

# Ventilating Patient with Refractory Hypercarbia: Use of APRV Mode

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## ABSTRACT

A 70-year-old patient referred to our critical care unit with the diagnosis of type II respiratory failure with shock. Patient was a known case of COPD for last 20 years. His chest radiology revealed bilateral infiltrates. Patient was managed conservatively in the form of antibiotics, vasopressor and ventilatory support with SIMV/VC mode. After ventilation with SIMV/VC mode for half an hour his blood gases revealed increasing PaCO<sub>2</sub> levels. The same result was obtained with PC mode and ASV and his PaCO<sub>2</sub> level reached above 170 mmHg. Then APRV mode was tried with modified settings. The results obtained were satisfactory and in next 24 hours PaCO<sub>2</sub> decreased to <66mmHg along with an increasing P/F ratio. APRV is not recommended as primary mode of ventilation in COPD but in resistant cases it can be helpful as it improves alveolar recruitment and pressure support is added to reduce hypercapnia.

**Keywords:** COPD, Hypercapnia, SIMV, Respiratory failure

## CASE REPORT

A 70-year-old male patient referred with the diagnosis of acute exacerbation of chronic obstructive pulmonary disease (COPD) with pneumonia and shock in our ICU. On admission his blood gases revealed PaO<sub>2</sub>- 102 mmHg, PaCO<sub>2</sub>- 84.2 mmHg, pH- 7.244, HCO<sub>3</sub><sup>-</sup>- 35.3 meq/l. Initially patient was managed with broad spectrum antibiotics, bronchodilators and vasopressor and other supportive treatment along with mechanical ventilation with SIMV/VC mode with settings: Pressure support (PS)- 16, positive end expiratory pressure (PEEP) - 6, FiO<sub>2</sub> - 50%

After ventilation for about 1 hour his blood gas analysis (ABG) was done, which showed PaCO<sub>2</sub> 73.2 mmHg and PaO<sub>2</sub> 79.4 mmHg [Table/Fig-1]. Then we switched over to PSIMV mode and ABG was done after one hour, which showed PaCO<sub>2</sub> 173 mmHg and PaO<sub>2</sub> 85 mmHg [Table/Fig-1].

Still patient's ventilation was not improved. PSIMV mode resulted in poor outcome with a PaCO<sub>2</sub> 173mmHg, the similar results were obtained when we shifted to ASV mode and patient's saturation started falling.

So, PSIMV mode was again used but this time along with sedation and neuromuscular blocker. Despite all these efforts and different mode and strategy, the ventilation was not sufficient and PaCO<sub>2</sub> did not decrease.

Finally all sedation and paralysis was stopped and patient was tracheostomised. APRV mode without inverse ventilation was started along with pressure support. P high was 35 mmHg, P low was 5 mmHg with I:E ratio 1:4 with theoretically working as PSIMV mode with a time cycled breath allowing spontaneous effort in between the cycle. After one hour of ventilation, the PaCO<sub>2</sub> levels came down to 156 mmHg from 173 mmHg. In next few hours PaCO<sub>2</sub> levels further reduced to 90 mmHg and P/F ratio 122 and in

next 24 hours PaCO<sub>2</sub> level was 62mmHg with a PaO<sub>2</sub> 97.6 mmHg [Table/Fig-1]. Patient improved over a week with same ventilation strategy and weaned off from ventilator.

## DISCUSSION

COPD is an obstructive airway disease characterized by increased airway resistance secondary to secretions, oedema, increased bronchial muscle tone facilitating premature airway closure at the level of small airways, resulting in poor ventilation of these regions and hence low ventilation perfusion (V/Q) ratios. This forces inspired air to other regions which may be ventilated in excess of their perfusion which is measured as dead space. Such patients have to hyperventilate excessively to maintain PaCO<sub>2</sub> resulting in alveolar hypoventilation resulting in CO<sub>2</sub> retention [1].

Airway pressure release ventilation (APRV) is a ventilation mode which produces alveolar ventilation as an adjunct to continuous positive airway pressure (CPAP) [2]. The variable P high, the set airway pressure is transiently released to a lower level P low, after which it is quickly restored to re-inflate the lungs. APRV allows spontaneous breathing at anytime during the respiratory cycle. In the absence of spontaneous breathing APRV mode is similar to pressure controlled inverse ratio ventilation. APRV mode is mostly used and most studied mode in patient with acute respiratory distress syndrome (ARDS). It has not been much studied in patient with (COPD).

