ACUTE RESPIRATORY DISTRESS SYNDROME: RAPID AND SIGNIFICANT RESPONSE TO VOLUME-CONTROLLED INVERSE RATIO VENTILATION

- A Case Report -

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Abstract

Pulmonary complications following cardiopulmonary bypass (CPB) are relatively common, with up to 12% of patients experiencing acute lung injury (ALI). The treatment for ALI or acute respiratory distress syndrome (ARDS) is primarily supportive with specific modes of mechanical ventilation. We report a 46-year-old man with ARDS after cardiac surgery whose arterial oxygenation was surprisingly improved 1 hour after using volume-controlled inverse ratio ventilation (VC-IRV).

Key words: pulmonary complication, CABG, treatment of ARDS.

Introduction

Pulmonary complications following cardiopulmonary bypass (CPB) are relatively common, with up to 12% of patients experiencing acute lung injury (ALI). The overall goals of mechanical ventilation in ARDS are: to maintain acceptable gas exchange and to minimize the occurrence of adverse effects associated with its application.

A growing consensus currently supports the use of low tidal volume ventilation, with positive end expiratory pressure (PEEP)1,2,4. Current clinical practice with known or suspected lung injury is, however, to limit inflation pressure. The inverse ratio ventilation (IRV) is a mode of mechanical ventilation in which the inspiratory time is prolonged (I≥E) and has the advantage over the conventional use of (E ≥ I and extrinsic-PEEP) in that unacceptable increases in peak airway pressures and peak alveolar pressures can be avoided3.
Case Report

A 46-year-old man, with a 5 year history of ischemic heart disease, hypertension and severe opium addiction and a 15 year history of heavy smoking, with EF = 50% and three vessels disease, was operated for coronary artery bypass graft (CABG) surgery, under cardiopulmonary bypass (CPB). The operation was uneventful.

After operation, in the ICU, because of bleeding with chest tube drainage in place, patient was sedated. Following reduction of drainage, weaning of patient from ventilator was gradually started. The day after, patient was completely weaned and was extubated.

Thirty six hours after extubation, however, he suddenly had an acute onset of severe arterial hypoxemia resistant to oxygen therapy. Meanwhile, the hemodynamics were stable, ECG did not show arrhythmia or ischemia, echocardiography did not show evidence of left ventricular failure. At this time (arterial pressure of oxygen) PaO₂/FIO₂ (fraction of inspiratory oxygen) was<100 (Table1-ABG1).

The patient was rapidly intubated, sedated and ventilated with conventional ventilator support that is usually used following surgery (Volume cycled, SIMV, RR = 12/min, Vt = 10cc/kg, FiO₂ = 50%, I:E = 1:2, PS = 15 cm H₂O, PEEP = 5 cm H₂O) following cardiac surgery (Table1-ABG2).

Chest x-ray revealed bilateral infiltration of the lungs (3 quadrant at first and 4 quadrant 1 hour later).

Based on Murray lung injury score that awards points for affected quadrants on chest x-ray, PaO₂/FiO₂ ratio, amount of PEEP applied and static compliance of the lungs (Table 2) our patient’s score was greater than 2.5, which confirmed severe ARDS. The ventilator’s settings were therefore changed to high PEEP level with low tidal volume. With this setting arterial oxygenation was improved, PaO₂ reached to 61-63 mmHg and O₂ saturation reached to 93-94% (Table 1-ABG3).

### Table 1

<table>
<thead>
<tr>
<th>ABG results (arterial blood gas)</th>
<th>ABG Result 1</th>
<th>ABG Result 2</th>
<th>ABG Result 3</th>
<th>ABG Result 4</th>
<th>ABG Result 5</th>
<th>ABG Result 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.34</td>
<td>7.39</td>
<td>7.31</td>
<td>7.48</td>
<td>7.35</td>
<td>7.44</td>
</tr>
<tr>
<td>PO₂</td>
<td>50</td>
<td>37</td>
<td>69</td>
<td>115</td>
<td>182</td>
<td>66</td>
</tr>
<tr>
<td>O₂ saturation</td>
<td>82</td>
<td>68</td>
<td>92</td>
<td>98</td>
<td>100</td>
<td>94</td>
</tr>
<tr>
<td>PCO₂</td>
<td>41</td>
<td>32</td>
<td>39</td>
<td>35</td>
<td>37</td>
<td>35</td>
</tr>
<tr>
<td>HCO₃</td>
<td>22</td>
<td>19</td>
<td>26</td>
<td>26</td>
<td>20</td>
<td>23</td>
</tr>
<tr>
<td>Total CO₂</td>
<td>23</td>
<td>20</td>
<td>27</td>
<td>27</td>
<td>21</td>
<td>24</td>
</tr>
<tr>
<td>B.E</td>
<td>-3</td>
<td>-5</td>
<td>3</td>
<td>4</td>
<td>-4</td>
<td>0</td>
</tr>
</tbody>
</table>

ABG result 1 - despite O₂ supplementation,
ABG result 2 - after re-intubation
ABG result 3 - with high PEEP levels and low tidal volume,
ABG result 4 - with IRV
ABG result 5 - with supplemental O₂ the day of discharge from ICU.
ABG result 6 - without supplemental O₂ on the day of discharge from ICU.

