High-frequency oscillation in adolescents

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Background. High-frequency oscillation (HFO) is a widely used lung-protective ventilatory strategy in paediatric and neonatal acute lung injury. Its safe and effective use has been hindered by inadequate recruitment of the lung during oscillation and, until recently, the lack of an adequately powered oscillator for use in adult practice.

Methods. We present data from three adolescents with severe acute respiratory distress syndrome (ARDS) who received HFO with the Sensormedics 3100B oscillator after failure of conventional mechanical ventilation. A manual recruitment manoeuvre was used in all patients prior to mechanical ventilation (conventional or HFO) and following tracheal suctioning or disconnection from the ventilator. Changes in oxygenation index were used to assess therapy.

Results. All patients showed at least a 25% reduction in oxygenation index within 2 h of HFO, with return to conventional ventilation after 27–65 h.

Conclusions. We found HFO, in conjunction with manual recruitment and prone positioning, to be a well-tolerated mode of ventilation in adolescents with ARDS and who were unresponsive to conventional ventilation. Given this success we hope to renew interest in this method for adults with ARDS, together with concurrent use of manual recruitment.

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The mortality of acute respiratory distress syndrome (ARDS) over the last three decades has remained greater than 40%.1 However, lung-protective ventilatory strategies using low tidal volumes have been shown recently to reduce mortality, possibly by reducing ventilator induced lung injury.1

High-frequency oscillation (HFO) is an effective low tidal volume ventilatory strategy widely used in paediatric and neonatal acute lung injury.2–3 Until recently, its use in adult practice has been limited by the lack of an adequately powered oscillator. There are now some studies demonstrating its safe and effective application in adult acute lung injury.4–6 Successful use of HFO has also been hindered by failure to adequately recruit the lung during oscillation.7–8 Current practice is to use HFO to achieve, then maintain recruitment.4–5 Our practice is to use a high-pressure, rapid manual recruitment manoeuvre initially, and subsequently to use HFO with lower recruitment pressures. The use of lower pressures on HFO may be associated with less haemodynamic compromise and barotrauma. Manual recruitment manoeuvres in humans have only been described previously in conjunction with conventional ventilation.9

In this report we demonstrate the successful management of ARDS in three adolescents using a combination of rapid manual recruitment manoeuvres and HFO with the Sensormedics 3100B oscillator (Yorba Linda, CA). Given the size of our patients, this method could be used generally for adults.

Methods and results

We present data from three adolescents with severe ARDS (oxygenation index greater than 30 and $P_{aO_2}/F_{IO_2}$ ratio less than 100) admitted to our Paediatric Intensive Care Unit (Table 1). All patients received HFO because conventional mechanical ventilation was unsuccessful.
Ventilatory strategy on conventional ventilation

Patients had been ventilated using the Servo 300A ventilator (Siemens-Elema, AB) in pressure-control mode without inverse I:E ratios, aiming for low tidal volumes (6 ml kg⁻¹) and tolerating a minimum pH of 7.20. Increases in PEEP and $F_{IO2}$ were used to improve oxygenation. All patients were nursed prone, interspersed with daily 4-h breaks in the supine position. Sedation was with i.v. morphine, oral clonidine, and i.v. lorazepam.

Changes in oxygenation index (mean airway pressure (mm Hg) $\times F_{IO2} \times 100/\{P_{aO2} (kPa) \times 7.5\}$), were used to assess response to therapy. Failure of conventional ventilation was defined as $P_{aO2}$ less than 8 kPa with an $F_{IO2}$ greater than 0.7, peak inspiratory pressure greater than 30 cm H₂O and PEEP greater than 10 cm H₂O for at least 1 h despite placing prone, neuromuscular block and manual recruitment (see below).

Manual recruitment manoeuvre

A manual recruitment manoeuvre was done before mechanical ventilation (conventional or HFO) and following tracheal suctioning or disconnection from the ventilator. This involved sustaining manual inflation of the lungs with an $F_{IO2}$ of 1.0 using a Mapleson C circuit (Intersurgical, Berkshire), with the inflating pressure measured with a manometer connected by a sidearm between the tracheal tube and reservoir bag. The inflating pressure was increased to a maximum of 50 cm H₂O until oxygen saturations increased to a stable value; this recruiting pressure was then maintained for 30 s after which the tracheal tube was clamped to prevent derecruitment whilst connecting the patient to the ventilator. The process took 1–2 min. The initial mean airway pressure on HFO was set 5 cm H₂O below the recruiting pressure (Fig. 1A). Patients were treated with a 10 ml kg⁻¹ bolus of i.v. crystalloid if hypotension prevented a full recruitment manoeuvre; thereafter, the manoeuvre was re-attempted.

