

Comparison of two ventilation modes in post-cardiac surgical patients

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ABSTRACT

Background: The cardiopulmonary bypass (CPB)-associated atelectasis accounted for most of the marked post-CPB increase in shunt and hypoxemia. We hypothesized that pressure-regulated volume-control (PRVC) modes having a distinct theoretical advantage over pressure-controlled ventilation (PCV) by providing the target tidal volume at the minimum available pressure may prove advantageous while ventilating these atelectic lungs. **Methods:** In this prospective study, 36 post-cardiac surgical patients with a $\text{PaO}_2/\text{FiO}_2$ (arterial oxygen tension/Fractional inspired oxygen) < 300 after arrival to intensive care unit (ICU), ($n = 34$) were randomized to receive either PRVC or PCV. Air way pressure (P_{aw}) and arterial blood gases (ABG) were measured at four time points [T1: After induction of anesthesia, T2: after CPB (in the ICU), T3: 1 h after intervention mode, T4: 1 h after T3]. Oxygenation index (OI) = $[\text{PaO}_2 / \{\text{FiO}_2 \times \text{mean airway pressure } (P_{\text{mean}})\}]$ was calculated for each set of data and used as an indirect estimation for intrapulmonary shunt. **Results:** There is a steady and significant improvement in OI in both the groups at first hour [PCV, 27.5(3.6) to 43.0(7.5); PRVC, 26.7(2.8) to 47.6(8.2) ($P = 0.001$)] and second hour [PCV, 53.8(6.4); PRVC, 65.8(7.4) ($P = 0.001$)] of ventilation. However, the improvement in OI was more marked in PRVC at second hour of ventilation owing to significant low mean air way pressure compared to the PCV group [PCV, 8.6(0.8); PRVC, 7.7(0.5), $P = 0.001$]. **Conclusions:** PRVC may be useful in a certain group of patients to reduce intrapulmonary shunt and improve oxygenation after cardiopulmonary bypass-induced perfusion mismatch.

Key words: Atelectasis, mechanical ventilation, pressure-controlled ventilation, pressure-regulated volume-controlled ventilation

INTRODUCTION

Pulmonary dysfunction is a frequent postoperative complication after cardiac surgery with cardiopulmonary bypass (CPB), and atelectasis is thought to be one of the main causes.^[1,2] Development of atelectasis is associated with decreased lung compliance and impairment of oxygenation.^[3] Post-CPB-induced lung atelectasis accounts for most of the marked post-CPB increase in intrapulmonary shunt and hypoxemia.^[4,5] Mechanical ventilation can be harmful to these atelectic lungs, especially when high tidal volumes and pressures that cause lung over distension are used.^[6,7] Pressure-controlled ventilation

(PCV) needs frequent titration of the inspiratory pressure to deliver a set tidal volume, whereas pressure-regulated volume-controlled (PRVC) ventilation mode provide the target tidal volume with the lowest possible airway pressure. In view of this distinct theoretical advantage of PRVC ventilation over PCV, we hypothesize that PRVC would result in lower intrapulmonary shunt with improvement in oxygenation compared to pressure-controlled (PC) mode of ventilation.

METHODS

After approval from institutional review board and obtaining informed patient consent from each individual, 36 patients scheduled to undergo first time elective coronary artery bypass grafting (CABG) surgery were enrolled prospectively in the study. Exclusion criteria included pre-existing pulmonary disease, associated significant valvular heart disease requiring valve repair/replacement, known pulmonary arterial hypertension (estimated pulmonary arterial pressure of >35 mmHg on trans thoracic echocardiography), poor left ventricular function (ejection fraction $< 35\%$), congestive

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heart failure, preoperative renal failure (serum creatinine >1.8 mg/dL) morbid obesity (body mass index >35) and smoking history in previous 2 months. Any patient re-explored because of excessive bleeding was also excluded.

