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Effects of hypercarbia on arterial oxygenation during one-lung ventilation: prospective randomized crossover study

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Background: This study aimed to evaluate the effects of hypercarbia on arterial oxygenation during one-lung ventilation (OLV).

Methods: Fifty adult patients undergoing elective video-assisted thoracoscopic lobectomy or pneumonectomy were enrolled. Group I patients (n = 25) were first maintained at normocarbia ($PaCO_2$: 38-42 mmHg) for 30 min and then at hypercarbia (45–50 mmHg). In Group II patients (n = 25), $PaCO_2$ was maintained in the reverse order. Arterial oxygen partial pressure (PaO_2), respiratory variables, hemodynamic variables, and hemoglobin concentration were compared during normocarbia and hypercarbia. Arterial O_2 content and O_2 delivery were calculated.

Results: PaO_2 values during normocarbia and hypercarbia were 66.5 ± 10.6 and 79.7 ± 17.3 mmHg, respectively (mean difference: 13.2 mmHg, 95% CI for difference of means: 17.0 to 9.3, P < 0.001). SaO_2 values during normocarbia and hypercarbia were 92.5 ± 4.8% and 94.3 ± 3.1% (P = 0.009), respectively. Static compliance of the lung (33.0 ± 5.4 vs. 30.4 ± 5.3 ml/cmH$_2$O, P < 0.001), arterial O_2 content (15.4 ± 1.4 vs. 14.9 ± 1.5 ml/dl, P < 0.001) and O_2 delivery (69.9 ± 18.4 vs. 65.1 ± 18.1 ml/min, P < 0.001) were significantly higher during hypercarbia than during normocarbia.

Conclusions: Hypercarbia increases PaO_2 and O_2 carrying capacity and improves pulmonary mechanics during OLV, suggesting that it may help manage oxygenation during OLV. Therefore, permissive hypercarbia may be a simple and valuable modality to manage arterial oxygenation during OLV.

Keywords: Arterial oxygen partial pressure; Carbon dioxide; Hypercarbia; One-lung ventilation; Shunt; Thoracic surgery.

Introduction

Presently, many thoracic surgeries require one-lung ventilation (OLV) to improve the operation field and expedite the operation. During OLV, maintenance of adequate arterial oxygenation is a major concern to anesthesiologists. In previous studies, 4–27% of patients undergoing OLV developed arterial hypoxemia [1–3]. Because a collapsed lung is not ventilated but perfused, a transpulmonary shunt is inevitably developed, which leads to impairment of oxygenation. In addition, atelectatic and hypoventilated areas are increased in the dependent ventilated lung by the positional effects during thoracic surgery with OLV in the lateral position, which contributes to ventilation/perfusion mismatch and decreases arterial oxygen partial pressure (PaO_2).

Hypoxic pulmonary vasoconstriction (HPV) is a physiologic mechanism that decreas-

es blood flow to hypoxic or atelectatic lung regions by arteriolar vasoconstriction via a pathway involving nitric oxide and/or cyclooxygenase synthesis inhibition [4]. HPV diverts pulmonary blood flow from poorer ventilated areas to better areas of the lung, reducing the shunt fraction, and improving oxygenation [5,6]. Although HPV reduces shunt flow, 15–40% of pulmonary blood shunts to the left heart during OLV [7]. Recommendations to prevent arterial hypoxemia during OLV include the use of high inspired fraction of oxygen (FiO_2), application of positive end-expiratory pressure (PEEP) to the dependent lung, and continuous positive airway pressure (CPAP) to the non-dependent lung [8]. However, these techniques are inadequate for maintaining adequate oxygenation in some patients undergoing OLV.

In clinical practice, many patients have hypercarbia due to decreased minute ventilation and increased dead-space ventilation during OLV compared to two-lung ventilation (TLV). Carbon dioxide (CO_2) is a potent vasodilator in cerebral and systemic circulation [9,10]. However, the effect of CO_2 on pulmonary circulation is unclear. In previous studies [10–14], the effects of CO_2 on pulmonary vessels varied by the experiment species and pulmonary vascular tone. Most previous studies were conducted on ventilated lungs, but studies OLV subjects are rare. If CO_2 dilates the pulmonary artery in the ventilated lung or constricts the pulmonary artery in the non-ventilated lung, then hypercarbia may increase arterial oxygenation during OLV. If CO_2 has the opposite effects, then hypercarbia should be avoided.

