Original Article

Effects of volume-controlled ventilation *vs.* pressure-controlled ventilation on respiratory function and inflammatory factors in patients undergoing video-assisted thoracoscopic radical resection of pulmonary carcinoma

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Background: The best ventilation approach for patients undergoing video-assisted thoracic surgery (ATS) for pulmonary carcinoma remains undefined. This study aimed to assess hemodynamics, airway pressure, arterial blood gas, and inflammatory factors in patients undergoing VATS for pulmonary carcinoma under volume-controlled ventilation (VCV) or pressure-controlled ventilation (PCV).

Methods: This was a prospective study of 60 patients with pulmonary carcinoma treated at a tertiary center in 2015–2016. The subjects were randomized to the VCV or PCV group after anesthesia and total lung ventilation (TLV). Hemodynamics and blood gas parameters were compared between the two groups pre-OLV (one-lung ventilation) (T1) and after 30 (T2), 60 (T3), and 120 (T4) minutes of OLV. Radial artery blood was collected to measure interleukin (IL)-6, IL-10, and tumor necrosis factor (TNF)-α levels.

Results: Hemodynamic and blood gas parameters were similar between the two groups (all P>0.05). During OLV, airway resistance (RAW) was significantly lower in the PCV group compared with the VCV group at T2 (26.0±3.8 *vs.* 29.9±7.3 cmH_2O/L/s), T3 (26.0±3.7 *vs.* 30.2±7.7 cmH_2O/L/s), and T4 (25.8±4.1 *vs.* 29.6±6.7 cmH_2O/L/s). Similar trends were found for peak pressure (Ppeak) and plateau pressure (Pplat). Mean pressure (Pmean) was similar between the two groups. Compared with the PCV group, TNF-α and IL-6 levels in the VCV group were significantly increased (all P<0.05). The levels of the anti-inflammatory mediator IL-10 were higher in the PCV group compared with the VCV group.

Conclusions: PCV for OLV during radical resection of pulmonary carcinoma by VATS could reduce Ppeak and downregulate pro-inflammatory factors, likely decreasing airway injury.

Keywords: Volume-controlled ventilation (VCV); pressure-controlled ventilation (PCV); hemodynamics; arterial blood gas; inflammation

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Introduction

With the improvement of thoracoscopy and minimally invasive treatment technologies, radical resection of pulmonary carcinoma by video-assisted thoracic surgery (VATS) is currently a mature technology. In order to create an appropriate visual field for surgery, radical resection of pulmonary carcinoma by VATS requires one-lung ventilation (OLV); however, during OLV, increased intrapulmonary shunt and airway pressure could induce or aggravate lung injury. During OLV, the patients are prone to suffer from hypoxemia due to abnormal pulmonary blood oxygenation, with an incidence of approximately 5% (1).

Volume-controlled ventilation (VCV) and pressure-controlled ventilation (PCV) are the two ventilation modes often used for OLV. VCV ensures stable and precise ventilation volume, but higher peak pressure (Ppeak) may lead to barotrauma and non-uniform gas distribution. On the other hand, PCV improves arterial oxygenation and has a rapidly decelerating flow pattern, but is also associated with lung injury due to traction forces on the lung and alveoli (2). The best approach for OLV remains controversial (3-10).

Inadequate mechanical ventilation-associated hypoxemia could induce the release of multiple cytokines and inflammatory mediators, causing ventilator-associated lung injury (11). Circulating neutrophils are activated by these cytokines and inflammatory factors, leading to delayed apoptosis and continuous release of proteolytic enzymes, reactive oxygen species (ROS), and additional inflammatory factors (11-13). Increased local inflammation may also lead to epithelial cell apoptosis and subsequent immune cell recruitment (14). OLV leads to increased levels of interleukin (IL)-1β, IL-6, and IL-8, which are characteristic of inflammatory responses (15).

Based on the above, the aim of the present study was to assess which ventilation mode (VCV or PCV) is more advantageous in terms of respiratory pressure, arterial blood oxygenation, and inflammatory factors, during OLV for patients undergoing radical resection of right pulmonary carcinoma.

