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Abstract

Introduction: Since its initial description lung protective strategy using tidal volumes of less than or equal to 6ml/kg predicted body weight, with high respiratory rates and maintaining plateau pressures less than or equal to 30cmH2O has been the standard ventilator management for ALI/ARDS patients, where a large number of randomized controlled clinical trials have proven it to reduce the mortality rates [1].

Airway pressure release ventilation (APRV) has been successfully used in neonatal, pediatric, and adult forms of respiratory failure. Experimental and clinical use of APRV has been shown to facilitate spontaneous breathing and is associated with decreased peak airway pressure and improved oxygenation.

Methods: Our study was conducted on twenty patients admitted in the Critical Care Department, Cairo University Hospital with proven diagnosis of ALI/ARDS, who were ventilated sequentially for twelve hours using Volume Control-Assisted Controlled mechanical ventilation with lung protective strategy settings and Airway pressure release ventilation. Every three hours haemodynamic variables, arterial blood gases, lung mechanics and need for sedation were assessed. Aiming to compare and evaluate the two modes as regards haemodynamic effects, impact on arterial blood gases, lung mechanics and need for sedation while maintaining adequate oxygenation.

Results: We did not demonstrate any significant change in the haemodynamic variables (heart rate, mean blood pressure, central venous pressure, and pulmonary capillary wedge pressure) between both modes of ventilation. (p-value >0.05).

Partial pressure of carbon dioxide, acid-base status, and serum bicarbonate level did not change significantly between the two modes of mechanical ventilation (p-value >0.05), there was a significant decrease in partial pressure of oxygen with APRV compared to twelve hours of controlled mechanical ventilation with lung protective strategy settings and APRV comparing to CMV to 16.6±4.3 cmH2O decreasing highly significant 20.1% reduction in Hypoxic Index after twelve hours of APRV following twelve hours of CMV (CMV 275±80.7, APRV 219.9±68.6) p-value 0.001, there was a significant increase in respiratory rate and minute ventilation by 29.3% and 26.7% respectively during the twelve hours of APRV compared to twelve hours of CMV with respiratory rate increase from 21.5±6.9 with CMV to 27.8±6.2 APRV, p-value 0.007. There was no significant changes in dynamic compliance between both modes of ventilation (53±25.8 CMV, 52.9±26.9 APRV) p-value >0.718. Highly significant decrease of 38.9% in peak airway pressure was noted during ventilation with APRV compared to CMV, (APRV 19.8±4, 32.4±6.7 CMV), p-value 0.0001, associated with that there was also a highly significant 19% decrease in mean airway pressure during ventilation with APRV compared to CMV with lung protective strategy (CMV 18.9±3.7, APRV 15.3±3.3), p-value 0.0001. The need for sedation by propofol during the twelve hour period of APRV significantly decreased by 56.2% compared to the twelve hours of application of lung protective ventilation using CMV, where the dosage of propofol decreased from 1.6±0.5mg/kg/hr with CMV to 0.7±0.8mg/kg/hr with APRV, p-value 0.0001.

Conclusion: APRV can be used safely as one of the optimum ventilatory strategies in patients with ALI/ARDS as it decreases airway pressures significantly and decreases the need for sedation while maintaining adequate oxygenation without altering haemodynamics.

Key Words: Acute lung injury – Airway pressure release ventilation.

Introduction

SINCE its initial description in 1967, several definitions have been proposed for the diagnosis of The adult Respiratory Distress Syndrome (ARDS) and for acute lung injury (ALI). At the present, the most commonly used definitions are those proposed by the American-European Consensus Conference [1]. ALI is defined as a “syndrome of inflammation and increasing permeability that is associated with a constellation of clinical, radiographic and physiologic abnormalities that cannot be explained by, but may coexist with left atrial or pulmonary capillary hypertension. Based on the severity of hypoxemia, ARDS is defined as a severe form of
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ALI {i.e., an arterial partial pressure of oxygen/fraction of inspired oxygen (PaO₂/FiO₂) ratio less than 200 rather than 300mmHg} [1].

The overall goals in mechanical ventilatory support in parenchymal lung injury are to provide adequate gas exchange while minimizing any potentially iatrogenic lung injury. Although many variables can be monitored during this process, clinical decisions generally involve balancing four important factors: Arterial pH, arterial hemoglobin saturation (SaO₂), lung stretch, and lung exposure to oxygen [2].

