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Comparison of the Sensormedics® 3100A and Bronchotron® Transporter in a Neonatal Piglet ARDS Model

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Summary. The Sensormedics® 3100A (Cardinal Health, Dublin, OH) (HFOV) and the Bronchotron® (Percussionaire, Sandpoint, ID) (HFPV) are high-frequency ventilation devices used to support neonatal respiratory failure; however, a comparison of the devices, with respect to gas exchange at similar ventilator settings, has not been previously studied. Thus, we compared the ability of HFOV to that of HFPV to provide oxygenation and ventilation during acute lung injury in a newborn animal model. Using a saline lung lavage model, 12 neonatal piglets were randomized to initial support with either the HFOV or HFPV with settings adjusted to achieve $PaCO_2$ of 45–60 mmHg. After stabilization, ventilator settings and arterial blood gases were serially recorded for 30 min. Animals were then crossed over to the alternative device set to deliver the same V_t, MAP, and F for an additional 30 min with the same parameters recorded. We found that the ΔP needed to generate adequate V_t on HFPV (35 ± 7 cmH_2O) trended higher versus HFOV (31 ± 7 cmH_2O $P = 0.09$) when the devices were matched for V_t, F, and MAP. No significant differences in ventilation ($PaCO_2 = 50 \pm 10.7$ mmHg vs. 46 ± 10 mmHg, $P = 0.22$) or oxygenation ($PaO_2 = 150 \pm 76$ mmHg vs. 149 ± 107 mmHg, $P = 0.57$) between the devices were found. We conclude that HFPV ventilates and oxygenates as well as HFOV at equivalent ventilator settings. HFPV may require larger ΔP's to generate equivalent V_t. **Pediatr Pulmonol. 2009; 44:693–700.** © 2009 Wiley-Liss, Inc.

Key words: high-frequency ventilation; high-frequency oscillatory ventilation; high-frequency percussive ventilation; tidal volume.

INTRODUCTION

High-frequency ventilation (HFV) is commonly used to treat neonatal patients with respiratory failure as either primary therapy or rescue strategy following failure of conventional mechanical ventilation (CMV).[1] It is also commonly used for rescue therapy in older children and adults with severe hypoxic respiratory failure.[2] Common modes of HFV used in the United States are high-frequency oscillatory ventilation (HFOV) and high-frequency jet ventilation (HFJV).

Unfortunately, children whose care has escalated to HFV often cannot be safely converted back to CMV, and therefore are placed at significant risk of deterioration or death if transport is required.[3,4] HFOV has been shown in several studies to improve pulmonary outcomes in premature infants when used for initial ventilatory management of respiratory distress syndrome.[5–9] While HFOV is generally effective in improving oxygenation and ventilation, its utility in transport is limited because of its size, weight, and electromagnetic interference. Some case studies have described the use of HFJV in transport, but the bulkiness of the device makes it cumbersome for travel.[10,11] The Bronchotron® (Percussionaire, Sandpoint, ID) is a light, portable, pneumatically powered,

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Percussionaire® (Sandpoint, ID) Corporation provided a loaner Bronchotron® for use in this study. Percussionaire® had no input or influence on the contents of this article.

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pressure-limited, time-cycled, high-frequency percussive ventilator (HFPV) which is gaining favor in a number of centers for intrahospital and interhospital transport of neonates. One center recently reported their experience of 134 infants in whom 96% were successfully transported using HFPV with improvement in oxygenation, ventilation, and acid–base status during the time that the patients were on the transport ventilator.[12]

We are unaware of any studies comparing the gas exchange of HFOV to HFPV. Anecdotal transport experience led us to believe that HFPV was more efficient in gas exchange than HFOV. There is also limited data on the comparison of gas exchange at different levels of mean airway pressure (MAP) on HFOV or HFPV. Therefore, a randomized, controlled crossover study using a neonatal porcine lung injury model was performed to determine whether gas exchange was similar between the Bronchotron® HFPV and the Sensormedics® 3100A (Cardinal Health, Dublin, OH) HFOV when the devices were matched for tidal volume (V_t), frequency (F), fraction of inspired oxygen (FiO_2), and MAP and to determine whether gas exchange on each ventilator was different at two different levels of MAP.