Methods

Patients

This was a prospective pilot study of 60 patients with pulmonary carcinoma consecutively enrolled and treated at the Jiangsu Cancer Hospital between May 2015 and March 2016. Inclusion criteria were: (I) VATS radical resection of right pulmonary carcinoma; (II) American Society of Anesthesiologists physical status (ASA) I–II; (III) at least 2 h of OLV. Exclusion criteria were: (I) any immune, endocrine, neurologic, or psychological disease; (II) history of pulmonary lobectomy; (III) serious cardiac, lung, renal, or hepatic dysfunction; (IV) tracheotomy; or (V) forced exhaled volume in 1 second (EEV1) <70%.

The protocol was approved by the ethics committee of the Jiangsu Cancer Hospital {No. NJU [2017] 549}. Informed consent was obtained from each patient or from their nearest relatives.

Grouping and intervention

Before anesthesia, all patients received 0.1 g of phenobarbital and 0.5 mg of atropine by intramuscular injection. The right internal jugular vein was cannulated and a radial artery puncture made under local anesthesia. General anesthesia was induced in all patients with midazolam (Nhwa Pharma. Co., Jiangsu, China) (0.08 mg/kg), propofol (1–2 mg/kg) (Corden Pharma S.P.A., Caponago, Italy), fentanyl (3–4 μg/kg) (Yichang HumanWell Pharmaceutical Co., Ltd., Hubei, China), and cisatracurium (0.15 mg/kg) (Jiang Su Heng Rui Medicine Co., Ltd., Lianyungang, China). The patients were intubated with double-lumen endobronchial tubes (Covidien LLC., Cornamaddy, Ireland) (#37–39 in males and #35–37 in females). Anesthesia was maintained with a continuous infusion of cisatracurium (7–10 μg/kg/min), propofol (6–12 mg/kg/min), and remifentanil (0.1–0.2 μg/kg/min). Intravenous fentanyl 50–100 μg was administered to maintain arterial pressure at ±20% of baseline level. The tube position was adjusted with a fiber bronchoscope, and total lung ventilation-VCV (TLV-VCV) was performed using an anesthesia machine (Ohmeda 7900; Datex Ohmeda, Helsinki, Finland). Parameters were: tide volume, 8 mL/kg; respiratory rate, 12/min; inspiratory/expiratory ratio (I:E), 1:2; fraction of inspired oxygen (FiO_2), 1.0; and oxygen flow, 1 L/min.

After TLV and before OLV, the patients were randomized to the VCV or PCV group using sealed sequential envelopes prepared by a statistician using a random number table. During OLV, the tidal volume (VT) in the VCV group was 6 mL/kg. In the PCV group the airway pressure was adjusted to achieve a VT of 6 mL/kg, and the PCV mode was used for ventilation. $P_{ET}CO_2$ was maintained at 30–45 mmHg. The PEEP value was 0 in both

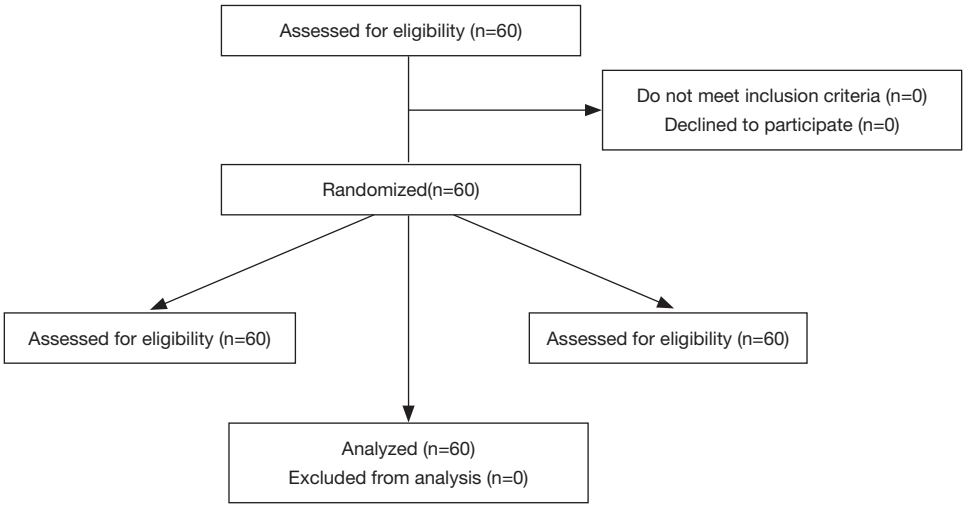

Figure 1 Study flowchart.