APRV mode has traditionally been used in ARDS patients as an inverse mode of ventilation with prolonged inspiratory time and short expiratory time, but for COPD settings were modified to increase expiratory time (I:E ratio=1:4) to facilitate CO<sub>2</sub> removal in the setting of increased airway resistance, also P low was kept as 5mmHg to avoid lung collapse during prolonged T low. But with the same settings in PCV mode we failed to achieve the desired results.

Presently, APRV is not considered as a mode of ventilation in COPD as due to prolong T high, hypercapnia occurs. However, in APRV the lung is ventilated at lower mean airway pressure near to low inflection point and it reduces over distension of alveoli as occurs in COPD. It improves oxygenation by better lung recruitment and promotes gaseous exchange. Hypercapnia can be prevented with modifying the ventilatory settings such as adding pressure support (i.e. bi-level mode) or changing I:E ratio as in our case [2]. Better results with

	ABG on SIMV/VC	ABG on PSIMV	ABG on APRV
PaCO <sub>2</sub>	73.2mmHg	173mmHg	62mmHg
PaO <sub>2</sub>	79.4mmHg	85mmHg	97.6mmHg
pH	7.326	7.013	7.38
SO <sub>2</sub>	95.9%	92.5	97%
HCO <sub>3</sub> <sup>-</sup>	37.4	43	35

[Table/Fig-1]: ABG on different modes of ventilation

APRV mode can be backed up with as it preserves unsupported spontaneous breathing, no sedation and neuromuscular blockade is required in APRV mode to maintain patient ventilator synchrony, patient can breathe during any phase of the ventilatory cycle which mainly occurs at high CPAP level resulting in less barotrauma and V/Q matching [2,3] and mean airway pressure is lower than control mode which is the key determinant of gas exchange.

APRV targets all determinants of CO<sub>2</sub> removal such as improved pulmonary mechanics in terms of pressure volume relationship which maintains lung volumes at optimal FRC, promoting alveolar recruitment [4], hence decreases physiologic dead space ventilation [4-6]. In addition it preserves patient's spontaneous breathing efforts leading to CO<sub>2</sub> washout benefiting patients with both hypoxic and hypercarbic respiratory failure [1].

Few studies have been done on use of APRV in COPD but much more work needs to be done to arrive at conclusion [7].

## CONCLUSION

APRV is not used as primary mode of ventilation in COPD as it can lead to hypercapnia. However, APRV can be used in patients with COPD who are not sufficiently ventilated by other modes. The

settings should be modified to prevent hypercapnia. APRV should not be used in every patient of COPD rather it should be used in selected patient with proper precautions to prevent hypercapnia and over inflation. APRV may be more helpful in patients with COPD combined with ARDS.

## REFERENCES

- [1] Spurzem JR, Rennard SI. Pathogenesis of COPD. *Semin Respir Crit Care Med.* 2005;26(2):142-53.
- [2] Modrykamien A, Chatburn RL, Ashton RW. Airway pressure release ventilation: An alternative mode of mechanical ventilation in acute respiratory distress syndrome. *Cleve Clin J Med.* 2011;78:101-01.
- [3] Stock MC, Downs JB, Frolicher DA. Airway pressure release ventilation. *Crit Care Med.* 1987;15:462-66.
- [4] Maung, AA, Luckianow G, Kaplan LJ. Lessons learned from airway pressure release ventilation. *Journal of Trauma and Acute Care Surgery.* 2012;72(3):624-28.
- [5] Habashi MN. Other approaches to open-lung ventilation: Airway pressure release ventilation. *Crit Care Med.* 2005;33(3 Suppl):
- [6] Varpula T, Valta P, Niemi R, Takkinen O, Hynynen M, Pettilä V. Airway pressure release ventilation as a primary ventilatory mode in acute respiratory distress syndrome. *Acta Anaesthesiologica Scandinavica.* 2004;48(6):722-31.
- [7] Neumann P, Golisch W, Strohmeyer A, Buscher H, Burchardi H, Sydow M. Influence of different release times on spontaneous breathing pattern during airway pressure release ventilation. *Intensive Care Med.* 2002;28(12):1742-49. Epub 2002 Oct 10.

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Date of Submission: **Aug 20, 2015**  
Date of Peer Review: **Sep 23, 2015**  
Date of Acceptance: **Oct 11, 2015**  
Date of Publishing: **Jan 01, 2016**

FINANCIAL OR OTHER COMPETING INTERESTS: None.