

High Frequency Oscillatory Ventilation: disease specific clinical management strategies

Introduction

High Frequency Oscillatory Ventilation (HFOV) has been in use for treatment of various forms of respiratory failure for several years. While the National Institute of Health's sponsored HiFi Trial did not demonstrate a benefit to its use, much was learned during that study which has altered the ways we apply this therapy today. One of the more poignant lessons learned from that study was that HFOV used in different ways, produced significantly different results. The success of HFOV is a direct result of the development of specific strategies for each patient. The discussion which follows describes the aspects of HFOV that affect oxygenation and ventilation (*CO₂ elimination*) and how they are adjusted when managing infants with specific pathologies.

Management of the patient requiring high frequency oscillatory ventilation is an art, based on a few simple principles. These basic principles will lead the clinician to choices which are primarily related to the pulmonary pathophysiology of the underlying disease.

HFOV effectively decouples oxygenation from ventilation in that changes made to alter oxygenation have little effect on CO₂ removal; conversely, changes made to effect a CO₂ change have little effect on oxygenation.

Oxygenation

To manipulate oxygenation, the clinician adjusts the patient's lung volume—as there is a close relationship between lung volume and surface area for gas exchange, and, in fact, a near linear relationship between lung volume and oxygenation. Because lung volume is established with Mean Airway Pressure (MAP) during HFOV, the MAP adjustment will have the most profound effect on oxygenation. As the mean airway pressure is raised and lowered, the clinician has the ability to control lung inflation and thereby optimize the alveolar surface area. For many disease states this optimum Mean Airway Pressure will correspond to an AP chest film which reveals eight to nine ribs to the level of the patient's diaphragm.

Oxygenation will also be affected to a lesser degree by manipulations in the amplitude (*Delta-P*) of the oscillatory wave form. Amplitude, which most closely corresponds to the tidal volume delivered by conventional ventilation, can affect oxygenation when inflation of the patient's lungs by MAP is marginal.

The frequency (*rate*) of ventilation will have the least effect on oxygenation and is generally not manipulated to improve oxygen delivery and/or reduce shunt.

Ventilation

Control over arterial PCO₂ and thus acid-base balance is achieved by manipulating the ventilator's oscillatory tidal

volume. In HFOV, CO₂ elimination is more closely tied to delivered volume (V) and less to frequency (f) whereby it is considered proportional to V²xf. This differs from conventional ventilation where CO₂ elimination is more linearly proportional to Vxf. The impact of this difference is that when clinicians are managing the arterial PCO₂ of a patient on HFOV, they need to be primarily concerned with the effect these ventilator manipulations have on tidal volume and can effectively give less attention to the direct impact of frequency on the PaCO₂ elimination equation. Using the 3100A HFOV, the three parameters which impact this now all-important “tidal volume” are:

1. Amplitude (Delta-P)
2. Frequency (Hz)
3. % I-Time

These controls are found grouped together on the control panel of the ventilator.

Primary manipulations in PaCO₂ are made with the amplitude or power control. Increasing the amplitude (*using the power knob*), will increase the displacement of the oscillating diaphragm, raising tidal volume delivered to the patient. This is then measured as an increased pressure swing amplitude at the airway opening and results in a lower PaCO₂.

Counter to intuition, lowering the frequency will also increase delivered tidal volume and lower PaCO₂. This happens in the 3100A, as at a lower frequency, the inspiratory time increases by several thousands of a second (*because inspiratory time is a fixed percentage of total cycle time*). This additional inspiratory time will result in more tidal volume delivery to the patient by either allowing for more piston displacement or more likely, allowing the piston to remain in the forward position longer (*at the same amplitude*) which results in more volume transfer across the endotracheal tube.

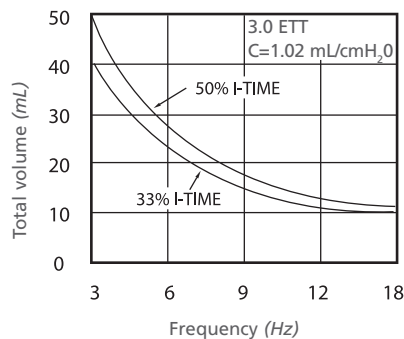


Figure 1: Distal tidal volume vs. frequency at maximum power

Finally, in the more extreme cases (*large patients with severely elevated physiologic dead space*), % inspiratory time can be increased to improve CO₂ elimination. As with the impact of slowing the frequency, increasing the percentage of inspiratory time allows a longer inspiratory phase, thus maximizing the 3100A's delivered tidal volume. Inspiratory time can be adjusted from 33 to 50 percent in 1 percent increments.

Initialization decisions

To switch a patient to HFOV from conventional ventilation, the clinician must consider three aspects of the candidate prior to making the decision regarding initial ventilator settings:

1. Current MAP
2. Disease pathology
3. Inflation of lung fields

Current conventional settings are used as a reference point and the patient's Mean Airway Pressure (MAP) on the conventional ventilator is assessed. The second aspect of the candidate which must be taken into account is the condition of the patient's lung fields prior to initiation of HFOV. If the patient receiving conventional ventilation has been under ventilated and is thus poorly inflated, the starting point on HFOV will be slightly higher (*2 cmH₂O*) than it would be with a patient with adequately inflated lungs.

The final consideration will be disease pathology. What follows is a disease-pathology-specific guide to initial settings which may be used as a starting point for HFOV in the neonatal intensive care setting.