### Table 2

<table>
<thead>
<tr>
<th>Points</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>All four</th>
</tr>
</thead>
<tbody>
<tr>
<td>CXR No of quadrants</td>
<td>No infiltration</td>
<td>One</td>
<td>Two</td>
<td>Three</td>
<td>&lt;100</td>
<td></td>
</tr>
<tr>
<td>PaO₂/FiO₂</td>
<td>≥300</td>
<td>225 to 299</td>
<td>165 to 224</td>
<td>100 to 174</td>
<td>&lt;100</td>
<td></td>
</tr>
<tr>
<td>PEEP cmH₂O</td>
<td>≤5</td>
<td>6 to 8</td>
<td>9 to 11</td>
<td>12 to 14</td>
<td>&gt;15</td>
<td></td>
</tr>
<tr>
<td>Cstat</td>
<td>≥80</td>
<td>60 to 79</td>
<td>40 to 59</td>
<td>20 to 39</td>
<td>≤19</td>
<td></td>
</tr>
</tbody>
</table>

Final score = aggregate Sum/No. of components assessed.
(0 = No lung injury; 0.1 to 2.5 = Acute lung injury; >2.5 = Adult respiratory distress syndrome).
The same ventilation was maintained for the following 48 hour, but PaO₂ did not increase and emphysema began to show.

With this development, the ventilator’s settings were modified to deliver inverse ratio ventilation (IRV) with reduction in ventilator rate and PEEP discontinued. An hour later, PaO₂ reached up to 115 mmHg and arterial O₂ saturation (SaO₂) of 98% (Table 1-ABG 4).

Weaning of patient was started, sedative drugs were tapered and surprisingly in 8 hour patient’s status normalized and he was extubated. Emphysema was reduced in 48 hour. Patient was discharged from ICU with acceptable ABG (Table 1-ABG 5 & 6), chest-x-ray (chest-x-ray 3) and stable hemodynamics (MAP = 83mmHg, HR = 87 beat/min, no serious arrhythmias and EF = 45%).

**Discussion**

Patients undergoing cardiac surgery experience physiologic stresses from anesthesia, surgical manipulation, and CPB. ARDS may develop as a sequel of CPB, or, more commonly, in the postoperative patient with cardiogenic shock, sepsis, or multiple organ failure.

The treatment for ALI or ARDS is primarily supportive with mechanical ventilation, a procedure allowing time for treatment of the underlying cause of lung injury and for natural healing. Low tidal volume ventilation should be applied to all patients with ARDS unless more efficacious strategy is demonstrated.

For more than two decades, PEEP has been used to improve arterial oxygenation in patients with ARDS. Indeed, several recent studies have found improved hemodynamic performance and fewer pulmonary complications using high PEEP levels with tidal volumes as low as 6 ml/kg in these patients.

Due to the increased physiologic dead space of patients with ARDS, ventilator rates greater than 20-25 breath/min are often required to normalize PaCO₂ and pH, unless excessive intrathoracic gas trapping occurs, leading to development of auto-PEEP which has the potential of adverse effects including barotraumas, hemodynamic instability, increased work of breathing, and decreased efficiency of diaphragmatic...
contractility. We also used this method (tidal volume = 6 ml/kg, PEEP + 8-15 cm H₂O, ventilator rate + 18-20 breath/min) on our patient but it could not increase PaO₂ to more than 69-74 mmHg.

Because of the preceding event and the start of emphysema (adverse effect of auto-PEEP), we changed the mode to volume-controlled-inverse ratio ventilation (VC-IRV). Other investigations have concluded that the effect of reduced expiratory time ventilation (VC-IRV) is similar to the use of PEEP. However, a growing oxygenation during volume-controlled VC-IRV, is considered the effect of reduced expiratory time ventilation (VC-IRV). Other investigations have changed the mode to volume-controlled-inverse ratio ventilation (VC-IRV) is recommended. Other investigations have been directed on the late effects of emphysema (adverse effect of auto-PEEP), we have observed that unacceptable increases in peak airway pressures and peak alveolar pressures can be avoided.

In our case, we could not highly improve arterial oxygenation with using of high PEEP levels and low tidal volume, but with the use of VC-IRV, oxygenation was rapidly and significantly improved while acceptable peak airway pressure was maintained.

Although beneficial effects of VC-IRV are known, and studies have been directed on the late effects of this mode of ventilation, yet the rapid and significant effect on oxygenation has not been reported.

Conclusions

In a situation where acceptable arterial oxygenation cannot be achieved with PEEP less than 15 cmH₂O, or when the use of PEEP is associated with excessive plateau pressure, the volume-controlled inverse ratio ventilation (VC-IRV) is recommended.

References