

Biphasic positive airway pressure and Airway pressure release ventilation

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Abstract

Biphasic positive airway pressure (BIPAP) and Airway pressure release ventilation (APRV) are two commonly used modes of ventilation. BIPAP is widely used as a 'universal mode of ventilation' for both invasive and noninvasive mechanical ventilation. Understanding the functioning of this mode helps in ventilation with this mode alone for all levels of support. APRV is a variant of BIPAP and offers benefits for protective lung ventilation. Allowing patient's spontaneous respiration unhindered at all phases of ventilation is the hallmark of these modes. The ventilatory characteristics, difference from the conventional pressure controlled ventilation (PCV), ventilatory settings, technique of weaning, indications, contraindications, advantages and disadvantages of these two modes of ventilation are described in this article.

Keywords: Airway pressure release ventilation, Biphasic positive airway pressure, Mechanical ventilation.

Introduction

Biphasic positive airway pressure (BIPAP) and Airway pressure release ventilation (APRV) are being increasingly used in intensive care units (ICU). It is important to understand the definitions, technicalities, advantages, disadvantages, indications and the method of use of these modes of ventilation. Several terminologies are used to describe the same modes of ventilation. BIPAP, BiPAP (registered trademark for noninvasive ventilation mode by Respironics Inc.), Bilevel, DUOPAP are all synonymous and have been coined separately for legal reasons. Similarly the term APRV has been often used in American journals but from the ventilation characteristics BIPAP would have been a better term.¹

BIPAP is described as a universal mode of ventilation because this mode can be used across the whole spectrum from completely controlled mechanical ventilation to completely spontaneous breathing. It

can also be used for noninvasive ventilation. BIPAP was first described in 1985 by Baum M and Benzer H.² APRV can be regarded as a variant of BIPAP. Stock MC and Downs JB first described APRV in 1987.³

BIPAP and APRV – how are they different?

BIPAP and APRV are both pressure-controlled modes of ventilation and allow spontaneous breathing at two levels of pressure (or continuous positive airway pressure, CPAP). BIPAP is essentially a CPAP system with time cycled changes between two different CPAP (or simply pressure) levels – P_{HIGH} and P_{LOW} (Figure 1). The flow is mechanically generated alternating between two pressure levels P_{HIGH} and P_{LOW} , and by the action of the inspiratory muscles during spontaneous ventilation. The spectrum of BIPAP is described and classified by Hörmann *et al* (Figure 2) as (a) IPPV (Intermittent Positive Pressure Ventilation) – BIPAP: There is no spontaneous respiration. Ventilation is pressure-controlled and time cycled (b) SIMV (Synchronised Intermittent Mandatory Ventilation) – BIPAP: Patient breathes spontaneously only on the P_{LOW} .

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Increased pressure at the upper level P_{HIGH} delivers a machine generated breath similar to the mandatory breath (c) ‘Genuine’ – BIPAP: Patient breathes spontaneously at both P_{HIGH} and P_{LOW} (d) CPAP: In this situation P_{HIGH} equals P_{LOW} . Patient takes over total ventilation.⁴ Thus we realise that BIPAP is a single mode of ventilation that can be used from initiation of mechanical ventilation till the end of weaning.

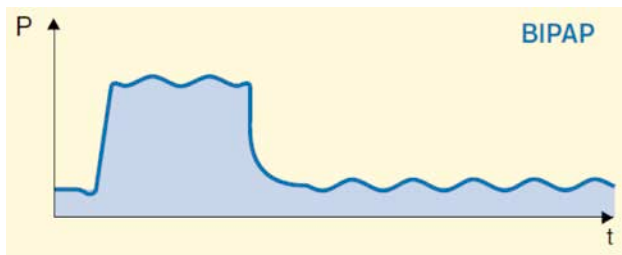


Figure 1: BIPAP mode allowing unrestricted spontaneous respiration during P_{HIGH} and P_{LOW}

