

High-Frequency Percussive Ventilation Revisited

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High-frequency percussive ventilation (HFPV) has demonstrated a potential role as a rescue option for refractory acute respiratory distress syndrome and as a method for improving inhalation injury outcomes. Nevertheless, there is a lack of literature examining the practical application of HFPV theory toward either improving gas exchange or preventing possible ventilator-induced lung injury. This article will discuss the clinically pertinent aspects of HFPV, inclusive of high- and low-frequency ventilation. (J Burn Care Res 2010;31:510–520)

A mode of mechanical ventilation known as high-frequency percussive ventilation (HFPV) has demonstrated a potential role as a salvage option for refractory acute respiratory distress syndrome (ARDS) and as a method for improving inhalation injury outcomes.^{1–13} Nevertheless, there is a lack of literature regarding the practical application of HFPV theory toward improving gas exchange. Furthermore, no discussion has been held regarding the possible risk of HFPV-associated ventilator-induced lung injury (VILI).

We will review the evidence-based support for HFPV as a prelude to a discussion of the underpinning concepts and clinical indications for its use. This will be followed by a brief discourse on the risks and benefits of HFPV-administered high- and low-frequency ventilation. The article will, throughout its sections, condense this evolving understanding into our institutional approach to both ventilator setting selection and ongoing investigation.

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HFPV: A BRIEF HISTORY

The volumetric diffusive respirator (VDR-4; Percussionaire, Corp., Sandpoint, ID) is the only ventilator designed to administer HFPV. Forebears of the VDR included airway hygiene devices that promoted airway clearance through to-and-fro “percussive” bursts of air.¹⁴ It was in carrying forward this percussive nomenclature that the mode was eventually coined HFPV. The ventilator works by basically emitting small high-frequency (HF) pulses of gas (high frequency is typically defined as ≥ 60 breaths/min) that accumulates or stacks to form a “low”-frequency tidal volume (V_t) breath. HFPV, in many ways, emulates a typical pressure-limited, time-cycled waveform (Figure 1A–D).

Predicated on its hygienic effects, HFPV was initially favored by inpatient burn units where percussive airflow facilitated the evacuation of airway debris originating from inhalation injury.¹⁵ The accrued clinical experience also revealed that HFPV conferred distinct advantages to conventional ventilator modalities. In unison with the concepts underlying HF jet ventilation and HF oscillatory ventilation (HFOV), HFPV seemed to augment gas exchange through the combined use of both HF V_t breaths that are smaller than dead space and low-frequency ventilation (Table 1 compares and contrasts currently available modes of HF ventilation).^{1–13,16–20}

HFPV: A CONCISE REVIEW OF THE EVIDENCE

As is often the case in intensive care, clinical use of HFPV preceded the groundwork sequential in vitro and in vivo validation studies. Nevertheless, the in

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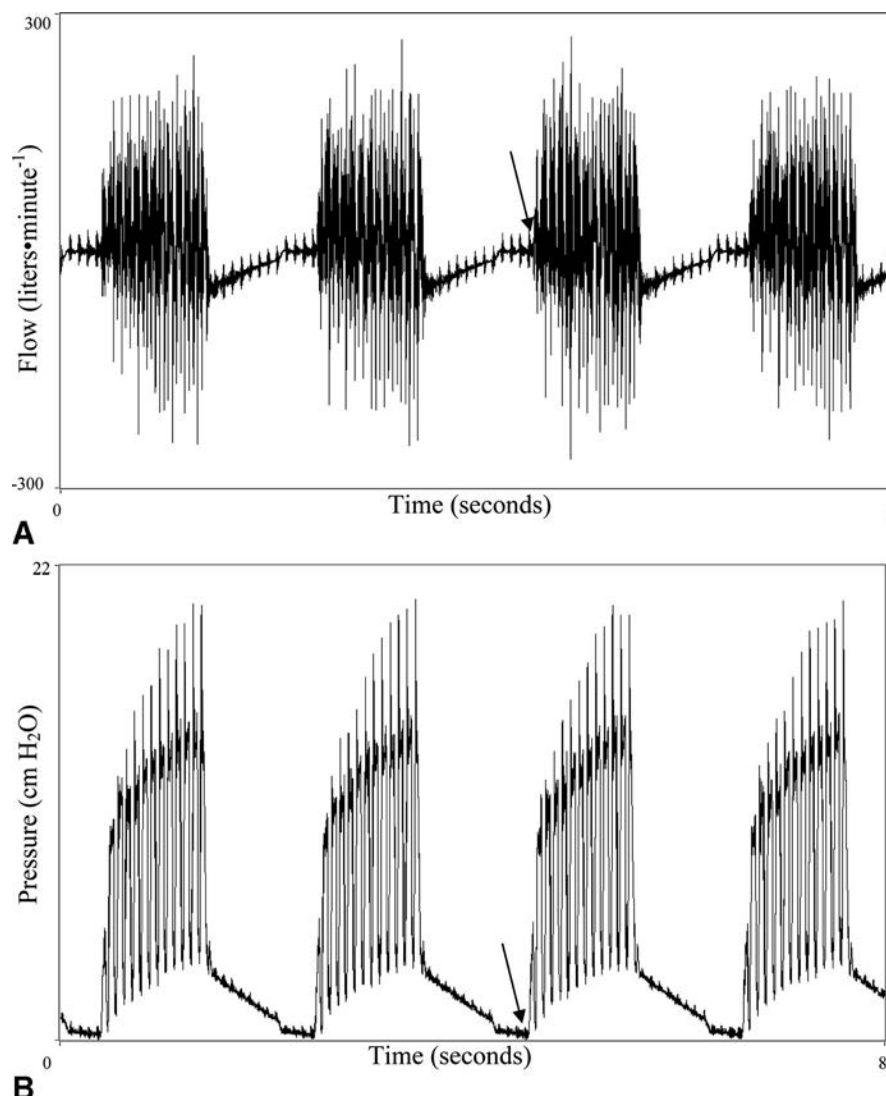