MATERIALS AND METHODS

All procedures were carried out according to a protocol approved by the Wilford Hall Medical Center Institutional Animal Care and Use Committee.

Materials

Lung Injury Model

Fifteen-term gestation, 3- to 5-day-old, unweaned Yorkshire neonatal piglets (mean weight: 4 kg, range 3.0–5.0 kg) were used. Each study piglet was initially anesthetized with isoflurane (3.5–4.5%), intubated with a 3.5 cuffed endotracheal tube (ETT) placed under a radiant warmer and conventionally ventilated. Femoral venous and arterial catheters were placed for access and arterial blood gas analysis, respectively. Continuous sedation and paralysis were provided with fentanyl (45–90 mcg/kg/hr) and pancuronium (0.1–0.3 mg/kg/hr) infusions, respectively. Lung injury was produced with saline lavage (8 ml/kg dwells for 30 sec). Saline dwells were continued until arterial PaO_2 decreased below 60 mmHg on FiO_2 of 1.0 (average 15 ± 7 dwells per study animal). Between each dwell, injury was also induced with volutrauma and atelectatruama using the MVP-10 IMV ventilator (Cardinal Health) for 1–3 min with the following settings: PIP 13–20 cmH_2O, PEEP 0–5 cmH_2O, I-Time 0.35 sec, E-Time 0.65–1.7 sec, (RR 30–60) flow 8 lpm, and FiO_2 1.0 ($V_t = 10.3 \pm 2.1$ ml/kg). Attempts were made to keep the MVP-10 settings constant, making no adjustment for $PaCO_2$, but the ventilator settings occasionally needed to be adjusted in order to resuscitate the pig after saline lavage.

ABBREVIATIONS

ABG	Arterial blood gas
ARDS	Acute respiratory distress syndrome
C	Compliance
CMV	Conventional mechanical ventilation
ΔP	Proximal pressure amplitude
F	Frequency
FiO_2	Fraction of inspired oxygen
HFOV	High-frequency oscillatory ventilation
HFPV	High-frequency percussive ventilation
HFV	High-frequency ventilation
IMV	Intermittent mandatory ventilation
MAP	Mean airway pressure
MVI	Modified Ventilatory Index
OI	Oxygenation Index
PEEP	Positive end expiratory pressure
PIP	Peak inspiratory pressure
$PaCO_2$,	Arterial partial pressure of carbon dioxide
PaO_2	Arterial partial pressure of oxygen
R	Resistance
RR	Respiratory rate
V_t	Tidal volume

MVP 10 (Cardinal Health)

The MVP-10 is a pneumatically driven conventional mechanical ventilator used for conventional transport of neonates. It has the ability to generate CPAP or IMV breaths and has the following parameters: flow rate, PIP, PEEP, I-time, E-time, and FiO_2.

Bronchotron® (Percussionaire) (Fig. 1)

The Bronchotron ® is a pneumatically powered, pressure-limited, time-cycled, HFPV that was developed in the mid-1980s by Dr. Forrest Bird. The ventilator has an internal pneumatic timing cartridge which cycles high-pressure gas supply at a frequency of 3–10 Hz. Rate and amplitude are adjustable while the I-time is not, instead being dependent on the resistance and compliance of the respiratory system as well as the set breath rate. The high-frequency output pulses from the timing cartridge then enter a sliding piston mechanism through a venturi cavity in its central axis (Phasitron®) (Percussionaire). This piston/venturi acts as an inspiratory and expiratory valve. In the inspiratory phase, the pulse of gas is augmented by additional entrained gas proportional to the pressure difference before and after the venturi.[13] This mechanism limits the tidal volumes seen by the neonatal lungs which can be a cause of ventilator-induced lung injury.[14,15] During expiration (between pulses from the timing cartridge), there is no gas inflow or entrainment, the piston springs back opening an exhalation port, and gas is allowed to exit the patient through an adjustable resistor that provides PEEP. The device uses 12 lpm of gas flow. The ventilator can be used in CMV mode, high-frequency

