Corrective Measures for Compromised Oxygen Delivery During Endotracheal Tube Cuff Deflation With High-Frequency Percussive Ventilation

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OBJECTIVE: To determine the effect of endotracheal-tube cuff deflation on airflow and $F_{IO_2}$ during high-frequency percussive ventilation (HFPV), and explore methods of correcting the cuff-deflation-associated decrease in mean airway pressure and $F_{IO_2}$ at the carina. METHODS: Using a mechanical lung model in our respiratory research laboratory, we measured circuit pressure near the connection to the endotracheal tube ($P_{vent}$), mean airway pressure ($P_{aw}$), pulsatile tidal volume ($V_T$), and $F_{IO_2}$ at the artificial carina. During cuff deflation we manipulated the pulsatile frequency, pulsatile flow, and the HFPV integral nebulizer. We then assessed 4 methods of correcting the decreased $F_{IO_2}$ and airway pressure during cuff deflation: (1) oxygen delivery at the inspiratory fail-safe valve, (2) oxygen delivery at the T-piece between the HFPV and the endotracheal tube, (3) continuous activation of the HFPV’s integral nebulizer, and (4) oxygen insufflation into the suction channel of the endotracheal tube. RESULTS: Cuff deflation reduced $P_{vent}$, $P_{aw}$, pulsatile $V_T$, and $F_{IO_2}$. Increasing the pulsatile flow and decreasing the pulsatile frequency further reduced $F_{IO_2}$ during cuff deflation. Injecting supplemental oxygen at the inspiratory fail-safe valve provided the best $F_{IO_2}$ increase. Injecting oxygen at the T-piece provided the second best $F_{IO_2}$ increase. Continuous activation of the integral nebulizer provided the third best $F_{IO_2}$ increase. Oxygen insufflation to the suction channel was least effective in correcting the $F_{IO_2}$ decrease caused by cuff deflation. CONCLUSION: Cuff-deflation-associated $F_{IO_2}$, $P_{aw}$, and pulsatile $V_T$ compromise can be partially corrected by any of the 4 methods we studied. Injecting supplemental oxygen at the inspiratory fail-safe valve is the most effective method.

Key words: percussive ventilation, high-frequency ventilation, endotracheal tube, cuff deflation. [Respir Care 2007;52(3):271–277]

Introduction

High-frequency percussive ventilation (HFPV) uses a device called a phasitron to create high-frequency pulses of gas. The key component of the phasitron is a flow-driven sliding Venturi device (Fig. 1) that overlays a high-frequency pulsatile waveform (100–900 cycles/min) on a lower-rate (5–30 cycles/min), conventional, time-cycled, pressure-limited “phasic” waveform breath. Patients with acute respiratory distress syndrome, whose compromised oxygenation and ventilation may be recalcitrant to conventional ventilation modes, may benefit from this combination of high-frequency and phasic conventional breaths. However, severe progressive acute respiratory distress syndrome may result in physiologic dead space and carbon dioxide accumulation that exceeds HPFV’s capacity to restore clinically acceptable ventilation.

Endotracheal-tube (ETT) cuff deflation during HPFV has been recommended as a means of enhancing carbon dioxide clearance and might have other benefits (Table 1). However, a lack of literature examining the objective gas flow and high-frequency waveform changes that can occur during cuff deflation limits our comprehen-
The purpose of this investigation was 2-fold: to determine the effect of cuff deflation on HFPV waveform and gas flow, and to explore methods of correcting the protocol-disclosed cuff-deflation-associated decrement in fraction of inspired oxygen (FIO2).

Methods

We connected an HFPV device (VDR-4 [volumetric diffusive respirator], Percussionaire, Sandpoint, Idaho) to a mechanical test lung (5600i, Michigan Instruments, Grand Rapids, Michigan) through an artificial trachea intubated with an 8.0-mm inner diameter (10.8-mm outer diameter) cuffed ETT (Hi-Lo, Mallinckrodt, St Louis, Missouri) (Fig. 2). The artificial trachea, intended to approximate the tracheal diameter of an average adult patient,6–8 was composed of 2-mm thick noncompliant plastic tubing (length 26 cm, outer diameter 22 mm, inner diameter 18 mm). The ETT cuff was completely deflated to allow gas flow around the ETT; if the cuff is only partially deflated, there can be confounding effects on mean airway pressure or altered waveform transmission and gas flux.