We hypothesized that hypercarbia increases arterial oxygenation compared to normocarbia during OLV. The primary purpose of this prospective, randomized crossover study was to evaluate the effects of hypercarbia on arterial oxygenation during OLV.

Materials and Methods

The clinical research was done following the ethical principles for medical research involving human subjects in accordance with the Helsinki Declaration 2013.

This prospective, randomized crossover study was approved by the Institutional Review Board of Chonbuk National University Hospital and registered with the WHO International Clinical Trials Registry Platform (KCT0003185). Written informed consent was obtained from all participants. Fifty adult patients who were assigned American Society of Anesthesiologists physical status I or II, and who underwent elective video-assisted thoracoscopic lobectomy or pneumonectomy due to lung cancer were enrolled in the study. Patients who presented cardiac arrhythmia, heart failure, chronic obstructive pulmonary disease, restrictive

pulmonary disease or increased intracerebral pressure were excluded. Arterial oxygenation can be greatly affected by surgical process such as ligation of the pulmonary vessels in the collapsed surgical lung. Therefore, we divided the patients into two groups by order of intervention, although it was a crossover comparison. After initiation of OLV, Group I patients were first maintained at normal arterial CO_2 partial pressure (normocarbia, $PaCO_2$: 38–42 mmHg) for 30 min (OLV-1) and then at high $PaCO_2$ (hypercarbia, 45–50 mmHg) for 30 min (OLV-2). In Group II patients, $PaCO_2$ was maintained in the reverse order (OLV-1, hypercarbia; and OLV-2, normocarbia). Subjects were randomly assigned using a computer-generated block randomization scheme to one of two groups (1 : 1 allocation ratio).

The anesthetic regimen was standardized for all patients. After placement of the electrocardiogram, pulse oximetry, non-invasive blood pressure, bispectral index (BIS) and peripheral nerve stimulator, anesthesia was induced with 1.0–1.5 mg/kg propofol, 4–6 ng/ml effect-site concentration of remifentanil, and 1.0 mg/kg rocuronium. Remifentanil was administered using a Minto model effect-site target-controlled infusion pump (Orchestra® Base Primea, Fresenius Vial, France). Patients were manually ventilated using a face mask with sevoflurane (4.0 vol% in 50% oxygen) until a train-of-four count of 0 in the peripheral nerve stimulator was obtained. Female and male patients were intubated with a 35 or 37 Fr. and 37 or 39 Fr. left-sided double-lumen tube (Shiley™ Endobronchial tube left, Covidien, USA), respectively. The double-lumen tube was positioned using a fiberoptic bronchoscope. After induction of anesthesia, a 20-gauge arterial catheter was inserted into the brachial artery in the non-dominant hand. The brachial artery catheter was connected to the FloTrac™ transducer (Edwards Lifesciences, USA) coupled to both an anesthesia workstation (Primus Infinity® Empowered, Dräger, Germany) and EV1000™ (software version 1.5, Edwards Lifesciences) for hemodynamic measurements, including invasive blood pressure (IBP), cardiac index (CI), stroke volume variation (SVV), and systemic vascular resistance index (SVRI). The right subclavian vein was catheterized under ultrasono-guide for intravenous fluid line, central venous blood gas analysis, and central venous pressure (CVP) measurement. Pressure transducers were zeroed at the cardiac level to atmospheric pressure.

Lungs were mechanically ventilated with 0.5 of FiO_2 using a tidal volume (TV) of 6–8 ml/kg predicted body weight, an inspiratory to expiratory ratio of 1 : 2, an inspiratory pause of 25% of total inspiration time, and 5 cmH_2O of PEEP. The ventilatory rate was adjusted to maintain normocapnia. Anesthesia was maintained with sevoflurane and remifentanil. Fresh gas flow was fixed 3 L/min. The end-expiratory sevoflurane concentration was fixed

to 1.0 vol%. Arterial blood pressure was kept within 20% of preanesthetic values by adjustment of remifentanil concentration. Initially, the end-tidal CO_2 ($ETCO_2$) and BIS value were maintained at 35–38 mmHg and 40–60, respectively. If the patient showed > 60 BIS value, the patient received midazolam and was excluded from the data analysis.

