

Measurement of Pulsatile Tidal Volume, Pressure Amplitude, and Gas Flow During High-Frequency Percussive Ventilation, With and Without Partial Cuff Deflation

Patrick F Allan MD, Jefferson R Thurlby MD, and Gregory A Naworol RRT

OBJECTIVE: With a high-frequency percussive ventilator and a mechanical lung model, to measure tidal volume (V_T), pulsatile pressure amplitude (difference between peak and nadir pulsatile pressure [ΔP]), and mean airway pressure (\bar{P}_{aw}) at various pulsatile frequencies, pulsatile inspiratory-expiratory ratios (I:E_p), and pressures (measured at the interface between the pulse-generator and the endotracheal tube [P_{vent}]). **METHODS:** With the endotracheal tube inside an artificial trachea, we manipulated the high-frequency percussive ventilation settings and adjuncts, including pulsatile frequency, I:E_p, and P_{vent} by manipulating pulsatile flow. We also studied the effects of partially deflating the endotracheal tube cuff. We measured \bar{P}_{aw} , pulsatile pressure amplitude at the carina (ΔP_c), and pulsatile V_T at the carina. With the cuff partly deflated, we measured the fraction of inspired oxygen (F_{IO_2}) in the gas efflux above and below the cuff. **RESULTS:** Increasing the pulsatile frequency from 300 cycles/min to 600 cycles/min and changing the I:E_p from 1:3 to 1:1 significantly reduced V_T ($p < 0.001$). \bar{P}_{aw} and ΔP_c were unaffected by the change in pulsatile frequency or I:E_p, except when we did not preserve the pulsatile flow. The measured V_T range was from 19.1 mL (at 600 cycles/min) to 47.3 mL (at 300 cycles/min). Partial cuff deflation did not significantly reduce \bar{P}_{aw} or ΔP_c , but it did significantly reduce V_T and F_{IO_2} . **CONCLUSION:** During high-frequency percussive ventilation, the pulsatile frequency is inversely related to V_T . Partial cuff deflation reduces the delivered F_{IO_2} . *Key words:* volumetric diffusive ventilation, volumetric diffusive respiration, high-frequency percussive ventilation, high-frequency ventilation, tracheal tube, cuff leak. [Respir Care 2007;52(1):45–49]

Introduction

High-frequency percussive ventilation (HFPV) is used extensively in burn units for severe inhalation injury and as an advanced ventilator mode for patients with acute respiratory distress syndrome who fail conventional ventilation.^{1–11} HFPV can be viewed as a hybrid ventilation mode in which a high-frequency (100–900 cycles/min) pulsatile waveform is integrated with a lower-frequency (5–30 cycles/min), conventional, time-cycled, pressure-limited breath with a phasic waveform (Fig. 1). Superimposition of the pulsatile waveform at higher frequencies (300–900 cycles/min) on a conventional pressure-cycled breath is thought to promote additional oxygen diffusion, while at lower frequency (100–300 cycles/min) convective alveolar ventilation and airway mucociliary clearance are enhanced.^{11–13} In addition, with high-frequency breath-stacking, HFPV may maintain airway pressure in the presence of large air leaks. With that in mind, partial deflation

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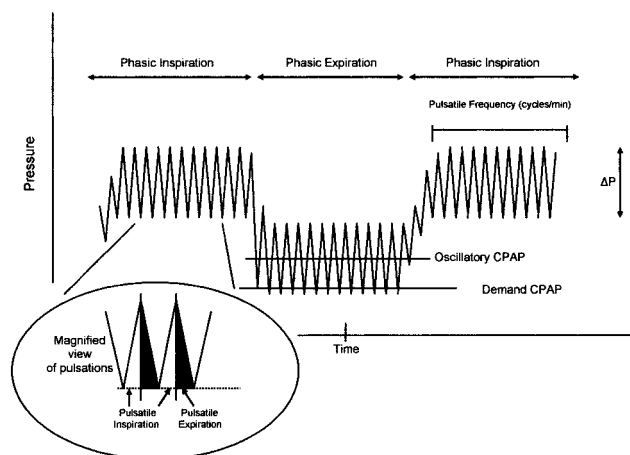


