Use of Continuous Positive Airway Pressure in Critically Ill Patients

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Introduction

Positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) are probably the most frequently used ventilatory treatments in critically ill patients. In a recent international survey, PEEP was used in more than 90% of patients with acute respiratory distress syndrome (ARDS) and in more than 50% of patients with an exacerbation of chronic obstructive pulmonary disease (COPD) [1]. PEEP is a technique in which airway pressure is maintained above atmospheric pressure at end expiration by pressurization of the ventilatory circuit, whereas during CPAP, pressure is applied to spontaneous breathing throughout the entire respiratory cycle. Many pathological conditions benefit from the application of PEEP or CPAP, as shown by the pioneering work of Poulton and Oxon [2] and Barach and colleagues [3] who demonstrated that application of positive pressure to the airway can effectively treat patients with cardiogenic pulmonary edema. Nowadays, CPAP or PEEP are used in various forms of acute respiratory failure to improve respiratory mechanics, gas exchange, and cardiac performance.

Pathophysiology

In patients with acute exacerbation of COPD, hypercapnia and respiratory acidosis are consequences of hypoventilation, often requiring mechanical ventilation to be reversed. Alveolar hypoventilation develops in spite of an increased respiratory drive and respiratory muscle activity, which are ineffective in increasing tidal ventilation due to the impairment of respiratory mechanics. The result is rapid shallow breathing, with low volume ventilation not compensated by the increase in respiratory rate because of the concomitant increase in dead space ventilation. The mechanical derangement of the respiratory system in patients with acute exacerbations of COPD is mainly due to dynamic hyperinflation, which reduces the force-generating capacity of the diaphragm, and increased airway resistance. When the load becomes excessive, respiratory muscle fatigue can develop. In these patients, it was initially thought that PEEP produced little or no additional benefit. Subsequently, it was recognized that, particularly in the presence of airflow obstruction, the lungs may fail to deflate to functional residual capacity at end-expiration [4]. As a consequence, alveolar pressure remains positive to an extent that depends on the volume of trapped air, a phenomenon referred to as intrinsic PEEP (PEEP). In this context, application of external PEEP replaces the amount of pressure that must be gener-
ated by the inspiratory muscles to offset PEEP, needed to initiate inspiratory flow or trigger the ventilator [5]. This benefit is evident during spontaneous breathing or with any patient-triggered mode of ventilation: CPAP or PEEP has been shown to reduce dyspnea, work of breathing, respiratory drive, inspiratory muscle effort, and to improve triggering function and patient-ventilator interaction [5–9]. However, if PEEP exceeds PEEPi, it may further hyperinflated the lungs, increasing the risk of barotrauma, with additional mechanical disadvantage to the diaphragm, and hemodynamic impairment [9]. PEEP should be titrated according to a precise evaluation of the level of PEEPi, which is difficult to evaluate when respiratory muscle activity is present [10].

Hypoxemic acute respiratory failure is caused by intrapulmonary shunt and venous admixture, ensuing from an acute reduction of aerated lung volume due to lung edema and/or atelectasis. As a consequence, functional residual capacity is decreased and respiratory mechanics impaired (i.e., reduced lung compliance). The reversal of hypoxemia requires interventions that recruit more aerated lung units for ventilation. In this context, PEEP can improve arterial oxygenation by increasing functional residual capacity, by reducing venous admixture, improving respiratory mechanics, and reducing the work of breathing [11, 12].

In cardiogenic pulmonary edema hypoxemia is often associated with hypercapnia secondary to respiratory muscle fatigue. The reduction of lung compliance and the increase in airway resistance increase the work of breathing [13]. Thus, the inspiratory muscles have to generate large negative swings in pleural pressure leading to an increase in left ventricular transmural pressure and afterload [14]. The reduction in cardiac output compromises oxygen delivery to the respiratory muscles, and may create a vicious cycle. In this setting, the application of CPAP has several beneficial effects on the cardiovascular system. A CPAP-induced increase in intrathoracic pressure decreases left ventricular transmural pressure (i.e., afterload), and improves left ventricular performance [14]. This effect of CPAP on afterload is primarily related to the improvement in respiratory mechanics that leads to a reduction in the inspiratory negative swings of intrathoracic pressure [13].

### Clinical Applications of CPAP

#### Hypercapnic Acute Respiratory Failure

The application of CPAP in acute exacerbations of COPD reduces the threshold load imposed on the inspiratory muscles by PEEPi, although it does not reduce hyperinflation and its related negative effects on the diaphragm’s force-generating capacity. The addition of CPAP to spontaneous breathing is effective in reducing dyspnea, decreasing the work of breathing, and improving cardiac function. During triggered modes of ventilation, PEEP reduces the patient’s inspiratory effort and ventilatory drive, and facilitates triggering of the ventilator by reducing both inspiratory effort and the delay between onset of inspiratory effort and initiation of ventilator assistance delivery, improving patient ventilator interactions, and reducing ineffective inspiratory efforts [5–8, 15]. Despite a large body of physiologic studies, no randomized, clinical trial has evaluated the effectiveness of CPAP in improving outcomes in ventilated patients with COPD. Nevertheless, many trials have demonstrated the effectiveness of non-invasive ventilation with 4–5 cmH₂O PEEP and pressure support in patients with COPD exacerbation and acute hypercapnia [16, 17].