Is lung protective ventilation beneficial for patients without acute respiratory distress syndrome?

Positive pressure mechanical ventilation can lead to ventilator-associated lung injury as a result of alveolar strain and cyclic alveolar atelectasis. Risk factors for ventilator-associated lung injury in patients without acute respiratory distress syndrome include high tidal volume (>6 ml/kg predicted body weight), acidaemia, blood product transfusion and restrictive lung disease (Gajic et al, 2004).

There is a convincing body of improved outcome evidence supporting the use of lung protective ventilation strategies in the intensive care unit and operating theatre and the use of low tidal volume (6–8 ml/kg predicted body weight) in slowing or halting progression to acute respiratory distress syndrome. However, perioperative physicians and intensivists underuse low tidal volume at the onset of mechanical ventilation. There is little evidence-based guidance for the most appropriate way to ventilate patients with healthy lungs (Serpa Neto et al, 2012).

Evidence supporting use of lung protective ventilation in patients without acute respiratory distress syndrome

Gajic et al (2004) demonstrated that 24% of intensive care unit patients with normal lungs who received mechanical ventilation for 2 days or longer developed acute respiratory distress syndrome within the first 5 days of mechanical ventilation. A multivariate analysis revealed an increased risk of developing acute respiratory distress syndrome with the use of volumes greater than 6 ml/kg (odds ratio 1.3 for every 1 ml > 6 ml/kg predicted body weight, $P<0.001$) (Gajic et al, 2004).

A meta-analysis, which included 15 randomized controlled trials and five observational studies, showed that mechanical ventilation with low tidal volume (around 6 ml/kg) was associated with better clinical outcomes (Serpa Neto et al, 2012). A trial looking at conventional ventilation (tidal volume 10–12 ml/kg) *vs* lung protective ventilation (6–8 ml/kg) in intensive care patients without acute respiratory distress syndrome demonstrated that at 12 hours post initiation of ventilation, the levels of inflammatory markers in bronchoalveolar lavage were significantly higher in the conventional tidal volume group (Pinheiro de Oliveira et al, 2010).

Evidence opposing use of lung protective ventilation in patients without acute respiratory distress syndrome

Lung protective ventilation may cause atelectasis, hypoxaemia and hypercapnia and may require permissive hypercapnia ventilation strategies, which can lead to acidaemia, cardiovascular and neurological dysfunction (Hickling et al, 1990). On the other hand, a high respiratory rate strategy in an attempt to maintain adequate minute ventilation may lead to development of intrinsic positive end-expiratory pressure and dynamic hyperinflation; this in itself can have adverse cardiorespiratory effects (Wolthuis et al, 2009).

Animal work suggests that even lung protective ventilation in the absence of primary pulmonary insult could potentially induce ventilator-associated lung injury. Whether this has an impact on clinical outcomes is unknown. This model offers opportunities to study and understand the pathophysiology behind the contribution of mechanical ventilation to acute respiratory distress syndrome in healthy lungs (Wolthuis et al, 2009).

Conclusions

It would be a reasonable strategy to use lung protective ventilation (tidal volume 6–8 ml/kg) and maintain low plateau airway pressures in all patients whether they are at risk of acute respiratory distress syndrome or not. This ventilation strategy minimizes alveolar distension, which is the primary cause of ventilation-associated lung injury (grade 2B). Although lung protective ventilation can lead to atelectasis and hypoxaemia, the application of positive end-expiratory pressure and recruitment manoeuvres with positive inspiratory pressure can prevent these consequences.

Adequately powered and accurately reported randomized trials are needed to determine whether lung protective ventilation using low tidal volume should be individualized for a specific subgroup of surgical and intensive care mechanically ventilated patients. BJHM

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