Eur Respir J 1999; 14: 733–734
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European Respiratory Journal
ISSN 0903-1936

High frequency oscillation *versus* conventional ventilation: is one superior?

R.M. Kacmarek

High frequency oscillation (HFO) in many centres has become the standard of care for the ventilatory management of the sickest of neonates and infants. Indeed, numerous animal studies identify both the oxygenation and ventilation benefits of HFO [1–5]. In addition, a number of randomized, prospective trials [6–9] comparing HFO and conventional ventilation as well as numerous case series [10–12] preformed in neonatal and paediatric patients have shown gas exchange advantages with HFO.

The article in this issue by GOMMERS *et al.* [13] of B. Lachmann's group presents a serious challenge to the superiority of HFO in ventilating the surfactant deficient lung. This group demonstrated in a rabbit surfactant deficient lung model that the use of HFO with a high lung volume strategy plus surfactant resulted in no better gas exchange than conventional mechanical ventilation (CMV) plus surfactant. They also observed, consistent with the data of others [3–5] that without surfactant HFO with a high-lung volume strategy could maintain oxygen tension in arterial blood (P_{a,O_2})/inspiratory oxygen fraction (F_{I,O_2}) ratios ≥46.55 kPa (≥350 mmHg). Whereas, CMV without surfactant failed to maintain ventilation and oxygenation and resulted in the death of all animals in this group prior to the end of the study [13].

The question that is stimulated by the data of GOMMERS *et al.* [13] must be: is HFO as a mode of ventilation superior to CMV or it is the approach used to provide HFO *versus* the approach most commonly used to provide CMV that is responsible for the improved oxygenation with HFO? It could be speculated that the benefit in oxygenation observed in many HFO studies [1–12] is not a result of the use of HFO per se but a result of the use of lung recruitment maneuvres along with a lung protective approach to ventilation. This may be especially true for the application of a sustained high inflation pressure to open the lung and then the provision of ventilation that avoids overdistention and repetitive opening and closing of unstable lung units with each breath.

As pointed out by GOMMERS *et al.* [13] their data differs from that of Froese et al. [14] regarding the use of CMV and HFO following surfactant administration. The contrasting outcomes of these two studies are most probably a result of the differences in tidal volume (V_T) during CMV following surfactant administration. GOMMERS *et al.* [13] used a V_T of 10 mL·kg body weight^{-1}, whereas, FROESE *et al.* [14] used a V_T of 20 mL·kg body weight^{-1}. It is well established that large V_T's during CMV result in ventilator induced lung injury [15]. It can be argued that the negative results with CMV in the study of FROESE *et al.* [14] study as well as the results of other trials [3, 5] may be a result of lung injury induced by the approach used during CMV.

What is more intriguing is to speculate about the results from trials comparing CMV and HFO without surfactant [1, 2, 4, 13]. The data in these groups from the study of GOMMERS *et al.* [13] are similar to those published by others [1, 2, 4]. Animals ventilated with HFO following induction of lung injury do better than animals ventilated with CMV. In order to identify the reasons why this is so, the approaches used to ventilate these animals in both the HFO and CMV groups must be carefully reviewed. In the HFO without surfactant group GOMMERS *et al.* [13] used an initial mean airway pressure (MAP) of 20 cmH_2O (lung recruitment) slowly decreasing the MAP over the next 4 h, while in the CMV group MAP was about 12 cmH_2O but more importantly (PEEP) was set at only 6 cmH_2O without the use of a recruitment maneuvre. In the HFO group, MAP was lowered over time to 12 cmH_2O without lose of oxygenation (46.55 kPa ≥350 mmHg) $P_{a,O_2}/F_{I,O_2}$). Based on this data in the HFO group, a PEEP of ~12 cmH_2O would be needed to prevent derecruitment in the CMV group. Would the results in the CMV group without surfactant have been different if a lung recruitment maneuvre was preformed, if PEEP was set at a level that prevented derecruitment during exhalation and if V_T was set to prevent overdistension? The current author recently presented preliminary data in a large sheep (30 kg) surfactant deficiency model utilizing such an approach and were able using lung recruitment maneuvres and appropriately set PEEP to maintain $P_{a,O_2}/F_{I,O_2}$ ratios >53.2 kPa (>400 mmHg) [16]. AMATO *et al.* [17] demonstrated dramatic improvements in survival using this approach to CMV in a randomized comparison to high V_T settings (12 mL·kg body weight^{-1}) and low PEEP (8–10 cmH_2O) without lung recruitment. In addition, RANIERI *et al.* [18] recently demonstrated a reduction in both pulmonary and systemic inflammatory mediators in patients with adult respiratory distress syndrome (ARDS) using this strategy, whereas patients ventilated with set PEEP based on oxygenation and large V_T's (12 mL·kg body weight^{-1}) demonstrated increases in both pulmonary and systemic inflammatory mediator levels.

Correspondence: R.M. Kacmarek, Respiratory Care, Ellison 401, Massachusetts General Hospital, 55 Fruit Street, Boston, MA 02114, Fax: 617 7244495.

In small animals as well as in neonates and infants a "high lung volume" strategy during CMV has been avoided based on the laboratory finding primarily of McCulloch *et al*. [3] and Kolton *et al*. [1]. However, in these studies, low PEEP was applied (≤8 cmH_2O) and neither V_T nor peak alveolar pressure defined. It may be that in neonates and infants a "high lung volume" ventilation strategy with CMV is simply not possible. However, this has not been adequately tested. In adults lung recruitment maneuvres with a lung protective ventilatory strategy during CMV does work.

High frequency oscillation is currently being trialed in adults [19]. One can only hope that future trial designs include lung recruitment maneuvres and lung protective ventilation strategies both in the conventional mechanical ventilation and high frequency oscillation arms, to ensure that the actual benefit or lack of benefit of high frequency oscillation can be identified. Similarly, in infants and neonates, it would be interesting to see if the results of future clinical trials would be different if conventional mechanical ventilation were applied with the same approach as high frequency ventilation. In none of the currently published randomized clinical trials was a lung recruitment and lung protective strategy used in both the conventional mechanical ventilation and high frequency ventilation arms [6–9, 20–24].

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