Review article

High frequency oscillatory ventilation in adult patients with acute respiratory distress syndrome

Balasaheb Pawar, Prasad Rajhans*, Sameer Jog, Prasad Akole, Bhagyashri Bhurke, Divyesh Patel, Monika Kothari

Email: dmhicv@gmail.com

Abstract

Clinical trials on the use of mechanical ventilation in Acute Respiratory Distress Syndrome (ARDS) showed that use of low tidal volumes reduces the mortality significantly. Another approach that proved beneficial was to open the lung and keep it open. Theoretically, this can be best achieved with High Frequency Oscillatory Ventilation (HFOV). HFOV provides oscillations at three to ten Hertz in adults. Both inspiration and expiration are active. Gas exchange occurs even though the tidal volume delivered during each oscillation is less than the anatomical dead space. Furthermore, the potential adverse effects of the conventional ventilation such as alveolar overdistension, and repeated opening and collapse of alveoli are reduced. Many investigators have studied the potential benefit of HFOV in neonatal and paediatric population but the evidence for its use in adult population is limited. A lot of work is being done and two large ongoing trials OSCAR and OSCILLATE should give better idea regarding its use in adults. This review article mainly focuses on the principles and practices of HFOV in adults and current evidence regarding its use in adults.

Keywords: Acute respiratory distress syndrome, Ventilator induced lung injury, High frequency oscillatory ventilation.

Introduction

Acute respiratory distress syndrome (ARDS) is a clinical condition that is associated with high mortality.

Balasaheb Pawar, MD, IDCCM
Consultant Intensivist, Department of Intensive Care Medicine

Prasad Rajhans, MD, FICCM
Chief Intensivist, Department of Intensive Care Medicine

Sameer Jog, MD, IDCCM
Consultant Intensivist, Department of Intensive Care Medicine

Prasad Akole, DNB, IDCCM
Consultant Intensivist, Department of Intensive Care Medicine

Bhagyashri Bhurke, MD, IDCCM
Consultant Intensivist, Department of Intensive Care Medicine

Divyesh Patel, MD, IDCCM
Associate Intensivist, Department of Intensive Care Medicine

Monika Kothari, MD, IDCCM
Associate Intensivist, Department of Intensive Care Medicine

Deenanath Mangeshkar Hospital, Erandawane Pune

Mechanical ventilation for ALI/ARDS has evolved over last two decades. A large number of clinical and preclinical trials have generated substantial evidence to emphasise the way in which mechanical ventilation is conducted with an impact on clinical outcome. The deleterious effects of mechanical ventilation are well understood and accepted world over. These deleterious effects include ventilator-induced lung injury (VILI). VILI can occur when overdistension of alveoli with high volumes and pressures disrupt the alveolar epithelial membrane causing injury. Repeated opening and closing of partially collapsed alveoli due to inadequate end-expiratory alveolar recruitment can disrupt both the alveolar epithelial and capillary endothelial membranes (atelectrauma) and contribute to lung injury. Alveolar injury caused due to overdistension or from repetitive opening and closing also initiates a systemic inflammatory

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response, which can eventually lead to failure of other organ systems and ultimately to mortality.[1] Thus the aim of lung protective ventilation is not only to improve gas exchange but also to prevent VILI.

High Frequency Oscillatory Ventilation (HFOV) theoretically appears ideally suited to support these principles of lung protective ventilation. It provides a relatively high mean airway pressure, which may open slow-recruiting compartments and keep open fast-collapsing portions of the lungs to recruit the lung. The very small tidal volume (V_T) provided by HFOV should minimise the risk of overdistension during inspiration while the higher mean airway pressure should reduce derecruitment during expiration. HFOV is a technique that was first introduced into neonatal and paediatric intensive care. High frequency positive pressure ventilation was first developed in the 1960s.[2] The potential of high frequency ventilation in humans has been studied since the observation that adequate gas exchange occurred in panting dogs with tidal volumes lower than the anatomic dead space.[3,4]

TECHNICAL ASPECTS OF HFOV

Three different types of high frequency ventilation (HFV) are in use: High Frequency Jet Ventilation (HFJV), High Frequency Percussive Ventilation (HFPV), and High Frequency Oscillatory Ventilation (HFOV). This article is limited to HFOV.