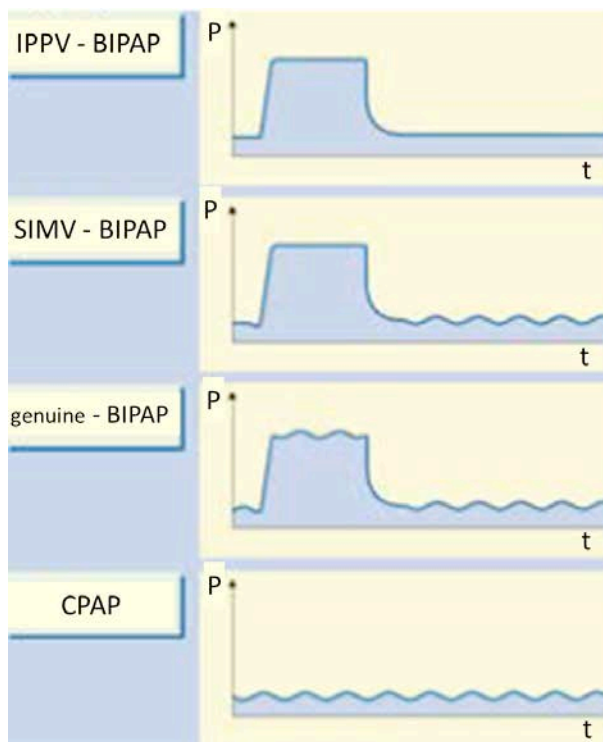


Figure 2: Spectrum of BIPAP

APRV is a variant of BIPAP (Figure 3). While there are no restrictions on the duration of lower pressure level P_{LOW} (i.e., T_{LOW}) in BIPAP, in APRV the duration of T_{LOW} is typically ≤ 1.5 s. The patient is at P_{HIGH} for the majority of the time during a respiratory cycle and only transiently (for duration of T_{LOW}) the pressure is ‘released’ to P_{LOW} . Hence the

nomenclature ‘Airway Pressure Release Ventilation’. It becomes evident that if the patient is not breathing spontaneously, APRV is inverse ratio ventilation. The feature common to BIPAP and APRV is that they allow unrestricted spontaneous breathing in any phase of mechanical ventilatory cycle. This is different from the conventional pressure-controlled ventilation in which the expiratory valve remains closed during a mandatory positive breath. Even if the patient attempts to forcefully exhale during inspiration (such as coughing), the ventilator abruptly stops inspiration to open the expiratory valve to allow expiration. In BIPAP and APRV, the expiratory valve is very sensitive and opens to even a slight increase in airway pressure. Similarly, the inspiratory valve is controlled precisely to provide exactly the right amount of flow to maintain a constant airway pressure.

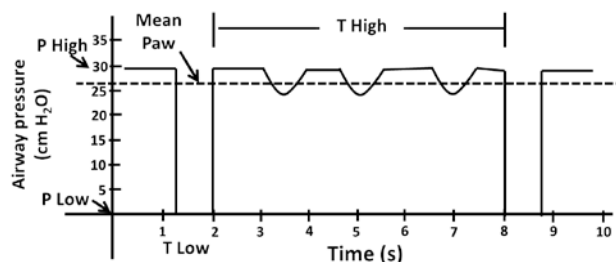


Figure 3: Airway Pressure Release Ventilation (APRV)

Initiation from P_{LOW} to P_{HIGH} and release from P_{HIGH} to P_{LOW} are synchronised with patient’s breaths in modern ventilators.

Setting the controls for BIPAP and APRV

There are essentially four settings to be set for BIPAP and APRV: P_{HIGH} , P_{LOW} , T_{HIGH} (duration of P_{HIGH}) and T_{LOW} (duration of P_{LOW} allowing CO_2 elimination). For BIPAP, a ventilation pressure P_{HIGH} of 12-15 cm H_2O above PEEP (positive end expiratory pressure) is recommended. The tidal volume should then be noted and P_{HIGH} adjusted if necessary. P_{LOW} is nothing but PEEP and set according to the PEEP requirements. T_{HIGH} and T_{LOW} are set to obtain a respiratory rate of 10-12 breaths/minute and an inspiratory:expiratory ratio (I:E ratio) of 1:2.