Ventilatory strategy on HFO

Initial HFO settings were as follows: bias flow 40 litre min⁻¹, $F_{IO2}$ 1.0, 33% inspiratory phase, and mean airway pressure set as above. Frequencies between 6 and 8 Hz were used and the amplitude was varied between 50 and 90 cm H₂O to maintain the $P_{aCO2}$ between 4 and 9 kPa. Sodium bicarbonate was not used to correct pH. Oxygen saturations between 88 and 92% were maintained by initially reducing $F_{IO2}$ to less than 0.6 and then reducing the mean airway pressure in 2 cm H₂O decrements as the $P_{aO2}$ and oxygenation index improved. Patients continued to be nursed prone as during conventional ventilation. Nitric oxide was not used. Sedation and paralysis were as above, with all three patients requiring paralysis for the first 24 h on HFO.

Conventional ventilation was started again when the $F_{IO2}$ was below 0.5 and mean airway pressure approximately 18–20 cm H₂O.

## Results

Figure 1 shows mean airway pressure and oxygenation index for the first 40 h of HFO. In all patients there was at least a 25% reduction in oxygenation index within 2 h of HFO. Overall the oxygenation index declined progressively from the initial median value of 32 on starting HFO to a median value of 8 after 30 h of HFO, with return to
conventional ventilation after 27–65 h. None of the patients had cardiovascular changes during manual lung recruitment or HFO despite requiring inotropic support for the underlying condition (Table 1). No patient developed air leak and all patients survived to discharge from hospital.

**Discussion**

HFO is an established treatment for both neonatal and paediatric acute lung injury which allows effective oxygenation and ventilation whilst minimizing ventilator-induced lung injury. Large tidal volume ventilation causes cyclic opening and closing of atelectatic alveoli which may cause lung damage and cause the release of local and systemic inflammatory mediators. HFO differs from conventional ventilation by delivering an almost constant distending pressure with active inspiratory and expiratory phases and, maximizing carbon dioxide removal primarily through facilitated diffusion, allowing the use of lower tidal volumes. This causes less stretch and shear injury and alveolar over distension. Following initial discouraging results from early HFO trials in neonates over a decade ago, it is now recognized that the success of HFO depends on both the operator and the oscillatory recruitment strategy.

With the Sensormedics 3100B oscillator, HFO can be applied to adults. This model has several modifications from its predecessor used in paediatrics (Sensormedics 3100A); it can achieve greater bias flow of up to 100 litre min⁻¹, has a greater amplitude, and possesses a more powerful electromagnet allowing faster acceleration to the required amplitude.

Three groups have recently the successful use of HFO in adults with ARDS.

The patients in Fort and colleagues' and Mehta and colleagues' studies showed significant improvement in oxygenation indices only after 12 and 48 h of HFO, respectively. Our patients showed a sustained improvement in oxygenation index following initiation of HFO with a 25% decrease within the first 2 h. Our ventilatory strategy differed from the other reports in three respects.

First, we used a manual recruitment manoeuvre to rapidly (1–2 min) recruit the lungs at initiation of HFO, setting the initial mean airway pressure at 5 cm H₂O less than the recruiting pressure. This was then reduced progressively maintaining oxygen saturations between 88 and 92% at an FIO₂ less than 0.6. In Fort's patients, the initial mean airway pressure was set at 2–3 cm H₂O above that of conventional ventilation and increased over a 3-h period, whilst on HFO, aiming for similar oxygen saturations. In Mehta’s study, the initial mean airway pressure was set at 5 cm H₂O above that of conventional ventilation and increased over a 3-h period, whilst on HFO, aiming for similar oxygen saturations. The recruiting pressures we used were greater than the initial mean airway pressures used during HFO in these two studies. We suggest this allowed us to rapidly and safely ‘open’ the lung, and then apply lower mean airway pressures that could be rapidly reduced resulting in the dramatic decrease in the oxygenation index described (Fig. 1).

Secondly, we repeated recruitment after circuit disconnection, tracheal suctioning, or arterial desaturation to restore airway opening.

Thirdly, we used greater oscillatory frequencies than that reported by both Fort and colleagues and Mehta and colleagues (6–8 vs 3–5 Hz) without experiencing severe hypercarbia (all Pco₂ values were below 9 kPa throughout HFO). The rationale for this was that lower frequencies during HFO result in higher alveolar tidal volumes, which may potentiate lung injury.

Our criteria for failed conventional ventilation included peak pressures that were less than that described by Fort and co-workers (32 and 40 vs ≥65 cm H₂O), with a similar
range of oxygenation indices. This would result in earlier institution of HFO in some patients, using HFO to protect the lung rather than as salvage therapy. Indeed Patient 1 in our report who had the longest period of conventional ventilation pre-HFO (50 h) was treated for twice as long as the other two patients, whilst a significant discriminating feature between the survivors and non-survivors in both Fort and colleagues' and Mehta and colleagues' studies was the duration of conventional ventilation before HFO. Furthermore, we used the prone position for all our patients.

In conclusion, we found HFO in conjunction with manual recruitment to be a well-tolerated mode of ventilation in adolescents with ARDS who did not respond to conventional ventilation. Ventilator strategy may be important in achieving success with HFO. Given the success of HFO we hope to renew interest in this method for adults with ARDS with concurrent use of manual recruitment.

References
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