CRITICAL CARE

COMMENTARY

High-frequency oscillatory ventilation with tracheal gas insufflation: the rescue strategy for brain-lung interaction

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See related research by Vrettou et al., http://ccforum.com/content/17/4/R136

Abstract

The occurrence of moderate to severe acute respiratory distress syndrome due to traumatic brain injury is not uncommon and is associated with an extremely high incidence of morbidity and mortality. Owing to the complex interaction between the lung and brain, protective ventilation for the lung with lower tidal volume and higher positive end-expiratory pressure with or without mild hypercapnia might be harmful for the brain, and maintaining normocapnia or mild hypocapnia by increasing tidal volume or respiratory rate (or both) with lower positive end-expiratory pressure levels for protecting the brain might lead to ventilator-induced lung injury. Balancing the end-point between lungs and brain becomes a challenging issue, and non-conventional modes of mechanical ventilation might play a role in the more difficult clinical cases. In this commentary, the authors discuss the rationale, based on the physiologic principle of targeting both vital organs, of applying high-frequency oscillation and tracheal gas insufflation in acute respiratory distress syndrome patients with traumatic brain injury.

Introduction

In this issue of *Critical Care*, Vrettou and colleagues [1] introduce the alternative rescue strategies of high-frequency oscillation (HFO) and tracheal gas insufflation (TGI) in acute respiratory distress syndrome (ARDS) patients with traumatic brain injury (TBI). A quarter of patients with TBI develop subsequent pulmonary dysfunction and are associated with high mortality. The main mechanisms of aggravating lung injury are stimulation of systemic inflammatory response and decreasing of lung tolerance to stress [2]. The evidence from animal models is that massive brain injury can increase the risk of ventilator-induced lung injury (VILI) [3-5].

The ventilator management in ARDS comprises low tidal volume (V_T) of 6 mL/kg ideal body weight, limited plateau pressure of the respiratory system at 30 cm H_2O, and maintenance of the partial pressure of arterial oxygen (PaO_2) at between 55 and 80 mm Hg. This strategy may cause permissive hypercapnia to negatively affect intracranial pressure (ICP) by cerebral vasodilation in patients with severe TBI. Additionally, the increased ICP in combination with low PaO_2 might promote cerebral tissue hypoxia. Therefore, a low V_T strategy may result in a progressive deterioration of neurological outcome.

The main end-points of mechanical ventilator management in severe TBI are maintaining the partial pressure of carbon dioxide ($PaCO_2$) at between 31 and 35 mm Hg and maintaining PaO_2 at more than 60 mm Hg or 100 mm Hg [6,7]. Trying to ventilate TBI patients with V_T to keep that range of $PaCO_2$ can aggravate VILI in patients either at risk of ARDS or with ARDS [8] (Figure 1). It was recently shown that higher V_T ventilation can be associated with higher morbidity and mortality in patients with non-previously injured lungs [9]. Consequently, most of the clinical trials about protective ventilation in ARDS exclude patients with TBI.

HFO is an alternative approach in ventilating patients with severe ARDS. Several studies have reported a clear benefit in terms of improvement of oxygenation, prevention of further lung injury from low V_T ventilation, and ability to recruit the lungs compared with conventional ventilation. However, two recent large randomized control trials have shown clear evidence that applying HFO

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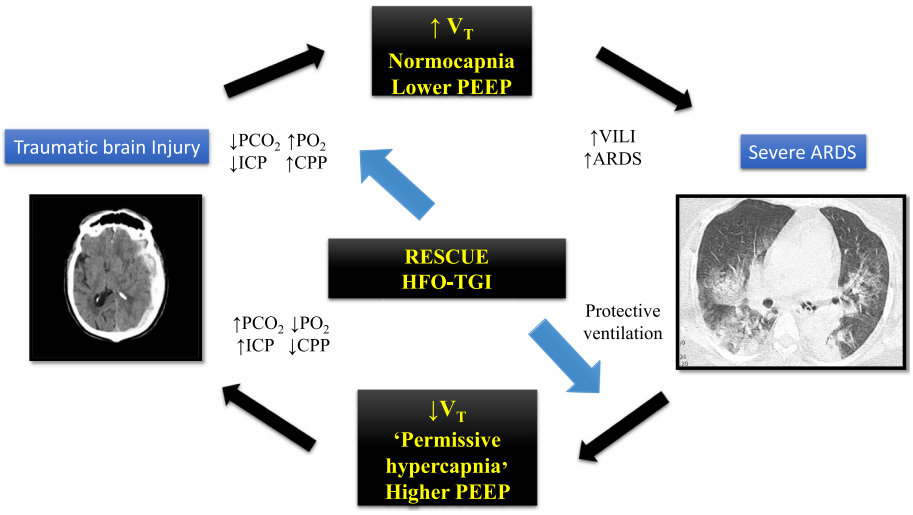