Anesthesia and mechanical ventilation

All chronic medications and concurrent diseases were recorded. Pre-medication included tablet alprazolam 0.5 mg orally night before surgery and ranitidine 150 mg orally on the morning of the surgery. General anesthesia was carried out using a balanced anesthetic technique with propofol (1-2 mg/kg), midazolam (0.05-0.1 mg/kg), fentanyl (5-10 mcg/kg), and vecuronium (0.12 mg/kg) intravenously, and maintenance of anesthesia was performed using sevoflurane (0.5-1 MAC) with 50% oxygen in air and supplemental doses of fentanyl (1-2 mcg/kg), midazolam (1-2 mg), and vecuronium (1-2 mg) hourly. After tracheal intubation, mechanical ventilation was started with volume-controlled ventilation (VCV) using tidal volume (TV) of 10 ml/kg and a respiratory rate (RR) to maintain end tidal carbon dioxide of 30-36 mmHg. The inspiratory/expiratory ratio (I/E) was 1:2. Crystalloid solutions (8-10 ml/kg) were used as maintenance fluid intraoperatively. A base line ABG (T1) was obtained before commencing CPB.

Cardiopulmonary monitoring

Monitoring included continuous electrocardiogram (ECG), pulse oximetry (SpO₂), end tidal carbon dioxide (EtCO₂), arterial blood gas (ABG), invasive mean arterial pressure (MAP), central venous pressure (CVP), cardiac output (CO) using the PiCCO technique (Pulsion medical systems, Munich, Germany), urine output and core temperature by electronic thermometers.

Surgical and cardio pulmonary bypass technique

CABG was performed through a median sternotomy with heparinization under CPB using aortic and two-stage atrio-venous cannulation. CPB was initiated using a membrane oxygenator with a non-pulsatile flow rate of 2.2 to 2.4 lit/min/m² and a mean arterial pressure of 50 to 80 mmHg. Moderate systemic hypothermia around 30 °C was induced during CPB. Myocardial protection was achieved with anti-grade hyperkalemic blood cardioplegia (4:1 blood: Crystalloid) at 4–10°C and topical ice slush. The lungs were not ventilated during CPB and connected to bain circuit with a basal oxygen flow of 50 ml/min. Before discontinuation of CPB, lungs were manually inflated until visible atelectasis disappeared. CPB was terminated with the same ventilator setting as before CPB. Inotropes, vasodilators, intravenous fluid, blood (set transfusion trigger was 10 g/dl), and blood products were given as indicated. The left internal mammary artery was used and the left pleura were routinely opened in all patients.

Intensive care unit management

Patients were transferred to the intensive care unit (ICU) with manual ventilation by a bain circuit with an oxygen flow of

10l/min. Upon arrival in the ICU, all patients were connected to VCV with the same intra operative settings, and as soon as hemodynamic stability had been achieved (defined as MAP ≥ 70 mmHg, CVP, equal to baseline value or cessation of systemic blood pressure fluctuations with respiration, heart rate 70-100 beats per minute), a fresh blood gas sample was obtained (T2) to estimate intrapulmonary shunt by obtaining PaO₂/FiO₂ ratio (P/F) and this ratio was used to identify our study subjects, and patients having a P/F ratio < 300 mmHg (*n* = 34) were randomly assigned to receive either PRVC ventilation (Group PRVC, *n* = 17) or PCV (Group PCV, *n* = 17) by using the sealed envelope technique. All the study subjects were sedated and paralyzed with a continuous infusion of atracurium (0.5 mg/kg/h) and midazolam (1 mg/h) for 2 h. Continuous analgesia was provided with fentanyl 1 mcg/kg every hourly for 24 h for postoperative pain relief. The dose of fentanyl was adjusted to keep the visual analog score <4 after tracheal extubation. Tramadol (1 mg/kg) was supplemented if further analgesia is needed. All patients were ventilated using the Siemens Servo 300 ventilator with the following variables TV, 10 ml/kg; RR to achieve EtCO₂ of 30-36 mmHg; positive end expiratory pressure (PEEP), 3 mmHg; upper pressure limit (P_{max}), 35 mmHg; I:E, 1:2. The delivery of target TV (10 ml/kg) in PCV mode was assured by adjusting the pressure control (above PEEP) knob at first and second hours of ventilation after recording airway pressure on the display monitor of the ventilator. The third, fourth and fifth ABG was recorded at first hours, second hours (T3 and T4) of study intervention and at 12 h postextubation (T5), respectively. A period of 15 min was allowed before data collection during which the patients were not disturbed by nursing procedures. All the blood gas samples (T2 to T4) were analyzed for indirect estimation of intrapulmonary shunt by using the shunt equation,^[8] oxygenation index (OI) = [arterial oxygen tension (PaO₂) / {fraction of inspired oxygen (FiO₂) × mean airway pressure (P_{mean})}].