Fig. 1. High-frequency percussive waveform superimposed on a conventional inspiration and expiration. ΔP = pulsatile pressure amplitude. CPAP = continuous positive airway pressure.

of the endotracheal tube (ETT) cuff has been recommended as an adjunct to assist in ventilation and secretion clearance during HFPV.^{11,12} However, there has been no detailed study of the effects of partial cuff deflation during HFPV on the characteristics of the high-frequency waveform and gas flow.

In HFPV, the air pulses/percussions are created by a device called a phasitron, which has a component that slides back and forth to generate a high-frequency pulsatile flow, in which the frequency and amplitude are conjoined or coupled (Fig. 2).^{11,12} Increasing the pulsatile frequency or changing the pulsatile inspiratory-expiratory ratio ($I:E_p$) from 1:3 to 1:1 may decrease the pulsatile pressure amplitude or pulsatile flow, and vice versa.¹¹⁻¹³ The additional tidal volume (V_T) generated by the pulsatile waveform is thought to be less than the anatomical dead space.¹¹⁻¹³ However, to our knowledge no study has directly isolated and measured the pulsatile volume or investigated the interaction between HFPV settings and pulsatile V_T , mean airway pressure (\bar{P}_{aw}), and pulsatile pressure amplitude (difference between peak and nadir pulsatile pressure [ΔP]).

To determine the relationship between the various aspects of the HFPV waveform, we adjusted the HFPV settings of pulsatile frequency, $I:E_p$, and the internal pressure measured by the HFPV at the interface of the ETT and the HFPV circuit (P_{vent}) to study the behavior of V_T , ΔP , and \bar{P}_{aw} . We also examined the effects of partial cuff deflation on distal airway pressure, V_T , and the fraction of inspired oxygen (F_{IO_2}) distal and proximal to the cuff in the artificial trachea.^{11,12}

Methods

An HFPV device (VDR-4 [volumetric diffusive respirator], Percussionaire, Sandpoint, Idaho) was attached to a

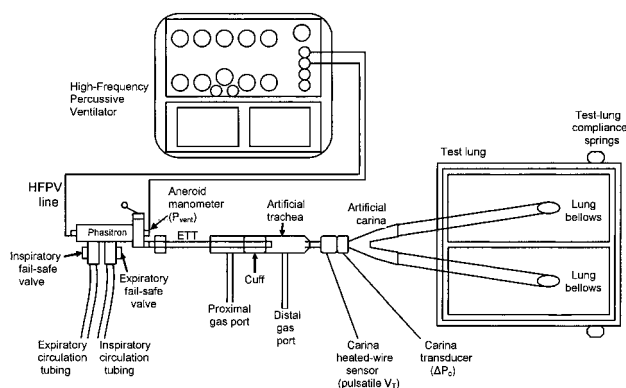


Fig. 2. Schematic of our setup with a high-frequency percussive ventilator (HFPV) and test lung. Not shown are the accessory flow tubing, expiratory circuit, or 1-L anesthesia reservoir bag. ETT = endotracheal tube. P_{vent} = pressure measured at the interface between the pulse-generator and the endotracheal tube. Pulsatile V_T = pulsatile tidal volume. ΔP_c = difference between cyclic pulsatile peak and nadir pressure measured at the carina.

test lung (5600i, Michigan Instruments, Grand Rapids, Michigan) through an artificial trachea intubated with an 8.0-mm cuffed ETT (see Fig. 2). The artificial trachea is composed of 0.5-cm thick noncompliant plastic tubing, 22 cm in length and 9 cm inner diameter. The test lung was set to a compliance of 0.03 L/cm H_2O . For all the experiments, the HFPV settings were maintained at a constant P_{vent} of either 20 cm H_2O or 40 cm H_2O , by adjusting pulsatile flow. Similar separate trials were also performed without adjusting the pulsatile flow. The phasic I:E was set with full "counterclockwise" inspiratory time while the expiratory time was turned off. The HFPV system includes an aneroid manometer that measures the pressure within the HFPV circuit between the phasitron and the attachment to the ETT (Fig. 3).

