Is airway pressure release ventilation, a better primary mode of post-operative ventilation for adult patients undergoing open heart surgery? A prospective randomised study

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ABSTRACT
Context: Cardiopulmonary bypass (CPB) induced acute lung injury is accounted for most of the post-operative pulmonary dysfunction which leads to decreased compliance and hypoxemia. Airway Pressure Release Ventilation (APRV) as compared to other modes of ventilation has shown to improve gas exchange in Acute lung injury (ALI)/Acute respiratory distress syndrome (ARDS) lungs.

Aims: We hypothesized APRV as a better primary mode of postoperative ventilation in adult post-cardiac surgery patients.

Methodology: The study included 90 postoperative surgical patients, which were randomized into three groups: SIMV-PC(P), APRV(A), and SIMV-VC(V) with 30 patients in each group.

Subjects and Methods: Lung compliance and serial arterial blood gas were assessed at regular intervals. PaO₂/FiO₂ ratio (a measure of oxygenation) and lung compliance were used as an indirect indicator for improvement in lung function. Hemodynamic parameters were closely observed for all the patients.

Statistical Analysis Used: Statistical analysis was done using ‘R’ software.

Results: There was a statistically significant improvement in PaO₂/FiO₂ ratio in the APRV group as compared to other groups. There was also an improvement in lung compliance after 6 h of ventilation and lesser duration of ventilation in the APRV group. However, it was not statistically significant.

Conclusions: Our study suggests that APRV can be a useful alternative primary mode of ventilation to improve lung compliance and oxygenation in adult post-cardiac surgical patients.

Keywords: APRV, cardiac surgery, mechanical ventilation

INTRODUCTION
Post-operative pulmonary dysfunction has been a significant cause of morbidity after cardiac surgery. The acute inflammatory response induced by CPB is often a major cause of acute lung injury. Various physiologic, biochemical, and histological changes have been found to occur after CPB leading to an increase in epithelial-capillary-endothelial permeability, eventually leading to lung damage.[1]

CPB induced lung injury is due to the activation of the complement system, leucocytes, endothelial cells, and other soluble inflammatory mediators. It is characterized by

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After endotracheal intubation, mechanical ventilation was initiated in all patients with SIMV-VC mode with a tidal volume of 6–8 ml/kg, initial respiratory rate of 12/min, titrated accordingly to maintain etco2 of 30–35 mm Hg and I:E ratio of 1:2. Anesthesia was maintained with isoflurane 0.5–1% with oxygen and air. Intravenous fluids were administered according to the central venous pressure and urine output.

Surgical technique
Open heart surgery was performed through a median sternotomy with heparinisation under CPB. Membrane oxygenator was used with a non-pulsatile flow of 2.2 to 2.4 liters/min/m², maintaining a mean arterial pressure of 50–80 mmHg. Moderate hypothermia of 28–30°C was induced. Adequate myocardial protection strategies were employed. Before weaning from CPB, lungs were recruited by manual ventilation. Inotropes, vasodilators, IV fluids were administered as indicated.

Post-operative management
90 patients who met the inclusion criteria were randomized into 3 groups i.e., ‘P’, ‘A’, ‘V’ as SIMV-PC, APRV, and SIMV-VC, respectively, consisting of three different ventilator strategies postoperatively with identical general ventilator measures and routine care. After the patient was shifted to the postoperative ICU randomized ventilator strategy was started accordingly and was maintained until the weaning criteria were met. Peak and plateau pressures were noted for all the patient's intraoperatively.

Ventilator strategies
All the patients were ventilated with the Dragger EVITA-4 machine. The pressure–volume loops were observed for all the patients. The goals of ventilation were to maintain: PaO2/FiO2 of ≥300 PaCO2 between 35 and 45 mm Hg, peak pressure ≤35 mm Hg, and plateau pressure ≤25 mmHg.

SIMV-PC mode: P inspiratory was set according to the plateau pressure recorded intraoperatively and was titrated according to the desired tidal volume. PEEP of 5 cm H₂O, pressure support of 10 cm H₂O, I:E ratio of 1:2 were uniformly applied to all the patients. Respiratory rate was initially set at 14–16 breaths per minute.

APRV mode: P high was set below the higher inflection point and above the lower inflection point and was titrated according to the desired tidal volume. P low was kept 0 for all the patients. T high initially was set as 3.6 s and T low as 0.6 s which correlated with an I:E ratio of 6:1 and respiratory rate of 15–16/min. During weaning in APRV P High was lowered 2 or 3 cm of H₂O pressure at a time, and

APRV was first described in 1987. It is a relatively new mode of ventilation in use as there has been a lot of debate about its advantages and disadvantages. It is identical to the inverse ratio, pressure-controlled ventilation with unsupported spontaneous breathing. It is based on the principle of the open lung approach.