In accordance with lung protective strategy, P_{HIGH} should ideally be set below the higher inflection

point on the static volume pressure curve, while P_{LOW} should be above the low inflection point. To improve oxygenation with BIPAP parallel increase in P_{HIGH} and P_{LOW} and/or an increase in I:E ratio (increasing T_{HIGH} and decreasing T_{LOW}) are effective. The measures to increase ventilation (and thereby reduce $PaCO_2$) whilst maintaining the same mean airway pressure are (a) increase ventilator pressure gradient by increasing upper pressure and if necessary reducing the lower airway pressure (altering lower and upper pressure in opposite directions) and (b) shorten the T_{LOW} and T_{HIGH} times (which increases the rate of mechanical breaths). T_{HIGH} should be set such that lungs are completely inflated as indicated by an end-inspiratory phase of no flow when spontaneous breathing is absent, and T_{LOW} should be set such that expiration is complete and intrinsic or autoPEEP is absent as indicated by no gas flow at the end of expiration.

With APRV, P_{HIGH} is usually set at a level between 20 and 30 cm H_2O or at the level of plateau pressure while on volume controlled ventilation. P_{LOW} is set between 0 and 5 cm H_2O initially. T_{HIGH} is set at 4–6 s and T_{LOW} at 0.2–0.8s. T_{LOW} is set such that expiratory flow ends at 50–75% of peak expiratory flow. To correct poor oxygenation with APRV, increase either P_{HIGH} by 2 cm H_2O increments or T_{HIGH} by 0.2 s or both to increase mean airway pressure. To correct poor ventilation (1) increase P_{HIGH} (to increase the pressure gradient determining the flow and so, a larger tidal volume is achieved) and decrease T_{HIGH} (to increase the number of releases or breaths) thus increasing minute ventilation without altering mean airway pressure as it is the mean airway pressure that determines oxygenation (2) increase T_{LOW} by 0.05–0.1s increments (3) decrease sedation to allow spontaneous ventilation which will increase the overall minute ventilation.

Weaning with BIPAP and APRV

The inspiratory O_2 concentration is reduced gradually to less than 50%. In BIPAP mode, the I:E ratio is reduced until inspiratory time is shorter than the expiratory time. P_{LOW} is reduced gradually to 5–7 cm H_2O . P_{HIGH} is also gradually reduced so that the difference in pressure between P_{HIGH} and P_{LOW} is 5–8 cm H_2O . The respiratory rate of the mechanical

breaths is reduced gradually to 6–8 breaths/min by increasing T_{HIGH} and T_{LOW} . When P_{HIGH} becomes equal to P_{LOW} , automatically this becomes CPAP mode.

The ‘Drop and stretch method’ is the popular method of weaning with APRV. The level of P_{HIGH} is gradually reduced (‘drop’) and the number of releases is reduced by increasing the T_{HIGH} (‘stretch’) until the mode is converted to CPAP as a method of spontaneous breathing trial before extubation.

Combination modes with BIPAP and APRV

The combinations of BIPAP and APRV with pressure support ventilation (PSV) and automatic tube compensation (ATC) are available. Addition of PSV above P_{HIGH} can increase airway pressure and result in over distention of the lungs. Addition of PSV to APRV reduces the benefits of spontaneous breathing by altering the normal sinusoidal flow of spontaneous breath to a decelerating assisted mechanical breath. The use of ATC during BIPAP or APRV may help overcome the artificial airway resistance during spontaneous breathing.