Figure 1. The 8-second graph of (A) high-frequency (HF) flow, (B) HF pressure, (C) low-frequency tidal volume (V_t), and (D) low-frequency flow to a test lung during HFPV (settings of 6 Hz, HF inspiratory/expiratory ratio of 1:1, low-frequency inspiratory and expiratory time of 2 seconds, peak inspiratory pressure of 20 cm H₂O, without applied positive end-expiratory pressure). The graphs demonstrate an increase in HF flow and pressure over the duration of a set inspiratory time to achieve a cumulative low-frequency V_t and flow. The end-expiration or inspiration-onset period is indicated by the arrow. Reproduced with permission from the American Association of Respiratory Care and Allan.²¹

vitro studies provide the only insight into the promising effects of HFPV relative to those offered by conventional low V_t ventilation. For example, recent inhalation injury and aspiration pneumonitis models have shown that HFPV fosters improvements in oxygen and CO₂ tensions while, to a similar extent, attenuating lung inflammation and histological lung injury.^{16,17}

Translating in vitro findings to the bedside, both case-control analyses and prospective studies have associated HFPV with beneficial physiologic and clinical outcomes. Pediatric and adult inhalational injury

studies have linked HFPV to an improvement in static lung compliance, ventilation, oxygenation index, and oxygen tension/fraction of inspired oxygen ratio over that offered by conventional ventilator modalities. HFPV has also been associated with a decrease in the incidence of ventilator-associated pneumonia and an improvement in mortality among inhalational injury patients. Importantly, these aims have been accomplished without invoking an increased incidence of hemodynamic instability or pulmonary barotrauma (ie, pneumothoraces).¹⁻⁶ The mode has also been shown to be safe in head trauma-associated elevated

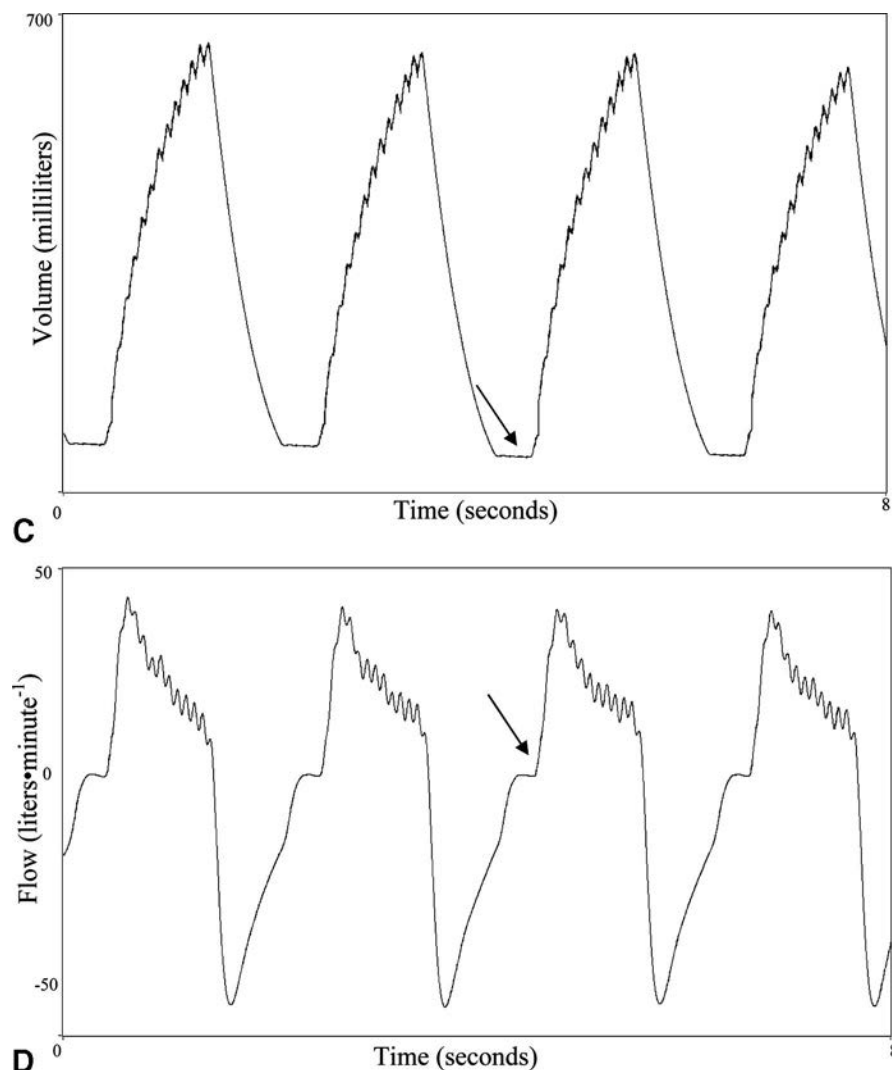


Figure 1. (Continued).

intracranial pressure cohorts.^{19,20} Similar to the effects of HFPV use during inhalation injury, HFPV may favorably affect oxygenation and ventilation when used as a salvage modality for trauma-associated ARDS.⁷⁻¹² However, none of the ARDS-related studies demonstrated a significant change in length of hospital or intensive care unit stay, ventilator-associated pneumonia, or mortality.

