Airway Pressure Release Ventilation in Acute Respiratory Distress Syndrome

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KEYWORDS

• APRV • Airway pressure release ventilation • ARDS • ALI
• PEEP • Alveolar derecruitment

Patients with acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) exhibit multiple areas of lung collapse, often in the dependent regions. The ensuing decrease in gas-exchanging alveoli leads to pulmonary shunting and hypoxemia. Conventional mechanical ventilation strategies can exacerbate this pulmonary pathology through ventilator-induced lung injury (VILI) by both overdistention as well as mechanical shear during repeat openings of the normal, more compliant alveoli. The current accepted lung protective ventilation strategy uses low tidal volume ventilation with moderate positive-end expiratory pressure (PEEP) (ARDSnet).

However, airway pressure release ventilation (APRV) is also increasingly used as an alternative mode of ventilation for both salvage therapy in hypoxemic respiratory failure as well as an alternate means of lung protection in patients with ALI/ARDS. In this article, the authors review the basic principles of APRV, discuss the theoretical and published experimental benefits of APRV, as compared with conventional modes of ventilation, and review the available human clinical data.

WHAT IS APRV?

Stock and Downs first described APRV in 1987 as a modified form of continuous positive airway pressure (CPAP) to enhance oxygenation by augmenting alveolar recruitment.[1] Because fairly high CPAP levels are used, CO_2 clearance must be supported by intermittently releasing the airway pressure and allowing ventilation, hence, the appellation airway pressure release ventilation. The APRV respiratory cycle is, therefore, divided into 2 time periods: a longer T_{high} period at the higher airway

The authors have nothing to disclose.
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Crit Care Clin 27 (2011) 501–509
doi:10.1016/j.ccc.2011.05.003 **criticalcare.theclinics.com**
0749-0704/11/$ – see front matter

pressure (P_{high}) and a much shorter (typically 0.4–0.8 seconds) T_{low} period during which pressure is released to P_{low} to allow for CO_2 clearance (**Fig. 1**). Because most of the respiratory cycle (80%–95%) is spent at P_{high}, the generated mean airway pressure is higher than in conventional ventilation modes without a corresponding increase in peak airway pressure.[2] The short release times retain a residual volume of air, thus, creating intentional auto-PEEP. Both the higher mean airway pressure and the auto-PEEP prevent collapse of alveoli and progressively recruit additional lung units to participate in gas exchange by matching the regional time constant variations in injured segments of lung.

It is essential to note that patients may breathe at any point in the phase cycle because of the unique valve construction of ventilators capable of providing APRV. In this way, APRV is fundamentally different from traditional cyclic ventilation where patients are constrained to either inspiration or exhalation occurring only during predefined periods. A full discussion of managing ventilator settings in APRV is beyond the scope of this article, but several reviews are available.[2–4]

Mechanics and Hemodynamic Effects of APRV

Oxygenation in APRV occurs by a combination of several mechanisms: (1) alveolar recruitment, (2) intrinsic PEEP, and (3) increased pulmonary and systemic blood flow.[5–8] CO_2 clearance occurs as a result of the combined effects of P_{high}, T_{high}, and T_{low} on recruiting alveoli and maintaining the lung at volumes approaching total lung capacity (TLC). At P_{high}, the chest wall resistance, compliance and elastance in concert with elastic recoil of the lung are balanced against the applied airway pressure. When that airway pressure is released, forces tending to expel gas are unopposed, generating gas flow rates that routinely approach negative 80 L/min (see **Fig. 1**). Thus, ventilation may occur over a short period of time, allowing a short T_{low} to be employed.

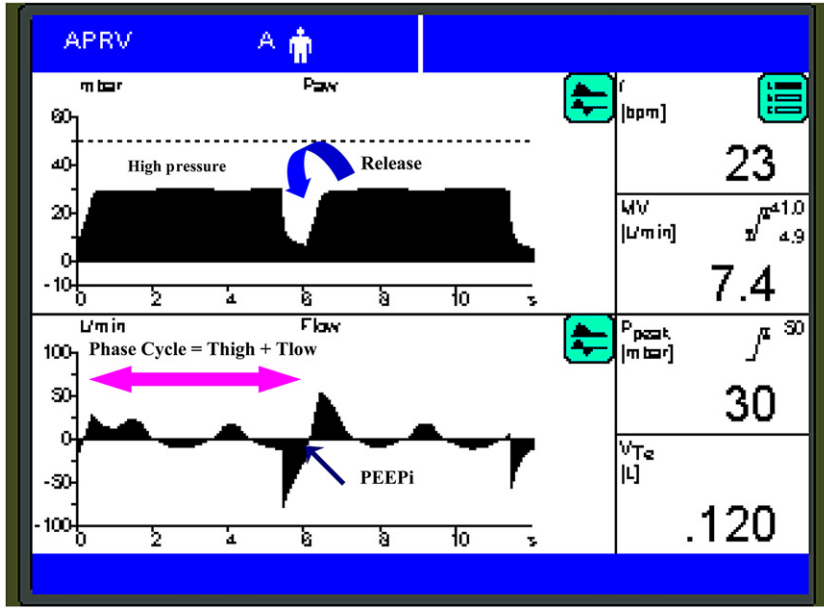

Fig. 1. Airway pressure release ventilation.