Table 1 Patient characteristics

Parameter	PCV	VCV	P
Age (years)	54.1±8.6	55.7±9.4	>0.05
Gender (male)	15 (50.0%)	12 (40.0%)	>0.05
Height (cm)	165±6	167±7	>0.05
Weight (kg)	63.8±7.0	66.2±11.0	>0.05

PCV, pressure-controlled ventilation; VCV, volume-controlled ventilation.

groups. FiO_2 was 1.0 during OLV, with an oxygen flow rate of 1 L/min. All procedures were carried out by the same group of surgeons.

Measurements

Electrocardiogram (ECG), heart rate (HR), and arterial pressure were continuously monitored. Arterial blood oxygen saturation (SpO_2), Ppeak, mean pressure (Pmean), plateau pressure (Pplat), airway resistance (RAW), partial oxygen pressure (PaO_2), and end tidal CO_2 pressure ($P_{ET}CO_2$) were recorded at (I) 10 min after TLV and before OLV (T1); (II) 30 min after OLV (T2); (III) 60 min after OLV (T3); and (IV) 120 min after OLV. Serum IL-6, IL-10, and tumor necrosis factor (TNF)-α levels were measured in serum from fasting radial artery blood (3 mL) collected at T1, T2, T3, and T4, respectively. ELISA kits (Nanjing Jiancheng Biotech, Nanjing, China) were used according to the manufacturer's instructions.

Statistical analysis

Continuous variables underwent testing for normality using the Shapiro-Wilk test. Normally distributed continuous data were expressed as mean ± standard deviation (SD) and analyzed by Student's t test or repeated measure ANOVA with the Tukey's post hoc test, as appropriate. Categorical data were presented as frequencies and analyzed by the Chi-square test. Data were analyzed with SPSS 19.0 (IBM, Armonk, NY, USA). Two-sided $P<0.05$ was considered statistically significant.

Results

Patients

Figure 1 presents the study flowchart. Sixty patients were assessed for eligibility and randomized 1:1 to the PCV and VCV groups, respectively. No patient was excluded. There were 33 males and 27 females, aged from 18 to 70 years. There were no significant differences in gender, age, and weight between the two groups (all $P>0.05$) (*Table 1*).

Hemodynamics and arterial blood gas

There were no significant differences in hemodynamic and blood gas parameters between the two groups (all $P>0.05$) (*Table 2*). In addition, during OLV, no patient suffered from severe hypoxia (SpO_2 <90%). Anesthesia and surgery were successful in all patients. No incident or complication occurred.

Table 2 Hemodynamics and arterial blood gas parameters in the VCV and PCV groups

Parameter	Mode	T0	T1	T2	T3	T4
HR (bpm)	PCV	78.70±14.70	72.73±10.67	78.40±11.72	74.66±8.96	70.03±11.61
	VCV	73.33±11.99	70.96±13.90	73.93±13.10	71.0±12.10	67.40±10.38
DBP (mmHg)	PCV	77.48±8.58	71.31±10.28	68.51±10.51	71.00±7.04	74.93±7.07
	VCV	75.63±9.36	71.20±10.49	69.80±9.87	68.93±7.86	71.06±7.43
SBP (mmHg)	PCV	147.37±15.36	120.65±18.55	114.48±13.78	119.20±2.56	125.37±1.96
	VCV	145.70±15.83	123.80±22.83	120.90±17.06	120.43±2.77	124.83±2.78
$P_{ET}CO_2$ (mmHg)	PCV	–	37.50±3.35	37.30±3.34	37.26±3.75	35.26±3.73
	VCV	–	38.03±3.52	37.76±3.32	37.20±3.10	35.80±3.65
PaO_2 (mmHg)	PCV	86.93±30.92	448.33±72.50	167.76±89.01	262.36±108.41	401.93±91.77
	VCV	87.83±20.10	432.53±99.39	187.83±77.61	263.06±96.81	437.36±83.69
$PaCO_2$ (mmHg)	PCV	39.93±4.45	41.56±4.24	42.86±5.00	42.46±4.83	42.43±5.02
	VCV	38.63±7.85	41.96±3.87	43.73±3.73	44.03±5.13	43.30±8.38

PCV, pressure-controlled ventilation; VCV, volume-controlled ventilation; HR, heart rate; DBP, diastolic blood pressure; SBP, systolic blood pressure; $P_{ET}CO_2$, end tidal CO_2 pressure; PaO_2, partial oxygen pressure; $PaCO_2$, partial CO_2 pressure. All P>0.05.