• pH: 7.20-7.45.
• SaO₂: >88%.
• p plat: <35cmH₂O.
• FiO₂: <0.6.

Balancing these four clinical goals of pH, SaO₂, lung stretch, and FiO₂ constitutes the art of mechanical ventilation in parenchymal lung injury [1].

Low-tidal-volume ventilation (Lung protective strategy) should be implemented in the context of a broader strategy of critical care management in a patient with acute lung injury or ARDS. An initial tidal volume of 6ml per kilogram of predicted, not actual, body weight should be used, as in the ARDSNet trial [1].

APRV is a ventilatory support pattern that provides a moderately high (i.e., 15 to 25cm H₂O) level of continuous baseline airway pressure that is interspersed with the brief deflation (release) periods. Alveolar recruitment is maintained by the baseline pressure application while the ventilatory support is provided through two mechanisms. First, the periodic brief (<1.5sec) deflation provide some level of bulk flow gas transport. Second, spontaneous ventilation is permitted during baseline and the release phases.

Aim of this work was to compare and evaluate conventional lung protective strategy versus APRV in acute lung injury.

Patients and Methods

Our study was conducted on 20 patients who were admitted to Critical Care Department, Cairo University Hospital from August 2006 to April 2008, they all had documented ALI and ARDS, and required invasive mechanical ventilation.

All patients were ventilated using the Evita 4 (Drager, Lubeck, Germany) ventilator.

All 20 patients were subjected to the following 12 hours to ventilation with APRV.

When changing from VC-CMV to APRV, the settings of the conventional lung protective ventilation serve as a guide to the APRV settings, where the optimum PEEP measured in conventional lung protective ventilation is set as the Phigh on APRV settings.

Results

A- Demographic data:
- Our study was conducted on 11 males (55%), and 9 females (45%), who had a mean age of 55.6±15.2 (range between 22 to 75 years), mean ideal body weight of 68±8.5kg.
- All patients had ALI/ARDS with varying aetiologies.
- There were 12 mortalities (60%) from the total of 20 patients, cause of all mortalities was septic shock and multiple organ dysfunction.

B- Effects on haemodynamic variables:
- Mean blood pressure:
  Comparison of effects of twelve hours of VC-CMV and twelve hours of APRV on mean blood pressure measurements:

  During the application of twelve hours period of lung protective ventilation and APRV to all study patients, we found out that there was no statistical significant difference in measured mean blood pressure between the two groups (p-value 0.804), also there was no statistical significant difference across time (p-value 0.477) (i.e., mean blood pressure did not change over time) moreover the interaction of groups and time indicates that there is no statistical significant difference (p-value 0.924) i.e., the groups are not changing over time and are not changing in different ways (Fig. 1).

  - Heart rate:

    During the application of twelve hours period of lung protective ventilation and APRV to all study patients, we found out that there was no statistical significant difference in measured heart rate between the two groups (p-value 0.73 1), also there was no statistical significant difference of heart rate across time (p-value 0.286), moreover the interaction of groups and time indicates that there is no statistical significant difference (p-value 0.924) i.e., the heart rate of both groups are not
changing over time and are not changing in different ways (Fig. 2).

- **Central venous pressure:**
  Regarding the central venous pressure measurement of all study patients during twelve hours period of conventional lung protective ventilation and APRV, we found that there was no significant time effect (p-value 0.207) i.e., the central venous pressure did not change over time in both groups independently. There was no statistical significant difference between the two modes (p-value 0.761), and there was no significant difference between in the interaction of both groups and time (p-value 0.368), which means that both groups are not changing over time or in different ways (Fig. 3).

- **Pulmonary capillary wedge pressure:**
  Comparison of effects of twelve hours of VC-CMV and twelve hours of APRV on pulmonary capillary wedge pressure measurements:
  On evaluating the pulmonary capillary wedge pressure of all study patients who were subjected to twelve hours period of conventional lung protective ventilation and twelve hour period of APRV, we found that there was no statistical significant difference between groups (p-value 0.313), but there was a statistical significant time effect (p-value 0.0001) which means that pulmonary wedge pressure increased over time. Moreover there is no statistical significant difference in the interaction of groups with time (p-value 0.788) (Fig. 4).

