



HIGH-FREQUENCY OSCILLATORY VENTILATION IN ADULTS

by David Wheeler RRT, NPS

The intent of this column is to impart a basic description of High Frequency Oscillatory Ventilation (HFOV), for the adult patient. Typically HFOV has been clinically applied in the neonatal ICU, yet there is growing interest in HFOV for the adult with ALI and ARDS. I would like to pique your intellectual curiosity and trust that the informed reader will use this brief note as a catalyst to further exploration of this most interesting method of mechanical ventilation.

High-frequency oscillatory ventilation (HFOV) is characterized by the very rapid transfer of extremely small volumes of gas superimposed on a system with a high mean airway pressure. Low volumes with high MAPs are characteristics that craft HFOV as the archetypal lung-protective ventilatory mode. These characteristics coupled with evidence based rationale for lung protection and successful clinical application of HFOV in the neonate has generated interest in HFOV for adults with ARDS and ALI.

In adult applications, HFOV creates extremely rapid pressure oscillations at a frequency that is typically around 3 and 10 Hz., (180 – 600 cycles). These oscillatory waves are superimposed on a constant mean airway pressure generally at a higher level, (3-5 Cm. H₂O), than typically applied in conventional mechanical ventilation. The oscillations are produced by a piston pump that effects both an active inspiration and expiratory phase. The pressure oscillations are significant

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proximally, however, they are attenuated to a significant degree as they move downstream. Indeed, this attenuation is so effective that there is virtually no “air movement” at the alveolar level.

Unique and rather distinctive flow profiles and gas exchange mechanisms are native to HFOV. These flow profiles result from extreme gas mixing in the airways due to the high energy inherent in the remarkable cycle frequencies and flow rates. The flow velocity profiles of HFOV include bulk convection, Taylor dispersion, asymmetric velocity profiles (coaxial flow velocities), pendelluft phenomena, cardiogenic mixing, molecular diffusion, Brownian movement and collateral ventilation. The flow velocity profiles of HFOV are not mutually exclusive and are perhaps best described as synergistic mechanisms of gas transport that improve ventilation-perfusion matching and oxygenation.

The mechanisms influencing gas flow, assimilation and pressure distribution during HFOV are linked via the principal mechanical character of the patient-ventilator system. Bulk convective gas movement plays a contributory role to ventilating the

proximal airways. However, Taylor dispersion where the longitudinal dispersion of gas in a diffusive process is amplified by radial transport mechanisms when laminar flow is applied with or without turbulent eddies is the more significant contributor to total gas transport.

Asymmetric velocity profiles or coaxial flow velocities, wherein fresh gas particles flow centrally down the length of the airway and the returning alveolar peripheral gas particles diffuse towards the outer airway wall on their journey “outward”, is one of the very interesting flow velocities of HFOV. This “coaxial” movement enhances “axial” gas transport with the returning alveolar gas traveling the airway wall towards the mouth and the central gas streaming towards terminal airways. This asymmetric velocity profile is a phenomenon that is intensely apparent at airway bifurcations. The bifurcation skews the inspiratory profile in contrast to the more symmetric expiratory velocity profile. The airway bifurcation phenomenon directs machine delivered gas to the alveoli along a cone in the center of the airway whilst exhaled gas moves out of the system via the outer airway wall. This asymmetric / coaxial flow profile is critical to the longitudinal convective gas transport mechanism and collective gas exchange of HFOV.

Pendelluft phenomena is a form of gas mixing between adjacent alveoli with incongruent time constants. Pendelluft literally translated means “air swing” and this serves as a practical image to illustrate this important flow velocity of HFOV. The pendelluft effect of HFOV creates some semblance of equilibration of gas in alveoli with disparate time constants. Of note is the notion that the inherent asymmetries in inertance and compliance of adjacent units have greater influence on pendelluft flow than do asymmetries in resistance of these units.

Collateral ventilation allows for lateral air movement between acinar units. It is thought that this collateral ventilation occurs via the pores of Kohn and canals of Lambert. Some argue that time constant inequalities and phase lags between lung regions may facilitate flow between adjacent lung units. The thoughtful clinician will appreciate the notion that gas exchange during HFOV may be distinctly improved by the flow between asynchronous adjoining airways.