HFOV is driven by a piston pump, which causes a diaphragm attached to it to oscillate at rapid frequencies (for adults, typically 3–10 Hz, or 180–600 breaths/min (*Figure 1*). A bias flow of gas continuously flows through the circuit which is typically set between 20 to 40 L/min. The diaphragm moves forward and backward as it oscillates. Each forward movement generates and delivers a small tidal volume (usually 1 – 2 mL/kg) to the patient creating an inspiration. Backward movement of the diaphragm is active and constitutes expiration. Thus, in HFOV, expiration is active and theoretically results in improved carbon dioxide (CO_2) elimination and reduced gas trapping.

Terminology

Pressure amplitude: The actual distance that the piston pump and the diaphragm move during oscillation is called the pressure amplitude (P). It usually ranges from 60 to 90 cm H_2O in adults. The set power on the ventilator determines this pressure amplitude. The oscillations produce a visible wiggle of the patient's body. Most commonly, when HFOV is first applied to a patient, P is initially set to a level that results in oscillatory patient movement down to the mid-thigh level.

The oscillatory pressure amplitude (ΔP) is measured in the ventilator circuit and does not represent actual pressure oscillations in the airways. The endotracheal tube and larger airways attenuate the oscillatory pressures and thus, the pressure swings in the alveoli are much less. The amplitude of oscillation and thus, the tidal volume are titrated to achieve acceptable CO_2 elimination.

Frequency: The speed of oscillation is set by manipulating the frequency. One Hertz is equal to 1 breath/s (60 breaths/min). A frequency of 5 Hz is equal to 5 breath/s or 300 breaths/ min. As the frequency increases, the time available for inspiration is reduced which in turn reduces the piston excursion provided the inspiratory to expiratory time ratio remains unchanged. Thus, the amplitude and the delivered tidal volume reduce. Thus, it can be said that tidal volume is inversely proportional to the frequency.

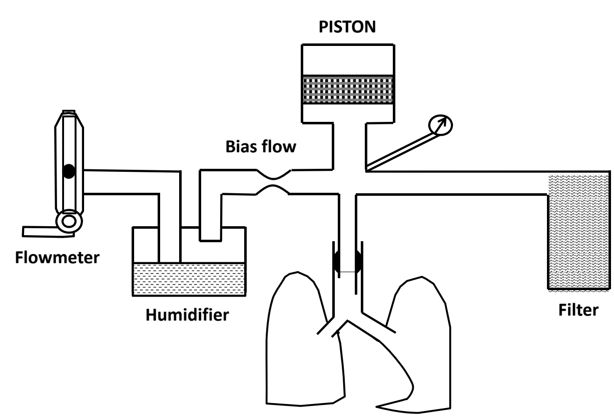

Figure 1: Schematic diagram representing the major functioning parts of the high frequency oscillatory ventilator

Mean Airway Pressure or Continuous Distending Pressure : This is the pressure which is set to keep the alveoli open. It is bias flow dependent and also can adjusted with Mean Airway Pressure (mPaw) knob on the machine.

Bias flow : This is continuous flow of gas (air and oxygen) in the circuit. The 0 to 60 L/min is the range available but usually 20 to 40 L/min is used in clinical practice.

Inspiratory time : Inspiratory time is set as a percentage of one oscillatory cycle time. Usually it is kept fixed at 33%.

Mechanisms of gas transport during HFOV

Tidal volumes delivered using the 3100B are not measured and are estimated to be 1 to 2 ml/kg (approximately the volume of anatomic dead space). Gas transport occurs in the larger airways by *convection* (mass movement of molecules) while it is mainly diffusion in the alveoli (movement due to the inherent movement of the molecules). The airways in between have a mixture of *convection and diffusion.*[5,6]

In the larger airways, during high frequency oscillation, gases tend to flow in a *coaxial* fashion where in the gases in the centre tends to move towards the alveoli and the gases towards the periphery tends to move outward. *Bulk flow* (movement of molecules down a concentration gradient) ensures gas delivery to proximal alveoli. Some mixing of fresh and residual gas can occur to dispersion. Gases can also flow between alveoli due to *pendelluft flow.*

Collateral ventilation and cardiogenic mixing also play a role. *Molecular diffusion* may be augmented at the alveolar level secondary to added kinetic energy from the oscillations. The importance of each of these mechanisms is debated, and it has been suggested that a combination of all the above factors may occur simultaneously during HFOV.[5-8]