C- **Arterial Blood Gases Variables:**

- **Partial pressure of oxygen:**
  Comparison of effects of twelve hours of VC-CMV and twelve hours of APRV on partial pressure of oxygen:
  During the application of twelve hours period of conventional lung protective ventilation and APRV to all study patients, we found out that there was statistical significant reduction in measured partial pressure of oxygen in the second group (p-value 0.046) (Table 1).

- **Partial pressure of carbon dioxide:**
  Comparison of effects of twelve hours of VC-CMV and twelve hours of APRV on partial pressure of carbon dioxide:
  On evaluating the partial pressure of carbon dioxide of all study patients who were subjected to twelve hours period of conventional lung protective ventilation and twelve hour period of APRV, we found that there was no statistical significant difference between groups (p-value 0.907) (Table 2).

- **Acid-base balance:**
  Comparison of effects of twelve hours of VC-CMV and twelve hours of APRV on acid-base balance:
  On evaluating the acid-base balance of all study patients who were subjected to twelve hours period of conventional lung protective ventilation and twelve hour period of APRV, we found that there was no statistical significant difference between groups (p-value 0.247) (Table 3).

D- **Effects on respiratory parameters and lung mechanics:**

- **Minute ventilation:**
  Comparison of effects of twelve hours of VC-CMV and twelve hours of APRV on minute ventilation:
  On evaluating the minute ventilation of all study patients who were subjected to twelve hours period of conventional lung protective ventilation and twelve hour period of APRV, we found that there was statistical significant increase in the second group (p-value 0.008) (Table 4).

- **Dynamic compliance:**
  Comparison of effects of twelve hours of VC-CMV and twelve hours of APRV on compliance:
  On evaluating the respiratory system compliance of all study patients who were subjected to twelve hours period of conventional lung protective ventilation and twelve hour period of APRV, we found out that there was no statistical significant difference between the two groups (p-value: 0.718) (Table 5).

- **Airway resistance:**
  On evaluating the airway resistance of all study patients who were subjected to twelve hours period of conventional lung protective ventilation and twelve hour period of APRV, we found that there was no statistical significant difference between the interaction of groups and time (p-value 0.249), there was no significant time effect, (p-value 0.612), which means that the airway resistance did not change over time (Fig. 5).

- **Peak airway pressure:**
  Comparison of effects of twelve hours of VC-CMV and twelve hours of APRV on peak airway pressure:
  On evaluating the peak airway pressure of all study patients who were subjected to twelve hours period of conventional lung protective ventilation and twelve hour period of APRV, we found that there was high statistical significant reduction in the second group (i.e; the two groups are significantly different from each other; the two lines are rather far apart), (p-value 0.0001) (Fig. 6).
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**Mean airway pressure:**
Comparison of effects of twelve hours of VC-CMV and twelve hours of APRV on mean airway pressure measurements:

On evaluating the mean airway pressure of all study patients who were subjected to twelve hours period of conventional lung protective ventilation and twelve hour period of APRV, we found that there was high statistical significant reduction in the second group (i.e.; the two groups are significantly different from each other; the two lines are rather far apart), \(p\)-value 0.0001). There was a highly statistically significant time effect, i.e., the two groups do change in mean airway pressure over time \(p\)-value: 0.0001). Moreover the interaction of time and groups is statistically significant \(p\)-value: 0.011) which means that groups are changing over time but also changing in different ways (Fig. 7).

**Respiratory rate:**
- **Comparison of effects of twelve hours of VC-CMV and twelve hours of APRV on respiratory rate:**

During the application of twelve hours period of lung protective ventilation and APRV to all study patients, we found out that there was statistical significant increase in the respiratory rate in the second group \(p\)-value 0.007) i.e.; the 2 groups are significantly different from each other; the two lines are rather far apart. The interaction of groups and time indicate that there is statistically significant difference which means that the groups are changing over time but are changing in different ways \(p\)-value: 0.02). Moreover there is statistical significant difference in respiratory rate with time in each group independently \(p\)-value: 0.001) (Fig. 8).

