± 6	0.288
ASA, 1/2	14/6	11/9	15/5	0.377
Smoking, former smoker/never smoker	4/16	6/14	4/16	0.799
Preoperative PaO ₂ (mmHg)	99 ± 15	99 ± 16	99 ± 18	0.995
Preoperative PaCO ₂ (mmHg)	44 ± 7	41 ± 5	42 ± 4	0.247
Preoperative FEV ₁ (% of predicted)	109 ± 16	108 ± 14	108 ± 15	0.983
Preoperative FVC (% of predicted)	103 ± 13	102 ± 9	99 ± 14	0.627
Preoperative FEV ₁ /FVC ratio (% of predicted)	79 ± 6	77 ± 5	80 ± 8	0.336

Preoperative DLCO (% of predicted)	99 ± 20	99 ± 19	99 ± 16	> 0.999
Pulmonary function test, obstructive/restrictive/normal	1/1/18	1/0/19	1/1/18	0.053
Pathology, adenocarcinoma/NSCLC/BAC/Sqcc	16/1/3/0	15/2/2/1	17/2/1/0	0.920
Operative region, RUL/RML/RLL/LUL/LLL	8/2/0/4/6	4/2/7/5/2	5/3/7/3/2	0.070
Operation time (min)	159 ± 55	141 ± 36	141 ± 40	0.330
Anesthetic time (min)	206 ± 50	196 ± 43	196 ± 38	0.728
One lung ventilation duration (min)	131 ± 40	121 ± 34	123 ± 38	0.665
Infused volume of lactated Ringer's solution (ml)	848 ± 344	748 ± 324	810 ± 353	0.649
Infused volume of 6% hydroxyethyl starch 130/0.4 (ml)	15 ± 67	20 ± 89	0	0.597
Propofol (mg)	1617 ± 643	1575 ± 525	1484 ± 235	0.691
Remifentanil (µg)	1356 ± 527	1247 ± 550	1406 ± 457	0.679

Data are presented as mean ± SD or number of patients. BAC, bronchioloalveolar carcinoma; DLCO, carbon monoxide pulmonary diffusing capacity; FEV1, forced expiratory volume in 1 s; FVC, forced vital capacity; LLL, left lower lobe; LUL, left upper lobe; NSCLC, non-small-cell lung carcinoma; RLL, right lower lobe; RML, right middle lobe; RUL, right upper lobe; Sqcc, squamous cell carcinoma.

Table 2. Respiratory data.

	TLV	OLV 30	OLV 50	OLV 70	OLV end
Tidal volume (ml)					
Traditional ventilation	549 ± 104	483 ± 114†‡*	480 ± 121†‡	493 ± 127†‡*	503 ± 91†
Protective ventilation	450 ± 71	322 ± 62†	324 ± 56†	326 ± 51†	421 ± 71
Protective ventilation with recruitment maneuver	420 ± 51	313 ± 35†	308 ± 39†	305 ± 41†	405 ± 65
Respiratory rate (breaths/min)					
Traditional ventilation	12 ± 2	10 ± 2	10 ± 3‡*	11 ± 2‡*	10 ± 2
Protective ventilation	13 ± 2	13 ± 3	14 ± 2	15 ± 2	13 ± 1
Protective ventilation with recruitment maneuver	14 ± 2	14 ± 2	14 ± 2	15 ± 2	13 ± 2
Peak inspiratory pressure (cmH ₂ O)					
Traditional ventilation	17 ± 2	26 ± 2†‡*	26 ± 2†‡*	25 ± 2†*	19 ± 5
Protective ventilation	18 ± 3	22 ± 4†	23 ± 4†	24 ± 4†	21 ± 4†
Protective ventilation with recruitment maneuver	18 ± 3	22 ± 4†	23 ± 4†	22 ± 3†	20 ± 3
Plateau pressure (cmH ₂ O)					