After changing to the lateral position, the double-lumen tube position was reconfirmed using a fiberoptic bronchoscope. An arterial and central venous blood sample was obtained in the lateral position with two-lung ventilation after an alveolar recruit maneuver. After arterial blood gas analysis, the difference between $PaCO_2$ and $ETCO_2$ was evaluated. During OLV, TV was not changed. The FiO_2 was initially set at 0.5 and adjusted to maintain arterial O_2 saturation above 90%. The FiO_2 was not changed during the study period. If the patients showed pulse oximetric oxygen saturation (SpO_2) lower than 90% in FiO_2 0.5, the FiO_2 was increased and the patients were excluded from the data analysis. The ventilatory rate was adjusted to maintain the preset target $PaCO_2$. In all patients, normocarbia and hypercarbia periods were stable for 30 min because HPV reaches a plateau by 20–30 min [15,16].

The primary endpoint of the study was PaO_2 in normocarbia and hypercarbia during OLV. The PaO_2 was recorded at TLV and at normocarbia and hypercarbia during OLV. At the time of measurement, the following respiratory and hemodynamic variables were recorded: expiratory TV, ventilatory rate, peak inspiratory pressure (P_{IP}), plateau pressure (P_{PL}), IBP, CVP, CI, SVV, and SVRI. The dynamic (C_{dyn}) and static compliances (C_{stat}) were calculated using the following equations: $C_{dyn} = TV/ P_{IP} - PEEP$ and $C_{stat} = TV/ P_{PL} - PEEP$. Arterial blood gas, hemoglobin (Hg) concentration, and lactate concentration were also recorded. Central venous blood gas analysis was performed to measure oxygen partial pressure ($PcvO_2$) and saturation ($ScvO_2$) of central venous blood. Arterial O_2 content (CaO_2) was calculated by the following equation: $CaO_2 = 1.39 \times Hg$ concentration $\times SaO_2 + 0.0031 \times PaO_2$. Oxygen delivery ($DO_2$) was calculated by the following equation: $DO_2 = CaO_2 \times CO$.

Statistical analysis

The sample size was predetermined by paired t-test sample size test using SigmaPlot 13.0 (Systat Software Inc., USA) based on the assumption that a pilot study of ten patients for PaO_2 difference between hypercarbia and normocarbia during OLV, which was the primary endpoint, showed an average of 10 mmHg and a standard deviation of 18 mmHg. For PaO_2 difference, a value of ≥ 10 mmHg was considered as clinically significantly different. It

was determined that 34 patients were required to obtain a difference in mean PaO_2 of 10 mmHg for an expected standard deviation of 20 mmHg with a significance level of 0.05 (α = 0.05) and a power of 80% (β = 0.20). To allow for attrition, the sample size was increased to 50 patients.

The PaO_2 measured for OLV-1 and OLV-2 were analyzed via linear mixed-effects (LME) modeling using SPSS 23.0 (IBM Corp., USA). The linear mixed model included the variables id (random effect), presence of hypercarbia (OLV-1 and OLV-2), the sequence of ventilation (OLV-1 first versus OLV-2 first) and interaction between the presence of hypercarbia and the sequence of ventilation. The LME modeling produced a restricted maximum likelihood estimation fit. The LME modeling was used to assess whether there was a differential carryover effect of the first given treatment. Patient and clinical characteristics were analyzed with unpaired t-tests or Chi-square tests. The blood gas analysis, hemodynamic variables, and respiratory variables were compared with unpaired t-tests between Group I and II. Data are presented as the mean ± SD. The α value adjustment with Bonferroni correction was made to compensate for multiple comparisons within primary outcomes. The α value was adjusted to 0.016 instead of 0.05. The P values were compared with this adjusted α value in interpreting primary outcome measures. Otherwise, P values < 0.05 indicated statistical significance.