**E- Need of sedation requirments:**

On evaluating the sedation dosage requirements of all study patients who were subjected to twelve hours period of conventional lung protective ventilation and twelve hour period of APRV, we found that there was statistical significant difference between groups \(p\)-value 0.0001) which means that the second group used a significantly less sedation dose than the 1\textsuperscript{st} group. There was a significant time effect, \(p\)-value 0.0001) which means that the sedation dosage changes significantly over time (i.e.; increased in the 1\textsuperscript{st} group, and decreased in the 2\textsuperscript{nd} group). Moreover regarding the interaction of groups and time there was a statistically significant difference between both groups. \(p\)-value 0.0001), which means that the groups are changing over time and changing in different ways (Fig. 9).
Table (1): Comparison of mean (±SD) values of partial pressure of oxygen during twelve hours VC-CMV and APRV.

<table>
<thead>
<tr>
<th>No.</th>
<th>Time</th>
<th>1st group Mean ± SD mmHg</th>
<th>2nd group Mean ± SD mmHg</th>
<th>p-value between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>3</td>
<td>144.6±45.6</td>
<td>132.4±41.0</td>
<td>0.046</td>
</tr>
<tr>
<td>20</td>
<td>6</td>
<td>164.1±49.3</td>
<td>134.1±52.7</td>
<td>0.907</td>
</tr>
<tr>
<td>20</td>
<td>9</td>
<td>164.9±45.5</td>
<td>127.9±47.6</td>
<td>0.907</td>
</tr>
<tr>
<td>20</td>
<td>12</td>
<td>166.1±46.3</td>
<td>130.8±47.9</td>
<td>0.907</td>
</tr>
</tbody>
</table>

Table (2): Comparison of mean (±SD) values of partial pressure of carbon dioxide during twelve hours VC-CMV and APRV.

<table>
<thead>
<tr>
<th>No.</th>
<th>Time</th>
<th>1st group Mean ± SD mmHg</th>
<th>2nd group Mean ± SD mmHg</th>
<th>p-value between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>3</td>
<td>36.9±7.2</td>
<td>36.7±8.6</td>
<td>0.907</td>
</tr>
<tr>
<td>20</td>
<td>6</td>
<td>35.8±5.8</td>
<td>36.2±8.1</td>
<td>0.907</td>
</tr>
<tr>
<td>20</td>
<td>9</td>
<td>36.7±5.4</td>
<td>36.8±8.1</td>
<td>0.907</td>
</tr>
<tr>
<td>20</td>
<td>12</td>
<td>36.5±5.7</td>
<td>37.3±7.7</td>
<td>0.907</td>
</tr>
</tbody>
</table>

Table (3): Comparison of mean (±SD) values of acid-base balance during twelve hours VC-CMV and APRV.

<table>
<thead>
<tr>
<th>No.</th>
<th>Time</th>
<th>1st group Mean ± SD mmHg</th>
<th>2nd group Mean ± SD mmHg</th>
<th>p-value between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>3</td>
<td>7.4±7.0</td>
<td>7.4±5.9</td>
<td>0.247</td>
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<tr>
<td>20</td>
<td>6</td>
<td>7.4±5.5</td>
<td>7.5±5.1</td>
<td>0.247</td>
</tr>
<tr>
<td>20</td>
<td>9</td>
<td>7.4±5.5</td>
<td>7.5±5.0</td>
<td>0.247</td>
</tr>
<tr>
<td>20</td>
<td>12</td>
<td>7.4±6.1</td>
<td>7.5±5.1</td>
<td>0.247</td>
</tr>
</tbody>
</table>

Table (4): Comparison of mean (±SD) values of minute ventilation during twelve hours VC-CMV and APRV.