Oxygenation and ventilation during HFOV

HFOV differs from conventional mechanical ventilation and there is decoupling of oxygenation and ventilation. Oxygenation is determined by the set fraction of inspired oxygen (FiO_2), mean airway pressure (mPaw) and the shunt fraction as in conventional mechanical ventilation. The higher mPaw inflates the lung, optimises the alveolar surface area for gas exchange, reduces intrapulmonary shunting of blood and thus improves oxygenation *(Figure 2)*. There is continuous flow of gas past the resistance (inflation) of the balloon on the mean airway pressure control valve (bias flow). The mPaw is changed by either adjusting the bias flow or the inflation of the balloon control valve (mPaw Adjust).

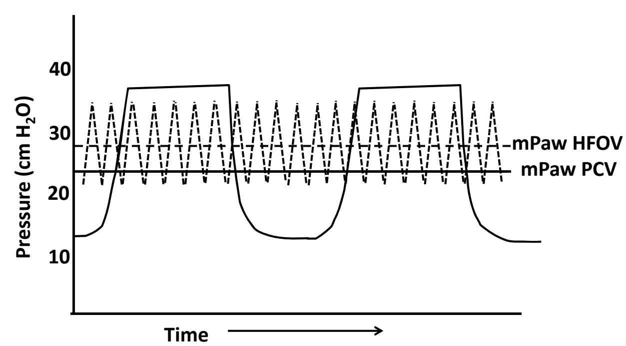

Figure 2: Comparison between the pressure swings on conventional ventilation and HFOV.

Ventilation is primarily a function of amplitude and frequency. The power setting on the HFOV determines the extent through which the piston moves and in turn tidal volume. Thus, tidal volume is directly proportional to amplitude. Increase in amplitude will increase the ventilation and thus CO_2 clearance. Increase in frequency decreases the range of piston movement and in turn decreases tidal volume. CO_2 clearance is inversely proportional to frequency.

Evidence for HFOV use in Adults

The use of HFOV has been extensively studied in the neonates and children with encouraging results. Evidence for HFOV in adults with lung injury is limited. HFOV has mostly been investigated as a rescue therapy for adult patients with ARDS who fail conventional mechanical ventilation.

Two case series with a total of 41 ARDS patients suggested that HFOV may be beneficial in these patients.[9,10] Mehta and colleagues[10] studied 24 patients with severe ARDS (lung injury score = 3.4 ± 0.6), PaO_2/FiO_2 ratio = 98.8 ± 39.0) failing conventional ventilation (ongoing hypoxaemia or high plateau pressures), and showed that HFOV could achieve an improvement in the PaO_2/FiO_2 ratio within 8 hours. Fort and colleagues studied 17 patients with severe ARDS (lung injury score = 3.81 ± 0.23, PaO_2/FiO_2 ratio = 68.6 ± 21.6) failing conventional ventilation and found similar improvements in oxygenation.[9] Both studies suggested that mortality improved in patients who had fewer pre-oscillator ventilator days. Although refractory hypoxaemia can be problematic in patients with ARDS, often the cause of the patient's death is multiple organ failure.

The Multicenter Oscillatory Ventilation for ARDS randomised 150 patients to HFOV (initial frequency of 5 Hz, mPaw > 5 cm H_2O than mPaw on Conventional Ventilation) and Conventional Ventilation (Tidal Volume of 6-10 ml of actual body weight). All the patients were on conventional ventilation for average 2-4 days before randomisation. 30 days ventilation free survival was not significantly different in two groups, but there was nonsignificant trend towards low mortality in HFOV arm compared to Conventional Ventilation.

In an updated review of eight randomised controlled trials, pooled results suggest that high frequency oscillation improves oxygenation and reduces the risk of treatment failure (refractory hypoxaemia, hypercapnoea, hypotension or barotrauma) as well as hospital or 30 day mortality compared with conventional mechanical ventilation in patients with ARDS.[12] Ongoing OSCILLATE trial aims to evaluate early use of HFOV. Until the results of this trial are available, use of HFOV currently remains mainly a rescue modality for refractory hypoxaemia.[13]

PRACTICAL CONDUCT OF HFOV

Initiation of HFOV

HFOV may be considered in ARDS patients who are failing on the conventional ventilator. In the absence of more substantial evidence, HFOV is used as a rescue modality when $FiO_2 > 0.7$ and PEEP >14 cm H_2O. Studies support the use of HFOV early in the course of disease as increasing number of days on conventional ventilation prior to HFOV is associated with poor outcome.