Gas particle interchange as a result of Brownian motion contributes to the diffusion of gases in the respiratory tract. In equilibrated HFOV, there is virtually zero gas flow in the alveolar region due to the extraordinarily high total cross-sectional area. The principal means of gas movement in the alveolar zone is molecular diffusion and gas transport that is articulated by Fick’s law. Cardiogenic oscillation as an adjunct to HFOV



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has yet to be enumerated. Some have suggested that cardiogenic oscillation may facilitate almost half of the oxygen uptake in HFOV. It has been put forth that the cadenced contractions of the heart may facilitate peripheral gas flow within adjoining parenchymal zones. Be reminded cardiogenic oscillation remains a subject for further investigation.

The cogent reader will be reminded that CO₂ elimination is critically dependent on the net oscillatory volume during HFOV. The fact that in HFOV there is an active expiratory phase contributes to the prevention of gas trapping and dynamic hyperinflation while promoting CO₂ elimination. The co-attendance of Taylor dispersion and molecular diffusion are essential for net gas transport during HFOV. In fact, exclusive of molecular diffusion and Brownian movement; every flow velocity profile of HFOV necessitates the initiation of some form of convective gas motion however, "bulk convection" is inconsequential to gas transport during HFOV.

Regarding the clinical application of HFOV in the adult population the piston pump oscillates at frequencies between 3 and 10 Hz., (180 and 600 cycles/min.) The oscillating diaphragm cycles at extreme frequencies to create pressure oscillations in the patient ventilator system. These oscillations generate the unique flow velocity profiles described previously. The unique flow velocity profiles are responsible for gas exchange and create the greater context of a stable lung field. Tidal volumes are dependant on and proportional to, the driving pressure and the inspiratory/expiratory ratio. Tidal volumes are inversely proportional to respiratory frequency. The unique flow velocity profiles coupled with the resistance of the endotracheal tube will yield noteworthy attenuation of the oscillation pressure in the distal airway.

In adult patients rates of 3 to 6 Hz. and driving pressures from 40 to 90 cm H₂O may need to be employed to achieve CO₂ clearance. Applying an optimal tidal volume strategy will mechanically splint patient airways. The inspiratory and expiratory cycles should be equivalent, (1:1). This will establish an unvarying oscillatory environment and stabilize both the lung field and mean airway pressure. With an active expiration there will be minimal derivation in mean pressure between airway opening and lung. It has been postulated that to amplify potential lung recruitment and lung volumes an ideal MAP may be 1.5 times the lower inflection point. This idealized MAP correlates with the point of maximum curvature on the deflation limb of the pressure-volume curve. The fine tuning of the MAP will create the greater context for optimizing volume delivery. Studies have demonstrated that tidal volume has a greater effect on net gas exchange than frequency during HFOV.

The manipulation of frequency may, at times, seem counter-intuitive. Higher frequencies will deliver lower tidal volume as well as diminish the degree of alveolar pressure swings. Again, the optimal frequency in HFOV may be in the 3 – 6 Hz. range and remains a point of discussion by many clinicians.

The collective impedance of the Patient-ventilator system is a significant factor in the net efficacy of ventilation during HFOV. Impedance incorporates patient-ventilator system elastance, resistance, and inertance. The collective impedance is an impediment to flow and if impedance increases a higher driving pressure may be required to negate its dampening effects.

The advantage of HFOV as a low volume high MAP lung protective strategy may yield lower incidences of VILI. The slight pressure swings, minute volumes and unique flow characteristics of HFOV enhance lung recruitment and create higher end-expiratory lung volumes. The stable MAP stabilizes the lung field and eliminates the potential for cyclical recruitment-derecruitment of the lung. Studies have demonstrated fewer histologic changes and lung inflammation with HFOV initiated earlier in the course of ARDS. HFOV has a role in the attenuation of VALI. There is literature to support the notion that HFOV decreases oxygen use, days on mechanical ventilation, and total hospital length of stay. However, many questions regarding the exact role of HFOV in ALI and ARDS remain unanswered.

The clinical use of HFOV has been associated with increases in central venous pressure and pulmonary artery occlusion pressure. Additionally, HFOV has been associated with clinically irrelevant decreases in CO that were perhaps due to a decrease in venous return. The informed clinician will be aware that patients transitioning from conventional means of ventilation to HFOV are satisfactorily volume repleted. The only contraindication to HFOV is in pathologies with exceedingly prolonged expiratory time constants such as severe obstructive lung disease or asthma.

HFOV is a valuable addition to the clinical cache of the informed clinician. HFOV has tremendous potential in the adult setting and has yet to be fully developed as a primary therapeutic option in many centers. Theoretically, HFOV is the ultimate lung protective strategy and this potential merits further investigation and discussion. This has been a very brief review of this most interesting and complex topic and I trust an issue I will revisit in the near future.

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