The primary outcome is to compare the OI measured at the following predetermined time intervals: After admission to the ICU, after first and second hours of assigned modes of ventilation and 12 h after extubation. The secondary outcome of the study are duration of mechanical ventilation, and failure to comply with the assigned mode of ventilation (Peak inspiratory pressure more than 30 mmHg to deliver the set tidal volume) between two ventilation strategies.

Inotropic agents were used to maintain a MAP of more than 70 mmHg after preload (8-10 mmHg), and heart rate was optimized (70-100 beats per minute). Nitroglycerin was infused at a range of 0.5–1.5 mcg/kg/min for 24 h postoperatively. After 2 h of intervention modes of ventilation, all patients were assessed every 30 min to facilitate weaning from ventilator support. The decision to extubate a patient was at the discretion of the consultant anesthetist, who is unaware of the study hypothesis but not

blinded to the assigned mode of ventilation and followed a predefined protocol^[9] along with standard hemodynamic criteria (a MAP \geq 70 mm Hg, pulse rate $<$ 100 beats per minute, and a blood loss less than 100 ml/h and decreasing from the chest drains). All patients were weaned on pressure support mode of ventilation. After extubation, patients were placed on 50% high flow oxygen masks. Duration of mechanical ventilation was recorded in hours from the time of admission to the ICU to the time of successful extubation (defined as absence of re intubation in next 24 h). Chest radiographs were obtained preoperatively, 2 h after assigned mode of ventilation in the ICU, on postoperative day 1, and after extubation. The findings of the radiograph were evaluated by an independent observer. Major post-operative complications (pneumothorax, lobar collapse, atelectasis, pulmonary edema and bronchospasm) were also recorded.

Statistical analysis

All values are reported as mean (standard deviation) unless otherwise stated. Numerical data were compared using Student’s unpaired *t* test. Categorical data were analyzed with a Chi-square test or Fisher’s exact test as appropriate. Comparisons of different time points versus baseline measures within the groups were performed by analysis of variance for

repeated measurement followed by Scheffe’s post test. All statistical analyses were done using the SPSS 14.0 software (statistical packages for Social Science, Chicago, IL, USA). A “*P*” value $<$ 0.05 was considered statistically significant.

RESULTS

In total 80 patients were screened during the study period. Twelve patients did not match the inclusion criteria. Thirty-two patients were excluded upon arrival into the ICU as they have a P/F higher than 300 from T2 blood sample. Thirty six patients were included in the study but two more patients excluded shortly thereafter because of recurrent ventricular fibrillation (*n* = 1) leading to cardiac arrest and death, and severe left ventricular dysfunction (*n* = 1) as evidenced by trans-esophageal echocardiography leading low output syndrome and oliguria [Figure 1, Consort diagram]. So in total 34 patients completed the study protocol.