Diffuse disease

In diffuse alveolar disease, the management goal is to recruit gas exchange surface area by opening alveoli and increasing lung volume. In the premature infant, the MAP is usually started at 1 to 2 cmH₂O greater than that set on the conventional ventilator. The frequency used is 10 to 15 Hz and is maximized to 15 Hz for the premature infant less than 1000 grams. The starting mean airway pressure must be sufficient to inflate the lungs regardless of the MAP on conventional ventilation so that an infant with a lung white-out and poor ABGs may require even higher MAPs. The amplitude (*Delta-P*) will be set to achieve the least chest wall movement

which will ensure minimum damage to the delicate architecture of the premature infant's lungs and the desired 40 to 50 mmHg PaCO₂. This will usually be at a Delta-P (*amplitude*) of 20 to 25 cmH₂O. For term and near-term infants with diffuse alveolar disease, the MAP is started at 2 to 4 cmH₂O greater than conventional ventilation and with frequency set at 10 Hz. The amplitude is set to achieve adequate chest wall movement, also near 25 cmH₂O.

Pulmonary interstitial emphysema (PIE)

Management of the premature infant with Pulmonary Interstitial Emphysema is slightly different. The mean airway pressure should be set equal to, or slightly less than, that on conventional ventilation. The reason for this is that with all airleak syndromes, the goal of optimizing alveolar surface area is diminished, settling for an adequate alveolar surface area. This will allow acceptable but not optimal oxygenation and ventilation, while speeding resolution of the air leak syndrome. The frequency is set at 10 to 15 Hz (*use the higher end of the range for the smaller infants*) and the amplitude (*Delta-P*) set to achieve minimal chest wall movement. This will require the clinician to allow elevated PaCO₂ levels (*50 to 60 mmHg*) while maintaining the pH above 7.25, which seems to be an acceptable compromise in the management of airleak syndromes.

Gross airleak

For the management of gross airleak in the premature infant, the MAP is set 1 cmH₂O greater than on conventional ventilation. Frequency is in the 10 to 15 Hz range depending on patient's size and amplitude set for minimal chest wall movement. An important point is that the initial MAP in airleak syndromes is dependent on the inflation of the non-airleak lung. The volume of this lung must be normalized to attain adequate gas exchange. If atelectasis occurs in the good lung, a minimal leak may have to be allowed while re-opening the lung and re-expanding it to eliminate the atelectasis.

Airleak in the term and near term infant is divided into two categories. Gross airleak with adequate inflation, and gross airleak with poor inflation. For gross airleak with poor inflation in the term and near-term infant, the MAP will be set 1 to 2 cmH₂O greater than on conventional ventilation. Frequency of 10 Hz and

amplitude to obtain adequate chest wall movement is used. For gross airleak with adequate inflation in the term and near-term infant, the MAP will be set equal to CMV with frequency of 10 Hz and an amplitude to achieve adequate chest wall movement.

Non-homogeneous lung diseases

The next category of lung pathology faced is commonly secondary to meconium aspiration. This results in a non-homogeneous lung disease pattern. When this non-homogeneous pathology occurs with air trapping, the MAP is usually initiated equal to CMV with the frequency in the 6 to 10 Hz range. The amplitude is set to achieve good chest wall movement. The reason for the lower range of frequency is to allow more time for the delivery of larger tidal volumes (*hence pressure swings*) distally in an effort to overcome some of the airway obstruction present in this disease pathology.

For non-homogenous lung disease with diffuse haze, the MAP is set 2 to 4 cmH₂O greater than conventional ventilation. Frequency again should be in the 6 to 10 Hz range and amplitude set to achieve good chest wall movement.

Focal pneumonia is another pathology frequently encountered in newborns with respiratory failure. For this pathology the MAP is set initially 1 cmH₂O greater than on CMV. Frequency is adjusted at 8 to 10 Hz and amplitude to achieve good chest wall movement

Pulmonary hypoplasia

The final pathology discussed is pulmonary hypoplasia. The two different presentations of this disease are each managed slightly differently. In Uniform Pulmonary Hypoplasia such as occurs in hydrops, the MAP is set at the same level as on conventional ventilation and then increased to achieve maximum oxygen saturation. If MAP is increased 5 to 6 cmH₂O without an increase in oxygen saturation, recheck the x-rays for lung and vessel positioning. Frequency is set at 10 to 15 Hz and amplitude set to achieve minimal chest wall movement, to minimize the lung injury for which patients with this pathology are at risk.

In Non-Uniform Pulmonary Hypoplasia such as occurs frequently in congenital diaphragmatic hernia, the MAP is started equal to or greater than CMV, dependent on the contra-lateral lung. The MAP should be initiated in the 10

to 12 cmH₂O range and increased in 1 cmH₂O increments to optimize the lung volume of the unaffected lung. This lung must be adequately inflated. This can frequently be difficult to assess radiographically as the clinician may not be certain to what degree this lung is also hypoplastic. To adjust the mean airway pressure after the initial setting, it should be increased slowly while observing cardiac performance. When over inflation occurs, the mediastinum will usually be shifted away from its optimal position, thus compromising cardiac filling and output. Frequency in this pathology is at 10 Hz and amplitude set to achieve adequate chest wall movement.

Summary

These guidelines for managing newborns on HFOV represent experience gained from many years with this technology. While HFOV seems to produce uniformly predictable outcomes by following the steps outlined, each infant and their disease process must be evaluated individually and the treatment strategy tailored to that particular patient for successful integration of high frequency ventilation into their care.

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