Indications and contraindications

BIPAP being a universal mode of ventilation can be used for any indication for mechanical ventilation. APRV is predominantly used for ventilation in Acute Lung Injury (ALI), Acute Respiratory Distress Syndrome (ARDS) and atelectasis after major surgery. APRV is not a right choice in situations of cerebral oedema, increased intracranial pressure (where increase in $PaCO_2$ secondary to reduced exhalation time and diminished spontaneous ventilation can be detrimental), chronic obstructive pulmonary disease (COPD) and neuromuscular diseases.

Advantages

1. Increased patient motivation – It is less stressful for patients to be able to breathe spontaneously at any point of time. It also increases the patient motivation to breathe spontaneously.
2. Decreased sedation requirements – Since patient can breathe spontaneously at all phases of respiratory cycle patient’s inspiratory efforts need not be suppressed with sedatives.⁵

3. Benefits of unrestricted spontaneous breathing – Spontaneous breathing draws the inspired gas to the nondependent lung regions by using patient's own respiratory muscles and the corresponding pleural pressure changes. This happens without raising the airway pressure to dangerous levels. The gas distribution in the lungs is more physiological. This results in improved oxygenation.⁶⁻⁹
4. Beneficial haemodynamic effects and improved regional blood flow – These effects are seen predominantly secondary to the favourable intrathoracic pressure changes resulting from spontaneous ventilation.¹⁰⁻¹²
5. Constant lung recruitment by APRV – APRV avoids repetitive inflation and deflation of the lung and provides 'open-lung approach' for ventilation. By applying P_{HIGH} for a T_{HIGH} (80–95% of the cycle time), the mean airway pressure is increased. There is improved ventilation-perfusion matching, decreased intrapulmonary shunt and decreased dead space.^{7,9,13}
6. APRV may reduce the risk of ventilator associated pneumonia (VAP) in trauma patients with pulmonary contusion.¹⁴

Having described the advantages, it should be emphasised that no reported studies have shown improved mortality with the use of BIPAP or APRV. There are contradictory reports showing increased oxygen consumption due to spontaneous breathing efforts during APRV.¹⁵⁻¹⁶ Breaths that occur at the P_{LOW} or during the pressure-release transition from the P_{HIGH} to P_{LOW} may have higher work of breathing in APRV.¹⁷ There may be patient-ventilator asynchrony as airway pressure would be decreasing and gas flow would be escaping the circuit just as the patient was trying to inspire.¹⁸ This might be a reason in favour of synchronising mandatory breath cycling with a spontaneous expiration during T_{HIGH} .

Complications of BIPAP and APRV

Most complications with BIPAP are minor and are described when used as a noninvasive mode of ventilation. They are actually the complications resulting from the patient-ventilator interface (mask). Pneumothorax has been reported in two adolescents receiving BIPAP ventilation.¹⁹

Inadequate ventilation resulting in retention of carbon dioxide and generation of autoPEEP resulting in pneumothorax have been described with APRV.²⁰

Conclusion

BIPAP reduces complexity of mechanical ventilation and can be used as a sole mode from initiation of ventilation to weaning of the patients from mechanical ventilation. By covering the entire treatment spectrum right through the course of therapy, BIPAP offers great flexibility. The utility of APRV in conditions such as ALI and ARDS is becoming more evident. Despite increasing evidence of improving oxygenation, haemodynamics, patient comfort and safety, many are largely unfamiliar with it and has not gained popularity as a ventilator mode. However, the perception that it is only a rescue therapy for the 'difficult to oxygenate' patient is changing.

References

1. Henzler D. What on earth is APRV? *Critical Care* 2011; **15**:115.
2. Baum M, Benzer H, Putensen C, Koller W, Putz G. Biphasic Positive Airway Pressure (BIPAP) - a new form of augmented ventilation. *Anaesthesist* 1989; **38**: 452-8.
3. Stock MC, Downs JB, Frolicher DA. Airway pressure release ventilation. *Crit Care Med* 1987; **15**: 462-6.
4. Hörmann C, Baum M, Putensen C, Mutz NJ, Benzer H. Biphasic Positive Airway Pressure (BIPAP) - a new mode of ventilatory support. *Eur J Anaesthesiol* 1994; **11**: 37-42.
5. Rathgeber J, Schorn B, Falk V, Kazmaier S, Spiegel T, Burchardi H. The influence of controlled mandatory ventilation (CMV), intermittent mandatory ventilation (IMV) and biphasic intermittent positive airway pressure (BIPAP) on duration of intubation and consumption of analgesics and sedatives: A prospective analysis in 596 patients following adult cardiac surgery. *Eur J Anaesthesiol* 1997; **14**:576-82.
6. Putensen C, Zech C, Wrigge H, Zinserling J, Stüber F, Von Spiegel T, *et al.* Long term effects