Results

Of the 50 allocated surgical patients, five patients, whose pulmonary artery was ligated before final measurement, and five patients, who showed arterial oxygen saturation less than 90% despite FiO_2 1.0, thus, requiring CPAP to the non-dependent lung, were excluded from the analysis (Fig. 1). During OLV, 40, four, and six patients were maintained with FiO_2 0.5, 0.8, and 1.0, respectively. No patient required transfusion during the operation. The demographic and preoperative clinical characteristics of the patients are shown in Table 1. The differential carryover effect of a preceding ventilation technique over the following ventilation technique and interaction between presence of hypercarbia and the sequence of ventilation were statistically insignificant (P = 0.771 and P = 0.713).

The $PaCO_2$ values during normocarbia and hypercarbia were 38.8 ± 2.6 and 48.2 ± 2.4 mmHg (mean ± SD), respectively. In both groups, PaO_2 was significantly decreased to convert from TLV to OLV, but PaO_2 was higher during hypercarbia than normocarbia. PaO_2 values during normocarbia and hypercarbia were 66.5 ± 10.6 and 79.7 ± 17.3 mmHg, respectively, (mean difference: 13.2 mmHg, 95% CI for difference of means: 17.0 to 9.3, P
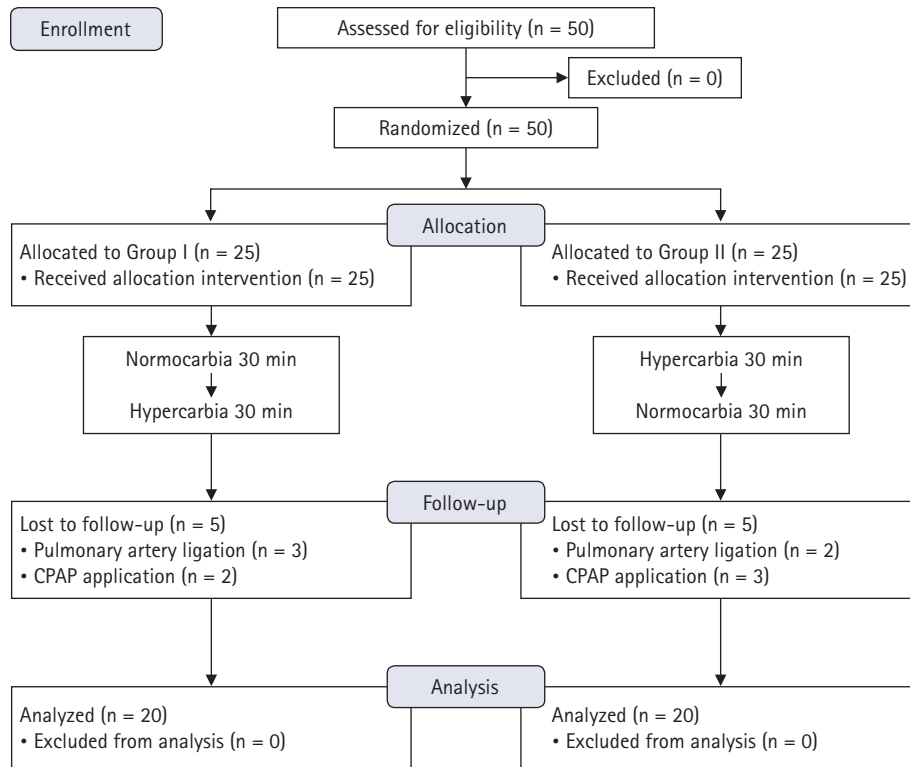
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Fig. 1. CONSORT flow diagram. Group I patients were maintained at normocarbia (PaCO$_2$: 38–42 mmHg) after then maintained at hypercarbia (PaCO$_2$: 45–50 mmHg). In Group II patients, PaCO$_2$ were maintained in the reverse order. CPAP: continuous positive airway pressure.

