Right Ventricular Function During One-Lung Ventilation: Effects of Pressure-Controlled and Volume-Controlled Ventilation

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Objectives: **To test the effects of pressure-controlled (PCV) and volume-controlled (VCV) ventilation during one-lung ventilation (OLV) for thoracic surgery on right ventricular (RV) function.**

Design: **A prospective, randomized, double-blind, controlled, crossover study.**

Setting: **A single university hospital.**

Participants: **Fourteen pairs of consecutive patients scheduled for elective thoracotomy.**

Interventions: **Patients were assigned randomly to ventilate the dependent lung with PCV or VCV mode, each in a randomized crossover order using tidal volume of 6 mL/kg, I: E ratio 1: 2.5, positive end-expiratory pressure (PEEP) of 5 cm H_2O and respiratory rate adjusted to maintain normocapnia.**

Measurements and Main Results: **Intraoperative changes in RV function (systolic and early diastolic tricuspid annular velocity (TAV), end-systolic volume (ESV), end-diastolic volume (EDV) and fractional area changes (FAC)), airway pressures, compliance and oxygenation index were recorded. The use of PCV during OLV resulted in faster systolic (10.1 ± 2.39 vs. 5.8 ± 1.67 cm/s, respectively), diastolic TAV (9.2 ± 1.99 vs. 4.6 ± 1.42 cm/s, respectively) ($p < 0.001$) and compliance and lower ESV, EDV and airway pressures ($p < 0.05$) than during the use of VCV. Oxygenation indices were similar during the use of VCV and PCV.**

Conclusions: **The use of PCV offers more improved RV function than the use of VCV during OLV for open thoracotomy. These results apply specifically to younger patients with good ventricular and pulmonary functions.**

KEY WORDS: Thoracic surgery, one-lung ventilation, pressure-controlled ventilation, volume-controlled ventilation, right ventricular function.

ONE-LUNG VENTILATION (OLV) provides excellent operative conditions for thoracic procedures but may impair cardiac and right ventricular (RV) function indices, increasing postoperative morbidity and mortality rates.[1–2] In the authors' previous study, they demonstrated that RV ejection fraction and cardiac index parameters were reduced significantly after the initiation of OLV due to concomitant increases in right ventricular afterload, stroke work, and end-diastolic volume augmented by increased airway pressures.[2] This may be harmful to patients with advanced obstructive lung diseases and those with pulmonary hypertension. Therefore, lowering airway pressures may enhance RV function.

Volume-controlled ventilation (VCV) is less commonly used for OLV during thoracic procedures because it may increase airway pressure, thus impeding RV function.[3] Pressure-controlled ventilation (PCV) may be useful during OLV in lowering airway pressure and intrapulmonary shunt.[4–5] Some investigators demonstrated improved oxygenation during the use of PCV for OLV,[4–5] whereas others found no significant difference in arterial oxygenation between PCV and VCV during OLV.[6–8]

The advantages of PCV over VCV during OLV include reductions in intrapulmonary shunts and mean and peak airway pressures,[9] with the latter limiting the risks of barotrauma and impaired RV function. The effects of PCV and VCV on RV function during OLV for thoracic surgery have not been studied yet.

The authors hypothesized that the use of PCV during OLV would be associated with better-preserved RV function than the use of VCV. The authors, therefore, compared the effects of PCV and VCV during OLV on RV function, respiratory mechanics, arterial oxygenation, and postoperative complications in patients scheduled for open thoracotomy.

METHODS

Following institutional review board (IRB) approval and written informed consent, 28 consecutive patients aged 18 to 65 years, (American Society of Anesthesiologists physical class [II–III]) scheduled for elective open thoracotomy, in which the duration of OLV was expected to exceed 1.5 hours, were included in this controlled, randomized, prospective, double-blind crossover study. The study was registered with www.clinicaltrials.gov (ref.NCT01763879).

Patients were excluded if they had decompensated cardiac disease (New York Heart Association class > II), forced vital capacity (FVC) or forced expiratory volume in 1 second (FEV_1) < 50% of predicted values, hepatic or renal disease, an absence of sinus rhythm such as atrial fibrillation and ventricular arrhythmias, mean pulmonary artery pressure > 30 mmHg, asthma, body mass index > 35 kg/m^2, or a previous history of pneumonectomy, bilobectomy, or lobectomy.