Adequate volume status must be ensured. At some centres, administration of volume bolus before connecting to HFOV is a standard practice. The patient's airway is suctioned and patency confirmed. If bronchoscopy is indicated, it must be performed before connecting to HFOV. Calibration and performance check of the high frequency oscillator must be conducted as per manufacturer's instructions.

A lung recruitment manoeuvre (RM) is conducted. It is performed typically for three situations, first at the time of initiation of HFOV, second for desaturation following suctioning, disconnection or change in patient position and the third for persistent hypoxaemia. RM is contraindicated in haemodynamically unstable patients, patients with airleak without intercostal drainage *in situ* and in the event of any complication during prior RM. During RM, the FiO_2 is set to 1.0 and oscillations are paused. mPaw is raised to 40 cm H_2O over 10 seconds and maintained at that level for 40 seconds. The mPaw is then lowered to 35 cm H_2O or to the initial mPaw, if RM was performed for disconnection or desaturation.

The mPaw is initially set at 5 cm H_2O above the mPaw during conventional ventilation. Power is set at 6 to begin with and further adjusted to get oscillations from clavicular to mid-thigh level. Frequency is set at 5 Hz and then adjusted according to $PaCO_2$ levels.

Some authors suggest to set frequency depending upon initial pH (<7.10 = 3.5 Hz; 7.10-7.19 = 4 Hz; 7.20-7.35 = 5 Hz; >7.35 = 6 Hz).[14] Inspiratory time is usually kept fixed at 33%. FiO_2 is set at 1.0 at the initiation of HFOV and subsequently decreased to safe limit of 0.6 as early as possible accepting a PaO_2 of 60 mm Hg and SpO_2 of 88 to 90%.

Monitoring and troubleshooting

Standard critical care monitoring of vital signs is of paramount importance. Hypotension at the time of initiation can be easily treated with fluid boluses. Chest wiggle must be observed in the initial period and followed thereafter. Absence of wiggle on one side occurs due to ipsilateral pneumothorax or lung collapse. Asymmetric wiggle occurs in endobronchial intubation particularly after change in patient position.

Monitoring of arterial blood gases (ABG) need to be more frequent in the initial period and thereafter must be monitored every 6 hours. ABG is also indicated after any change in the settings or any clinically important event. A chest X-ray is obtained within first hour of initiation and then depending upon clinical indication. If posterior ends of 8-9 ribs are seen, then it is considered to be adequate inflation. Hyperinflation can compress the vascular bed leading to $\dot{V}/\dot{Q}$ mismatch.

If oxygenation does not improve and FiO_2 requirement continues to be high, then RM is repeated. The mPaw is increased 2-3 cm H_2O at a time till improvement in oxygenation is seen. An mPaw of 40 cm H_2O and above is used in a very few patients with severe ARDS. High $PaCO_2$ and respiratory acidosis is dealt with in three ways: Increase amplitude first, decrease frequency and if necessary allow a small cuff leak.

Weaning of HFOV

Weaning from HFOV should begin from the time of initiation. Once the target oxygenation is achieved, FiO_2 is decreased by 0.05 to 0.1 every 10 to 15 min. The first aim is to achieve the safe limit of 0.6 and then decrease further till 0.4. After this, mPaw is decreased by 2 cm of H_2O at a time. Once the FiO_2 of 0.4 and mPaw of 20 cm H_2O is reached with acceptable oxygenation, the patient may be changed back to conventional ventilation.

Potential complications and drawbacks of HFOV

Due to use of high mPaw during HFOV, barotrauma and haemodynamic compromise are potential complications. Volume preloading is advised by some authors before switching to HFOV from conventional ventilation.[12] Requirement of heavy sedation and paralysis is another problem. Daily interruption of sedation is often not possible.

Suctioning of airway should be as minimal as possible. It can be achieved with closed suction systems but their role in preventing derecruitment is not proven. Procedures such as bronchoscopy and tracheostomy are not advisable while the patient requires high mPaw. Complications such as pneumothorax and collapse cannot be diagnosed by auscultation and Chest X-ray (CXR) is needed.