<table>
<thead>
<tr>
<th>No.</th>
<th>Time</th>
<th>1st group Mean ± SD mmHg L/min</th>
<th>2nd group Mean ± SD mmHg L/min</th>
<th>p-value between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>3</td>
<td>8.6±2.2</td>
<td>10.2±2.4</td>
<td>0.008</td>
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<tr>
<td>20</td>
<td>6</td>
<td>8.5±2.4</td>
<td>10.3±3.2</td>
<td>0.008</td>
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<tr>
<td>20</td>
<td>9</td>
<td>8.3±2.2</td>
<td>11.1±2.7</td>
<td>0.008</td>
</tr>
<tr>
<td>20</td>
<td>12</td>
<td>8.6±2.4</td>
<td>10.9±2.2</td>
<td>0.008</td>
</tr>
</tbody>
</table>
ARDS: All these patients were diagnosed to have ALI/ARDS by Sydow  

The two modes of ventilation indicate that there was no statistical significant difference in mean blood pressure (MBP) between both modes of ventilation (12.5 ± 13.2 CMV, 13.2 ± 14.7 APRV) p-value 0.313.

In our study the insignificant change in MBP between the two modes of ventilation indicate that optimum levels of PEEP and Peak airway pressure were used to maintain optimal levels of oxygenation without affecting haemodynamics.


Regarding heart rate evaluation there was no significant change in heart rate between the two ventilator modes during the twelve hour period of ventilation (101.2 ± 14.7 CMV, 100.7 ± 14.9 APRV) p-value 0.731.

In concordance to our results studies conducted by Sydow [3]; Putensen [4]; Wrigge [5]; Varpula [6] and Dart [7], showed no statistical significant change in heart rate readings between the two modes of ventilation p-values >0.05.

When taking measurements of central venous pressure in all study patients, we found that there was no significant change between both modes of ventilation during the twelve hour period of ventilation (7.95 ± 2.1 CMV, 7.8 ± 1.3 APRV) p-value 0.761.

Similar results were reached during previous clinical studies conducted by Sydow [3]; Putensen [4]; Wrigge [5]; Varpula [6] and Dart [7], which showed no statistical significant change in central venous pressure readings between the two modes of ventilation p-values >0.05.

During the twelve hour period of ventilation with CMV with lung protective strategy and APRV, there was no statistical significant difference in pulmonary capillary wedge pressure (PCWP) between both modes of ventilation (12.5 ± 2.7 CMV, 13.2 ± 2.3 APRV) p-value 0.313.

Similar results were reached during previous clinical studies conducted by Sydow [3]; Putensen [4]; Wrigge [5], Varpula [6] and Dart [7], which showed no statistical significant change in pulmonary capillary wedge pressure readings between the two modes of ventilation as all intravascular volumes were optimized to maintain haemodynamics and adequate tissue perfusion p-values >0.05.

It was clear from the above results that APRV can safely enhance haemodynamics with ALI/ARDS patients and in the same time has a beneficial effect on arterial blood gases and hypoxia index.

<table>
<thead>
<tr>
<th>Number</th>
<th>Time</th>
<th>1st group Mean ± SD cmH2O</th>
<th>2nd group Mean ± SD cmH2O</th>
<th>p-value variable interaction of between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>3</td>
<td>45.3 ± 21.9</td>
<td>53.0 ± 27.5</td>
<td>0.718</td>
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<tr>
<td>20</td>
<td>9</td>
<td>51.5 ± 25.8</td>
<td>52.6 ± 27.8</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>12</td>
<td>53.0 ± 25.8</td>
<td>52.9 ± 26.9</td>
<td></td>
</tr>
</tbody>
</table>

**Discussion**

In our present study, twenty patients were admitted to the Critical Care Department, Cairo University Hospital between August 2006 to April 2008, it included 11 males (55%), and 9 females (45%), who had a mean age of 55.6 ± 15.2 (range between 22 to 75 years), mean ideal body weight of 68 ± 8.5 kg.

**All these patients were diagnosed to have ALI/ARDS:**

In the present work, on evaluating the mean arterial blood pressure during the twelve hours of conventional lung protective ventilation and APRV, we found that there was no statistical significant change in mean blood pressure (MBP) between the two modes of ventilation (94.5 ± 12.6 CMV, 94.8 ± 13.8 APRV) p-value 0.804.

In our study the insignificant change in MBP between both modes of ventilation indicates that optimum levels of PEEP and Peak airway pressure were used to maintain optimal levels of oxygenation without affecting haemodynamics.