Although intriguing, these clinical reports are limited by small sample sizes, retrospective constructs, lack of an adequate comparator (ideal body weight-defined protective lung ventilation), poorly-defined criteria for patients who “fail” conventional modes, inadequate final outcome measures, and conflicting results. Despite these deficiencies, the salutary effects of HFPV in aggregate suggest that this modality may be most applicable to trauma-related ARDS cohorts,

ARDS recalcitrant to lung protective ventilation, and inhalation injury.

HIGH-FREQUENCY VENTILATION CONCEPTS

Although one does not need to scrutinize all of the endorsing principles of HF ventilation, some familiarity is helpful in developing a bedside approach. It is important to recognize that HFOV was the mode examined by the vast majority of the HF literature. Nevertheless, recent work at our institution has revealed that HFPV models many aspects of the oscillatory waveform (eg, waveform responses to loading conditions and frequency-dependent behavior), allowing for the extrapolation of oscillator-derived findings to HFPV.²¹

Table 1. Comparison of the three most common modes of high-frequency ventilation

	HFPV Via VDR-4	HFOV 3100B	HFJV
Requires a large bias gas flow rate	No	Yes	No
Requires continuous electrical power	No	Yes	Variable
Functions as an attachment to a conventional ventilator	No	No	Yes
Requires external compressed air gas source	Variable (only when FiO ₂ <1.0 required)	Yes (uses a second compressed gas flow for oscillator cooling)	Yes
Uses a singular sustained mean airway pressure	No	Yes	Variable (dependent on CV and HFJV setting or device)
Includes bulk tidal volume ventilation	Yes	No	Variable (dependent on CV and HFJV setting or device)
Includes “power” or ΔP option to augment high frequency tidal volume	No	Yes	No
Allows for high frequency inspiratory:expiratory ratio adjustment	Yes	Yes	Yes
Designed to permit spontaneous respiration	Yes	No	Variable (dependent on CV and HFJV setting or device)
Allows for endotracheal tube cuff deflation	Possibly (see cuff deflation section of text)	Yes	No
Compels all ventilator gas flow through a heated humidifier	No	Yes	No

HFPV, high frequency percussive ventilation; *VDR-4*, volumetric diffusive respirator-4; *HFOV*, high frequency oscillatory ventilation; *HFJV*, high frequency jet ventilation; *CV*, conventional ventilator.

Conventional precepts of dead space and effective alveolar ventilation have limited application to HF.^{22–24} Instead, a more suitable starting point is to conceptualize the lung as motionless. When the apneic lung is inflated with a continuous laminar flow of oxygen, there results a distal-to-proximal gas concentration gradient beginning at the alveolar–arterial interface and ending at the central bronchial structures. Diffusion then becomes the principal means of gas interchange. HF Vt markedly enhance this diffusion gradient by repeatedly delivering fresh gas to the proximal bronchial tree. In general, bulk or convective gas flow may be the primary determinant of gas motion at low HF rates, but as the HF rate increases, factors such as inertance, turbulent airflow with eddy formation, asymmetric central-to-airway wall flow velocities, radial transfer, and distal pressure sweep become involved.^{25–29} The extent to which each element contributes to the efficacy of HF ventilation in general, or is advantageous to HFPV in particular, is unclear, and thus only a few of the salient tenets will be explored in this article. Comprehensive topical reviews discussing the dynamic properties of HF flow have been published.^{24–29}

Inertance is seldom evaluated in mechanical ventilation text as it rarely exerts a significant effect at conventional rates. Gas viscosity, the length and diameter of the conducting tube complex, and HF rate comprise the essential elements of inertance. Similar to the physics of inertia, inertance can be conceptualized as the pressure cost of accelerating gas flow to ever-higher frequency levels or, put more simply, a HF rate-related barrier to Vt transmission. Inertance effects couple frequency and Vt such that an increase in HF rate will result in an exponential decrease in Vt (Figure 2).²¹ It follows that to magnify each Vt and, thus, CO₂ excretion, one must reduce the HF rate setting.

It is important to note that, when compared with conventional ventilation, HF-mediated CO₂ removal depends to a larger degree on Vt than on frequency. Indeed, clinical studies have suggested that the efficiency of CO₂ clearance equates to the product of frequency and the squared value of each Vt.²⁵ The origin of this ventilatory response is multifactorial. By using aerosolized radioisotope techniques in dogs, Venegas et al demonstrated that increases in HF rate