Traditional ventilation	17 ± 2	23 ± 3†‡*	23 ± 3†‡*	23 ± 3†*	18 ± 5
Protective ventilation	17 ± 3	20 ± 3†	21 ± 4†	21 ± 4†	19 ± 4
Protective ventilation with recruitment maneuver	17 ± 2	20 ± 4†	21 ± 4†	20 ± 4†	19 ± 4
End-tidal CO ₂ (mmHg)					
Traditional ventilation	31 ± 3	32 ± 3	32 ± 4	32 ± 3	30 ± 4
Protective ventilation	33 ± 4	36 ± 5	35 ± 4	36 ± 4	34 ± 5
Protective ventilation with recruitment maneuver	33 ± 3	35 ± 2	35 ± 3	35 ± 3	34 ± 3
PaO ₂ /FiO ₂ ratio					
Traditional ventilation	425 ± 133	170 ± 54†	198 ± 63†	241 ± 82†	341 ± 105‡
Protective ventilation	442 ± 156	150 ± 58†	193 ± 89†	209 ± 100†	454 ± 123
Protective ventilation with recruitment maneuver	475 ± 151	202 ± 91†	218 ± 102†	241 ± 121†	428 ± 122
PaCO ₂ (mmHg)					
Traditional ventilation	38 ± 4	43 ± 5†	41 ± 7	40 ± 4	41 ± 4
Protective ventilation	40 ± 4	49 ± 7†	47 ± 6†	47 ± 5†	45 ± 5†
Protective ventilation with recruitment maneuver	42 ± 5	48 ± 6†	47 ± 4†	46 ± 5	46 ± 6

SaO ₂ (mmHg)					
Traditional ventilation	100 ± 1	99 ± 2	99 ± 1	99 ± 2	99 ± 1
Protective ventilation	100 ± 1	98 ± 2†	98 ± 6	99 ± 2†	100 ± 1
Protective ventilation with recruitment maneuver	100 ± 1	99 ± 2	99 ± 2	99 ± 1	100 ± 1
V _D /V _T (%)					
Traditional ventilation	19 ± 7	25 ± 6	24 ± 10	21 ± 6	25 ± 8
Protective ventilation	18 ± 8	27 ± 5†	26 ± 5	23 ± 5	23 ± 8
Protective ventilation with recruitment maneuver	21 ± 9	27 ± 5	25 ± 6	24 ± 5	25 ± 7

Data are presented as mean ± SD. OLV; one lung ventilation. TLV, during two lung ventilation and before the start of OLV; OLV 30, at 30 minutes after the start of OLV; OLV 50, at 50 minutes after the start of OLV; OLV 70, at 70 minutes after the start of OLV; OLV end, at 10 minutes after the end of OLV. †*P*-value < 0.05: compared to the value of TLV within the group. ‡*P*-value < 0.05: compared to the protective ventilation group. **P*-value < 0.05: compared to the protective ventilation with recruitment maneuver group.

Table 3. Hemodynamic data.

	TLV	OLV 30	OLV 50	OLV 70	OLV end
Heart rate (beats/min)					
Traditional ventilation	66 ± 10	69 ± 11	65 ± 8	65 ± 10	71 ± 13
Protective ventilation	67 ± 12	71 ± 9	70 ± 11	68 ± 11	69 ± 11
Protective ventilation with recruitment maneuver	62 ± 7	68 ± 9	65 ± 10	67 ± 9	65 ± 8
Mean arterial pressure (mmHg)					
Traditional ventilation	77 ± 14	79 ± 10	82 ± 10	87 ± 13†	82 ± 10
Protective ventilation	79 ± 20	78 ± 10	84 ± 12	96 ± 12	88 ± 14
Protective ventilation with recruitment maneuver	77 ± 11	80 ± 10	81 ± 11	83 ± 10	88 ± 10†
Cardiac index (l/min/m ²)					
Traditional ventilation	3 ± 1	3 ± 1	3 ± 1	3 ± 1	3 ± 1
Protective ventilation	3 ± 1	3 ± 1	3 ± 1	3 ± 1	3 ± 1
Protective ventilation with recruitment maneuver	3 ± 1	3 ± 1	3 ± 1	3 ± 1	3 ± 1

Data are presented as mean ± SD. OLV; one lung ventilation. TLV, during two lung ventilation and before the start of OLV; OLV 30, at 30

minutes after the start of OLV; OLV 50, at 50 minutes after the start of OLV; OLV 70, at 70 minutes after the start of OLV; OLV end, at 10 minutes after the end of OLV. †*P*-value < 0.05: compared to the value of TLV within the group.

Table 4. Intraoperative events and postoperative complications.

	Traditional ventilation (n = 20)	Protective ventilation (n = 20)	Protective ventilation with recruitment maneuver (n = 20)	P-value
Intraoperative PaO ₂ less than 80 mmHg	1	4	2	0.478
Intraoperative SpO ₂ less than 95%	0	1	0	> 0.999
Intraoperative peak inspiratory pressure more than 30 cmH ₂ O	11†	6	3	0.034
Intraoperative plateau pressure more than 25 cmH ₂ O	7	4	4	0.602
Occurrence of postoperative complication	3	1	2	0.863
Abnormal finding of chest X-ray				
Atelectasis	9	8	6	0.713
Pulmonary edema	0	1	1	> 0.999
Consolidation	2	4	2	0.710
Subcutaneous emphysema	1	0	3	0.310

Data are presented as number of patients. † P -value < 0.05: comparison between the traditional ventilation and the protective ventilation with recruitment maneuver group.