Fig. 1. The Bronchotron® HFPV. The percussion knob primarily sets the breath frequency and the flowrate knob primarily sets breath size; however, adjusting either knob can affect both parameters. The Phasitron® acts as a pneumatic clutch for the breaths coming from the Bronchotron®. It has an inspiratory valve and a PEEP valve. The ETT fits on the right hand side of the Phasitron®.

mode, or a combination of the two (high frequency with IMV sigh breaths); however, it was used only in high-frequency mode in our experiment as it is often used in that way for neonatal transports and that was our particular study interest. On the face of the ventilator, there is an operational pressure dial which was set at 35 psi during the experiment, a flowrate dial, which adjusts the gas flow to the Phasitron® thus affecting the pulse amplitude, and a ''Percussion'' dial, which adjusts the rate of the oscillations. An adjustable PEEP valve is located on the Phasitron® body itself and is used to set the MAP.

Sensormedics 3100A (Cardinal Health)

The 3100A is a high-frequency oscillatory ventilator developed in the early 1980s. It uses an electromagnetically driven piston to change the volume, and thus the pressure inside of a rigid circuit containing a continuous bias flow of fresh gas. By this mechanism, small tidal volume breaths are sent into the patient's lungs. It runs exclusively in high-frequency mode. It delivers pressure-limited, time-cycled breaths at a rate of 3–15 Hz. The following parameters were adjusted during these experiments: MAP, ΔP, and Hz. The I:E ratio and bias flow, while adjustable, were set at 33% and 20 lpm, respectively.

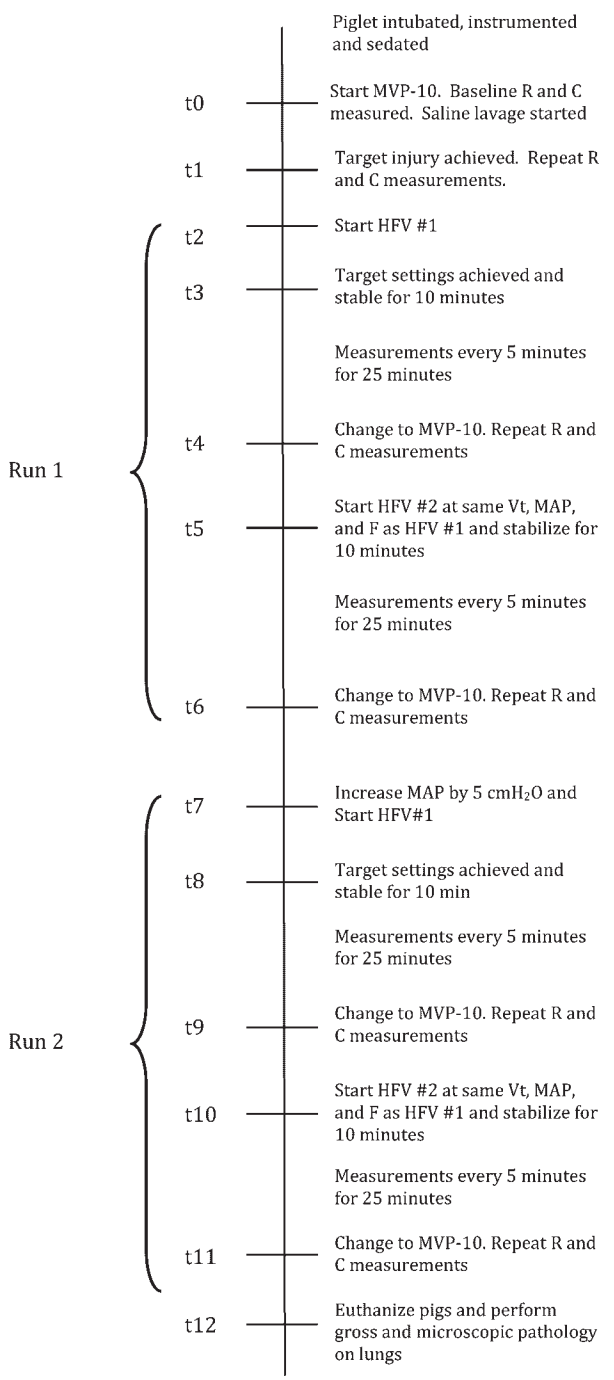
Florian® Neonatal Respiratory Monitor (Acutronic® Medical Systems, Zug, Switzerland)