Figure 1 The effect of mechanical ventilation setting and acid-base disturbance on the brain and lung in acute respiratory distress syndrome (ARDS) and traumatic brain injury. CPP, cerebral perfusion pressure; HFO-TGI, combination of high-frequency oscillation with tracheal gas insufflation; ICP, intracranial pressure; PCO_2, partial pressure of carbon dioxide; PEEP, positive end-expiratory pressure; PO_2, partial pressure of arterial oxygen; VILI, ventilator-induced lung injury; V_T, tidal volume.

in moderate to severe ARDS is not superior to low V_T ventilation [10] and might even be harmful in terms of in-hospital mortality [11]. Nevertheless, the first trial did not specify whether the TBI subgroup was included or excluded, and the second one clearly excluded the TBI subgroup. So we still have a question: are there any places for HFO? Vrettou and colleagues [1] make an effort to answer this question in this issue of *Critical Care*. The authors demonstrate the beneficial effect of the combination of HFO with TGI (HFO-TGI) on oxygenation, hemodynamics, cerebral perfusion pressure (CPP), and ICP in severe ARDS patients with TBI. The authors aimed to achieve optimal oxygenation and lower-to-normal $PaCO_2$, avoiding possible adverse effects such as the increase of ICP and decrease of CPP.

Commentary

From previous study, HFO has been shown to improve oxygenation but also has been reported to increase CO_2 retention. Another limitation is that the increase of intrathoracic pressure in HFO might lead to a decrease of cardiac output and consequently a worsening of CPP. Mentzelopoulos and colleagues [12] have shown that, in patients with early severe ARDS, HFO-TGI (compared with HFO) can improve oxygenation and conventional mechanical ventilation without having adverse effects on $PaCO_2$ and hemodynamics. TGI can enhance CO_2 elimination by (a) improving the CO_2 washout from the proximal part of the anatomic dead space, (b) increasing lung volume and recruiting non-aerated lung regions by generating additional expiratory flow resistance, and (c) increasing the lung compliance, thus promoting a less injurious ventilation and better CO_2 removal [13]. In another study, the authors demonstrated that additional recruitment maneuver can improve oxygenation and lung compliance without disturbing hemodynamics in ARDS without TBI [14]. HFO-TGI, compared with conventional ventilation, improves lung compliance and PaO_2/fraction of inspired oxygen ratio while decreasing the plateau pressure of the respiratory system and the incidences of higher ICP and lower CPP. Furthermore, the improvements in ICP and CPP were demonstrated 4 hours after applying HGO-TGI. The main mechanisms of improvement of cerebral hemodynamics during HFO-TGI are (a) the lowering of mean tracheal pressure to below the actual mean airway pressure displayed by ventilator and (b) lung recruitment without over-distension by the previously mentioned mechanisms.

However, the application of TGI can lead to bronchial mucosal damage from the jet stream and retention of secretion. These complications are not reported in this study. However, the authors applied HFO-TGI for less than 12 hours. The recruitment maneuver during pre-HFO-TGI might lead to transient hypotension and is not life-threatening. An alternative could be the use of extracorporeal CO_2 removal, but this technique might pose a risk in patients with TBI [15].

Conclusions

HFO-TGI may be considered an alternative rescue ventilation strategy in patients with TBI and severe ARDS. This method might provide non-injurious ventilation for the lung and optimal gas-exchange to protect the brain. When these techniques are used, an intensive

monitoring of the respiratory function as well as brain physiology is mandatory.

Abbreviations

ARDS: acute respiratory distress syndrome; CO_2: carbon dioxide; CPP: cerebral perfusion pressure; HFO: high-frequency oscillation; HFO-TGI: combination of high-frequency oscillation with tracheal gas insufflation; ICP: intracranial pressure; $PaCO_2$: partial pressure of carbon dioxide; PaO_2: partial pressure of arterial oxygen; TBI: traumatic brain injury; TGI: tracheal gas insufflation; VILI: ventilator-induced lung injury; V_T: tidal volume.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

Both authors contributed to manuscript preparation and read and approved the final manuscript.

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Published: 27 Aug 2013

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10.1186/cc12862
Cite this article as: Pelosi and Sutherasan: High-frequency oscillatory ventilation with tracheal gas insufflation: the rescue strategy for brain-lung interaction. *Critical Care* 2013, 17:R179