Ventilator Management in Brain-Damaged Patients with ARDS

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Introduction

Acute lung injury/Acute respiratory distress syndrome (ALI/ARDS) is a permeability pulmonary edema characterized by refractory hypoxemia leading to multiple system organ failure.1 The mortality rate of ARDS still remains more than 40% and survivors suffer significant decrements in their quality of life.2 In a retrospective analysis, 27% of subarachnoid hemorrhage (SAH) developed ALI, and the development of ALI was an independent bad prognostic factor.3 In an international observational study, high tidal volume was associated with the development of ALI after severe brain injury.4

Lung Protective Mechanical Ventilation in Brain-Damaged Patients

Alveoli remain permanently open at functional residual capacity (FRC) in healthy peoples. Only if volume is reduced forcibly to near residual volume, peripheral airways prompt to close in normal lungs. However, alveoli close at volumes above FRC in acute respiratory distress syndrome (ARDS)/acute lung injury (ALI). The collapsed alveoli increase ventilation/perfusion heterogeneity resulting in intrapulmonary shunt and severely impaired oxygenation. Numerous evidences show that inappropriate mechanical ventilator settings can produce further lung damage to the ARDS lungs, which has been defined as ventilator induced lung injury (VILI). Inappropriate mechanical ventilator settings can cause proinflammatory cytokines production,5 and can facilitate translocation of bacteria from the bronchial tree into the systemic circulation.

The two important aspects of VILI seem to be end-inspiratory overdistension and a low end-expiratory lung volume allowing repeated collapse and re-expansion with each respiratory cycle (tidal recruitment). Tidal recruitment is thought to result in high shear force on alveolar walls and small airways during inflation, especially at the interfaces between collapsed and aerated alveoli. Therefore, low tidal volume (6 mL per Kg of predicted body weight) and limitation of plateau pressure under optimal PEEP (less than 30 cm H2O) is a key of lung protective ventilator strategy.6

Low tidal volume without adequate PEEP may not be valid to improve ARDS patients' outcome. After 5 days of mechanical ventilation on zero PEEP in brain-damaged patients static elastance and minimal resistance were significantly increased compared with 8 cm H2O of PEEP applied patients.7 However, it is not clear how to select optimal PEEP level to prevent the bad tidal recruitment. Moreover, a safe upper limit for plateau pressure has not been determined in ARDS patients.8 One way to circumvent VILI may be alveolar recruitment maneuvers (ARM) in early stage of ALI. However, the application of early ARM with low tidal volume has not been proved its efficacy on the mortality reduction.9,10

PEEP Setting & Other Adjunctive Measures in a Patient with Increased Intracranial Pressure

Application of PEEP up to 25 cm H2O did not impair intracranial pressure (ICP), brain tissue oxygenation, or regional cerebral blood flow in a animal model with SAH.11 However, stepwise elevation of PEEP resulted in a significant decrease of mean arterial pressure and cerebral blood flow in patients with severe SAH.11 However, PEEP increasing up to 12 mm Hg did not significantly influence ICP.12 The effect of PEEP on ICP may be different depending on the lung elastance change induced by applied PEEP. When PEEP induced alveolar hyperinflation leded significant increase in ICP.13 Whereas when PEEP induced alveolar recruitment, ICP did not change.13 In another study, PEEP did not affect cerebral hemodynamics in patients with low respiratory system com-
Raising the head-of-bed by 30 degrees in euvolemic patients with traumatic brain injury and subarachnoid haemorrhage ameliorated the PEEP effect on ICP and CPP.\textsuperscript{15} Inspiration to expiration ratio did not cause significant changes in ICP or cerebral perfusion within $1:1$ to $1:2$.\textsuperscript{16}

**Conclusion**

In spite of the remarkable advance in the understanding of ARDS pathogenesis, the only effective therapeutic measure to decrease the mortality up to now is low tidal volume mechanical ventilation\textsuperscript{8} up to now. Most ARDS patients die of multi-organ failure rather than irreversible respiratory failure, indicating that ALI is closely associated with other organs by neurological, biochemical, metabolic, and inflammatory reactions. Although the careful use of induced hypocapnia to temporally decrease increased ICP remains a therapeutic tool in current practices, hyperventilation to induce hypocapnia may not be recommendable in brain-damaged patients with ARDS. Hyperventilation can induce ALI, or aggravate already developed ALI. And judicious application of PEEP increment is feasible in ventilating neurosurgical patients with acute intracranial pathology and concomitant acute respiratory distress syndrome (ARDS). However, it is not well addressed the optimal balance between brain and lung protection in patients with more refractory raised ICP.

**REFERENCES**