Demographic and baseline variables were not statistically different [Table 1]. All the patients were tracheally extubated within 13 h after the intervention and none of the patients

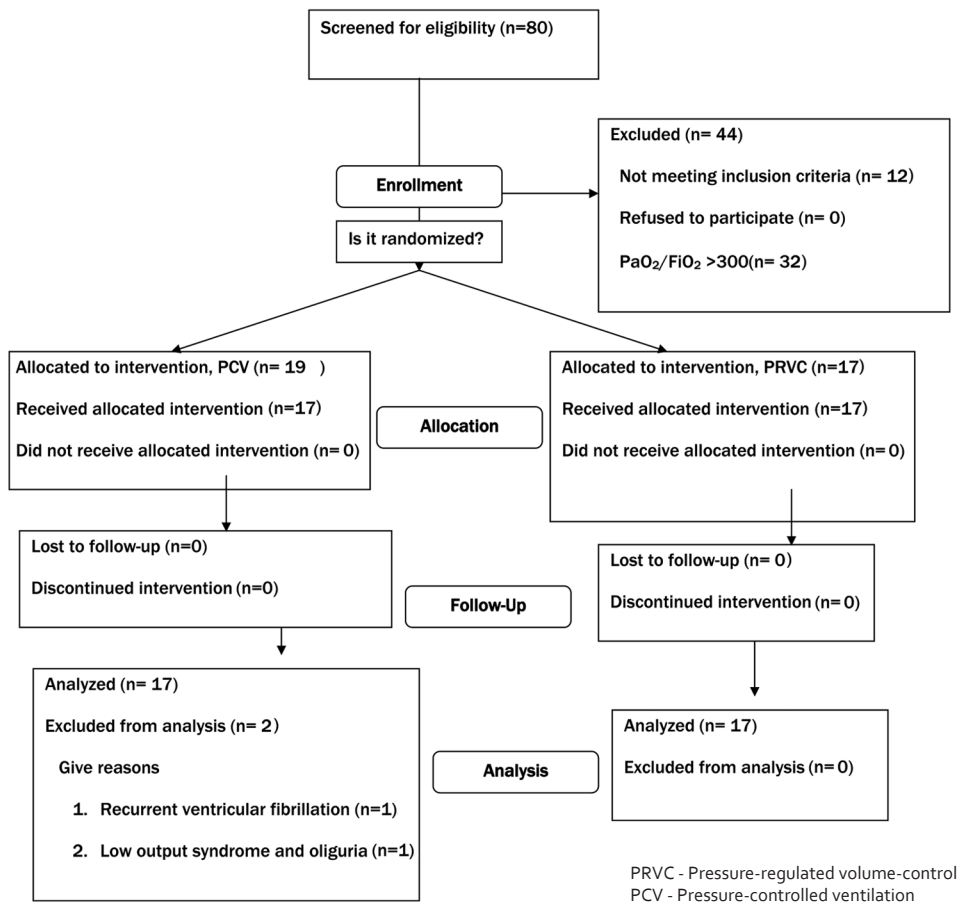


Figure 1: Consort diagram

required re-intubation during the study period [Table 2]. Operative data like CPB time, aortic cross clamp time, anesthesia time, inotropes used, fluids, and blood product transfusions are also comparable among the groups [Table 2].

The primary outcomes of the study are shown in Table 3 and Figure 2. The OI, mean air way pressure, and cardiac output were not significantly different between the groups on admission to ICU. After ventilating these patients for 2 h with the assigned study mode of ventilation (PCV or PRVC) both the groups showed a marked increase OI[PCV: 43.0(7.5), 53.8(6.4), $P = 0.001$; PRVC: 47.6(8.2)65.8(7.4),

$P = 0.001$] and a marked decrease in P_{mean} [PCV: 9.4(0.8),8.6(0.8), $P = 0.001$; PRVC: 9.2(0.7),7.7(0.5), $P = 0.001$] both at first hour and second hour of ventilation from base line values [OI:PCV, 27.5(3.6); PRVC, 26.7(2.8); P_{mean} :PCV, 9.0(0.7); PRVC, 8.8(0.6)]. The cardiac output did not changed significantly either within the group or between the groups ($P > 0.05$) at first and second hours of ventilation [Table 3].