Allocation

Allocated to Group I (n = 25)
• Received allocation intervention (n = 25)

Normocarbia 30 min / Hypercarbia 30 min

Allocated to Group II (n = 25)
• Received allocation intervention (n = 25)

Hypercarbia 30 min / Normocarbia 30 min

Follow-up

Lost to follow-up (n = 5)
• Pulmonary artery ligation (n = 3)
• CPAP application (n = 2)

Lost to follow-up (n = 5)
• Pulmonary artery ligation (n = 2)
• CPAP application (n = 3)

Analysis

Analyzed (n = 20)
• Excluded from analysis (n = 0)

Analyzed (n = 20)
• Excluded from analysis (n = 0)

Table 1. Demographic Data and Clinical Characteristics

	Group I (n = 20)	Group II (n = 20)	P value
Age (yr)	66.0 ± 8.0	62.7 ± 7.8	0.195
Sex (F/M)	7/13	7/13	0.740
Height (cm)	161.1 ± 6.6	162.5 ± 8.5	0.578
Weight (kg)	61.4 ± 9.2	66.4 ± 11.9	0.145
ASA PS (1/2)	3/17	3/17	0.658
Operation site (Left/Right)	5/15	7/13	0.730
Preoperative lung function			
FVC (L)	3.3 ± 0.6	3.3 ± 0.8	0.911
FEV1 (L)	2.5 ± 0.5	2.5 ± 0.6	0.823
FEV1/FVC (%)	76.2 ± 5.9	75.8 ± 8.1	0.856

Values are presented as mean ± SD or number of patients. Group I patients were first maintained at normocarbia (PaCO$_2$: 38–42 mmHg), then maintained at hypercarbia (PaCO$_2$: 45–50 mmHg). In Group II patients, PaCO$_2$ was maintained in the reverse order. ASA PS: American society of anesthesiologists physical status, FVC: functional vital capacity, FEV$_1$: forced expiratory volume for 1 sec.

Fig. 2. Arterial O$_2$ partial pressure was increased by changing the arterial CO$_2$ partial pressure (PaCO$_2$) from normocarbia to hypercarbia in 37 of 40 individual patients (93%). Group I patients were first maintained at normocarbia (PaCO$_2$: 38–42 mmHg), then maintained at hypercarbia (PaCO$_2$: 45–50 mmHg). In Group II patients, PaCO$_2$ were maintained in the reverse order.

0.04, but 7.35 ± 0.04 during hypercarbia (P < 0.001).

Although fixed TV was applied, P$_{IP}$ and P$_{PL}$ were significantly lower during hypercarbia than normocarbia in both groups. Based on these results, C$_{dyn}$ and C$_{stat}$ were higher during hypercarbia than normocarbia (Table 2). The hemodynamic variables, including IBP, HR, CI, CVP, SVV, and SVRI, as well as the Hg con-

< 0.001). Arterial O$_2$ saturation (SaO$_2$) values during normocarbia and hypercarbia were 92.5 ± 4.8% and 94.3 ± 3.1% (P = 0.009), respectively. For individual patients, PaO$_2$ was increased by changing the PaCO$_2$ from normocarbia to hypercarbia in 37 of 40 patients (93%) (Fig. 2). During normocarbia, pH was 7.42 ±
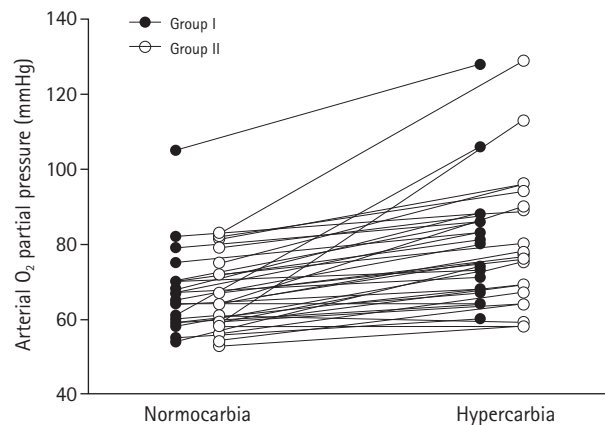
Table 2. Respiratory Variables during One-Lung Ventilation

	Normocarbia	Hypercarbia	P value
Tidal volume (ml)	413.0 ± 50.4		
Ventilatory rate (beats/min)	15.3 ± 2.5	9.4 ± 1.8	< 0.001
Peak inspiratory pressure (cmH$_2$O)	24.8 ± 3.7	21.5 ± 2.5	< 0.001
Plateau pressure (cmH$_2$O)	18.9 ± 2.3	17.7 ± 1.9	< 0.001
Dynamic compliance (ml/cmH$_2$O)	21.3 ± 3.2	25.3 ± 3.4	< 0.001
Static compliance (ml/cmH$_2$O)	30.4 ± 5.3	33.0 ± 5.4	< 0.001

Values are presented as mean ± SD.