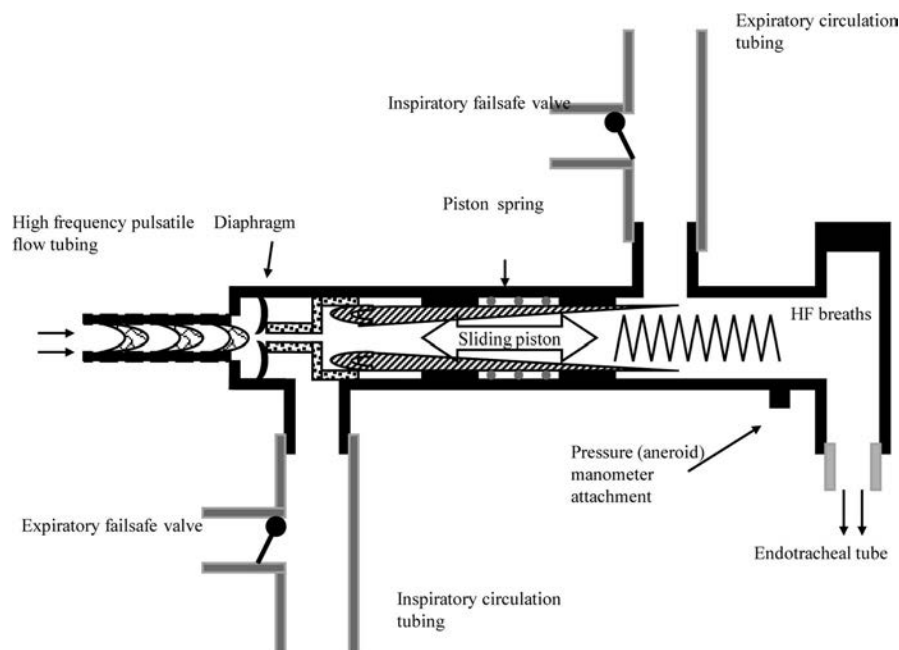


Figure 2. Simplified schematic of the flow amplifier with encased coaxial piston and circulation tubing/fail-safe valve sites. Bidirectional arrow depicts piston motion; HF, high frequency. Reproduced with permission from the American Association of Respiratory Care and Allan and Nawrool.⁴⁸

produced an increase in ventilation throughout the lung, indicating a diffuse dispersive method of gas delivery. In contrast, augmenting V_t mainly enhanced lung base inflation through a combination of both dispersive and convective effects.²⁸ These observations imply that regional recruitment is imperative to CO_2 elimination. In a seminal study, Spahn *et al*³⁰ showed that when the delivered V_t falls below ventilator dead space rebreathing volume, ventilation precipitously worsens. This observation pinpointed the importance of bulk V_t size, also known as convective gas flow, as a critical component of HF gas exchange. The latter investigators also established that V_t benefits may be compromised by unnecessary tubing placed between the HF mechanism and the endotracheal tube.

The ongoing exploration of HF-mediated gas flux still has to answer how increasing the HF rate brings about an improvement in oxygenation in the setting of acute lung and inhalation injury. What has been gleaned from recent models is that HF waveform behavior may vary depending on lung tissue properties.^{31–37} The HF waveform has a near-sinusoidal shape, which is described in part by a peak-to-trough pressure range. In normal lungs the HF pressure amplitude diminishes in size the further it travels from the endotracheal tube. This attenuation effect is a result of airway impedance and is one of the com-

monly cited reasons that HF breaths are considered less harmful than the considerable pressure and volume changes exerted during conventional ventilation. However, after inducing lung injury with a saline lavage in rabbits, Kamitsuka *et al*³⁷ noted that the usual drop in HF peak-to-trough pressure swings going from the trachea to alveolus may be partially reversed. In explanation, lavage-induced atelectasis leads to fall in regional tissue compliance. This change in tissue elasticity can evoke an HF resonance response that amplifies HF pressure swings at the alveolus. Ever-higher HF rates also seemed to widen alveolar pressure differentials.^{38,39} In theory, resonance and/or HF rate-mediated pressure magnification could facilitate gradual alveolar recruitment in previously atelectatic lung and form the basis of superior HF-mediated oxygenation. There is an important note of caution because alveolar pressure phenomena could be construed as detrimental to fragile tissue, in essence constituting a form of microbarotrauma or, alternatively, induce alveolar hyperinflation.^{35,40} Arguing against the latter concern and in favor of the beneficial effects of HF V_t is that HFPV has not been shown to aggravate tissue inflammation.^{16,17}

Escalating bronchiolar level resistance can dampen HF pressure swing transmission to the alveolus.^{38,39} Inhalation injury is mainly a conducting and peripheral airway insult which augments total airway resis-

tance. Therefore, the type of damage may inherently minimize HF-mediated alveolar trauma. However, the marginalizing effect of peripheral resistance on alveolar HF pressure fluctuations may also explain why HFOV (a mode that lacks a low-frequency bulk Vt component) may be relatively less effective at improving gas exchange in comparison with HFPV and conventional modalities.⁴¹ Although it is unclear why bulk Vt may be effective in this clinical context, the latter observation supports the continued use of low-frequency Vt in inhalation injury.

Finally, HF may be permissive of endogenous forms of gas movement. For instance, isotope washout studies revealed juxtacardiac ventilation at combinations of relatively low HF rate and Vt signifying a latent contribution from cardiogenic oscillations.^{27,28} Stroboscopic studies of the subpleural region have also discerned that pore-mediated interalveolar and interbronchiolar gas motion or pendelluft adds to HF gas flux.²⁹

LOW-FREQUENCY VENTILATION CONCEPTS

To the aforementioned HF mechanisms HFPV then adds the principal components of conventional ventilator methodology: low-frequency minute ventilation and mean airway pressure (Paw). Because of their relatively large volumes, low-frequency breaths are an effective means of evacuating dead space and improving ventilation. If excessive, low-frequency breaths may also inflict a variant of VILI known as volutrauma. The evidence-based corollary for this form of lung injury can be found in the ARDSNet trial. The latter study, limited to patients diagnosed with the conditions of acute lung injury (ALI) or ARDS, demonstrated that small Vt (4–8 ml/kg ml/kg ideal body weight, with a concurrent plateau pressure of ≤ 30 cm H₂O) resulted in improved outcomes.⁴² Peak airway or plateau pressure does not reflect the terminal airways but rather the average mechanical properties of the chest and airway complex and lung parenchyma. For that reason, lone HFPV airway pressure measurements may be a poor surrogate in determining the presence or scope of VILI.

