Commentary

The role of high-frequency oscillatory ventilation in paediatric intensive care

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See related research by Slee-Wijffels *et al*. in this issue [http://ccforum.com/content/9/3/R274]

Abstract

Mechanical ventilation during acute respiratory failure in children is associated with development of ventilator-induced lung injury. Experimental models of mechanical ventilation that limit phasic changes in lung volumes and prevent alveolar overdistension appear to be less damaging to the lung. High-frequency oscillatory ventilation, using very small tidal volumes and relatively high end-expiratory lung volumes, provides a safe and effective means of delivering mechanical ventilatory support with the prospect of reducing the development of ventilator-induced lung injury. Despite theoretical advantages and convincing laboratory data, however, the use of high-frequency oscillatory ventilation in the paediatric population has not yet been associated with significant improvements in clinically significant outcome measures.

Introduction

In this issue of *Critical Care*, Slee-Wijffels and colleagues [1] report on the use of high-frequency oscillatory ventilation (HFOV) as a rescue therapy for children with severe respiratory failure in the paediatric intensive care unit. They describe 51 children with severe respiratory failure, initially managed with conventional mechanical ventilation, who required HFOV as rescue therapy. In this retrospective study, the authors looked for differences between patients with a diagnosis of diffuse alveolar disease (DAD; 63% of the sample) and those with a diagnosis of small airway disease (SAD; 33% of the sample). Oxygenation index (OI) was significantly higher before and during HFOV in DAD patients than in SAD patients, whereas the arterial carbon dioxide tension before commencing HFOV was higher in SAD patients than in DAD patients. The overall survival rate was 64%, with 56% survival among DAD patients versus 88% in patients with SAD.

Slee-Wijffels and colleagues postulate that earlier instigation of HFOV may improve the outcome of children with respiratory failure secondary to DAD, and suggest that HFOV should be considered a rescue therapy for respiratory failure in SAD – a clinical condition in which HFOV is often avoided because of the perceived risk for worsening pulmonary overdistension.

Discussion

Acute respiratory distress syndrome (ARDS) is the most severe form of acute lung injury and is often quoted as having a mortality rate of around 30%. ARDS can be defined according to the American–European Consensus Conference Committee criteria: acute onset; presence of bilateral infiltrates on chest radiography; arterial oxygen tension/inspired fractional oxygen index <200; and absence of clinical evidence for left-sided heart failure. Treatment is largely supportive, with mechanical ventilation, and is associated with the development of so-called ventilator-induced lung injury (VILI).

There are a number of mechanisms that can lead to development of VILI. These include production of gross air leaks; diffuse alveolar injury due to overdistension; injury due to repeated cycles of recruitment/derecruitment, in which alveolar units open during inspiration and collapse again during expiration, resulting in the generation of high shear stress; and damage due to the release of inflammatory mediators in the lung. These processes are often referred to as 'barotrauma', 'volutrauma', 'atelectrauma' and 'biotrauma', respectively.

The lungs of patients with ARDS are almost inevitably heterogeneously damaged, and mechanical ventilation with normal or even low tidal volumes can lead to regional lung injury through the mechanisms described above. There is a

ARDS = acute respiratory distress syndrome; DAD = diffuse alveolar disease; HFOV = high-frequency oscillatory ventilation; OI = oxygenation index; SAD = small airway disease; VILI = ventilator-induced lung injury.

considerable amount of laboratory data suggesting that repetitive cycles of pulmonary recruitment and derecruitment are associated with demonstrable markers of lung injury. Similarly, experimental models of mechanical ventilation that limit phasic changes in lung volumes, prevent alveolar overdistension and reverse atelectasis appear histologically to be less damaging to the lung [2]. Recognition of the issues surrounding VILI has led to the development of various lung-protective ventilatory strategies, with the aim of reducing the magnitude of damaging cyclic alveolar fluctuations through the application of higher positive end-expiratory pressure levels and by reducing tidal volumes. These goals may be achieved using conventional ventilation, and in 2000 the ARDSNetwork investigators [3] reported a 9% decrease in absolute mortality in adult patients with ARDS using a lung-protective strategy involving the use of small tidal volumes (6 ml/kg predicted body weight) together with an average positive end-expiratory pressure of 10 cmH_2O. A similar strategy may be pursued using HFOV, where adequate gas exchange can be achieved while using extremely small tidal volumes in the range 1–3 ml/kg (often less than the anatomical dead space) and where it is possible to maintain relatively high end-expiratory lung volumes without inducing overdistension.

Many of the available data regarding the use of HFOV in the paediatric population are derived from case series in which the therapy was offered to children with severe respiratory failure secondary to diffuse alveolar disease and air leaks. These case series suggest that HFOV can be safely used as rescue therapy in this clinical setting, and that its use is associated with improvements in oxygenation and carbon dioxide clearance without worsening air leaks [4].

The first and largest randomized clinical trial examining the effects of HFOV in children was the cross-over study reported by Arnold and colleagues [5], in which 70 patients with DAD and/or air leaks were randomly assigned to receive either conventional ventilation, using a strategy that limited the peak inspiratory pressure, or HFOV. The study found no difference in terms of survival or in the duration of mechanical ventilation, but it did demonstrate that significantly fewer patients receiving HFOV remained dependent on supplementary oxygen therapy at 30 days. In that study the OI was shown to discriminate between survivors and nonsurvivors in the first 24 hours of therapy, and an OI of 42 or greater at 24 hours predicted mortality with an odds ratio of 20.8, sensitivity of 62% and specificity of 93%. This finding is consistent with that of Slee-Wijffels and colleagues [1], who noted a higher range of OI values in the DAD group, in which the mortality was higher than in the SAD group. Another significant finding of the study conducted by Arnold and coworkers was that, in a *post hoc* analysis, the benefits were not as great in the group that crossed over to the HFOV treatment arm, supporting suggestions, including those by Slee-Wijffels and colleagues [1], that HFOV may be more effective when used earlier in the disease process.

Conclusion

Despite considerable laboratory data supporting the use of HFOV in the treatment of severe respiratory failure in children, studies in the paediatric population have not been able to demonstrate any significant improvements in clinically significant outcome measures [6]. This may be due to the wide range of aetiologies of acute respiratory failure in the paediatric intensive care unit, together with a gradual trend toward the use of more protective conventional ventilation strategies that emphasize lung recruitment and minimize tidal volumes. The report by Slee-Wijffels and colleagues [1] serves to remind us of the continuing high mortality in children with severe respiratory failure secondary to DAD, and emphasizes the need for large-scale, prospective, randomized controlled trials to clarify fully the role of HFOV in its management.

Competing interests

The author(s) declare that they have no competing interests.

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