In all patients, standard monitors and state entropy (SE) and response entropy (RE) were applied. A thoracic epidural or paravertebral catheter was inserted, but no local anesthetics were infused during the study to avoid their effects on hypoxic pulmonary vasoconstriction.[10]

Anesthetic technique was standardized in all studied patients. Anesthesiologists who administered the anesthetic were not involved in assessing the patient. General anesthesia was induced with propofol (2-3 mg/kg) and fentanyl (2-3 μg/kg). Cisatracurium (0.2 mg/kg) was administered to facilitate the placement of a left-sided double-lumen tube, and the correct position of its tip was confirmed with a fiberoptic bronchoscope. Anesthesia, consisting of a 0.7 to 1.5 minimum alveolar concentration of sevoflurane, was administered to maintain SE values below 50 and the difference between RE and SE below 10. Fentanyl, 0.5μg/kg increments, was administered when the SE values were > 50,

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1053-0770/2601-0001$36.00/0

http://dx.doi.org/10.1053/j.jvca.2013.09.012

when the difference between RE and SE was >10, and when the mean arterial blood pressure (MAP) or heart rate was $>20\%$ of baseline values despite a target sevoflurane of MAC ≥ 1.5. The radial artery was catheterized, and cisatracurium increments were used to maintain surgical relaxation.

Both lungs of each patient were ventilated mechanically using VCV mode, which was performed using a semi-closed circuit anesthesia ventilator, Aisys™ (Datex-Ohmeda, GE Healthcare, Helsinki, Finland), with 0.5 inspired oxygen (F_IO2) in air and an actual delivered tidal volume (VT) of 8 mL/kg predicted body weight (PBW). PBW was estimated from the equation: PBW (kg) = 50 + 2.3 (height [in] − 60) for males and PBW (kg) = 45.5 + 2.3 (height [in] − 60) for females, an inspiratory-to-expiratory [I:E] ratio of 1:2.5 and a positive end-expiratory pressure (PEEP) of 5 cmH_2O, with respiratory rate adjusted to achieve a $PaCO_2$ of 35 mmHg to 45 mmHg. Peak airway pressure (Ppk) was limited to 35 cmH_2O, and a fresh gas flow (FGF) of 1.5 L/min to 1.8 L/min was used. A flow sensor (D-lite, GE, Helsinki, Finland) was connected between the tracheal tube and the "Y" piece of the respiratory circuit to measure the Ppk, plateau airway pressures (Ppl) and static compliance.

Transesophageal echocardiography was performed using a Philips iE33 Echocardiography System (Philips Technologies; Bothell, WA). RV function was evaluated by measuring both maximal systolic and diastolic tricuspid annular velocities (TAV). These parameters were recorded at the RV free wall from the deep transgastric long-axis apical 4-chamber views using pulsed-wave Doppler tissue imaging. RV end-systolic volumes (ESV) and end-diastolic volumes (EDV) were measured with 3-dimensional (3D) TEE volumetric quantification. Later, an independent trained operator with more than 10 years' experience and who was blinded to the study groups reviewed the echocardiographic results.

OLV was initiated after pleurotomy, and the patients were allocated randomly to 2 groups by drawing sequentially numbered, sealed, opaque envelopes containing a computer-generated randomization code. In the PCV-VCV group (n = 14), the dependent lung was ventilated with PCV for 30 minutes followed by the VCV, while in the VCV-PCV group (n = 14), PCV and VCV were applied in reverse order.

During the PCV period, the inspiratory pressure was adjusted to deliver an actual TV of 6 mL/kg PBW to the patient's dependent lung without exceeding a Ppk of 35 cmH_2O. During the VCV period, the patient's dependent lung was ventilated with an actual TV of 6 mL/kg PBW. F_IO_2, I:E ratio, PEEP, respiratory rate, and FGF (1.5-1.8 L/min) were maintained as during TLV, and the lumen of the nondependent lung was left open to air. Recruitment maneuvers for the dependent lung were repeated at 30-minute intervals by increasing the inspiratory pressure to 35 cmH_2O for 10 seconds.[2]

All surgical procedures were performed by the same surgeons. Intraoperative hypoxemia was defined as a decrease in arterial oxygen saturation to less than 90% and was treated by increasing F_IO_2 to 1.0. An addition of 2 cmH_2O of CPAP was considered if increasing F_IO_2 failed to correct hypoxemia.[2]

Patients were administered lactated Ringer's solution at a rate of 2 mL/kg/h during surgery. If MAP decreased below 20% of baseline values, 250 mL of a 5% plasma protein fraction were administered; if insufficient, patients were administered repeated doses of intravenous ephedrine (5 mg) or norepinephrine (5 μg) to maintain urine output ≥ 0.5 mL/kg/hour. Hemoglobin concentration ≥ 8 g/dL was maintained by administering red blood cell concentrates.