Table 3. Bispectral Index, Hemoglobin Concentration, and Hemodynamic Variables during One-Lung Ventilation

	Normocarbia	Hypercarbia	P value
BIS	51.5 ± 8.7	52.4 ± 7.7	0.382
Hemoglobin concentration (g/dl)	11.4 ± 1.1	11.5 ± 1.1	0.179
MAP (mmHg)	79.6 ± 11.5	79.7 ± 11.3	0.899
HR (beats/min)	68.2 ± 12.0	68.7 ± 11.5	0.575
CI (L/min/m^2)	2.6 ± 0.7	2.7 ± 0.7	0.170
CVP (mmHg)	8.6 ± 3.6	8.8 ± 3.9	0.183
SVV (%)	7.0 ± 2.9	7.3 ± 2.9	0.252
SVRI (dyne·sec·cm^5/m^2)	2150.5 ± 552.3	2120.2 ± 606.4	0.554
CaO$_2$ (ml/dl)	14.9 ± 1.5	15.4 ± 1.4	< 0.001
DO$_2$ (ml/min)	65.1 ± 18.1	69.9 ± 18.4	< 0.001

Values are presented as mean ± SD. BIS: bispectral index, MAP: mean arterial pressure, HR: heart rate, CI: cardiac index, CVP: central venous pressure, SVV: stroke volume variation, SVRI: systemic vascular resistance index, CaO$_2$: arterial oxygen content, DO$_2$: oxygen delivery.

Table 4. Oxygen Partial Pressure (PcvO$_2$) and Saturation (ScvO$_2$) of Central Venous Blood and Lactate Concentration

	Normocarbia	Hypercarbia	P value
PcvO$_2$ (mmHg)	38.8 ± 6.3	44.3 ± 4.7	< 0.001
ScvO$_2$ (%)	70.1 ± 7.4	74.9 ± 5.3	< 0.001
Lactate concentration (mmol/L)	1.1 ± 0.4	1.0 ± 0.4	< 0.001

Values are presented as mean ± SD.

centration and BIS during normocarbia, were comparable to hypercarbia in both groups. However, CaO$_2$ and DO$_2$ were significantly higher during hypercarbia than during normocarbia (Table 3). PcvO$_2$, ScvO$_2$, and lactate concentration were significantly different between normocarbia and hypercarbia in both groups (Table 4).

Discussion

Although OLV provides optimum surgical conditions during thoracic surgery, it is associated with impairment of gas exchange. In addition to arterial hypoxemia, hypercarbia is commonly developed during OLV. Because atelectasis may readily occur in the dependent lung, the application of PEEP is necessary to prevent atelectasis during OLV. Increased lung volume and PEEP elevate airway pressure. Increased airway pressure may impede perfusion of the dependent lung, leading to dead-space ventilation. In-

creased dead-space ventilation may cause hypoventilation and hypercarbia [17]. Additionally, anesthesiologists are apt to reduce TV to prevent increased PIP during OLV. For these reasons, hypercarbia is common in arterial blood gas analysis during OLV in clinical practice. In the present study, moderate hypercarbia increased PaO$_2$, SaO$_2$, CaO$_2$, DO$_2$, PcvO$_2$, ScvO$_2$, C$_{dyn}$, and C$_{stat}$ but decreased airway pressure and lactate concentration. These results were considered as positive effects on gas exchange during OLV.