Although the increase in OI was significant within the group, it reached significant level only at second hour of ventilation between the groups [PCV, 53.8(6.4) vs. PRVC, 65.8(7.4), $P = 0.001$] and was attributed to significantly low P_{mean} in the PRVC group at 2 h of ventilation [PCV, 8.6(0.8) vs. PRVC, 7.7(0.5), $P = 0.001$]. This beneficial effect of PRVC mode was continued till 12 h post extubation and reflected as higher $\text{PaO}_2/\text{FiO}_2$ ratio in the PRVC group compared to the PCV group [PCV, 376(20) vs. PRVC, 390(17)].

Table 1: Patient and clinical characteristics

Group	PCV (n = 17)	PRVC (n = 17)	P value
Age (year)	59.1 (6.9)	59.0 (7.2)	0.942
Males/Female	11/6	12/5	1.000
BMI (kg/m ²)	26.3 (1.7)	27.0 (2.1)	0.261
Smoking (Packs/day)			
Non smoker	5	3	0.766
Occasional Smoker	5	7	
≤1	5	4	
>1	2	3	
NYHA II/III	12/5	8/9	0.296
Ejection fraction (%)	45 (4.8)	42 (4.4)	0.115

Data are presented as mean (standard deviation) unless otherwise indicated, n = Number of patients, PCV = Pressure controlled Ventilation, PRVC = Pressure regulated Volume control, BMI = Body mass index, NYHA = New York Heart Association grading

Table 3: Post-operative variables

	T2	T3	T4	T5*	P value†
Oxygenation index					
PCV (n = 17)	27.5 (3.6)	43.0 (7.5)	53.8 (6.4)	376 (20)	0.001
PRVC (n = 17)	26.7 (2.8)	47.6 (8.2)	65.8 (7.4)	390 (17)	0.001
P value‡	0.666	0.181	0.001	0.035	
Mean air way pressure					
PCV (n = 17)	9.0 (0.7)	9.4 (0.8)	8.6 (0.8)	PE	0.001
PRVC (n = 17)	8.8 (0.6)	9.2 (0.7)	7.7 (0.5)	PE	0.001
P value‡	0.340	0.456	0.001		
Cardiac output					
PCV (n = 17)	4.61 (0.2)	4.45 (0.2)	4.55 (0.3)	PE	0.378
PRVC (n = 17)	4.46 (0.4)	4.21 (0.3)	4.44 (0.3)	PE	0.221
P value‡	0.387	0.149	0.541		
Arterial carbon dioxide tension (PaCO ₂)					
PCV (n = 17)	39.3 (4.1)	38.4 (4.0)	40.0 (4.6)	42.4 (2.8)	0.428
PRVC (n = 17)	39.0 (3.4)	37.3 (4.5)	39.6 (5.1)	42.2 (2.6)	0.138
P value‡	0.848	0.464	0.778	0.854	

Data are presented as mean (standard deviation) unless otherwise indicated, n= number of patients. *T5, Oxygen index was calculated by measuring [arterial oxygen tension (PaO₂) / fraction of inspired oxygen (FiO₂)] after extubation. †Comparison with in the group, among T2, T3, and T4; ‡Comparison between the groups. PE = post extubation cardiac output and mean airway pressure were not measured. Oxygenation index (OI) = [arterial oxygen tension (PaO₂) / {fraction of inspired oxygen (FiO₂) x mean airway pressure (P_{mean})}]. PCV = Pressure controlled ventilation, PRVC = Pressure regulated volume control, T2 = after cardio pulmonary bypass (in ICU), T3 = One hour after intervention mode

Table 2: Perioperative data

Group	PCV (n = 17)	PRVC (n = 17)	P value
Oxygenation index (T1)	50.2 (7.1)	46.5 (6.4)	0.121
Cardiac output (T1)	4.06 (0.4)	3.98 (0.4)	0.680
Duration of CPB (minutes)	112 (18)	120 (18)	0.222
Duration of ACC (minutes)	67 (11)	71 (13)	0.367
Number of graft	3.24 (0.5)	3.29 (0.8)	0.768
Anesthesia time (minutes)	200 (17)	205 (22)	0.445
MVD (hours)	9.0 (2.8)	8.0 (1.9)	0.120
Net fluid transfused (mL)	707 (91)	744 (101)	0.276
Net blood transfused (mL)	694 (143)	694 (143)	1.000
P_{mean} after induction (T1) (mmHg)	7.2 (1.1)	7.7 (1.1)	0.256
Inotropes used*			
Adrenaline (n)	2	5	0.238
Dobutamine (n)	16	17	0.103