At the end of surgery, the nondependent lung was re-expanded, TLV was resumed as before surgery, sevoflurane was discontinued, the residual neuromuscular block was antagonized, and the patient was extubated. Postoperative analgesia consisted of continuous epidural or paravertebral infusion of bupivacaine, 0.125%, and fentanyl, 2 μg/mL.

Computerized data were collected by an independent investigator blinded to patient allocation and not involved in patient management. Primary outcomes included changes in RV function (systolic and early diastolic TAV, EDV, ESV, and fractional area changes [FAC]). Secondary outcome variables included hemodynamic parameters (heart rate and MAP); Ppk; Ppl; static compliance of the respiratory system calculated as exhaled TV/Ppl (the sum of externally applied and intrinsic PEEP); the ratios of arterial tension to inspired fraction of oxygen (PaO_2/F_IO_2), arterial oxygen saturation (SaO_2) and carbon dioxide tension ($PaCO_2$); intraoperative need for CPAP during OLV; use of fentanyl, ephedrine, and norepinephrine; perioperative hypoxemia ($SaO_2 < 90\%$); and the rates of respiratory and circulatory failure and arrhythmias. Secondary outcomes also included length of hospital stay and postoperative complications such as the need for ICU admission, acute lung injury (ALI), pneumonia, atelectasis, and re-thoracotomy for air leakage as well as a 30-day mortality rate. RV function and hemodynamic, oxygenation, and ventilation variables were recorded after induction of anesthesia (postinduction), 30 minutes after the use of PCV and VCV during OLV (PC-OLV and VC-OLV, respectively), and 15 minutes after resuming TLV.

A previous study showed that the normally distributed mean systolic TAV in anesthetized patients was 7 cm/s (standard deviation [SD], 1.4 cm/s).[11] A priori power analysis indicated that a sample size of 13 pairs was sufficiently large to detect a 20% difference in mean systolic TAV after the start of OLV with a type-I error of 0.05 and a power of 90%. To compensate for patients dropping out during the study, more patients (10%) were added for a final sample size of 28 patients.

The carryover effect (ie, the persistence of the effect of the first intervention on the operative conditions into the second period) was avoided by comparing the effects of period (time effect) and the order of treatment using independent t-tests. Data were tested for normality using the Kolmogorov-Smirnov test. Fisher's exact test was used to compare categoric data. Repeated two-way ANOVA and paired t-tests were used to study changes in primary and secondary endpoints during each intervention. The Wilcoxon rank sum test was used to compare nonparametric values. Data were expressed as mean ± SD or number (%). A p value < 0.05 was considered statistically significant.

RESULTS

All 28 consecutive patients enrolled and scheduled for open thoracotomy (14 in the PCV-VCV group and 14 in the VCV-PCV group) completed the study. Their demographic and clinical characteristics, all of which were similar in the 2 groups, are presented in Table 1, including FEV_1, FVC, side of thoracotomy, underlying pathology, type of surgery and intraoperative fentanyl use, administration of ephedrine and norepinephrine, and durations of surgery and anesthesia.

The PCV-VCV and VCV-PCV groups also did not differ significantly in post-induction systolic and early diastolic TAV, ESV, EDV, FAC, HR, MAP, Ppk, Ppl, static compliance, PaO_2/F_IO_2 ratio, SaO_2, and $PaCO_2$.

Compared with the postinduction, VC-OLV and two-lung ventilation periods, the use of PC-OLV resulted in significantly faster systolic (7.1 ± 1.4, 5.8 ± 1.7, and 6.9 ± 1.5 *v* 10.1 ± 2.4 cm/s, respectively) and early diastolic (5.4 ± 1.4, 4.6 ± 1.4, and 5.7 ± 1.5 *v* 9.2 ± 1.9 cm/s, respectively), TAV values ($p < 0.001$ each) and significantly lower RV EDV values ($p < 0.005$) (Table 2).

Compared with the postinduction, PC-OLV, and two-lung ventilation periods, the use of VC-OLV resulted in significantly