The use of aerosolised medications in conjunction with HFOV is not very effective. Metered dose inhalers are largely ineffective with only about 25% of a nebulised medication being detectable at the end of the ET tube.[16] If flow driven nebulisers attached to the HFOV circuit are used, mPaw and amplitude will require adjustment of these variables to maintain stable HFOV settings. Aerosol generator that uses vibrational element (vibrating mesh nebuliser) to produce an aerosol has shown positive results.[15-17]

Patient transport during HFOV may be a significant logistical problem because there is no portable version of the equipment. However, an effective protocol for transport of patients on HFOV has been described.[18]

References

1. Slutsky AS, Tremblay LN. Multiple system organ failure. Is mechanical ventilation a contributing factor? *Am J Respir Crit Care Med* 1998; **157**:1721-5.

2. Henderson Y, Chillingsworth F, Whitney J. The respiratory dead space. *Am J Physiol* 1915; **38**:1-19.
3. Lunkenheimer PP, Frank I, Ising H, Keller, Dickhutt HH. Intrapulmonary gas exchange during simulated apnea due to transtracheal periodic intrathoracic pressure changes. *Anaesthetist* 1973; **22**:232-8.
4. Bohn DJ, Miyasaka K, Marchak BE, *et al.* Ventilation by high frequency oscillation. *J Appl Physiol* 1980; **48**:710-6.
5. Pillow JJ. High frequency oscillatory ventilation: mechanisms of gas exchange and lung mechanics. *Crit Care Med* 2005; **33**:S135-41.
6. Weavind L, Wenker O. Newer modes of ventilation: an overview. *Int J Anesthesiol* 2000; **4**:4.
7. Pillow JJ, Neil H, Wilkinson MH, Ramsden CA. Effect of I/E ratio on mean alveolar pressure during high frequency oscillatory ventilation. *J Appl Physiol* 1999; **87**:407-14.
8. MacIntyre NR. High frequency ventilation. *Crit Care Med* 1998; **26**:1955-6.
9. Fort P, Farmer C, Westerman J, *et al.* High frequency oscillatory ventilation for adult respiratory distress syndrome – a pilot study. *Crit Care Med* 1997; **25**:937–47.
10. Mehta S, Lapinsky SE, *et al.* Prospective trial of high-frequency oscillation in adults with acute respiratory distress syndrome. *Crit Care Med* 2001; **29**:1360–9.
11. Derdak S, Mehta S, Stewart TE, *et al.* The Multicenter Oscillatory Ventilation for Acute Respiratory Distress Syndrome Trial (MOAT) study investigators: High frequency oscillatory ventilation for acute respiratory distress syndrome in adults: a randomized, controlled trial. *Am J Respir Crit Care Med* 2002; **166**:801–8.
12. Sud S, Sud M, Friedrich JO, *et al.* High frequency oscillation in patients with acute lung injury and acute respiratory distress syndrome (ARDS): systematic review and meta-analysis *BMJ* 2010; **340**:c2327.
13. The Oscillation for ARDS Treated Early (OSCILLATE) Trial Pilot Study. Canadian Critical Care Trials Group. ClinicalTrials.gov Identifier. NCT00474656.
14. Fessler HE, Derdak S, Ferguson ND, *et al.* A protocol for high-frequency oscillatory ventilation in adults: Results from a roundtable discussion. *Crit Care Med* 2007; **35**:1649-54.
15. Garner SS, West DB, Bradley JW. Albuterol delivery by metered dose inhaler in a pediatric HFOV model. *Crit Care Med* 2000; **28**:2086-9.
16. Higgins J, Diebold A, Mellor S, *et al.* The evaluation of aerosolized albuterol deposition in-line via conventional ventilation versus HFOV. *J Resp Care* 2001; **46**:A1080.
17. Fink JB, Barraza P, Bisgaard J. Aerosol delivery during mechanical ventilation with high frequency oscillation: an in vitro evaluation. *Chest* 2001; **120**:S277.
18. Cartotto R, Ellis S, Gomez M, Cooper A, Smith T. High frequency oscillatory ventilation in burn patients with the acute respiratory distress syndrome. *Burns* 2004; **30**:453-63.