The Florian® has a hot wire anemometer that measures airflow, which is mathematically integrated by an internal algorithm to give measured tidal volume. It also has an internal pressure transducer. It operates in both conventional and high-frequency mode and has a sampling rate of 1 kHz. Pressure measurements (PIP, MAP, and PEEP) are recorded in both conventional and high-frequency mode to a 1 cmH_2O precision. We calculated ΔP from these values as PIP – PEEP. V_t is measured to a 0.1 ml precision.[16] We measured V_t just proximal to the ETT. MAP and ΔP were measured through a side port ETT adapter, so that consistency in the parameters' measurement between each ventilator would be preserved. Pulmonary mechanics (respiratory system R and dynamic compliance C) are internally calculated by the device in CMV mode from flow and pressure inputs.

Experimental Procedure

After intubation and instrumentation as described above, the respiratory system (lung and ETT tube) R and C were measured at baseline (t_0) (Table 1). Lung injury was induced as described above. After target lung injury was obtained (mean time to injury 93 ± 37 min), R and C values were again measured (t_1) and piglets were assigned to their initial study mode of HFV (either HFPV or HFOV) (t_2). The first high-frequency ventilator was initially set to a rate of 6–8 Hz to facilitate matching frequency on both ventilators, a MAP of 0–2 cmH_2O above the MAP on the MVP-10 and a V_t of 2–3 ml/kg with good chest wall vibration. These initial settings were determined to give blood gas values that were close to our goals during pilot animal experiments prior to commencing this study. Settings were further adjusted to give $PaCO_2$ values between 45 and 60 mmHg, and PaO_2 values between 60 and 80 mmHg on $FiO_2 = 1.0$. After achieving the goal blood gas values, ventilation was continued without further adjustment of the high-frequency ventilator for 10 min to ensure stability of the blood gases (t_3), after which ventilatory parameters (MAP, PIP, PEEP, V_t, and F) and ABGs were recorded every 5 min until six ABGs were obtained. Piglets were then briefly placed back on the MVP-10 and the respiratory systems R and C were again measured (t_4). Animals were converted to the secondary high-frequency ventilator and settings were adjusted to achieve the same MAP, V_t, and F as on the first HFV (t_5). Animals were allowed to stabilize for 10 min on this new ventilator and then ventilatory parameters (MAP, PIP,

TABLE 1— Schematic of Experimental Procedure



PEEP, V_t, and F) and ABGs were again recorded every 5 min until six ABGs were obtained. The piglet was then placed back on the MVP-10 and the respiratory systems R and C were again measured (t_6) (Run 1).

Next, in order to look at changes in gas exchange at a higher MAP, the piglets were put back on the first high-frequency ventilator and the MAP increased by 5 cmH_2O in an attempt to further recruit the lung (t_7). The ventilator settings were adjusted to closely match the V_t and F settings from Run 1. Ventilation was continued on these settings for 10 min (t_8) after which ventilatory parameters (MAP, PIP, PEEP, V_t, and F) and ABGs were again recorded every 5 min until six ABGs were obtained. Piglets were then briefly placed back on the MVP-10 for the measurement of respiratory systems R and C (t_9). Animals were then converted back to the secondary high-frequency ventilator at the same, V_t, and F (t_{10}). Animals were allowed to stabilize for 10 min on the second ventilator and then ventilatory parameters (MAP, PIP, PEEP, V_t, and F) and ABGs were again recorded every 5 min until six ABGs were obtained. The piglet was finally placed back on the MVP-10 and the respiratory systems R and C were measured once more (t_{11}). At the end of the protocol, all piglets were euthanized and the lungs harvested for gross and microscopic evaluation to determine for extent of lung injury.