HFPV Gas Flow and Waveform Experiments

The aneroid manometer (see Fig. 1) measures the pressure (Pvent) within the HFPV circuit near the attachment to the ETT. HFPV pulsatile flow was set to attain a Pvent of 20 cm H2O. As will be discussed in the Results section, cuff deflation markedly reduced Pvent, mean airway pressure (Paw, measured at the artificial carina), high-frequency tidal volume (VT, measured at the artificial carina), and FIO2 (measured at the artificial carina); therefore all the cuff deflation experiments were conducted with the pulsatile flow set to maximum. HFPV settings were chosen to isolate the sustained effects of changes in the high-frequency waveform and gas flow without the confounding effects of conventional breath cycling. Therefore the HFPV was set for maximum inspiratory time, and the expiratory time was turned off. Similarly, adjunctive programming settings, such as the convective pressure rise and oscillatory/demand continuous positive airway pressure were also turned off. Pulsatile frequency was set at 3 Hz or 10 Hz, at a high-frequency inspiratory-expiratory ratio of 1:3. The HFPV blender was set to deliver an FIO2 of 1.0.
Artificial airway resistance was set by the placement of resistors at the artificial carina, so that distal to the ETT tip the total airway resistance was 5 cm H₂O/L/s. Test lung compliance was set to 0.05 L/cm H₂O.

Pₜₜ was measured by a high-frequency neonatal heated-wire sensor (Florian, Acutronic Medical Systems, Hirzel, Switzerland) placed in-line at the artificial carina (distal to the tip of the ETT). FIO₂ at the artificial carina was measured by a gas analyzer (Datex-Ohmeda S/5, GE Healthcare, Waukesha, Wisconsin) with an in-line attachment.

**Redressing the Cuff-Deflation-Associated Decrement in Oxygen Delivery**

The first portion of the experiment disclosed cuff-deflation-associated reductions in carina FIO₂ and airway pressure, so we examined integral and adjunctive (ie, external to the HFPV circuit) methods to augment carina FIO₂ and airway pressure. For all the experiments involving adjunctive devices, the pulsatile flow was set to maximum, for 2 reasons: (1) titrating pulsatile flow to a preset P vent while using an adjunctive gas delivery device could confound determination of the independent effects on gas flow and airway pressure of each of the adjunctive devices, and (2) given the loss of airway pressure associated with cuff deflation, it was clinically relevant to ascertain the maximum sustained airway pressure and its associated gas flow for the combination of HFPV and the examined adjunctive methods of correcting the decreased FIO₂ and pressure.

We used 4 supplemental-gas-flow techniques:

1. Set the HFPV’s integral nebulizer to full “counterclockwise” activation, which diverts gas flow from the blender gas supply to the HFPV circuit.
2. Inject oxygen at the T-piece between the HFPV circuit and the ETT (see Fig. 1).
3. Inject oxygen at the inspiratory fail-safe valve (see Fig. 1).
4. Insufflate oxygen into the suction channel of the ETT (see Fig. 2).

A flow meter (Timeter Instruments, Lancaster, Pennsylvania) was attached to a hospital wall oxygen source via approximately 1.5 m of 5-mm inner-diameter plastic tubing, either directly to the inspiratory fail-safe valve or to the T-piece between the HFPV and the ETT. The wall oxygen flow was measured with a flow sensor (VT plus, BioTek, Winooski, Vermont) interposed in the tubing between the wall flow meter and the inspiratory fail-safe valve or T-piece. Supplemental oxygen was increased in increments of 5 L/min, from 0 L/min to 70 L-per-minute (L/min).

Insufflation was performed by connecting the oxygen source to the ETT’s suction channel, which runs from the proximal end of the ETT to 0.5 cm above the cuff, where it opens onto the external surface of the ETT, and delivering oxygen at 0–15 L/min.