APRV produces a release of airway pressure from an elevated baseline to simulate expiration. The elevated baseline pressure facilitates oxygenation, and the timed releases aids in carbon dioxide removal.

Literature has described many advantages of APRV over other modes of ventilation. Advantages being, lower airway pressures, lower minute ventilation, minimal adverse effects on the cardio-circulatory function, unsupported spontaneous ventilation, decreased sedation use, and near-total elimination of neuromuscular blockade.

Thus the authors hypothesized that APRV could be a better primary mode of postoperative ventilation in adult post-cardiac surgical patients.

SUBJECTS AND METHODS
After approval from the ethical committee and obtaining written informed consent from the patients, 90 patients were enrolled prospectively in the study.

Patients with pre-existing pulmonary disease (or) abnormal pulmonary function test, known pulmonary arterial hypertension (pulmonary arterial pressure >35 mm Hg on trans thoracic echocardiography), severe post-operative left ventricular/right ventricular dysfunction, preoperative renal failure, morbid obesity (BMI >35), smoking history in the previous 2 months and post-operative re-exploration were excluded from the study.

METHODOLOGY
Anaesthetic technique
All patients were premedicated with appropriate doses of tablet Alprazolam on the previous night and morning of the day of surgery. Under standard ASA and invasive arterial monitoring, all patients received general anesthesia with balanced anesthesia technique using propofol (1–2 mg/kg), midazolam (0.05–0.1 mg/kg), fentanyl (5–10 mcg/kg), and vecuronium (0.1 mg/kg).
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T Low was lengthened in 0.5–2.0 s increments, depending on patient tolerance. The goal was to arrive at straight CPAP usually at 12 cm of H₂O and then the clinician either委会 CPAP or simply extubated the patient at 6–10 cm of H₂O. Before switching to CPAP, P High often is approximately 14–16 cm of H₂O pressure and T High is at 12–15 s.

SIMV-VC mode: tidal volume was set with a target of 6-8 ml/kg and the initial respiratory rate was set as 14–16 per min. I:E ratio of 1:2, PEEP of 5 cm H₂O, and pressure support of 10 cm H₂O were uniformly applied to all the patients. FiO₂ was titrated according to the PaO₂/FiO₂ ratio in all the groups.

Lung compliance, serial blood gas analysis for PaO₂/FiO₂ ratio, plateau pressure, mean airway pressure, peak airway pressures were recorded at baseline, 30 min, 1 h (h), 2nd h, 4th h, 6th h, 8th h, 12th h, 16th h or till weaning (whichever was earlier). Continuous hemodynamic and hourly urine output monitoring was done for all the patients. Also, the duration of ventilation, ICU stay, hospital stay was recorded in all the groups.

General care
All the patients were nursed in 30° semi-recumbent positions. CPAP trial was given once the patient met the weaning criteria. Patients were extubated once fully conscious and after complete recovery of reflexes with no postoperative bleed and hemodynamic instability.

Other principles of treatment were hemodynamic management, regular ET suctioning, nebulization, and chest physiotherapy. Once extubated all patients were put on incentive spirometry and deep breathing exercises.

Statistical analysis
Statistical analysis was done using ‘R’ software. Categorical data were analyzed with a Chi-square test. Hemodynamic and ventilator parameters of various time intervals were compared using repeated measure ANOVA and the values are reported as mean ± standard deviation. A value of P < 0.05 was considered statistically significant.

RESULTS
Demographic and baseline variables were comparable among all the groups [Table 1]. Hemodynamic parameters and operative data like CPB time, aortic cross-clamp time, type of surgery, and duration of surgery were comparable among the groups [Table 2].

Post-operative data like systolic blood pressure, diastolic blood pressure, heart rate, central venous pressure, inotrope score, urine output, and sedation score were also comparable between the groups [Table 3].

The primary outcome, as depicted in Graph 1 and Table 4, shows an overall improvement in the PaO₂/FiO₂ ratio in the ‘A’ group which is statistically significant. Extubation time was also faster in the ‘A’ group. However, it is statistically insignificant [Table 2 and Graph 3].