Table 5. Cytokine level in BAL of dependent lung before/after one lung ventilation.

	Traditional ventilation (n = 20)	Protective ventilation (n = 20)	Protective ventilation with recruitment maneuver (n = 20)	P-value		
				T vs. P	P vs. PR	T vs. PR
TNF-a (pg/ml)	0.90 (0.42-1.65) / 1.16 (0.52-5.97)	0.72 (0.01-1.67) / 0.99 (0.5-6.2)	0.82 (0.03-1.67) / 0.57 (0.01-1.35)	> 0.999	0.049†	0.339
IL-1β (pg/ml)	0.61 (0.14-23.6) / 1.07 (0.24-23.58)	0.9 (0.13-3.77) / 0.77 (0.03-2.52)	1.29 (0.77-6.23) / 0.29 (0.12-1.4)	0.897	0.966	0.135
IL-6 (pg/ml)	2.96 (0.11-8.75) / 6.92 (0.32-26.08)	1.01 (0.01-5.78) / 2.58 (0.12-10.32)	2.84 (0.01-7.89) / 0.8 (0.01-7.78)	> 0.999	> 0.999	> 0.999
IL-8 (pg/ml)	43.62 (23.19-434.52) / 90.61 (18.3-432.49)	31.55 (2.75-245.45) / 28.89 (0.71-129.54)	93.18 (15.41-128.04) / 50.53 (11.55-106.82)	0.867	> 0.999	> 0.999
IL-10 (pg/ml)	0.01 (0.01-0.11) / 0.02 (0.01-0.33)	0.01 (0.01-0.03) / 0.02 (0.01-0.32)	0.03 (0.01-0.07) / 0.02 (0.01-0.18)	> 0.999	0.234	> 0.999

Data are presented as median (interquartile range). BAL; bronchoalveolar lavage, P: protective ventilation, PR; protective ventilation with recruitment maneuver, T; traditional ventilation. †P-value < 0.05: comparison between protective ventilation group and the protective ventilation with recruitment maneuver group.

Table 6. Cytokine level in BAL of non-dependent lung before/after one lung ventilation.

	Traditional ventilation (n = 20)	Protective ventilation (n = 20)	Protective ventilation with recruitment maneuver (n = 20)	P-value		
				T vs. P	P vs. PR	T vs. PR
TNF-a (pg/ml)	0.86 (0.03-2.49) / 6.97 (0.95-37.57)	0.05 (0.01-0.48) / 1.07 (0.13-5.79)	0.6 (0.3-1.57) / 8.93 (1.69-52.62)	> 0.999	> 0.999	> 0.999
IL-1 β (pg/ml)	1.28 (0.29-2.61) / 9.13 (1.42-38.27)	0.54 (0.03-2) / 2.79 (0.72-6.24)	1.29 (0.19-6.16) / 3.31 (1.96-12.75)	> 0.999	> 0.999	> 0.999
IL-6 (pg/ml)	3.02 (0.69-6.79) / 99.56 (13.79-218.4)	2.26 (0.09-3.47) / 6.11 (0.71-49.02)	2.57 (0.37-10.53) / 48.39 (14.13-145.75)	0.835	0.400	> 0.999
IL-8 (pg/ml)	76.54 (45.36-210.41) / 518.54 (179.31-831.79)	34.66 (9.37-158.41) / 88.19 (15.52-260.99)	33.74 (19.86-108.27) / 315.58 (99.37-801.91)	> 0.999	> 0.999	> 0.999
IL-10 (pg/ml)	0.02 (0.01-0.43) / 0.04 (0.01-0.86)	0.01 (0.01-0.03) / 0.02 (0.01-0.3)	0.02 (0.01-0.27) / 0.31 (0.02-1.53)	> 0.999	0.363	0.284