Figure 1 shows the phasitron and its housing and the HFPV circuitry. Gas from the HFPV blender is delivered in a high-frequency flow-interrupted manner against the sliding component, which consists of a blunt-ended conical plastic piece with a central distally expanding diameter hollow core, all contained within a specially constructed housing. The spring-loaded phasitron augments the frequency of the intermittent pulsatile flow delivered to it by superimposing additional spring-driven oscillations. The programmed intermittent airflow can be flow-regulated by the pulsatile flow dial; the higher the pulsatile flow, the more gas volume in each pulsatile breath delivered to the ETT. The pulsatile frequency is regulated by the pulsatile frequency control dial.

Pₜₜ is manipulated via (1) high-frequency breath-stacking and/or (2) adjusting the volume of gas delivered with each pulse (ie, changing the pulsatile flow). A third mechanism of augmenting gas delivery is inherent in the gas-flow properties of the sliding component of the phasitron. As gas flows from the HFPV, it encounters first the blunt and narrow core of the conical component, where the initial acceleration in gas flow to overcome the narrow orifice then decelerates as it travels through the distally widening component. This design, in theory, leads to a pressure drop across the orifice of the device, whereby further gas volume can be drawn in to be concurrently delivered with each pulse. According to the manufacturer, this mechanism is modulated by the airway pressure, frequency, and pulsatile inspiratory-expiratory ratio.

Two one-way flap valves (an expiratory and an inspiratory valve) allow for pressure release (expiratory fail-safe valve) and spontaneous-ventilation-triggered ambient-air entrainment (inspiratory fail-safe valve). The HFPV cir-
circuitry consists of standard corrugated tubing that links the inspiratory and expiratory circulation points of the phasitron housing. The tubing is interrupted by an in-line access point for a nebulizer, which draws gas from the oxygen blender. A 3-L anesthesia bag acts as an oxygen reservoir and a second one-way expiratory fail-safe valve.

All portions of the experiment were performed in triplicate. Data are reported as mean ± 1 standard deviation unless otherwise noted.

**Results**

**HFPV Gas Flow and Waveform Experiments**

During HFPV with $P_{vent}$ of 20 cm H$_2$O at 10 Hz and a blender $F_{O_2}$ of 1.0, full cuff inflation resulted in $F_{O_2}$ of 0.97 ± 0.00 at the carina, $V_T$ of 18.3 ± 0.76 mL, and $P_{aw}$ of 20.0 ± 0.00 cm H$_2$O at the carina. Cuff deflation during the same HFPV and test-lung settings reduced all the variables: $F_{O_2}$ 0.65 ± 0.00, $P_{vent}$ 8.0 ± 0.00 cm H$_2$O, $V_T$ 7.7 ± 0.62 mL, and $P_{aw}$ of 3.0 ± 0.00 cm H$_2$O. Maximizing pulsatile flow during cuff deflation permitted a $P_{vent}$ increase to 18 ± 0.00 cm H$_2$O, $V_T$ to 7.8 ± 0.22 mL, and $P_{aw}$ to 7.0 ± 0.00 cm H$_2$O. Maximum pulsatile flow, however, induced a marked reduction in carina $F_{O_2}$ to 0.55 ± 0.00. Table 2 lists the results for carina $F_{O_2}$, $P_{vent}$, and $P_{aw}$ at the 3 Hz and 10 Hz pulsatile frequency settings.

During cuff deflation, increasing the pulsatile flow further decreased mean carina $F_{O_2}$, relative to the carina $F_{O_2}$ reductions caused by decreasing pulsatile frequency (see Table 2).

**Redressing the Cuff-Deflation-Associated Decrement in Oxygen Delivery**

Attaching the in-line oxygen (set to zero flow) to the inspiratory fail-safe valve, thereby occluding the only access point for ambient-air entrainment, increased carina $F_{O_2}$ from 0.54 ± 0.00 to 0.99 ± 0.00. Injecting oxygen directly to the inspiratory fail-safe valve gave mean carina $F_{O_2}$ values of 0.95 ± 0.00 to 0.98 ± 0.00 with supplemental oxygen flow of as little as 5 L/min.