<table>
<thead>
<tr>
<th>Number</th>
<th>Time</th>
<th>1st group Mean ± SD cmH2O</th>
<th>2nd group Mean ± SD cmH2O</th>
<th>p-value variable with time</th>
<th>p-value interaction of group and time</th>
<th>p-value between groups</th>
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</thead>
<tbody>
<tr>
<td>20</td>
<td>3</td>
<td>33.2 ± 6.5</td>
<td>20.1 ± 4.1</td>
<td>0.0001</td>
<td>0.0001</td>
<td>0.0001</td>
</tr>
<tr>
<td>20</td>
<td>6</td>
<td>32.3 ± 6.9</td>
<td>20.1 ± 3.7</td>
<td></td>
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</tr>
<tr>
<td>20</td>
<td>9</td>
<td>31.9 ± 6.9</td>
<td>20.1 ± 3.6</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>20</td>
<td>12</td>
<td>32.4 ± 6.7</td>
<td>19.8 ± 4.0</td>
<td></td>
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</tr>
</tbody>
</table>
effect of the two modes of ventilation on the partial pressure of carbon dioxide in both groups which showed that partial pressure of carbon dioxide in all patients in our study showed no significant change between the two modes of mechanical ventilation (36.5±5.7 CMV, 37.3±7.7 APRV) p-value 0.907.

In close relation to our study Kaplan [8] conducted a study in 12 patients aiming to determine whether APRV can safely enhance haemodynamics with ALI/ARDS relative to pressure control and found out that there was no significant change in partial pressure of CO2 between both ventilator modes p-value >0.05.

This was also stated in studies conducted by Sydow [3], Putensen [4], Wrigge [5], Varpula [6] and Dart [7] p-value >0.05, and also from the effect of the two modes of ventilation on the Acid-base balance of the two groups of patients which showed that during the twelve hour period of ventilation by the two different modes of ventilation, there was no significant change in pH (7.4±6.1 CMV, 7.5±5.1 APRV) p-value 0.247.

Studies by Sydow [3], Putensen [4], Wrigge [5], Varpula [6] and Dart [7] showed same results as ours with no change in pH between the two modes of ventilation p-value >0.05.

As regard the effect of both types of ventilation on the partial pressure of oxygen and hypoxic index our results showed that during the twelve hour period of ventilation of all study patients with APRV there was a significant decrease in partial pressure of oxygen compared to twelve hours of controlled mechanical ventilation with lung protective strategy with a 21.3% decrease (CMV 166.1±46.3, APRV 130.8±47.9) p-value 0.046.

Associated with that, there was a highly significant 20.1% reduction in Hypoxic Index after 12 hours of APRV following twelve hours of CMV (CMV 275±80.7, APRV 219.9±86.8) p-value 0.001.

This significant decrease is attributed to decrease in mean airway pressures with APRV, and our study contains small sample size and our patients were only subjected to twelve hours of APRV.

In contrast to what seems disadvantageous in the above discussion, Hypoxic Index and partial pressure of oxygen after twelve hours of APRV increased significantly by 32.5% and 29.3% respectively compared to baseline readings before inclusion in our study (baseline pO2 101±31, APRV pO2 130.8±47.8), (baseline shunt fraction 166±57.2, APRV shunt fraction 220±86.8) p-values 0.0001.

Although target levels of pO2 and Hypoxic Index were not reached by APRV as in CMV, yet data recently discussed above denote sufficient and optimum level of recruitment and oxygenation with APRV compared to baseline readings before inclusion in our study.

A study conducted by Kaplan [8] and Varpula [6] showed no significant change in Hypoxic Index in patients with ALI/ARDS when ventilated with PCV versus APRV (PCV 168±24 versus APRV 182±18) p-value >0.05.

While a study conducted by B. Dart [7] (2005) on 46 trauma patients with ALI/ARDS showed a significant increase 13% in Hypoxic Index when using APRV (APRV 299±88, CMV 243±141) p-value <0.05, this may attributed to the aetiology of ALI/ARDS being trauma only, our patients included septic patients that had an exaggerated inflammatory response that had a more significant impact on the respiratory compliance and oxygen indices.

Wrigge [8] conducted a study on 30 patients with ALI/ARDS who were randomly assigned for PCV and APRV for 72 hours, his study resulted in a significant 30% increase in Hypoxic Index, this study goes hand in hand with the study conducted by Putensen [4] on 24 patients with ALI/ARDS using APRV showed increase in partial pressure of oxygen by 13% p-value <0.05.