Data are presented as mean (standard deviation) unless otherwise indicated, n = Number of patients T1, before commencement of cardiopulmonary bypass. *Inotropes used refers to the total period of 24 hours after surgery. PCV = Pressure controlled Ventilation, PRVC = Pressure regulated Volume control, BMI = body mass index, CPB = Cardiopulmonary bypass, ACC = Aortic cross clamp, MVD = Duration of mechanical ventilation, P_{mean} = mean air way pressure

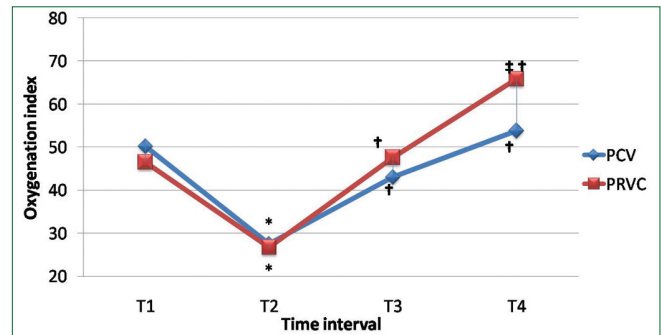


Figure 2: Oxygenation index [arterial oxygen tension (PaO₂) / {fraction of inspired oxygen (FiO₂) x mean airway pressure (P_{mean})}] at different time interval among the groups. T1 = before commencement of cardiopulmonary bypass, T2 = after cardio pulmonary bypass (in ICU), T3 = one hour after intervention mode, T4 = 2 h after intervention mode. * $P < 0.05$ versus to T1; † $P = 0.001$ versus T2; ‡ $P = 0.03$ versus PCV

None of the patients in any group had pulmonary complications such as pneumothorax, pleural effusion, pulmonary edema, lobar collapse, and bronchospasm, or chest x-ray evidence of atelectasis. All patients had good pain relief during the postoperative period.

DISCUSSION

In the current study, the patient population received two different kinds of mechanical ventilation (PCV or PRVC) for suspected atelectasis-induced perfusion mismatch following on pump CABG surgeries, and evaluated for resolution of atelectasis as evidenced by the OI. The result showed that both PCV and PRVC mode significantly improves OI and gas exchange in the post-CPB period. Further analysis revealed that this significance is mainly contributed by a surge in OI after switching from VCV to decelerating flow pressure limited ventilation. This finding is not surprising as studies have demonstrated an improvement in oxygenation and pulmonary mechanics in patients with ARDS who were switched from VCV to PCV.^[9-11] However, PRVC was found to be advantageous in later stages of ventilation as it results in significant lower mean airway pressure and improved OI compared with PCV mode [Table 3]. This can be explained by the fact that, although both PCV and PRVC use a decelerating flow pattern which has been shown beneficial in acute lung injury,^[10,12] PRVC combines the benefits of decelerating flow of PCV with a safety of a volume guarantee at a lowest possible titrated inspiratory pressure.

OI assessment is essential in the management of patients requiring mechanical ventilation. A decrease in the oxygen index (impaired gas exchange) may indicate the presence of atelectasis and reflect intrapulmonary shunt. The $\text{PaO}_2/\text{FiO}_2$ ratio and the $\text{PaO}_2/\text{PAO}_2$ ratios are the most common of these measurements but do not remain equally sensitive across the entire range of FiO_2 and do not account for changes in the functional status of the lung that result from alterations in positive end-expiratory pressure (PEEP), auto-PEEP, or other techniques for adjusting average lung volume during mechanical ventilation. So we used a new OI,^[8] which is based on the usual P/F ratio but takes into consideration some important mechanical ventilator support variables such as change in airway pressure, inspiratory time fraction, and tidal volume.