Presumably it is because the VDR originates from an era preceding the widespread appreciation of Vt effects that its transducers are not supplemented with volumetric sensors. The latter defect thwarts Vt data collection, and in consequence, clinicians may risk inflicting unappreciated volutrauma. To remediate this deficiency, we completed an in vitro validation of an in-line pneumotachograph flow sensor.²¹ An in vitro construct comprising an HFPV ventilator con-

nected to the flow sensor, an endotracheal tube, and a test lung in series was then used to evaluate Vt magnitude. Importantly, HF Vt were less than 120 ml/breath and thus unlikely to be associated with VILI effects in adults.^{28,29} In contrast, low-frequency ventilation-associated Vt may be larger than many realize. When using ALI-modeled conditions, we measured the Vt administered by HFPV across a typical range of adult settings to include the following: HF rate of 4 to 12 Hz, Paw of 10 to 30 cm H₂O, low-frequency respiratory rate of 10 to 20 breaths/min with corresponding inspiratory times of 1 to 3 seconds, and applied positive end-expiratory pressures (PEEPs) of 5 and 10 cm H₂O. HFPV delivered Vt extended from 607 to 3452 ml (mean Vt of 1337 ml; SD ± 700 ml; 95% confidence interval [CI] of 1175–1499 ml).²⁹ In a standard 70-kg ideal body weight man, these volumes would correspond to a mean 19.1 ml/kg. Practical ways to reduce HFPV-delivered low-frequency Vt and thus attenuate VILI risk will be discussed.

Regardless of the ventilator mode, Paw is the product of the inspiratory time, expiratory time, inspiratory pressure, and (PEEP or, in HFPV terminology, continuous positive airway pressure [CPAP]). Increasing the Paw through incremental adjustments in these variables (or a decrease in expiratory time) will reverse atelectasis and potentially improve patient oxygenation. The state-of-the-art precedence for inspiratory and PEEP settings is currently defined by ARDSNet, which provides both a tabular PEEP and plateau pressure limit (usually ≤ 30 cm H₂O) pathway. This approach (maintain a plateau pressure < 30 m H₂O) suffices in the vast majority of ARDS patients. However, as long as Vt are reigned in, the optimal inspiratory- and expiratory-time aspects of Paw are still open to additional exploration. Anecdotal experience, supplemented by both retrospective HFPV and prospective airway pressure release ventilation research, has shown that frequent brief expiratory times (ie, < 1 second) will minimize expiratory phase-associated derecruitment.^{12,43,44} If oxygenation remains inadequate, then the inspiratory interval is lengthened to increase the time available for Paw-mediated alveolar reexpansion.^{43,44}

In summary, an improved comprehension of HFPV physiology advocates a sequential approach to the selection of high- and low-frequency settings. The overarching clinical goal is to attain acceptable gas exchange while avoiding VILI. This is accomplished by exploiting both HF and Paw waveforms while containing low-frequency Vt.

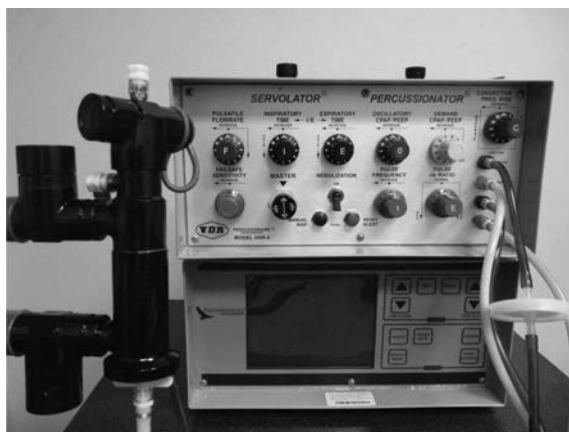


Figure 3. Photograph showing the volumetric diffusive respirator (VDR)-4 and flow amplifier (at left).

HFPV: ENGINEERING OVERVIEW AND SETTINGS

In brief, there are two physical components essential to the VDR: the HF ventilator itself and a tandem flow amplifier, given the proprietary label of “Phasitron” (Figures 3, 4). Small-bore plastic tubing from the ventilator delivers HF airflow to the amplifier where it impacts against a hollow, coaxial, spring-loaded sliding piston. A venturi-like aspect to the piston design draws additional gas from a ventilator circuit reservoir to boost the original ventilator-tubing gas flow before its final delivery to the patient.