.Injecting oxygen at the T-piece between the HFPV circuit and the ET& increased carina $F_{O_2}$ from 0.53 ± 0.00 at zero supplemental oxygen flow, to 0.89 ± 0.00 at 70 L/min supplemental oxygen flow and pulsatile frequency of 3 Hz, and to 0.90 ± 0.00 at 70 L/min supplemental oxygen flow and pulsatile frequency of 10 Hz. Slope analysis found a carina $F_{O_2}$ increase of 0.03 at 3 Hz, and 0.02 at 10 Hz, for each 5-L/min increase in supplemental oxygen.

Oxygen insufflation at 15 L/min marginally increased carina $F_{O_2}$ to 0.55 ± 0.00 at 3 Hz, and to 0.56 ± 0.00 at 10 Hz.

Continuous activation of the integral HFPV nebulizer improved carina $F_{O_2}$ to 0.63 ± 0.00 at 3 Hz, and to 0.65 ± 0.00 at 10 Hz (Fig. 3).

Occluding the inspiratory fail-safe valve (ie, connecting the oxygen tube to the inspiratory fail-safe valve but delivering zero oxygen flow) markedly reduced $P_{vent}$, $P_{aw}$, and carina $V_T$, regardless of the pulsatile frequency. Figures 4 and 5 show that, relative to cuff deflation with the maximum pulsatile flow baseline, occluding the inspiratory fail-safe valve produced a 57–71% reduction in $P_{vent}$ and $P_{aw}$, from $P_{vent}$ and $P_{aw}$ baseline values of 18 cm H$_2$O and 7 cm H$_2$O, to 6 cm H$_2$O and 2 cm H$_2$O at 10 Hz, and 7 cm H$_2$O and 2 cm H$_2$O at 3 Hz.

At 10 Hz, with 5-L/min increments in supplemental oxygen (from 5 L/min to 70 L/min) there was a linear increase in $P_{vent}$ and $P_{aw}$. At 60 L/min (see Fig. 3), both $P_{vent}$ and $P_{aw}$ plateaued, and the HFPV reservoir bag inflated, and oxygen vented from the expiratory valve at both the phasitron housing and the HFPV circuitry, which further increased venting of oxygen to the ambient air.
Slope analysis at 10 Hz with supplemental oxygen flows from 5 L/min to 60 L/min showed a $P_{vent}$ increase of 1.25 cm H$_2$O and a $P_{aw}$ increase of 0.42 cm H$_2$O per 5-L/min increase in supplemental oxygen flow. Carina $V_T$ was unchanged from the valve occlusion result to 70 L/min supplemental oxygen, at both frequencies (Fig. 6). There was also a linear increase in $P_{vent}$ and $P_{aw}$ at the 3 Hz frequency but no plateau effect was noted (see Figs. 4 and 5). Reservoir-bag inflation and expiratory-valve venting was noted at 65 L/min at 3 Hz.

Injecting supplemental oxygen at the T-piece also showed a linear relationship between the oxygen flow and $P_{vent}$ and $P_{aw}$. $P_{vent}$ increased 1.07 cm H$_2$O and $P_{aw}$ increased 0.29 cm H$_2$O for each 5-L/min increment in oxygen flow at 10 Hz. There was no plateau effect in $P_{vent}$ or $P_{aw}$ at either 3 Hz or 10 Hz. Carina $V_T$ was unchanged from the valve occlusion result to 70 L/min, at both frequencies. There was neither reservoir-bag inflation nor expiratory-valve venting at 3 Hz or 10 Hz with oxygen injection at the T-piece, regardless of the supplemental oxygen flow.

Insufflation and continuous activation of the integral nebulizer minimally improved $P_{vent}$ and $P_{aw}$ over the cuff-deflation baseline (see Figs. 4 and 5).

