THE USE OF HIGH FREQUENCY OSCILLATORY VENTILATION (HFOV) IN THE SURGICAL PATIENT

SUMMARY

High frequency oscillatory ventilation (HFOV) has emerged as an option for the treatment of patients with severe acute respiratory distress syndrome (ARDS). HFOV has been shown to be both safe and efficacious in improving oxygenation. The theoretical advantages of HFOV, however, have not yet translated clinically into improved outcomes when compared to conventional ventilation (CV) in the few trials published to-date.

RECOMMENDATIONS

➢ **Level 1**
  ➢ None

➢ **Level 2**
  ➢ HFOV should be considered in patients with oxygenation failure (FiO_2 ≥ 0.7, PEEP ≥ 15 cm H_2O) refractory to conventional mechanical ventilation.

➢ **Level 3**
  ➢ Initial HFOV settings should include the following: mean airway pressure (Paw) 5 cm H_2O above CV; FiO_2 1.0; Frequency 6 Hz; Inspiratory time 33%; Amplitude (ΔP) 90 cm H_2O; Bias flow 40 L/min.
  ➢ Clinical indications include treatment for refractory oxygenation failure and refractory bronchopleural fistula.

INTRODUCTION

Acute respiratory distress syndrome is a multi-factorial disease process that has a mortality rate of at least 30% (1). The mainstay of therapy is generally supportive mechanical ventilation. There is a significant decrease in lung compliance that often requires a considerable increase in ventilator settings. This increase in support often creates supranormal intrathoracic pressures. Recent literature has shown that these high pressures can induce additional lung injury known as ventilator induced lung injury (VILI) (1,2). This has prompted the search for alternative ventilation strategies that minimize VILI.

VILI may occur by multiple mechanisms. Overpressure in the alveolar unit causes air leakage termed "barotrauma". Overdistension of the alveoli, termed "volutrauma", causes both epithelial and endothelial damage. Repeated distension and collapse of the alveoli during the ventilatory cycle causes additional shear forces known as "atelectrauma". Finally, this repetitive cycle of injury leads to the release of inflammatory mediators that can also lead to multi-organ failure on a systemic level (1,3). The properties of HFOV can, in theory, minimize these mechanisms of injury. This has made it an attractive option for these clinical situations.

EVIDENCE DEFINITIONS
- **Class I:** Prospective randomized controlled trial.
- **Class II:** Prospective clinical study or retrospective analysis of reliable data. Includes observational, cohort, prevalence, or case control studies.
- **Class III:** Retrospective study. Includes database or registry reviews, large series of case reports, expert opinion.
- **Technology assessment:** A technology study which does not lend itself to classification in the above-mentioned format. Devices are evaluated in terms of their accuracy, reliability, therapeutic potential, or cost effectiveness.

LEVEL OF RECOMMENDATION DEFINITIONS
- **Level 1:** Convincingly justifiable based on available scientific information alone. Usually based on Class I data or strong Class II evidence if randomized testing is inappropriate. Conversely, low quality or contradictory Class I data may be insufficient to support a Level I recommendation.
- **Level 2:** Reasonably justifiable based on available scientific evidence and strongly supported by expert opinion. Usually supported by Class II data or a preponderance of Class III evidence.
- **Level 3:** Supported by available data, but scientific evidence is lacking. Generally supported by Class III data. Useful for educational purposes and in guiding future clinical research.

HFOV works by maintaining lung inflation at a constant elevated mean airway pressure (Paw) while using a piston to cycle the ventilation rate at several hundred times per minute. This results in a tidal volume that is often smaller than the anatomical dead space of the lungs. Recent studies, the most prominent being by the ARDS Network group, have shown that lower tidal volumes help to reduce volutrauma injury to the lungs (4). The smaller tidal volumes of HFOV fit well with this concept. HFOV also functions to minimize the cycle of alveolar distension and collapse by maintaining airway pressures throughout ventilation. The rapid pressure changes created by the oscillating piston are attenuated at the alveolar level thereby minimizing atelectrauma and improving alveolar recruitment (3). This effect is also improved by the use of periodic recruitment maneuvers while on HFOV. All of these attributes of HFOV would seem to make it an attractive therapy for severe ARDS patients.

Unfortunately, the theoretical advantages of HFOV have not yet been proven in the clinical setting. Current literature shows that HFOV is safe and improves oxygenation in ARDS patients. Current controlled trials show promising trends, but have yet to demonstrate improved outcomes with the use of this therapy.

LITERATURE REVIEW
In 2000, the ARDS Network published a comparison between low tidal volume (6 mL/kg) ("lung protective") and traditional tidal volume (12 mL/kg) ventilation. The result was a significant decrease in mortality in the low tidal volume group. While the optimal tidal volume strategy has not been determined, it is evident that there is certain pathology caused by over distention and collapse of alveoli during the respiratory cycle. This disruption of the functional pulmonary unit results in both local and systemic inflammation further exacerbating hypoxemia and multi-organ failure (4). In addition, studies by Ranieri et al. showed a decrease in white blood cells and select inflammatory mediators in blood and alveolar lavage samples in patients ventilated using lung protective strategies (5).