Statistics

The ventilators were randomized as to which HFV was used first to eliminate starting order as a confounder, but the data were analyzed by device regardless of order of use to determine if there was a statistically significant difference in the ability of the two devices to ventilate at equivalent settings. All statistics are reported as mean and SD. Primary outcomes were $PaCO_2$ and PaO_2 comparing the ventilators at each MAP level. Secondary outcomes were comparing the gas exchange of each ventilator at the different MAP levels, and comparing the ΔP required to generate equal V_t between ventilators at each MAP level and between MAP levels for each ventilator. Post hoc analysis compared PaO_2 over the six time points of the initially randomized HFV run only, comparing the six animals started on each HFV, reflecting rapidity of recruitment. A repeated measures analysis of variance was used to determine differences in outcome variables ($PaCO_2$, PaO_2, V_t, ΔP) over time. Power analysis determined a sample size of 12, provided 80% power to detect a 2 SD difference between groups, from baseline to end of study at the alpha level of 0.05.

RESULTS

Twelve piglets were included in the data analysis. Three animals were excluded including two piglets that died during induction of lung injury and one that failed to achieve target blood gas parameters.

Lung Injury

Significant lung injury was produced in all study animals as demonstrated by changes in R and C values, OI, and MVI pre- and post-saline lavage (Table 2). Lung C and

TABLE 2— Lung Injury Data, Mean (SD)

	Pre-lavage	Post-lavage	After Run 1	After Run 2
All animals				
C (ml/cmH_2O)	2.4 (0.6)	0.9 (0.3)	1.3 (0.8)	1.8 (0.5)
R (cmH_2O/L/sec)	113 (13)	156 (33)	143 (32)	128 (17)
OI	1.4 (0.8)	10.7 (4.0)		
MVI	30 (13)	109 (34)		
Bronchotron as first high-frequency device				
C (ml/cmH_2O)	2.3 (0.7)	1.0 (0.2)	1.5 (1.0)	1.7 (0.4)
R (cmH_2O/L/sec)	107.5 (15.6)	147 (26.1)	136 (35)	123 (19)
OI	1.4 (0.9)	9.8 (3.8)		
MVI	30 (17)	106 (24)		
3100A as first high-frequency device				
C (ml/cmH_2O)	2.6 (0.5)	1.0 (0.4)	1.2 (0.6)	1.8 (0.6)
R (cmH_2O/L/sec)	118 (7)	162 (40)	151 (31)	133 (16)
OI	1.5 (0.7)	11.5 (4.2)		
MVI	30 (10)	112 (44)		

OI = (MAP)(FiO_2)/(PaO_2); MVI = [(PIP)(rate)($PaCO_2$)]/1000.

R did not significantly change during Run 1. While R and C did improve with the increase in MAP between Run 1 and Run 2, the respiratory system was stable within clinically relevant ranges throughout Run 2 (Table 2). Additionally, there were no statistically significant differences between the matched MAP, V_t, and F settings between the different runs (Table 3 and Fig. 2). All animals had histological evidence of alveolar lung injury (hyaline membranes, intra-alveolar, and interseptal edema at necropsy).

$PaCO_2$ and PaO_2 Difference Between 3100A and Bronchotron® (Fig. 3)

For Run 1, at lower MAP, the mean $PaCO_2$ after 30 min on the Bronchotron® at stable settings was 49.7 ± 10.7 mmHg versus 45.5 ± 10.0 mmHg with the 3100A ($P = 0.22$). For Run 2, at higher MAP, the mean $PaCO_2$ after 30 min on the Bronchotron® with stable settings was 49.6 ± 11.1 mmHg vs. 46.8 ± 11.7 mmHg with the 3100A ($P = 0.33$). Similarly, the mean PaO_2 during Run 1, after 30 min on the Bronchotron® at stable settings, was 150 ± 76 mmHg vs. 149 ± 107 mmHg with the 3100A ($P = 0.57$). For Run 2, the mean PaO_2 with the Bronchotron® was 311 ± 125 mmHg vs. 297 ± 128 mmHg with the 3100A ($P = 0.99$). Comparing each ventilator to itself between runs (i.e., keeping V_t and frequency the same, but increasing the MAP by 5 cmH_2O), there was a significant change in the PaO_2 on both the Bronchotron ($P = 0.001$) and the 3100A ($P = 0.0004$), but there was no significant difference in the $PaCO_2$ on either the Bronchotron ($P = 1.0$) or the 3100A ($P = 0.64$).