The phasic I:E represents the conventional inspiratory and expiratory times upon which the pulsatile frequency is superimposed (see Fig. 1). To isolate and focus on the interaction of only the high-frequency aspects of the pulsatile volume, ΔP , and \bar{P}_{aw} , we turned off the convective pressure rise, oscillatory/demand continuous positive airway pressure, and the ventilator's nebulizer function.

The artificial trachea has 2 gas sampling ports, 5 cm proximal and distal to the ETT cuff (see Fig. 2). We measured F_{IO_2} and gas balance (ie, nitrogen and carbon dioxide) with a gas analyzer (Datex-Ohmeda S/5, GE Healthcare, Waukesha, Wisconsin) at both the sampling ports. F_{IO_2} on the HFPV was set to 1.0, which was externally verified with the gas analyzer.

The cuff was either kept fully inflated or set to partial cuff deflation, which was implemented by decreasing P_{vent} by 5 cm H_2O (from the 40-cm H_2O inflated-cuff baseline). We gradually deflated the cuff and then restored P_{vent} by increasing pulsatile flow.

To determine the origin of entrained air, we sequentially occluded the expiratory and inspiratory fail-safe valves and the circulation tubing to prevent ambient air from being entrained into the circuit, while retaining the 1.0 F_{IO_2} gas flow directly delivered by the high-frequency flow-interrupter to the phasitron (see Fig. 3). Occluding these circuit apertures allowed us to sequentially analyze ambient-air entrainment and delivered gas.

We measured exhaled V_T , \bar{P}_{aw} , and the high-frequency pressure amplitude with a neonatal high-frequency heated-wire sensor (Florian, Acutronic Medical Systems, Hirzel, Switzerland) placed in-line, distal to the tip of the ETT (location equivalent to the carina). Airway pressure amplitude and \bar{P}_{aw} were measured via the co-housed Florian manometer, which measured airway pressure via an in-line connection at the carina (ΔP_c). We studied pulsatile frequencies of 300 cycles/min and 600 cycles/min, and I:E_p settings of 1:1 and 1:3. Changing the I:E_p changes the registered pulsatile frequency, so when we changed the I:E_p we also changed the pulsatile frequency setting to maintain the original pulsatile frequency.

Each experiment was performed in triplicate. The data are expressed as mean \pm standard deviation values. We used analysis of variance testing for repeated measures to assess the differences at 300 cycles/min and 600 cycles/min. Differences were considered significant when $p < 0.05$.

Results

Effects of Pulsatile Frequency and I:E_p on V_T , \bar{P}_{aw} , and ΔP

At a set I:E_p of 1:3 and a P_{vent} of 20 cm H₂O, increasing the pulsatile frequency from 300 cycles/min to 600 cycles/min significantly reduced V_T , from 47.3 ± 0.76 mL at 300 cycles/min to 19.1 ± 1.94 mL at 600 cycles/min ($p < 0.001$). Increasing the pulsatile frequency from 300 cycles/min to 600 cycles/min caused no statistically significant change in ΔP_c or \bar{P}_{aw} . Adjusting the I:E_p to 1:1 while maintaining the same P_{vent} significantly reduced V_T , compared to the I:E_p 1:3 baseline at the same respective frequencies; the measured mean volumes were 43.2 ± 0.35 mL at 300 cycles/min ($p < 0.001$) and 17.8 ± 0.05 mL at 600 cycles/min ($p = 0.027$). Mean ΔP_c and \bar{P}_{aw} were not significantly different at I:E_p 1:1 than at I:E_p 1:3.