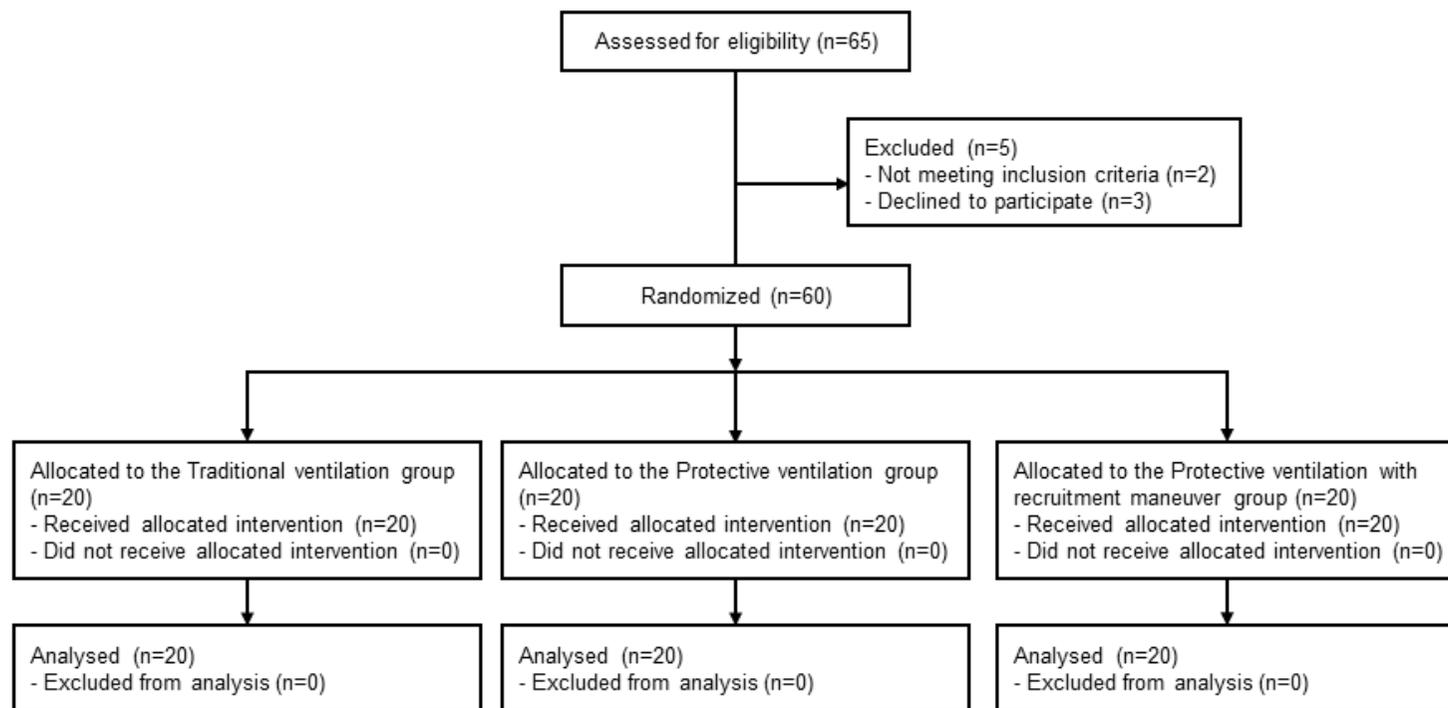
Data are presented as median (interquartile range). BAL; bronchoalveolar lavage, P: protective ventilation, PR; protective ventilation with recruitment maneuver, T; traditional ventilation.

Table 7. Cytokine level in serum before/after one lung ventilation.

	Traditional ventilation	Protective ventilation	Protective ventilation	P-value		
	(n = 20)	(n = 20)	with recruitment maneuver (n = 20)	T vs. P	P vs. PR	T vs. PR
TNF-a (pg/ml)	0.01 (0.01-1.65) / 0.38 (0.01-1.83)	0.01 (0.01-1.65) / 0.01 (0.01-2.06)	0.21 (0.01-2.42) / 0.01 (0.01-1.65)	> 0.999	0.599	0.743
IL-1 β (pg/ml)	0.2 (0.01-0.63) / 0.29 (0.01-0.97)	0.12 (0.01-0.52) / 0.07 (0.01-0.61)	0.27 (0.01-0.58) / 0.26 (0.01-0.5)	> 0.999	> 0.999	0.324
IL-6 (pg/ml)	0.21 (0.01-5.1) / 22.22 (6.5-66.2)	0.01 (0.01-1.31) / 8.13 (0.87-13.78)	0.01 (0.01-1.86) / 10.09 (3.76-40.89)	> 0.999	> 0.999	> 0.999
IL-8 (pg/ml)	4.04 (0.01-5.58) / 6.2 (3.61-9.99)	2.94 (0.01-5.29) / 6.04 (0.01-7.88)	4.76 (0.01-6.63) / 6.61 (2.17-7.42)	> 0.999	> 0.999	> 0.999
IL-10 (pg/ml)	0.45 (0.01-1.88) / 1.35 (0.01-3.34)	0.12 (0.01-1.22) / 0.18 (0.01-1.5)	0.38 (0.01-7.47) / 0.57 (0.01-3.58)	0.195	> 0.999	0.027†

Data are presented as median (interquartile range). P: protective ventilation, PR; protective ventilation with recruitment maneuver, T; traditional ventilation. †P-value < 0.05: comparison between the traditional ventilation and the protective ventilation with recruitment maneuver group

Figure 1. Flow diagram.