Analysis of ΔP Needed to Produce Similar V_t

We looked at what ΔP was needed on each ventilator to achieve similar V_t during the experiment (Fig. 3). For Run 1, mean ΔP on the Bronchotron® and the 3100A were 35 ± 7 cmH_2O versus 31 ± 7 cmH_2O, respectively ($P = 0.09$). For Run 2, mean ΔP on the Bronchotron® and the 3100A was 36 ± 3 cmH_2O vs. 32 ± 3 cmH_2O, respectively ($P = 0.10$). Furthermore, there was no significant difference in the ΔP needed to generate V_t from Run 1 to Run 2 on the Bronchotron ($P = 0.78$) or on the 3100A ($P = 0.27$).

Post Hoc Analysis of Rapidity of Oxygenation Improvement

Looking at only the first set of six time points of the six animals initially randomized to HFOV versus the six randomized to HFPV, mean PaO_2 at time point 1 was 79 ± 32 vs. 125 ± 58, respectively ($P = 0.13$) while at time point 6 it was 81 ± 28 versus 184 ± 75, respectively ($P = 0.01$). The interaction of time versus

TABLE 3— Matched Ventilator Parameters, Mean (SD)

	Run 1			Run 2		
	Bronchotron	3100A	*P*-value	Bronchotron	3100A	*P*-value
TV (ml/kg)	3.1 (0.8)	3.0 (0.7)	0.87	3.1 (0.7)	3.0 (0.6)	0.82
F (Hz)	7.2 (0.6)	7.2 (0.6)	0.97	7.2 (0.6)	7.3 (0.6)	0.90
MAP (cmH_2O)	8.5 (1.6)	8.4 (1.6)	0.88	13.6 (2.3)	13.6 (2.2)	0.98

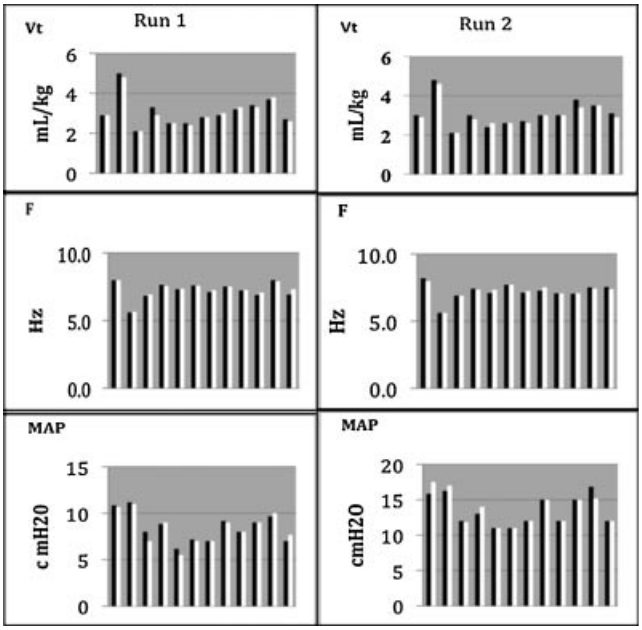


Fig. 2. Matched parameters on each ventilator for each of 12 subjects. Values for Bronchotron (black) and 3100A (white). Run I displayed on the left and Run 2 on the right.