The main cause of hypoxemia during OLV is the intrapulmonary shunt through the non-dependent, non-ventilated lung. The alveolar collapse in the non-dependent lung activates HPV, leading to an increase in resistance to flow in the dependent pulmonary artery, thus diverting more perfusion to the ventilated, dependent lung. In the present study, increased PaO$_2$ reflected as decreased intrapulmonary shunt during hypercarbia. Although the mechanism was not clarified, hypercarbia may increase pulmo-

nary vascular resistance in the non-dependent lung or decrease pulmonary vascular resistance in the dependent, ventilated lung. A pulmonary vasoconstrictor almitrine enhances HPV and prevents OLV-induced decrease in PaO_2 [18,19]. Inhaled nitric oxide has selective pulmonary vasorelaxation in the dependent, ventilated lung during OLV. Therefore, both almitrine and nitric oxide increase arterial oxygenation during OLV. Chuang et al. [11,20] reported that inhaled CO_2 has vasodilatory effects and reverses pulmonary hypertension induced by hypoxia in isolated perfused rat lungs. Previous studies have indicated that CO_2 is a mild vasoconstrictor during basal tone condition but is a potent vasodilator at high pulmonary vascular resistance [14,21–24]. Unfortunately, the present results did not provide a mechanism because pulmonary vascular resistance and pulmonary blood flow were not measured in both lungs.

In the present study, the pH value was decreased by 0.06–0.08 during hypercarbia compared to normocarbia. A low pH shifts the oxyhemoglobin dissociation curve to the right by Bohr effects. As the curve shifts to the right, the SaO_2 for given PaO_2 decreases, i.e., decreased Hg affinity for O_2 [25]. As the pH decreases from 7.40 to 7.35, like the decrease in the present study, Hg releases O_2 more readily to tissues, although oxygen uptake is reduced from the alveoli. Respiratory acidosis can potentiate HPV [26]. Although the increase of PaO_2 was not excluded by the effects of acidosis in our results, hypercarbia provides positive effects to the management of arterial hypoxemia during OLV in conjunction with increased CaO_2 and DO_2. Accordingly, blood lactate concentration was lower during hypercarbia than normocarbia in both groups, although the values were considered within normal ranges.

In the present study, P_{IP} and P_{PL} were significantly lower during hypercarbia than normocarbia in both groups although TV was not changed. Based on these results, C_{dyn} and C_{stat} were higher during hypercarbia than normocarbia. These results may reflect that increased CO_2 relaxes bronchiole and lung parenchyma. CO_2-dependent regulation of lung compliance and ventilation-perfusion matching have been explained by pH- and CO_2-dependent changes. Previous studies have reported that CO_2 relaxes lung parenchyma and increases lung compliance [27,28]. These hypercarbia effects provide positive effects on ventilation/perfusion matching and management of arterial hypoxemia during OLV. However, increased lung compliance during hypercarbia may be influenced by the respiratory rate change. The results require further studies to clarify the mechanism.

The key points of lung protective mechanical ventilation strategies of acute respiratory distress syndrome (ARDS) are low TV and increased PEEP. The unintended consequences of the protective ventilation are hypercapnia and hypercapnic acidosis owing to a reduction in minute ventilation and a worsening of ventilation/perfusion mismatch. Previously, acidosis has been permitted as an adverse side effect of protective ventilation. However, several studies have shown the ability of CO_2 to protect against lung injury and repair independently of low TV [29–33]. The concept has been changing from permissive hypercapnia to therapeutic hypercapnia in ARDS. OLV is associated with a high rate of postoperative pulmonary complications, and OLV is currently recognized as a risk factor for acute lung injury [34,35]. Although the pathophysiologic mechanism of acute lung injury after OLV is different for the ventilated and non-ventilated lung, hypercarbia may help prevent and/or repair acute lung injury after OLV.

There are two limitations to the current study. First, the results may be affected by the surgical process. To exclude this effect, patients were divided into two groups in a different order, although the study was designed for crossover comparison. Moreover, the study was discontinued if the pulmonary artery was ligated before the final measurement. Nevertheless, the results could be affected by operation. Therefore, it would be better if the study was performed before the operation. Second, the present results did not provide a mechanism that hypercarbia increased PaO_2 during OLV because the pulmonary vascular resistance and pulmonary blood flow were not measured in both lungs, as mentioned above. Further studies are needed to confirm the mechanism of hypercarbia.

In conclusion, hypercarbia increases PaO_2 and O_2 carrying capacity and improves pulmonary mechanics without significant hemodynamic changes during OLV. Thus, it may help manage oxygenation during OLV. Therefore, permissive hypercarbia may be a simple and valuable modality to manage arterial oxygenation during OLV.

Acknowledgements

Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

Author Contributions

Jun Ho Lee (Methodology; Writing – original draft)
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