Airway pressure and hospitalization times

During OLV, RAW was significantly lower in the PCV group compared with the VCV group at T2 (26.0±3.8 *vs.* 29.9±7.3 cmH_2O/L/s), T3 (26.0±3.7 *vs.* 30.2±7.7 cmH_2O/L/s), and T4 (25.8±4.1 *vs.* 29.6±6.7 cmH_2O/L/s). Similar trends were found for Ppeak and Pplat. However, Pmean values showed no significant differences at any time point between the two groups. Airway pressure data are detailed in *Table 3*. Meanwhile, hospitalization times were similar between the two groups, i.e., 8.467±1.14 and 8.433±1.10 days in the VCV and PCV groups, respectively (P>0.05).

Inflammatory factors

At T1, T3, and T4, TNF-α levels were 43.5±9.4, 43.9±14.7, and 44.8±1.9 pg/mL, respectively, in the PCV group; while 47.6±6.6, 51.7±9.1, and 52.5±1.7 pg/mL, respectively, were obtained in the VCV group. Similarly, IL-6 amounts at T1, T3, and T4 were 71.6±7.1, 71.0±10.0, and 71.9±6.8 pg/mL, respectively, in the PCV group; for 73.2±16.0, 77.1±8.8, and 77.9±8.8 pg/mL, respectively in the VCV group. The differences in the levels of these pro-inflammatory cytokines were statistically significant at T3 and T4 (all P<0.05). Meanwhile, there was a significant increase in IL-10 amounts in the PCV group compared with the VCV group at T4 (18.2±6.1 *vs.* 15.5±3.4 pg/mL, P<0.05). The levels of inflammatory factors are summarized in *Table 4*.

Discussion

The best ventilation approach for patients undergoing VATS for pulmonary carcinoma remains undefined (3-10). Therefore, this study aimed to assess hemodynamics, airway pressure, arterial blood gas, and inflammatory factors in patients undergoing VATS for pulmonary carcinoma under VCV or PCV. The results suggested that PCV for OLV during radical resection of pulmonary carcinoma by VATS could reduce Ppeak and the levels of pro-inflammatory factors, likely decreasing airway injury.

Licker *et al.* (11) indicated that high airway pressure during surgery is an independent risk factor for acute pulmonary injury. The objective of protective ventilation is to avoid overexpansion and collapse of the pulmonary alveoli, and to reduce shear stress induced lung injury by decreasing the VT and airway pressure (16-19). Considering the pathophysiological mechanism of lung injury during OLV, it was suggested that avoiding high airway pressure is necessary. Kim *et al.* (19) conducted a retrospective study comparing PCV and VCV during OLV in adult patients; the results indicated that PCV reduces the peak inspiratory pressure. In the present study, compared with VCV, the values of Ppeak, Pplat, and RAW were significantly decreased with the PCV mode. Montes *et al.* (20) showed

Table 3 Airway pressure between PCV and VCV

Parameters	Ventilation modes	T1	T2	T3	T4
Raw (cmH_2O/L/s)	PCV	15.1±3.6	26.0±3.8*	26.0±3.7$^{\Delta}$	25.8±4.1*
	VCV	15.1±3.2	29.9±7.3*	30.2±7.7$^{\Delta}$	29.6±6.7*
Ppeak (cmH_2O)	PCV	16.8±2.8	21.4±4.1	22.1±4.5*	21.8±4.6$^{\Delta}$
	VCV	16.7±3.1	24.6±4.5	25.0±4.2*	25.0±4.1$^{\Delta}$
Pplat (cmH_2O)	PCV	14.2±2.4	19.4±3.2$^{\Delta}$	20.3±3.7*	20.3±3.5*
	VCV	14.1±3.1	21.9±3.1$^{\Delta}$	22.6±3.0*	22.5±3.2*
Pmean (cmH_2O)	PCV	5.8±0.8	7.7±1.0	7.8±1.0	7.9±2.2
	VCV	6.3±1.2	8.1±1.2	8.2±1.1	8.1±1.2

*, P<0.05; $^{\Delta}$, P<0.01 *vs.* VCV group. PCV, pressure-controlled ventilation; VCV, volume-controlled ventilation; Ppeak, peak inspiratory pressure; Pplat, plateau pressure; Pmean, mean pressure.