The largest of the two randomized controlled trials using HFOV in the adult population was published by Derdak et al. in 2002 (6). The purpose of this study was to evaluate the safety and efficacy of HFOV. One hundred forty-eight adult patients with ARDS were randomized to HFOV versus conventional ventilation (CV). The HFOV group was exposed to significantly higher Paw and showed a significant improvement in PaO_2/FiO_2 ratio over the first 16 hours. This improvement, however, did not persist past 24 hours. Thirty day mortality was 37% in the HFOV patients and 52% in CV patients. This difference only trended towards significance (6,7). Of note, there was also a significant increase in pulmonary artery occlusion pressure (PAOP) and central venous pressure (CVP) measurements in the HFOV patients. There was no significant difference in complications during therapy. There was also an insignificant difference in total ventilator days (6). This paper concluded that HFOV is safe and efficacious in the adult population; however, the study was not powered to show a significant decrease in mortality. The only other randomized controlled trial was stopped prematurely secondary to enrollment issues. No major conclusions were reached in this limited trial of 61 patients (8).

Mehta and colleagues reported a retrospective review of 156 patients in which HFOV was used as rescue therapy in patients with ARDS. These patients had received CV for 5.6 ± 7.6 days prior to HFOV. Most patients showed significant improvement in both PaO_2/FiO_2 and oxygenation index. These effects persisted for greater than 72 hours. Twelve percent of these patients, however, failed HFOV secondary to oxygenation, ventilation, or hemodynamic difficulties. Of patients with pulmonary artery catheters, an increase in CVP and a decrease in cardiac output (CO) to low normal values were shown. PAOP showed a transient decline that lasted 6 hours. Thirty day mortality was 62% and the pneumothorax rate was 22% in these patients. In rescue efforts, HFOV has been shown to improve oxygenation with minor alteration in pulmonary artery catheter values. One of the independent risk factors for death identified was days on conventional ventilation prior to HFOV. This indicates a possible advantage to the earlier institution of HFOV therapy. The authors also concluded that HFOV is a safe and efficacious modality (9).

The alveolar recruitment accomplished by HFOV can be further augmented and maintained through the use of recruitment maneuvers. Ferguson et al. reported a pilot study of 25 patients transitioned to HFOV from CV. Recruitment maneuvers included pausing oscillations while FiO_2 was increased to 1.0 and Paw

was increased to 5 cmH$_2$0 above baseline. This combination showed a significant decrease in FiO$_2$ requirements within 12 hours of transition. No increase in complication rate was observed, however, overall mortality was 44% in the group. As with all of the studies involved, death was typically attributed to multi-organ failure (10).

There are several case reports describing the use of HFOV in patients with high output or refractory bronchopleural fistula (BPF). Ha and Johnson report a case of a 55 year old man developing a BPF after decortication for empyema (12). In this scenario, the BPF worsened significantly with the development of ARDS. The patient recovered after a 28 day course of HFOV therapy. Crimi et al describe treatment of six patients with unilateral lung injury treated with independent lung ventilation and HFOV therapy. Three of the six patients treated had concomitant BPF (13). HFOV is recommended as a second line of therapy for BPF.

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High Frequency Oscillatory Ventilation Algorithm

Suggested Indications
1. Oxygenation: $FiO_2 \geq 0.7$, $PEEP \geq 15$ cm H_2O
2. Ventilation: pH< 7.25 with TV $\geq$ 6mL/kg and peak pressure > 30cm H_2O

Relative Contraindications
1. Airflow obstruction
2. Intracranial hypertension

Initial Settings
1. Bias flow 40L/min
2. Inspiratory time 33%
3. mPaw 35 cmH$_2$O or 5cm above mPaw
4. FiO$_2$ 1.0
5. Amplitude (P) 90 cmH$_2$0 (oscillations to mid-thigh)
6. Frequency 6 Hz
7. Initial recruitment maneuver

OXYGENATION
SaO$_2$ $\geq$ 88%; PaO$_2$ $\geq$ 55cm $H_2$0 on initial settings? — Yes

No

Increase FiO$_2$ in .05 increments

SaO2, PaO2 acceptable? — Yes

No

FiO$_2$ = 1.0?

Yes

Increase mPaw in 3 cmH$_2$O increments

SaO2, PaO2 acceptable? — Yes

No

mPaw 45-50cmH$_2$O? — No

Yes

Consider alternate mode if FiO$_2$ 1.0 and mPaw 45-50 cmH$_2$O with unacceptable oxygenation

VENTILATION
pH > 7.25? — Yes

No

Decrease frequency in 0.5 Hz increments

pH acceptable? — Yes

No

Frequency 3 Hz?

Yes

Increase dP to raise pH

pH acceptable? — Yes

No

dP 100cm H_2O?

Yes

Institute 5cm cuff leak

pH acceptable? — Yes

No

NaHCO$_3$ gtt for pH <7.15

pH acceptable? — Yes

No

Consider alternate mode if pH <7.15

VENTILATOR WEANING

FiO$_2$ 0.4-0.5 mPaw 25 cmH$_2$O? — No

Yes

Stable 12 - 24 hours? — No

Yes

Hold transition

No

Ventilator Transition
PCV 15 cm H_2O
PEEP 15cm H_2O
FiO$_2$ 0.4-0.5
RR 20-25

Recruitment Maneuvers (RM)
1. FiO$_2$ 1.0 for 5 minutes
2. mPaw 5cm above baseline
3. Hold oscillations for 40 seconds
4. Increase mPaw in 5cm increments until oxygenation improved

Tenets
1. Use frequency as primary adjustment for pH
2. RM twice daily, upon circuit interruption, and prn for mPaw adjustments
3. Wean FiO$_2$ prior to mPaw
4. ABG as needed (no EtCO$_2$)
5. Monitor for barotrauma
6. Optimize fluid status

14.