This may be attributed to more hours of ventilation (72 hours) of APRV used for the patients in these studies compared to twelve hours in our study.

In our study, there was a significant increase in respiratory rate and minute ventilation by 29.3% and 26.7% respectively during the twelve hours of APRV compared to twelve hours of CMV with respiratory rate increase from 21.5±6.9 with CMV to 27.8±6.2 APRV p-value 0.007. Minute ventilation increased from 8.6±2.4 CMV to 10.9±2.2 APRV p-value 0.007. This significant increase was anticipated and was owed to increased spontaneous respiratory efforts due to decrease in sedation levels with APRV (minute ventilation did not exceed normal range).

Apparently this may seem disadvantageous but this goes hand in hand with lung protective ventilation strategy including ventilation with high respiratory rates targeting a normal pH and accept-
able pO₂. Especially that tidal volumes didn’t change significantly between the two modes of ventilation used in our study, \( p \)-value 0.742, so patients on APRV received tidal volumes of almost 6ml/kg ideal body weight which goes hand in hand with lung protective strategy.

Putensen and Wrigge [9] studies showed no significant change in minute ventilation between both groups of patients receiving APRV versus PCV for 72 hours, \( p \)-value >0.05. Yet there was a significant increase in spontaneous respiratory breath with APRV compared to PCV, \( p \)-value <0.05. This agrees with our study which states that increase in respiratory rate was owed to increase in spontaneous breathing efforts.

Assessment of dynamic compliance of the respiratory system of all study population we found that there was no significant changes between both modes of ventilation (53 ±25.8 CMV, 52.9 ±26.9 APRV) \( p \)-value >0.718.

A highly significant decrease of 38.9% in peak airway pressure was noted in our study during ventilation with APRV compared to CMV, (APRV 19.8±4, 32.4±6.7 CMV), \( p \)-value 0.001.

Associated with that there was also a highly significant 19% decrease in mean airway pressure during ventilation with APRV compared to CMV with lung protective strategy (CMV 18.9 ±3.7, APRV 15.3±3.3), \( p \)-value 0.0001.

These results are supported by the studies conducted by Kaplan [8] who showed a decrease in peak airway pressure by 34.2% and mean airway pressure by 33% with APRV, \( p \)-value <0.01.

The need for sedation by propofol during the twelve hour period of APRV significantly decreased by 56.2% compared to the twelve hours of application of lung protective ventilation using CMV, where the dosage of propofol decreased from 1.6 ± 0.5mg/kg/hr with CMV to 0.7 ±0.8mg/kg/hr with APRV, \( p \)-value 0.000 1.

In concordance to our results, the study conducted by Kaplan [8] showed a decrease in sedation requirements by 32% with APRV \( p \)-value <0.01.

Conclusion:

From our present study we conclude the following:

• Our study did not demonstrate any significant change in the haemodynamic variables (heart rate, mean blood pressure, central venous pressure, and pulmonary capillary wedge pressure) between both modes of ventilation.

• On evaluating the blood gases variables our study showed no change in partial pressure of carbon dioxide, serum bicarbonate level and pH values between the two modes of ventilation.

However, there was a significant increase in partial pressure of oxygen and in Hypoxic Index with APRV and VC-CMV compared to the baseline readings while being ventilated with nonprotective lung strategy, yet this increase was more significant with VC-CMV than APRV.

• We also demonstrated that there was an increase in respiratory rate and minute ventilation with APRV compared to VC-CMV, however there was no change in tidal volume between the two modes of ventilation.

• Effect of both modes of ventilation on respiratory mechanics in our study revealed significant increase in dynamic compliance with both modes of ventilation compared to the baseline readings with nonprotective lung strategy; however no significant difference was noticed between VC-CMV and APRV.

• Regarding peak airway pressure and mean airway pressure there was a significant decrease with APRV compared to VC-CMV.

• Our study demonstrated a significant decrease in need for sedation with APRV compared to VC-CMV.

Recommendations:

We recommend that APRV can be used safely as one of the optimum ventilatory strategies in patients with ALI/ARDS as it decreases airway pressures significantly and decreases the need for sedation while maintaining adequate oxygenation without altering haemodynamics.

References