국문 초록

폐 절제술을 받는 환자에서 작은 크기의 일회호흡량(tidal volume)과 호기말양압(positive end-expiratory pressure)을 적용하여 기계환기를 할 때 폐포끌어모으기 조작(recruitment maneuver)이 염증반응에 미치는 영향에 대해서는 분명히 밝혀진 바가 없다. 본 연구에서는, 폐 절제술을 받는 환자에서 작은 크기의 일회호흡량과 호기말양압과 함께 폐포끌어모으기 조작을 사용했을 때 전통적인 환기방법이나 폐보호 환기방법에 비해서 폐 및 전신 염증반응 줄어들 것으로 가정하였다.

비디오흉강경을 이용한 폐엽 절제술(video-assisted thoracoscopic lobectomy)이 예정된 환자 60 명을 세 군으로 무작위배정하였다. 세 군은 전통적인 환기(traditional ventilation)군, 폐보호 환기(protective ventilation)군, 폐보호 환기 및 폐포끌어모으기 조작(protective ventilation with recruitment maneuver)군이다. 양측폐환기(two lung ventilation)를 시행하기 위해 ‘전통적인 환기군’에서는 10 ml/kg 의 일회호흡량을 적용하였고 ‘폐보호 환기군’과 ‘폐보호 환기 및 폐포끌어모으기 조작군’에서는 8 ml/kg 의 일회호흡량과 5 cmH₂O 의 호기말양압을 적용하였다. 일측폐환기(one lung ventilation)를 시행하기 위해 ‘전통적인 환기군’에서는 10 ml/kg 의 일회호흡량을 적용하였고 ‘폐보호 환기군’과 ‘폐보호 환기 및

폐포끌어모으기 조작군'에서는 6 ml/kg 의 일회호흡량과 5 cmH₂O 의 호기말양압을 적용하였다. 폐포끌어모으기 조작은 '폐보호 환기 및 폐포끌어모으기 조작군'에서만 시행하였고 일측폐환기를 시작한 지 10 분 후에 시작하였다. 기관지내시경을 사용하여 의존 폐(dependent lung)와 비의존 폐(non-dependent lung)의 기관지폐포세척(bronchoalveolar lavage)을 하였으며, 일측폐환기를 시작하기 전과 일측폐환기가 끝난 후에 두 번 시행하였다. 기관지폐포세척을 행한 시점과 동일한 때에 혈액을 채취하였다. 기관지폐포세척액과 혈장 내의 사이토카인(cytokine) TNF- α , IL-1 β , IL-6, IL-8, IL-10 의 농도를 측정하였다.

의존 폐의 기관지폐포세척액 내 TNF- α 농도는 일측폐환기가 끝난 후 '폐보호 환기군'에서 '폐보호 환기 및 폐포끌어모으기 조작군'에 비교하여 높았다 (P = 0.049). 그러나, 의존 폐의 기관지폐포세척액 내 IL-1 β , IL-6, IL-8, IL-10 농도는 일측폐환기가 끝난 후 세 군간 유의미한 차이가 없었다. 비의존 폐의 기관지폐포세척액 내 TNF- α , IL-1 β , IL-6, IL-8, IL-10 농도는 세 군간 유의미한 차이가 없었다. 혈액 내 IL-10 농도는 일측폐환기가 끝난 후 '전통적인 환기군'에서 '폐보호 환기 및 폐포끌어모으기 조작군'에 비교하여 높았다 (P = 0.027).

폐포끌어모으기 조작을 폐보호 환기와 함께 사용하였을 때 비디오흉강경을 이용한 폐엽 절제술을 받는 환자에서 의존 폐의 염증반응이 폐보호 환기만을 적용했을 때에 비교하여 감소하였다. 또한, 폐포끌어모으기 조작을 폐보호

환기와 함께 사용하면 전신 염증반응이 전통적인 환기에 비해서 감소하였다. 따라서, 비디오흉강경을 이용한 폐엽 절제술을 받는 환자에서 폐보호 환기와 함께 폐포끌어모으기 조작을 시행하여 국소 및 전신 염증반응을 줄일 수 있을 것으로 기대된다.

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키워드: 염증, 일측폐환기, 폐수술요법, 환기

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