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Case Report

Anaesthesia Section

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 $PaCO_2$ levels. The same result was obtained with PC mode and ASV and his $PaCO_2$ level reached above 170 mmHg. Then APRV mode was tried with modified settings. The results obtained were satisfactory and in next 24 hours $PaCO_2$ decreased to <66mmHg along with an increasing P/F ratio. APRV is the 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_2- 102 mmHg, $PaCO_2$- 84.2 mmHg, pH- 7.244, HCO_3- 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_2- 50%

After ventilation for about 1 hour his blood gas analysis (ABG) was done, which showed $PaCO_2$ 73.2 mmHg and PaO_2 79.4 mmHg [Table/Fig-1]. Then we switched over to PSIMV mode and ABG was done after one hour, which showed $PaCO_2$ 173 mmHg and PaO_2 85 mmHg [Table/Fig-1].

Still patient's ventilation was not improved. PSIMV mode resulted in poor outcome with a $PaCO_2$ 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 $PaCO_2$ did not deceased.

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 $PaCO_2$ levels came down to 156 mmHg from 173 mmHg. In next few hours $PaCO_2$ levels further reduced to 90 mmHg and P/F ratio 122 and in next 24 hours $PaCO_2$ level was 62mmHg with a PaO_2 97.6 mmHg [Table/Fig-1]. Patient improved over a week with same ventilation strategy and weaned off from ventilator.

	ABG on SIMV/VC	ABG on PSIMV	ABG on APRV
$PaCO_2$	73.2mmHg	173mmHg	62mmHg
PaO_2	79.4mmHg	85mmHg	97.6mmHg
pH	7.326	7.013	7.38
SO_2	95.9%	92.5	97%
HCO_3-	37.4	43	35

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

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 PaCO2 resulting in alveolar hypoventilation resulting in CO_2 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_2 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

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_2 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_2 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.

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