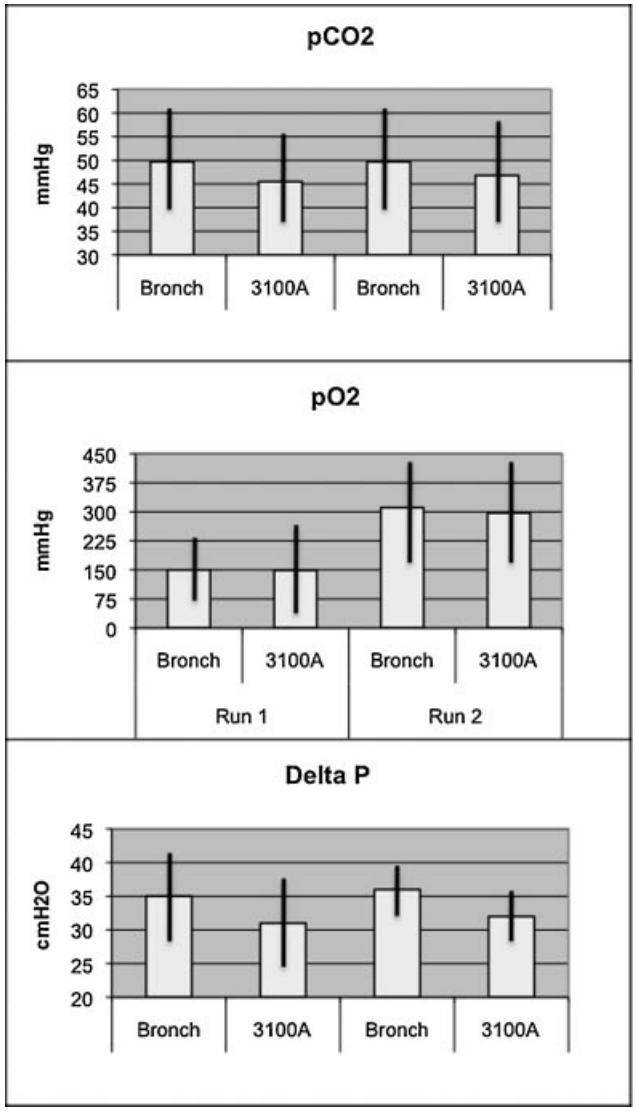


Fig. 3. Mean and SD of $PaCO_2$, PaO_2, and ΔP at time 6 for all study animals separated by ventilator and by run.

device was significant by repeated measures ANOVA ($P = 0.0003$) indicating that oxygenation improved more rapidly on HFPV than on HFOV during each animal's first half an hour on HFV after lung injury.

DISCUSSION

When matched for V_t, F, and MAP in a neonatal ARDS piglet lung injury model, we have shown that the HFPV and HFOV devices ventilate and oxygenate within comparable clinical parameters. Also, while oxygenation improved when the MAP increased (from Run 1 to Run 2) on each device, ventilation did not change for the same V_t and F. The improvement in oxygenation is presumably due to improved lung recruitment and decreased V/Q mismatch. The equivalence of ventilation between the two devices is consistent with the notion that CO_2 elimination during HFV is dependent only on V_t and F regardless of the mode of HFV or the MAP.[9,17] We are not aware of any other head-to-head animal studies comparing these two modes of HFV.

While HFOV has been studied in a number of different animal lung injury models, there are few relevant animal studies on HFPV. Of the studies that have been done, only larger animals have been used simulating adult ARDS and burn injury.[18] There have not been studies published to date using small animals more relevant to pediatric and neonatal lung injury. Additionally, the majority of human clinical studies done on HFPV have been case series involving pediatric and adult patients comparing it with conventional ventilation.[18] However, despite this lack of evidence, some have suggested that HFPV has become the standard of care at burn centers in the United States.[19] A randomized clinical trial involving eight infants done in 1988 comparing pressure volume curves on CMV to HFPV concluded that HFPV provides better sustained lung recruitment than CMV.[20] There has been one animal study comparing CMV, HFOV, and HFPV. This study concluded that HFPV use resulted in significantly less lung damage than either CMV or HFOV in an adult baboon model of smoke inhalational injury.[21]

In our study, we chose to match V_t instead of ΔP, as this is more consistent with the physiology of gas exchange in HFV as noted above. Matching F and V_t equalizes the confounding variables and allows a conclusion that any difference in $PaCO_2$ must be due to ventilator differences. While the I:E ratios of the two devices are unable to