**Discussion**

Cuff deflation lowers carina $F_{O2}$ due to ambient-air entrainment from the inspiratory fail-safe valve. Ambient-air entrainment is exacerbated by an increase in pulsatile flow and by a reduction in pulsatile frequency. Ambient air entrainment probably occurs due to a cuff-deflation-
induced reduction in HFPV circuit pressure. Hypothetically, the pressure loss from cuff deflation causes the inspiratory fail-safe valve to remain open, due to a lack of back pressure that would close the valve. Alternatively, or possibly in addition to the gas-flow changes caused by the open fail-safe valve, the increase in airflow from an increase in either pulsatile flow or gas volume per pulse at a reduced frequency may compound the pressure drop across the phasitron and increase ambient-air entrainment. In support of the primary intra-circuit origin of air entrainment, we found that only a relatively small contribution to total ambient-air entrainment occurs at the ETT cuff, as implied by the very small (0–1%) increase in carina \( F_{O_2} \) when we insufflated oxygen above the ETT cuff.

As the major source of ambient-air entrainment, the inspiratory fail-safe valve should be the optimal location to inject supplemental oxygen. In support of this theory, bias-flow supplemental oxygen injected at the inspiratory fail-safe valve maintained carina \( F_{O_2} \) at 1.0 and permitted a 22% increase in \( P_{vent} \) (approximately 22 cm H\(_2\)O at 10 Hz) in excess of those attained with HFPV with maximum pulsatile flow alone (approximately 18 cm H\(_2\)O at 10 Hz). This also translated into a potential 28% increase in \( P_{aw} \) relative to the cuff-deflation baseline at 10 Hz. Although a bias oxygen flow of 45–50 L/min permitted a restoration of \( P_{vent} \) to 20 cm H\(_2\)O at 10 Hz, the lowest acceptable gas flow is approximately 60 L/min at 10 Hz and 65 L/min at 3 Hz, because those are the flow ranges that permit inflation of the anesthesia bag, which indicates the complete oxygen flushing of ambient air from the HFPV circuit reservoir. Higher oxygen flow simply opens the expiratory fail-safe and circuit valve and vents gas to the ambient air, which explains the plateau effect of pulsatile pressure, \( P_{aw} \), and \( P_{vent} \). Intentional occlusion of the expi-
ratory fail-safe and/or circuit access points may achieve higher airway pressure, but airway edema, peri-cuff airway narrowing, or unintended cuff inflation could result in catastrophic lung hyperinflation. Similarly, though occlusion of the inspiratory fail-safe valve may provide a carinal $F_{\text{IO}_2}$ of 1.0, it also deprives the phasitron circuit of an important means to sustain and/or augment $P_{\text{vent}}$ and $P_{\text{aw}}$.

In summary, injecting oxygen at the inspiratory fail-safe valve or between the phasitron and the ETT are probably the best options for augmenting carina $F_{\text{IO}_2}$, $P_{\text{vent}}$ and $P_{\text{aw}}$ during cuff deflation.

Not only does cuff deflation reduce carina $F_{\text{IO}_2}$, the associated decrease in $P_{\text{aw}}$ may cause atelectasis or alveolar derecruitment and thus further reduce the patient’s oxygenation. Therefore our findings suggest not using cuff deflation in patients who require a high $P_{\text{aw}}$ to sustain alveolar recruitment (eg, cardiogenic and noncardiogenic pulmonary edema), at least until further investigations are done.

There are limitations in extrapolating our findings to animal models or clinical settings. A mechanical lung model does not replicate the dynamic in vivo changes in airway and lung impedance and thus may lead to poor modeling of in vivo gas-flow properties. It is possible that the aforementioned changes have prevented clinical reports of cuff-deflation-associated changes in oxygenation from being mentioned in the medical literature. However, when conducting the same protocol under high airway resistance (75 cm H$_2$O/L/s) and low mechanical lung compliance conditions (0.01 L/cm H$_2$O) there was no significant difference in the carina $F_{\text{IO}_2}$ results. Similarly, HFPV with partial cuff deflation (as opposed to full cuff deflation) significantly decreases carina $F_{\text{IO}_2}$. Respectful of these limitations, the present findings provide insight into HFPV-associated limitations to gas-flow delivery and a means of redressing circuit design or cuff-deflation-associated HFPV shortcomings.

**Conclusion**

Cuff deflation during HFPV reduces $F_{\text{IO}_2}$ as a result of pulsatile-flow/frequency-modulated ambient-air entrainment from the inspiratory fail-safe valve. Supplemental oxygen can restore $F_{\text{IO}_2}$.

**REFERENCES**