It is best to draw comparisons with conventional ventilator modes to understand the semantics of HFPV. Low-frequency ventilation settings include the inspiratory (I-time) and expiratory time (E-time) which, depending on the resulting total breath duration, will de-

cide the respiratory rate. During the inspiratory phase, the lung is inflated to a peak inspiratory pressure (PIP). The PIP is analogous to a conventional ventilator’s pressure limit. Once the I-time cycles off, the lung is then allowed to passively deflate to a demand CPAP (dCPAP). The dCPAP is identical to a conventional ventilator-applied PEEP. The two HF settings include the rate in breaths per minute and the HF inspiratory/expiratory ratio, which ranges from 1:1 to 1:3. Finally, if the clinician wishes to implement HF breaths during the expiratory phase as well, then the oscillatory CPAP (oCPAP) is activated. The oCPAP then causes the ventilator to superimpose HF breaths onto the fixed dCPAP flow.

No ALI/ARDS studies to date have produced a prospectively validated algorithm for choosing HFPV settings. Both high- and low-frequency ventilation principles are encapsulated in a protocol, which is currently undergoing clinical trials at our institution (Table 2). The protocol is also designed to lessen low-frequency V_t magnitude and attendant VILI risk.

When confronted with a decline in arterial oxygen tension, we chose to first exploit the HF ventilation feature by rapidly increasing the HF rate. As for the underlying low-frequency setting, the I:E ratio is also progressively inverted (using both a lengthening of I-time and a diminution in E-time) during the initial portions of the algorithm, whereas increases in inspiratory pressures (ie, PIP) are used later. PIP is largely maintained at ≤ 30 cm H_2O . This is in contrast to others who advocate the eventual use of PIP settings in excess of 80 cm H_2O , a pressure likely to be injurious to the lung.¹³ Once the required PIP exceeds 30 cm H_2O , consideration is given to either adjunctive therapies to improve oxygenation (eg, prone positioning) or alternative modes of mechanical ventilation where supplementary oxygenation methods have proven efficacy. Our approach to expiratory pressure (ie, CPAP) originates from a modified version of the ARDSNet PEEP methodology.^{45,46} To simplify CPAP choices, the two-part CPAP settings used with HFPV were combined: dCPAP and oCPAP were summed to arrive at a total CPAP level.

Certain restrictions were applied in an effort to avert large low-frequency V_t delivery. We allow no more than a 10 to 15-cm H_2O pressure gradient between PIP and end-expiratory pressure (ie, total CPAP) while simultaneously decreasing E-time (ie, usually to <1 second). Throughout our in vitro observations, the maintenance of a pressure gradient of 10 to 15 cm H_2O in conjunction with shortened E-time settings resulted in V_t of 330 ml (SD ± 118 ml; 95% CI 295–365 ml at a relatively low lung com-

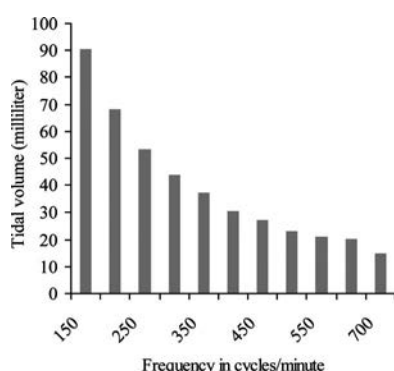


Figure 4. High-frequency (HF) tidal volume (V_t) as a function of frequency. See text for discussion. Reproduced with permission from the American Association of Respiratory Care and Allan.²¹

Table 2. Proposed approach to HFPV/VDR-4 management

Oxygenation Goal: PaO ₂ : >65 mm Hg or SpO ₂ [≤] 89%	Proposed Initial Settings	Strategy Sequence*															
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
FiO ₂	1.00	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7	0.8	0.9	0.9†	0.9†	1.0†	1.0†	1.0†	1.0†
PIP (cm H ₂ O)	20	15	15	20	25	25	30	30	30	30	30	35	35	35	35	40	[≤]40
oCPAP (cm H ₂ O)	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5
dCPAP (cm H ₂ O)	5–10	0	5	10	15	15	15	15	15	15	15	20	20	20	25	25	25
I-time (s)	2	1.0	1.0	1.0	2.0	2.0	3.0	3.5	3.5	4.0	4.5	5.5	6.0	7.0	>7.0	>7.0	>7.0
E-time (s)	2	2.0	2.0	2.0	1.0	1.0	0.7	0.7	0.7	Min	Min	Min	Min	Min	Min	Min	Min
H _{Fr} (cycles/min)	480	480	480	480	480	600	600	600	720	720	720	720	Max	Max	Max	Max	Max

Turn FiO₂ down from initial settings every 10 minutes as permitted to sustain clinically acceptable PaO₂ or SpO₂. Then change frequency and pressure settings to match the table according to the lowest FiO₂ achieved. Subsequently adjust settings per the table above for oxygenation every 2 hours if SpO₂ is >89% or every 5 to 10 minutes if <89% (see below for ventilation instructions). Note: at E-times ≤2 s, the lowest pressure during E-time may represent the combination of o/dCPAP and intrinsic-positive end-expiratory pressure (iPEEP) in which case dCPAP may be reduced to reach the desired total E-time pressure (eg, Column 7: oCPAP and dCPAP are set at 5 and 15 cm H₂O, respectively, but measured E-time nadir pressure is 25 cm H₂O indicating 5 cm H₂O of iPEEP—action: reduce the dCPAP until the desired total E-time pressure of 20 cm H₂O is attained).

Ventilation goal‡: The PaCO₂ required to sustaining a pH ≥7.15. Consider checking arterial blood gases or end-tidal CO₂ concentration 15 to 20 minutes after every change in settings.