We reasoned that maintaining an artificially constant P_{vent} by manipulating the pulsatile flow might confound the interpretation of pulsatile pressure and volume, so in a separate trial we evaluated the impact of increasing the pulsatile frequency without adjusting the pulsatile flow. In this trial we found a significant reduction in mean V_T at 300 cycles/min: 47.4 ± 0.61 mL with pulsatile flow main-

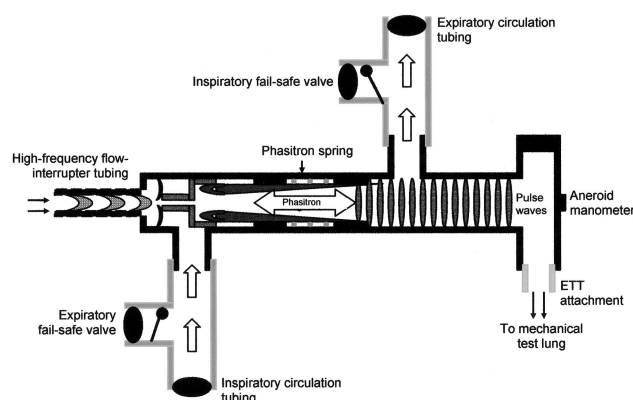


Fig. 3. Schematic of the pulse-generator (phasitron), circulation tubing, and fail-safe valves. The open arrows denote airflow through the circuit. The bidirectional arrow denotes the sliding movement of the phasitron component that creates the gas pulses. The black ovals indicate the ports that we occluded during the tests. ETT = endotracheal tube.

tenance, versus 42.9 ± 0.40 mL without pulsatile flow maintenance ($p < 0.001$), whereas there was no significant difference from the pulsatile-flow-preserved P_{vent} trials in the observed ΔP_c at 300 cycles/min or 600 cycles/min or in V_T at 600 cycles/min. However, \bar{P}_{aw} did significantly decrease with increasing frequency at both I:E ratios (3.25 ± 0.75 cm H₂O, $p < 0.001$) relative to the pulsatile-flow-preserved test.

Manipulation of P_{vent}

Increasing P_{vent} from 20 cm H₂O to 40 cm H₂O at a pulsatile frequency of 300 cycles/min increased \bar{P}_{aw} by 20 ± 0.0 cm H₂O and increased ΔP_c by 20.3 ± 5.5 cm H₂O ($p < 0.001$). V_T was significantly reduced at P_{vent} of 40 cm H₂O, at both 300 cycles/min ($V_T = 39.1 \pm 0.06$ mL, $p < 0.001$) and 600 cycles/min ($V_T = 15.0 \pm 0.91$ mL, $p < 0.001$) (Fig. 4). At the same high P_{vent} , adjusting the I:E_p to 1:1 did not significantly affect ΔP_c or \bar{P}_{aw} , relative to the I:E_p 1:3 setting. However, V_T at I:E_p of 1:1 was significantly reduced at 300 cycles/min ($V_T = 34.2 \pm 0.404$ mL, $p < 0.001$) and 600 cycles/min ($V_T = 13.8 \pm 0.70$ mL, $p = 0.007$). At the I:E_p 1:3 setting, increasing the pulsatile frequency from 300 cycles/min to 600 cycles/min significantly decreased V_T , from 38.4 ± 1.5 mL to 15.3 ± 2.2 mL ($p < 0.001$) but did not significantly change \bar{P}_{aw} or ΔP_c .

In a separate trial we evaluated the impact of increasing the pulsatile frequency without adjusting the pulsatile flow. We found no significant difference in ΔP_c or V_T , compared to the P_{vent} trials in which we preserved the pulsatile flow. As in the other trial, \bar{P}_{aw} significantly decreased (4.0 ± 0.0 cm H₂O) when we increased the frequency, compared to when the pulsatile flow was preserved