In this study we have made two assumptions: First, atelectasis is proportional to intrapulmonary shunt and second, intrapulmonary shunt is proportional to OI. Our study assumptions are well supported by previous studies.^[4,5,8,13-15] An improvement in cardiac output and change in regional circulation i.e. hypoxic pulmonary vasoconstriction (HPV) after surgery may bring an

improvement in OI by changing the intrapulmonary shunt fraction. In the current study, all the patients were on nitroglycerine infusion (0.1-0.2 mcg/kg/min) thus negating the HPV in any particular group. There is no appreciable change in cardiac output, either between the groups or within the group at first and second hours of assigned mode of ventilation. The initial improvement in the cardiac output after CPB and in ICU could be because of intra-compartmental transformation of fluids immediately after CPB and corrective surgical procedure.

The impact of ventilating the lung during CPB in order to minimize postoperative atelectasis was the subject of several investigations with conflicting result and had no significant sustained effect on postoperative pulmonary gas exchange.^[16-18] Recruitment maneuver with high inspiratory pressure or incremental PEEP may not be suitable in these groups of patients for fear of compromising arterial graft flow and adverse hemodynamic consequences.^[19-23] So a postoperative mechanical ventilation strategy aiming at optimizing both tidal volume and inflation pressure is ideal to ventilate these atelactic lungs in the post CPB period. In our study we preferred to use a physiological PEEP of 3 mmHg and a tidal volume of 10 ml/kg, as the entire study participants had “healthy” lungs and no acute lung injury risk factors. None of our patient failed to comply with the intervention mode and the highest inspiratory pressure required for delivering the target tidal volume (10 ml/kg) after CPB (in the ICU) is far below the maximum available pressure (28 mmHg in PCV and 27 mmHg in PRVC) mode.

PCV has been found useful in improving OI in post-cardiac surgical patients and most of the advantages of this mode of ventilation have been attributed to its ability to lower or limit the peak inspiratory pressure.^[24,25] P_{mean} was lower for all patients using the PRVC mode compared to the PCV mode, and reached statistical significance at second hour of ventilation without any change in alveolar ventilation as indicated by the comparable PaCO_2 [Table 3]. This low P_{mean} in the PRVC group is responsible for a higher OI at second hour of ventilation and this beneficial effect of PRVC is reflected even 12 h after extubation with a higher P/F ratio in the PRVC group compared to the PCV group.

There are very few reports on the influence of specific mode of ventilation on the duration of ventilation. Rappaport *et al.*^[11] reported shorter duration of ventilation when pressure-limited mode (decelerating flow) was compared with VC ventilation in adults but others did not obtain similar results in their study.^[26-28] The present study did not find a significant difference in duration of ventilation in two groups probably owing to small sample size [Table 1].

One of the contradicting findings in our study is the absence of radiological evidence of atelectasis despite a low OI on admission to ICU. The probable reason could be: First, we have obtained chest radiograph after 2 h of mechanical ventilation and these regular chest radiographs might have underestimated both the frequency and amount of atelectasis;^[5] second, in contrast to other study^[11] where the inclusion criterion was PaO₂/FiO₂ ratio < 150, our patients had only mild lung injury (PaO₂/FiO₂ ratio < 300).

We believe that the present study is the first to compare the use of PRVC and PCV modes in treating post-CPB atelectasis in adult cardiac surgical patients. As the pre intervention cardiac output, OI and P_{mean} are comparable in both groups; the rise in OI can be attributed to a significant low P_{mean} in the PRVC group. Because all our patients were sedated and paralyzed, these results may not be extrapolated to patient-triggered modes.

We conclude that during mechanical ventilation in post-cardiac surgical patients without pre-existing lung disease, PRVC mode may be superior to PCV mode in reducing intrapulmonary shunt by precise titration of inspiratory pressure.

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