Ventilation strategy sequence: Lower H_{Fr}, shorten I-time and increase RR; any change to improve ventilation may impair oxygenation:

1. Decrease the H_{Fr} in 60 to 120 cycles/min increments to no less than a 240 cycles/min. If inhalation injury occurs, keep ≥480 cycles/min.
2. Reduce I-time in 0.5 seconds increments to no less than a 1-second minimum. Keep E-time the same and, thus, increase the respiratory rate.
3. Increase PIP in 2 cm H₂O increments to no more than 35 cm H₂O.
4. Increase E-time in 0.5-second increments to no more than 3 to 4 seconds: Remember that increasing the E-time will decrease RR unless I-time is simultaneously shortened.
5. Decrease CPAP in 2-cm increments to what is deemed clinically appropriate to ensure oxygenation.
6. Do cuff deflations in 5-cm H₂O increments: cuff deflation is performed by increasing PIP by 5 cm H₂O and then releasing cuff pressure until PIP decreases by 5 cm H₂O. Repeat deflation as needed until goal is attained. Reconsider a deflation of more than 10 cm H₂O total.

* Increase high frequency rate and CPAP then lengthen I-time then shorten E-time then increase PIP. If transitioning from conventional ventilation to HFPV then match the VDR-4 mean airway pressure to that of the conventional ventilator using a test lung.

† Consideration should be given to adjunctive therapies to improve oxygenation (eg, inhaled nitric oxide and prostacyclin) or alternative modes (eg, HFOV). If adjunctive therapies do not help, then plans for venovenous ECMO should be implemented.

‡ Follow the reverse of the ventilation sequence if respiratory alkalosis develops—however, start at ventilation goal sequence 1 not at sequence 6 (eg, sequence 1. Increase the H_{Fr} in 60 to 120 cycles/min increments to no more than 720 cycles/min).

FiO₂, fraction of inspired oxygen; PIP, peak inspiratory pressure (use the pressure as registered by the aneroid manometer on top of the VDR); oCPAP, oscillatory continuous positive airway pressure (CPAP); dCPAP, demand CPAP; I-time, inspiratory time; E-time, expiratory time; H_{Fr}, high frequency rate (cycles/minute); max, maximal frequency; min, minimal E-time permitted.

pliance of 20 ml/cm H₂O with a high-fixed resistance of 10 cm H₂O s/L) to 457 ml (SD ± 102 ml; 95% CI of 426–488 ml at a relatively high lung compliance of 60 ml/cm H₂O with a fixed resistance of 7.5 cm H₂O s/L) which, in a 70-kg man, approximates ARDSNet goals (ie, 4.7–6.5 ml/kg; unpublished observation). It is interesting to note that animal model studies showing an equivalence in outcomes between HFPV and conventional low Vt ventilation used a similar 10 to 15-cm H₂O pressure gradient approach.^{16,17} At our institution, real-time confirmation of algorithm-directed Vt is obtained through the use of the previously described flow sensor. By using flow sensor measurements as a guide, clinicians can also deviate from the protocol as long as the measured Vt meet ARDSNet specifications.

Ventilator “liberation” is performed in reverse order once the underpinning disease state and ARDS have begun to recede. We often quantify the rapid

shallow breathing index during the course of daily spontaneous breathing trials to prognosticate the chance of successful extubation. This is accomplished by either simply inserting an in-line pneumotachograph or changing over to a ventilator that allows for minute ventilation measurement.

HFPV ADJUNCTS

Supplemental Ventilation Approaches: Partial Endotracheal Tube Cuff Deflation

Several authors have found that an endotracheal tube cuff deflation may facilitate the removal of tracheal and bronchial debris. A partial or complete endotracheal tube cuff deflation may also permit an additional means of CO₂ escape.^{47,48} Studies suggest that HF ventilation creates a CO₂ release response whereby oxygen-enriched gas from the ventilator flowing through the cen-

ter of the airway lumen is encircled by a countercurrent airway wall-based outflow of CO₂-replete gas.^{47,48} Although intuitive and clinically appealing, simple cuff deflation may also incur a penalty.

Because of a probable design flaw, the VDR amplifier assembly allows for spontaneous ventilation through the use of an “inspiratory fail-safe” flap-valve.¹⁴ Cuff deflation may lead to a pressure drop within the flow amplifier device, causing the latter valve to stay open. This breach has been shown to result in the entrainment of ambient 0.21 FiO₂ air. In addition, we have found that airway pressure is dissipated around the deflated cuff. Unfortunately the PIP, which is measured by a pressure transducer further mounted back at the ventilator circuit, does not reflect the actual airway pressure in this setting. Clinicians may have the device set to deliver a PIP of 30 cm H₂O but, depending on the extent of cuff deflation, $\leq 50\%$ of the registered pressure actually reaches the patient’s distal airways. Thus, the introduction of a cuff deflation imparts both a decrease in FiO₂ and airway pressure, which may lead to precipitous hypoxia in ARDS patients.^{47,48} HFPV studies that use cuff deflation as part of their protocol must also be interpreted in light of our *in vitro* findings, as their reported peak airway pressure and Paw data may have significantly overestimated the actual pressure delivered to the carina.

We have found that careful HFPV setting selection attains clinically sufficient ventilation in the vast majority of cases, often with the acceptance of permissive hypercapnia in the appropriate setting. This has resulted in the infrequent use of adjunctive cuff deflation. When deemed clinically necessary (eg, severe acidotic states), no more than a 5 to 10-cm H₂O cuff depressurization is used (Table 2 for instructions). As a final note on this topic, cuff deflation should not be dismissed from the HFPV armamentarium. Our *in vitro* studies have confirmed that cuff deflation may attenuate airway pressure and Vt delivery by permitting airflow to escape around the cuff to the ambient environment. In essence, cuff deflation may provide a previously unappreciated form of lung protection. We are conducting a pilot protocol to verify the gas exchange effects of a partial or complete cuff deflation.