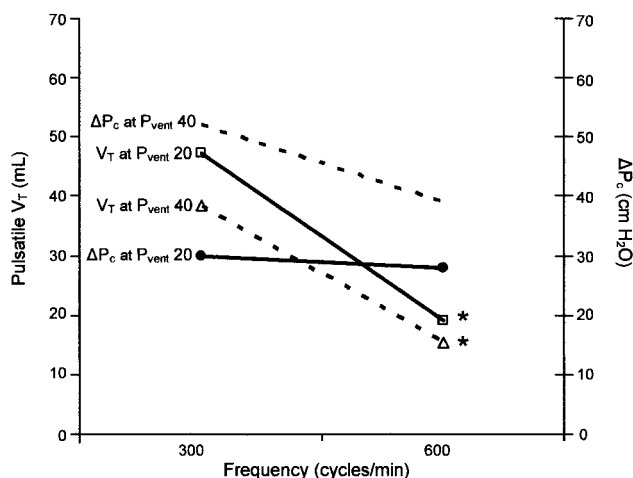


Fig. 4. Measured pulsatile tidal volume (V_T) and pulsatile pressure amplitude at the carina (ΔP_c) at P_{vent} (the pressure measured at the interface between the pulse-generator and the endotracheal tube) values of 20 cm H₂O and 40 cm H₂O, at 300 cycles/min and at 600 cycles/min. The pulsatile inspiratory-expiratory ratio was set at 1:3. * = Statistically significant reduction in measured V_T between 300 cycles/min and 600 cycles/min.

($p < 0.001$), but this occurred only when the frequency was increased with I:E_p 1:3: not at I:E_p 1:1.

Partial Cuff Deflation

Partial cuff deflation (5 cm H₂O P_{vent} decrease from 40 cm H₂O, followed by an increase in pulsatile flow to reestablish a P_{vent} of 40 cm H₂O) did not significantly change \bar{P}_{aw} or ΔP_c , relative to full cuff inflation, at either of the frequencies. V_T significantly decreased at 300 cycles/min ($V_T = 36.9 \pm 0.25$ mL, $p < 0.001$), but not at 600 cycles/min. During partial cuff deflation, neither changing the I:E_p from 1:3 to 1:1 nor increasing the pulsatile frequency significantly affected carinal \bar{P}_{aw} or ΔP_c , relative to the full-cuff-inflation baseline.

With full cuff inflation the proximal and distal port F_{IO_2} values were 0.21 and 1.0, respectively. This suggests that there was no entrained ambient air with the cuff inflated or efflux of gas around the inflated cuff to the proximal port. After cuff deflation the proximal and distal F_{IO_2} were 0.87 ± 0.01 and 0.87 ± 0.01 , respectively. This suggests that with partial cuff deflation, ambient air was being entrained into the test lung, either around the deflated cuff or through the circuit. When the oxygen analyzer attachment was substituted for the HFPV circuit manometer port attachment, the measured F_{IO_2} was 0.87 ± 0.01 . This suggests direct ambient-air entrainment into the HFPV circuit, and not from peri-cuff gas sampling.

To further study the origin of the entrained air, we sequentially occluded the expiratory and inspiratory fail-safe valves and the circulation tubing to prevent ambient-air

entrainment into the circuit during partial cuff deflation (see Fig. 3). Occluding the inspiratory fail-safe valve decreased P_{vent} by 15 cm H₂O. The F_{IO_2} measured at the distal and proximal ports after occluding the inspiratory fail-safe valve was 0.99 ± 0.0 . Occluding the expiratory fail-safe valve did not change airway pressure or oxygen concentration. The circuit was then restored, with the exception of a disconnection (not an occlusion) of the inspiratory circulation tubing, and exposure of the same port to F_{IO_2} of 0.21 from ambient air. The proximal and distal port F_{IO_2} decreased to 0.48 ± 0.02 and 0.56 ± 0.01 , respectively.

With occlusion of the inspiratory circulation tubing and the inspiratory fail-safe valve, the proximal and distal port F_{IO_2} increased to 0.81 ± 0.08 and 0.93 ± 0.04 , respectively. These findings are consistent with gas sampling through the oxygen reservoir contained within the expiratory circulation tubing (see Fig. 3). In summary, the "open circuit" effect of the inspiratory fail-safe valve appeared to be responsible for ambient-air entrainment intended to maintain aneroid manometer pressure during partial cuff deflation and, in so doing, compromised the delivered F_{IO_2} .

