



## APRV: A Ray of Hope Despite all of the Darkness

Authors

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A 60 year old male, known case of morbid obesity with obstructive sleep apnoea presented with complains of increasing breathlessness for 2 days. He was afebrile, drowsy and dyspnoeic, BMI was 44 Kg/m<sup>2</sup> (Obesity class III) with heart rate 116/min, blood pressure 170/90 mmhg, SpO<sub>2</sub> 64% on room air and Respiratory rate 26-30/min. On auscultation heart sounds were normal and bilateral air entry was decreased in lungs with presence of wheeze. Arterial blood gas was suggestive of type II respiratory failure with severe respiratory acidosis. Initially BIPAP was applied. However, the patient subsequently developed bradycardia. He was intubated and revived by Cardiopulmonary resuscitation as per ACLS protocol. Post resuscitation he was haemodynamically stable with GCS E<sub>2</sub>V<sub>T</sub>M<sub>3</sub>.

Diagnosis of severe Acute respiratory distress syndrome (ARDS) was made based on PaO<sub>2</sub>/FiO<sub>2</sub> of 78.75 as per Berlin definition.<sup>[1]</sup> Chest x-ray showed bilateral opacities in lung parenchyma. 2D-Echo was normal. He was initially ventilated with Volume control (VC) mode with 80% FiO<sub>2</sub>, 500ml TV, 24/min RR and PEEP of 14 cm of H<sub>2</sub>O. He was sedated and paralysed with infusions of Inj. Fentanyl and Inj. Atracurium. However hypoxia did not improve as PaO<sub>2</sub> was 58 mmhg but PCO<sub>2</sub> decreased to 48mmhg. Peak airway pressures were 40-42 cm of H<sub>2</sub>O.

Recruitment manoeuvre was tried with PEEP of 30 cm of H<sub>2</sub>O, however it was not fruitful as PaO<sub>2</sub> was same and SpO<sub>2</sub> was 86% with 80% FiO<sub>2</sub>. The mode of ventilator was changed to Pressure control (PC) with 75% FiO<sub>2</sub>, 22/12 cm of H<sub>2</sub>O PS/PEEP, 20/min RR. With PC mode, Peak airway pressures were similar and also PaO<sub>2</sub> was 55mmhg.

We considered placing the patient in prone position, as suggested by PROSEVA trial.<sup>[2]</sup> However as the patient was morbidly obese, the decision was deferred as it would have significantly affected the nursing care. Finally the patient was ventilated by APRV mode with initial settings: 70% FiO<sub>2</sub>, 30 cm of H<sub>2</sub>O P high, 6 cm of H<sub>2</sub>O P low, 5 sec T high, 0.5 sec T low. PaO<sub>2</sub> increased to 65 mmHg within 2 hours and SpO<sub>2</sub> increased to 90%. He was tracheotomised in i/v/o low GCS. By day 10 in ICU, with 55% FiO<sub>2</sub>, PaO<sub>2</sub> and SpO<sub>2</sub> increased to 70 mmhg and 94% respectively. P high was gradually decreased to 22 cm of H<sub>2</sub>O but P low was same. There was no need for paralysis with APRV mode and requirement for sedation was occasional. Peak pressures were 32-35 cm of H<sub>2</sub>O.

Patient was weaned from APRV to PSV mode with PS/PEEP of 20/8 cm of H<sub>2</sub>O and 50% FiO<sub>2</sub>. Finally shifted to portable BIPAP machine with similar settings. We believe since muscle

relaxation was not required with APRV mode, we could wean the patient early from the ventilator. However, we ultimately lost the patient to Septic shock with multi-organ failure.

Our experience is supported by a randomized controlled trial, in which 30 mechanically ventilated trauma patients were randomly assigned to either APRV or pressure-limited ventilation. APRV was associated with shorter duration of mechanical ventilation, a shorter ICU length of stay, and use of less sedatives and paralytics.<sup>[3]</sup>

Thus to conclude, morbidly obese patients with ARDS, in whom conventional modes of ventilation and other adjunctive therapies may be of questionable benefit can show good response to APRV mode.

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#### References

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