Table 4 Inflammatory factors in the VCV and PCV groups

Cytokine	Ventilation mode	T1	T3	T4
TNF-α (pg/mL)	PCV	43.5±9.4	43.9±14.7*	44.8±1.9$^{\Delta}$
	VCV	47.6±6.6	51.7±9.1*	52.5±1.7$^{\Delta}$
IL-6 (pg/mL)	PCV	71.6±7.1	71.0±10.0*	71.9±6.8$^{\Delta}$
	VCV	73.2±16.0	77.1±8.8*	77.9±8.8$^{\Delta}$
IL-10 (pg/mL)	PCV	15.7±5.0	17.6±8.4	18.2±6.1*
	VCV	14.6±3.4	15.4±3.3	15.5±3.4*

*, P<0.05; $^{\Delta}$, P<0.01 *vs.* VCV group. PCV, pressure-controlled ventilation; VCV, volume-controlled ventilation; TNF-α, tumor necrosis factor α; IL, interleukin.

that in patients with normal preoperative lung function, the effect of arterial oxygenation is not significantly influenced by PCV or VCV, while Ppeak was lower in the PCV group, corroborating the present study. Assad *et al.* (21) showed that PCV is superior to VCV because it provides ventilation with lower Ppeak.

A study by Tugrul *et al.* (5) indicated that PCV could improve oxygenation. Pardos *et al.* (10) considered that compared with PCV, an equal VT in VCV during OLV does not affect early postoperative arterial oxygenation and arterial oxygenation during OLV. In the present study, no significant differences between VCV and PCV were found during OLV, indicating that PCV was not superior to VCV in improving oxygenation. Body position, anesthesia methods, anesthetics, and hypoxic pulmonary vascular contraction (HPV) are associated with hypoxemia during OLV (10). Indeed, high concentrations of inhaled anesthetics reduce HPV, promote pulmonary shunt, and decrease PaO_2. Meanwhile, some intravenous anesthetics, such as propofol and remifentanil, do not reduce HPV and affect the intrapulmonary shunt (10). Thoracic epidural anesthesia blocks the sympathetic nervous system, expands pulmonary blood vessels, inhibits HPV, and changes the ventilation blood flow ratio (22). In patients with normal pulmonary function, PCV has no significant preoperative impact on PaO_2 compared to VCV (9). Compared with VCV, PCV improves postoperative oxygenation in elderly patients with abnormal pulmonary function (3). In the present study, PCV was used to reduce airway pressure and intrapulmonary venous-to-arterial shunt. In addition, the patients received intravenous anesthesia. No patient had hypoxemia during the operation.

The activation of cytokines and their cascade reactions during OLV are the main mechanisms leading to lung injury. TNF-α is produced by alveolar macrophages and promotes inflammation (23), through release of cytokines

such as IL-6, leading to pulmonary tissue damage (24). During the inflammatory response induced by mechanical ventilation, the levels of TNF-α and IL-6 are correlated to the degree of lung injury (25,26). The present study demonstrated that TNF-α and IL-6 levels were higher with VCV compared with PCV. Since ventilation time was relatively short, the lack of effect on hemodynamics and blood gas could be due to this short period. Meanwhile, the compensatory anti-inflammatory functions of the human body can induce the release of endogenous anti-inflammatory mediators such as IL-10, which can reduce lung tissue damage (27). In the present study, IL-10 levels in patients with PCV were higher than in patients with VCV after 120 min of OLV. Although the above data suggest decreased airway injury in the PCV group, further studies are required for confirmation. The clinical relevance of differences in airway pressure and cytokine levels remains unclear, since hospitalization times were similar between the two groups.

The present study was not without limitations. The sample size was relatively small. In addition, the patients underwent a single type of surgery. Only a small panel of inflammatory factors were examined in blood samples, which may constitute a systematic bias. Additional studies should be performed in bronchoalveolar lavage fluid specimens.

In conclusion, PCV for OLV during radical resection of pulmonary carcinoma by VATS could reduce Ppeak and the levels of pro-inflammatory factors, likely decreasing airway injury. These results could provide some reference about the most appropriate ventilation mode for such patients.

Acknowledgements

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

Ethical Statement: The protocol was approved by the ethics committee of the Jiangsu Cancer Hospital {No. NJU [2017] 549}. Informed consent was obtained from each patient or from their nearest relatives.

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