

High Frequency Jet Ventilation In Neonatal Respiratory Disease Unresponsive To High Frequency Oscillatory Ventilation

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BACKGROUND: Whilst high frequency oscillatory ventilation (HFOV) is a very effective therapy for neonatal respiratory disease, not all infants respond to its charms. Despite careful manipulation of mean airway pressure (PAW) and oscillatory frequency, some babies fail to improve on HFOV, and may go on to receive extracorporeal membrane oxygenation (ECMO), or to die if ineligible for ECMO. Whilst high frequency jet ventilation (HFJV) is known to be effective in rescuing infants failing on conventional mechanical ventilation (CMV), its role in the management of lung disease refractory to HFOV is less clear. A single report limited to 10 babies with chronic lung disease and nosocomial pneumonia has suggested that HFJV can be effective in rescuing infants who are oxygenating poorly on HFOV (Friedlich et al; J Matern Fetal Neonatal Med 2003; 13:398). We have had the opportunity to examine this question in a larger, more diverse group of neonates failing HFOV with refractory hypoxaemia and/or persisting respiratory acidosis.

AIM: To document the physiological responses and short and long term outcome when HFJV was applied to infants failing on HFOV.

METHODS: The Royal Children's Hospital, Melbourne, is a quaternary referral centre in which HFOV is delivered solely using the SensorMedics 3100A oscillator (SensorMedics Inc., Yorba Linda, CA), and HFJV using the Life Pulse Jet ventilator (Bunnell Inc., Salt Lake City, UT). In the study period (January 1994 - December 2002), we identified all infants who were judged by attending clinicians to be deteriorating on HFOV, and as a result were transferred to HFJV. This decision was based on the presence of one or more of: a) profound hypoxaemia ($\text{PaO}_2 < 50$ mmHg; $\text{FiO}_2 1.0$), b) respiratory acidosis ($\text{PaCO}_2 > 65$ mmHg with $\text{pH} < 7.3$), c) hemodynamic instability despite maximal inotropic support d) uncontrolled air leak, and e) progressive gas trapping. Management on HFJV was tailored to the underlying pathophysiology, with positive end-expiratory pressure set to maintain appropriate lung volume, and judicious use of CMV breaths. Ventilator settings, alveolar-arterial oxygen difference (AaDO_2) and PaCO_2 were recorded whilst on HFOV, and then at regular intervals after switching to HFJV. Gas exchange trends, ventilatory outcomes and survival were examined in the group as a whole, and also within the major diagnostic groups encountered, namely meconium aspiration syndrome (MAS), congenital diaphragmatic hernia (CDH) and chronic lung disease/pulmonary interstitial emphysema (CLD/PIE).

RESULTS: A total of 29 transitions from HFOV to HFJV were identified in 28 infants. Reason(s) for transition to HFJV included refractory hypoxia (n=12), respiratory acidosis (n=5), hemodynamic instability (n=4), uncontrolled airleak (n=6), and gas trapping (n=21). Initiation of HFJV allowed a reduction in PAW from 16 to 13 cm H_2O , and was associated with improvement

in both oxygenation and CO_2 clearance. In the group as a whole, AaDO_2 decreased from 500 ± 150 mm Hg (mean \pm SD) prior to commencing HFJV to 260 ± 170 mm Hg 72 hrs later ($p < 0.01$). Oxygenation improved in all but 3 infants after transition to HFJV. A significant improvement in CO_2 clearance was also noted, particularly in the first 12 hrs (PaCO_2 pre-HFJV 56 ± 17 mm Hg, decreasing to 44 ± 10 mm Hg at 12 hrs, $p < 0.01$). Whether the improvement in gas exchange ultimately led to survival was a function of the diagnosis, such that all 7 infants with MAS survived (1 needed ECMO), whereas survival was only 11% and 22% in the groups with CDH ($n=9$) and CLD/PIE ($n=9$), respectively. The MAS group had the most impaired oxygenation at the time of transition to HFJV, and showed the most significant improvement after HFJV was initiated. Infants with CLD/PIE were significantly older at the time of starting HFJV than the MAS group (median 21 days vs. 1 day, $p < 0.05$), and usually had severe cystic and or fibrotic changes on chest X-ray.

CONCLUSION: Initiation of HFJV in infants unresponsive to HFOV was consistently associated with better gas exchange, and particularly in those with MAS, ultimately led to survival. HFJV should be considered wherever HFOV is failing, and, if used, should be initiated before the lung becomes irretrievably damaged.