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- of spontaneous breathing during ventilatory support in patients with acute lung injury. *Am J Respir Crit Care Med* 2001; **164**: 43–9.
7. Wrigge H, Zinserling J, Neumann P, Defosse J, Magnusson A, Putensen C, *et al.* Spontaneous breathing improves lung aeration in oleic acid induced lung injury. *Anesthesiology* 2003; **99**:376–84.
 8. Sydow M, Burchardi H, Ephraim E, Zielmann S, Crozier TA. Long term effects of two different ventilatory modes on oxygenation in acute lung injury. Comparison of airway pressure release ventilation and volume controlled inverse ratio ventilation. *Am J Respir Crit Care Med* 1994; **149**:1550–6.
 9. Putensen C, Mutz N, Putensen-Himmer G, Zinserling J. Spontaneous breathing during ventilatory support improves ventilation/perfusion distribution in patients with ARDS. *Am J Respir Crit Care Med* 1999; **159**:1241–8.
 10. Kaplan LJ, Bailey H, Formosa V. Airway pressure release ventilation increases cardiac performance in patients with acute lung injury/adult respiratory distress syndrome. *Crit Care* 2001; **5**:221–6.
 11. Hering R, Veihofer A, Zinserling J, Wrigge H, Kreyer S, Berg A, *et al.* Effects of spontaneous breathing during airway pressure release ventilation on intestinal blood flow in experimental lung injury. *Anesthesiology* 2003; **99**:1137–44.
 12. Hering R, Zinserling J, Wrigge H, Varelmann D, Berg A, Kreyer S, *et al.* Effects of spontaneous breathing during airway pressure release ventilation on respiratory work and muscle blood flow in experimental lung injury. *Chest* 2005; **128**:2991–8.
 13. Cane RD, Peruzzi WT, Shapiro BA. Airway pressure release ventilation in severe acute respiratory failure. *Chest* 1991; **100**:460–3.
 14. Walkey AJ, Nair S, Papadopoulos S, Agarwal S, Reardon CC. Use of airway pressure release ventilation is associated with a reduced incidence of ventilator-associated pneumonia in patients with pulmonary contusion. *J Trauma* 2011; **70**:E42–E47.
 15. Field S, Kelly SM, Macklem PT. The oxygen cost of breathing in patients with cardiorespiratory disease. *Am Rev Respir Dis* 1982; **126**:9–13.
 16. Uyar M, Demirag K, Olgun E, CanKayali I, Moral AR. Comparison of oxygen cost of breathing between pressure support ventilation and airway pressure release ventilation. *Anaesth Intensive Care* 2005; **33**: 218–22.
 17. Kallet RH. Patient-ventilator interaction during acute lung injury, and the role of spontaneous breathing: part 2: airway pressure release ventilation. *Respir Care* 2011; **56**:190–206.
 18. Myers TR, MacIntyre NR. Does airway pressure release ventilation offer important new advantages in mechanical ventilator support? *Respir Care* 2007; **52**:452–60.
 19. Pribble CG, Berg MD. Pneumothorax as a complication of bilevel positive airway pressure (BiPAP) ventilation. *Clinical Intensive Care* 2000; **11**:145–7.
 20. Maung AA, Luckianow G, Kaplan LJ. Lessons learned from airway pressure release ventilation. *J Trauma Acute Care Surg* 2012; **72**:624–8.