Supplemental Oxygenation Approaches

Inhaled nitric oxide and prostacyclin could potentially be used in tandem with HFPV to enhance oxygenation. Nonetheless, there are no human or animal model studies that have explored HFPV in combination with either agent. Notably, we recently cared for a patient with an isolated chemical inhalation injury, which eventuated in ARDS.⁴⁹ The patient’s oxygen-

ation status failed to respond to a trial of HFPV at high-level settings. Administration of continuously nebulized prostacyclin led to an improvement in arterial oxygen tension within minutes of administration. We have recently replicated these results in a burn patient with severe inhalation injury (unpublished observation).

Basic Ventilator Adjuncts

For aerosol delivery, our mainstay device has been a low dead space, vibrating mesh-based system, which has shown some promise during oscillatory ventilation.⁵⁰ If the viscosity of the nebulized solution requires a jet nebulizer (eg, prostacyclin), then a low flow method may be used. Whatever the case, the aerosol device is always interposed between the amplifier and the endotracheal tube. Vigilance is required when adding any apparatus to the circuit as the increased dead space effect may undermine HF Vt magnitude, reduce effective ventilation, and produce elevated CO₂ levels. As a compensatory measure, we will often decrease the HF rate immediately after placement.

Finally, humidity-moisture exchangers or heated humidification are considered standard-of-care means of conditioning hospital gas flow during mechanical ventilation. A recent study demonstrated that the unique flow amplifier design and/or often-used heated-wire circuits may compromise gas flow heating and humidification.⁵¹ These findings have an even more poignant clinical relevance in burn populations. In the heated rooms commonly encountered in burn units, heated-wire humidifier circuits have been repeatedly found to significantly reduce the quantity of humidified gas delivered to the patient.³⁵ Thus, the hallmark friable and denuded airway mucosa seen with combustion product inhalation may endure additional harm from exposure to relatively dry ventilator breaths. Based on these results, we recommend the use of a nonheated wire active humidification system with gas flow derived from the jet nebulizer included in the VDR circuitry.

Spontaneous Ventilation During HFPV

Our experience has exposed two potential problems with HFPV. Because the ventilator implements a single HF flow to sustain a single inspiratory-phase PIP and expiratory-phase CPAP, the device can only partially compensate for spontaneous flow or pressure demands from the adult patient. As a result, both spontaneous respiration and coughing can frequently distort an HFPV waveform (Figure 5). Preliminary sensor readings also suggest that the HFPV flow rate (not to be confused with HF rate) may be less than

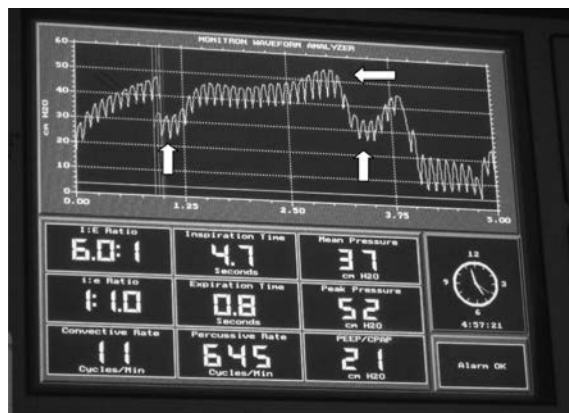


Figure 5. Pressure-time illustration of spontaneous breathing during high-frequency percussive ventilation (HFPV). The patient was set to a 4.7-second inspiratory time and 0.8-second expiratory time. The pressure-time curve shows negative- (vertical arrows) and positive-pressure (horizontal arrow) fluctuations resulting from spontaneous patient effort. Despite the peak inspiratory pressure being set to 35 cm H₂O, the pressure exceeded 50 cm H₂O or descended to below 25 cm H₂O during the course of a single breath.

the preferred 60 to 80 L/min used with conventional modes (Figure 1D). Inadequate compensation for patient effort can increase a given patient's work-of-breathing culminating in patient-ventilator dyssynchrony. Only additional selective study will clarify the presence, extent, and clinical impact of these concerns in adults.⁵² With the judicious use of intravenous sedation-analgesic combinations (usually fentanyl with the individual or combined use of midazolam and propofol and/or dexmedetomidine), patient-ventilator dyssynchrony has been infrequent and has not led to the discontinuation of HFPV at our institution. Importantly, HFPV-permissive spontaneous ventilation can be anticipated to improve end-organ perfusion and gas exchange, and as such, the avoidance of intravenous paralytic agents should be encouraged.⁴⁵

CONCLUSION

The underlying technology and practical approaches to HFPV continue to undergo evolution. With these improvements, we anticipate that HFPV will find a larger role in the care of select mechanical ventilation-dependent populations. The recent ability to measure V_t endows HFPV with the unique capacity to exploit both high- and low-frequency ventilation in order to favorably influence gas exchange while still adhering to a lung-protective, low-V_t ventilation strategy. Nevertheless, an additional prospective study is required to

define the optimal means of using HFPV in the broader scheme of acute lung and inhalation injury.

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