Discussion

Given the mechanical properties of the phasitron, the pulsatile frequency and amplitude are thought to be coupled.¹¹⁻¹² However, a significant increase in \bar{P}_{aw} and ΔP_c , implemented by increasing the P_{vent} , significantly reduced V_T (see Fig. 4), which suggests an uncoupling of pulsatile V_T and any resultant change in \bar{P}_{aw} or ΔP_c . Similarly, we found dissociation between ΔP and V_T as a function of increasing pulsatile frequency (see Fig. 4). This is probably because of the frequency and flow-dependent constituents of impedance—namely, flow-dependent resistance, frequency-dependent inertance, and lung compliance. For instance, by increasing pulsatile flow to sustain the elevated P_{vent} goal, V_T is compromised by the resultant increase in flow-dependent resistance. Conversely, ΔP is relatively preserved because of a distal decrease in lung compliance caused by the increase in \bar{P}_{aw} . On the other hand, with an increase in pulsatile frequency inertance effects then attenuate distal V_T and, possibly through an increase in airflow and thus flow-dependent resistance, prevent a similarly significant decrease in distal ΔP . Inertance represents the limits imposed by high-frequency volume accelerations; it is expressed as a proportionality constant of the difference in pressure across the mass of the airway and surrounding parenchyma and the mass of the administered gas. It also appears that an increase in pulsatile frequency may significantly compromise \bar{P}_{aw} , which implies that the bias flow and breath-stacking mechanisms

employed by HFPV may be insufficient to maintain \bar{P}_{aw} in the absence of augmented pulsatile flow.

Increasing the pulsatile flow compensates for the loss of pressure associated with partial cuff deflation. Given that pulsatile flow is drawn from the high-frequency flow-interrupter in the HFPV device, the reduced delivered F_{IO_2} was an unexpected and profound result. More revealing was that the HFPV circuit appeared to be the main, if not the only, source of ambient-air entrainment. The ambient-air entrainment may be a consequence of the loss of reflected \bar{P}_{aw} against the phasitron. With the reduction in back-pressure, the HFPV circuit may mechanically respond by permitting ambient-air supplementation to restore or compensate for this pressure change. As a caveat, the reduction in F_{IO_2} may also reflect the extent to which our ETT/artificial-trachea model permitted peri-cuff airflow, which would be anticipated to vary depending on the ratio of ETT or ETT cuff to trachea diameter and luminal airflow properties. The potential deleterious clinical effects of compromised F_{IO_2} suggest reconsideration of partial cuff deflation in patients who are difficult to oxygenate with a fully inflated cuff. We are currently exploring corrective options to restore distal F_{IO_2} delivery.

Limitations to our study include the lack of multi-point analog-to-digital-conversion sampling technology, which may improve pulsatile V_T and pressure amplitude resolution.¹⁴ Lung modeling may also (through its intrinsic mechanical properties) magnify or attenuate pulsatile flow changes, limiting extrapolation of our results. Our trials also did not explore the effects of HFPV-unique and advantageous programming to include expiratory pressure and oscillation and convective pressure rise, among others, as this experiment focused only on the high-frequency waveform characteristics of HFPV.

To the best of our knowledge this is the first study to directly measure high-frequency pulsatile V_T during HFPV, to establish the fundamental effects of HFPV settings adjustments on high-frequency waveform features, and to analyze those waveform features and gas flow during partial cuff deflation. Understanding the means of measuring and manipulating high-frequency pulsatile V_T sets a foundation for studying the optimal method of employing HFPV for lung-protective (low- V_T) ventilation in acute respiratory distress syndrome.

Conclusion

During HFPV, pulsatile frequency is inversely related to V_T . Partial cuff deflation caused an unanticipated reduction of delivered F_{IO_2} ; this requires clinical study.

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