Plateau pressure and peak airway pressure were comparable between the three groups [Graphs 4 and 5 respectively]. Although mean airway pressure was high in group ‘A’ due to its inherent ventilator property [Graph 6], the lung compliance showed an improving trend after 6 h of ventilation in group A [Graph 2] which is statistically insignificant [Table 5].
ICU and hospital stay did not vary much among all the groups [Table 2]. All patients had good post-op pain relief. None of the patients had complications that required interventions.

**DISCUSSION**

The postoperative lung dysfunction may be manifested as conditions ranging from subclinical functional changes in most patients to full-blown ARDS in <2% of cases after CPB.\(^2\)\(^,\)\(^,\)\(^,\)\(^,\)\(^,\)\(^,\)\(^,\)\(^,\)\(^,\)\(^,\)\(^,\)\(^,\) The causes of postoperative pulmonary dysfunction being an accumulation of extravascular fluid due to alterations in the alveolar-capillary membrane, alveolar collapse, decrease in functional residual capacity, retention of airway secretions, deteriorated lung mechanics leading to intrapulmonary shunting.\(^2\)

To our knowledge, very few studies have compared APRV with other conventional modes of ventilation as an initial mode of post-op ventilation in adult post-cardiac surgical patients undergoing open-heart surgery on cardiopulmonary bypass.

Our hypothesis for the potential benefits of APRV as a primary mode of postoperative ventilation in adult post-cardiac surgical patients is based on several experimental and clinical studies.\(^3\)\(^,\)\(^,\)\(^,\)\(^,\)\(^,\)\(^,\)\(^,\)\(^,\)\(^,\)\(^,\)\(^,\)\(^,\)

Literature has described two unique features of APRV as the most beneficial properties i.e., maintenance of

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**Table 3: Comparison of P values of post-operative data between the groups**

| Variables         | Baseline | 30 min | 1 h  | 2 h  | 4 h  | 6 h  | 8 h  | 12 h |
|-------------------|----------|--------|------|------|------|------|------|------|
| SBP               | 0.850    | 0.102  | 0.853| 0.361| 0.515| 0.318| 0.569| 0.202|
| DBP               | 0.148    | 0.439  | 0.201| 0.113| 0.297| 0.823| 0.703| 0.727|
| HR                | 0.802    | 0.791  | 0.484| 0.065| 0.144| 0.138| 0.456| 0.758|
| CVP               | 0.535    | 0.600  | 0.995| 0.501| 0.082| 0.070| 0.061| 0.327|
| INOTROPE SCORE    | 0.75     | 0.200  | 0.261| 0.259| 0.094| 0.095| 0.158| 0.792|
| URINE OUTPUT      | 0.173    | 0.778  | 0.263| 0.491| 0.273| 0.424| 0.077| 0.383|
| SEDATION SCORE    | 0.121    | 0.301  | 0.120| 0.780| 0.203| 0.231| 0.090| 0.132|
constant airway pressure and unsupported spontaneous ventilation.\(^3\)

Maintaining a constant airway pressure has been proven to be beneficial in many ways. It facilitates alveolar recruitment, enhances diffusion of gases, allows alveolar units with slow time constants to fill, preventing overdistension of alveoli and augmenting collateral ventilation.\(^3\)

Our results are consistent with this explanation as we found a statistically significant improvement in the PaO₂/FiO₂ ratio in the APRV group [Graph 1]. We also found a better improvement in lung compliance after 6 h of ventilation with the APRV group; however, this was not statistically significant [Graph 3].

Our results are also consistent with various other studies where APRV was compared to conventional modes of ventilation in patients with acute lung injury and severe ARDS who concluded that APRV was more efficient in decreasing atelectasis and improving the oxygenation.\(^9\)-\(^11\)

Asimakopoulos G \textit{et al}.\(^8\) evaluated V/Q matching in 24 patients with acute respiratory distress syndrome during airway pressure release ventilation with or without spontaneous breathing or during pressure support ventilation. They found that spontaneous breathing during APRV was associated with increases in right ventricular end-diastolic volume, stroke volume, cardiac index, and PaO₂ when compared with APRV without spontaneous breathing. They presumed that the uncoupling of spontaneous and mechanical ventilation during APRV improves V/Q matching in ARDS patients by recruiting non ventilated lung units.

Hemodynamic parameters like mean arterial pressure and central venous pressure were comparable in all the groups in our study.

