

High frequency oscillatory ventilation for adult ARDS: Is this the end of the road?

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Sir,

What is next for high frequency oscillatory ventilation (HFOV)? This very obvious question arises after negative results coming from two trials, namely the OSCAR trial (High-frequency oscillation for acute respiratory distress syndrome, 2013) [1] and the OSCILLATE trial (High-frequency oscillation in early acute respiratory distress syndrome, 2013) [2]. The results of the primary outcome of the OSCAR trial, namely all-cause mortality at 30 days, showed no significant difference; while the OSCILLATE trial rate of in-hospital mortality showed significantly higher mortality in the HFOV group in comparison with the control group [1, 2]. These results are disappointing for clinicians who have HFOV in their armamentarium for the management of moderate to severe acute respiratory distress syndrome (ARDS) in adults. However, as both trials possess the strength of being well-conducted prospective, randomized, controlled, multicenter, multinational studies, their results could be translated to general application in similar populations.

Despite the theoretical advantage of less volutrauma and atelectrauma by the use of HFOV, there is growing evidence that ventilator-induced lung injury (VILI) still occurs due to continuous overdistension with high pressure, as well as oscillation amplitude and frequency related biotrauma [3]. Is the VILI the only responsible factor for the failure of HFOV? Moreover, is this the end of the road for HFOV's journey in being used in the management of adult ARDS?

Looking back at the physiological studies regarding HFOV in adult ARDS, Papazian *et al.* found that 12 hours of ventilation with HFOV in the supine position did not improve the PaO₂/FiO₂ ratio, while using HFOV in the prone position improved the PaO₂/FiO₂ ratio significantly [4]. On the other hand, they also demonstrated that although there was a decrease in the level of interleukin-8, a marker of inflammation in broncho-alveolar lavage fluid, after prone positioning during conventional ventilation (CV), its level significantly increased when HFOV was used. Another physiological study, by the same group of investigators, re-confirmed that HFOV in the supine position did not improve the PaO₂/FiO₂ ratio after 12 hours [5]. The important finding of this study was that using HFOV in the supine position after 12 hours of

prone positioning with CV maintained the improvement in oxygenation related to prone positioning. However, using CV in supine position after 12 hours of prone positioning resulted in the decrease of PaO₂/FiO₂ ratio to the base line, i.e. the same as before prone positioning within the next 12 hours.

These two physiological studies demonstrate that HFOV may be not a good option for initial recruitment of collapsed alveoli (which are otherwise recruitable, as with prone positioning) despite there being a recruitment manoeuvre with a mean airway pressure of 45 cm H₂O for 40 seconds, performed at the beginning of HFOV as per their protocol [4, 5]. Indeed, it was prone positioning which was able to recruit collapsed alveoli, a benefit which could be continued with HFOV, even in the supine position.

Against the background of these physiological studies, we need more planned clinical studies in which we should achieve the recruitability benefit of prone positioning before HFOV use (meaning a shorter duration of hypoxemia and fewer chances of the occurrence of the proinflammatory effects of HFOV). Subsequently, one could assess the need for fluid resuscitation at the beginning of HFOV in order to mitigate the reduction of preload induced by a high mean airway pressure used during HFOV (i.e. less hemodynamic compromise and probably fewer chances of end-organ failure).

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