**Table 4: Comparison of PaO\(_2\)/FiO\(_2\) ratio between the groups**

|        | SIMV-PC | APRV  | SIMV-VC | \(P\) |
|--------|---------|-------|---------|-------|
| Baseline| 413±94  | 443±85| 410±47  | 0.078 |
| 30 min | 408±85  | 428±56| 391±45  | 0.069 |
| 1 hr   | 398±66  | 466±50| 380±46  | 0.002 |
| 2 h    | 382±63  | 456±60| 383±46  | 0.001 |
| 4 h    | 383±84  | 470±75| 369±86  | 0.003 |
| 6 h    | 384±129 | 469±205| 323±54 | 0.002 |
| 8 h    | 314±139 | 426±145| 298±170| 0.003 |

**Table 5: Comparison of Mean airway pressures, Peak airway pressures, Plateau pressures and lung compliance between the groups**

**Mean airway pressures**

|        | SIMV-PC | APRV  | SIMV-VC | \(P\) |
|--------|---------|-------|---------|-------|
| baseline| 9.2±1.3 | 14±2.7| 10.3±2.7| 0.001 |
| 30 min | 9.4±1.1 | 14±3.3| 10.0±2.2| 0.010 |
| 1 hr   | 9.7±1.2 | 13.9±3.2| 9.4±1.9| 0.001 |
| 2 h    | 9.8±1.3 | 13.5±3.7| 9.3±2.4| 0.001 |
| 4 h    | 9.7±0.8 | 14±3.6 | 9.8±2.1 | 0.010 |
| 6 h    | 9.6±1.1 | 14±3.8 | 10.7±2.0| 0.020 |
| 8 h    | 9.8±0.9 | 14±4.3 | 10±1.3  | 0.010 |

**Peak airway pressure**

|        | SIMV-PC | APRV  | SIMV-VC | \(P\) |
|--------|---------|-------|---------|-------|
| baseline| 21.8±3 | 19±3.8| 20.6±3.4| 0.070 |
| 30 min | 21.2±2.9| 19.2±3.6| 20.1±3.4| 0.082 |
| 1 hr   | 20.7±2.8| 18.8±3.7| 20.3±3.5| 0.083 |
| 2 h    | 20.4±3.2| 18.9±3.6| 19.9±3.0| 0.210 |
| 4 h    | 20.7±3.2| 19.3±3.9| 20.0±3.4| 0.415 |
| 6 h    | 20.3±2.9| 18.6±4.1| 20.8±2.9| 0.198 |
| 8 h    | 18.7±2.1| 18±3.1 | 19.8±1.6| 0.305 |

**Plateau pressure**

|        | SIMV-PC | APRV  | SIMV-VC | \(P\) |
|--------|---------|-------|---------|-------|
| baseline| 20.3±2.7| 18±3.4| 19.3±3.1| 0.070 |
| 30 min | 19.97±2.6| 18.3±3.5| 19.1±3.1| 0.140 |
| 1 hr   | 19.72±2.4| 18.3±3.1| 19.1±3.1| 0.190 |
| 2 h    | 19.47±2.8| 18.4±3.3| 18.6±2.6| 0.230 |
| 4 h    | 19.2±2.5| 18.5±3.8| 18.8±2.6| 0.710 |
| 6 h    | 19±2.8 | 18.3±4.6| 19.4±2.2| 0.670 |
| 8 h    | 18.4±2.5| 17.8±2.9| 18.6±2.1| 0.810 |

**Lung compliance**

|        | SIMV-PC | APRV  | SIMV-VC | \(P\) |
|--------|---------|-------|---------|-------|
| baseline| 38.3±6.9| 39.0±8.2| 41.3±6.9| 0.263 |
| 30 min | 39.2±7.1| 40.7±8.6| 42.6±6.6| 0.221 |
| 1 hr   | 40.9±7.5| 42.5±8.2| 44.7±5.9| 0.143 |
| 2 h    | 42.2±8.9| 44.0±9.3| 45.2±6.3| 0.386 |
| 4 h    | 41.3±6.8| 45.3±9.8| 46.5±7.3| 0.079 |
| 6 h    | 42.3±6.0| 49.2±9.3| 48.1±9.1| 0.059 |
| 8 h    | 45.0±8.9| 53.7±10.5| 48.7±10.5| 0.143 |
Also, we found a lesser duration of ventilation in the APRV group. This suggests that better improvement in PaO2/FiO2 and lung compliance could be the reason for lesser ventilator duration in the APRV group.

However, overall ICU duration, hospital stay, inotrope score did not vary much among all the groups.

CONCLUSIONS

We conclude that APRV may be a useful alternative primary mode of ventilation to improve lung compliance and oxygenation in adult post-cardiac surgical patients.

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Conflicts of interest
There are no conflicts of interest.

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