be matched, prior study has demonstrated that different I:E ratios did not change CO_2 elimination when V_t and F were controlled for.[22,23] Thus, the lack of a detectable difference in $PaCO_2$ implies the ventilators provide equivalent gas exchange. While many providers in a clinical setting will adjust the ΔP according to the visible oscillations of the patient's chest as a surrogate for V_t, we know that the relationship between V_t and ΔP is dependent on lung resistance and compliance (i.e., degree of illness) as well as the internal resistance and compliance of the ventilator circuit. For example, we noted that a Bronchotron® V_t of 2.5 cc/kg required a ΔP from 22 to 43 mmHg with similar ranges for the 3100A. Animal and test lung studies[24,25] comparing performance characteristics of various high-frequency ventilators have shown that, as well as changing with degree of illness, the relationship between V_t and ΔP also varies between HFV types when matched for lung resistance and compliance. If so, matching ΔP provided by different modes of HFV would not be equivalent to matching V_t (V_t has to be matched directly). In our study, we did observe a trend toward requiring a larger ΔP with the Bronchotron® to deliver the same V_t as the 3100A, and although not statistically significant, this concept deserves further study. We believe based on the studies[24,25] noted that this is a real effect but our study was not powered so as to overcome the confounding variation in R and C (affecting ΔP vs. V_t), which were matched between devices within each animal but varied widely between animals. We speculate the effect is due to differences in ventilator pressure waveforms, inspiratory times, and operating mechanisms.

The introduction of V_t measurements in CMV in recent years has contributed greatly to safe and effective patient management. Likewise, since HFV ΔP does not uniquely determine V_t, targeting volume during HFV could be expected to similarly improve ventilator management by limiting the V_t and thus decreasing the risks of hyperventilation in ELBW infants and by allowing rational choice of ΔP and F setting combinations to minimize volutrauma and avoid gas trapping. Air flow and volume measurements may also help detect and respond to R and C changes to the patient–ventilator system caused by clinical deterioration or improvement, ETT dislodgement or obstruction, surfactant administration, etc. However, measurement of V_t during HFV is much more challenging due to the small volumes involved and the rapid changes in airflow that occur during HFV. It has been recently reported in an observational study using a hot wire anemometer that V_t's of 1.6–1.8 ml/kg were needed to maintain normocapnia in premature infants ventilated at frequencies of 10–15 Hz.[26] Further development of routine clinical HFV V_t measurement is clearly warranted to enhance the safety and utility of these devices.

In CMV, the dependence of minute ventilation on V_t and rate but not MAP, and the effect of MAP on lung volume, lung resistance, and compliance, and thus on the relationship of V_t to ΔP are well understood. The effects of similar parameters in HFV have not been studied extensively. An early study[27] found that CO_2 elimination is similar at varying levels of MAP when V_t and F are held constant, using an experimental high-frequency device in a dog model. Equivalent results were found in normal and saline lavaged rabbits.[28] Similarly, our results with clinical HFV's showed that with V_t and F held constant, increasing MAP by 5 cmH_2O did not affect $PaCO_2$ levels for either ventilator. This again suggests that ventilation depends only on V_t and F, and not on MAP or type of HFV, although we did not test a sufficient range of MAP levels to exhaustively address this issue.

Theoretically, increasing HFV MAP and thus lung recruitment might be expected to change both lung R and C and therefore the relationship between V_t and ΔP, much as it does in CMV by changing the position on the compliance curve. Thus, clinically one might expect a change in $PaCO_2$ with increased MAP at the same ΔP setting. However, a human neonatal study[29] indicated that V_t measured with a pneumotachograph did not change when MAP was increased by 2 and then 5 cmH_2O at fixed ΔP in each infant. In contrast, the previously mentioned observational neonatal study[24] showed a significant positive association by multiple regression between MAP and V_t in patients managed clinically for normocapnia, not controlling for ΔP. Our study found no difference in ΔP at fixed V_t between two MAP levels for either HFV. We noted, however, that changing MAP in our model unexpectedly resulted in only small effects on respiratory systems R and C, so that any resulting effect on ΔP might not have been detectable. While a larger change in MAP may have resulted in a detectable change in both the previous study[29] and ours, we did not answer this question with our model. This important issue also deserves further study.

In conclusion, when matched for V_t, F, and MAP, the Bronchotron® and the Sensormedics® 3100A appear to be equally effective in ventilating and oxygenating a newborn piglet with ARDS. Increasing MAP on either ventilator increases PaO_2 but does not seem to have an affect on ventilation. Post hoc analysis leads us to hypothesize that HFPV may recruit the lung faster than HFOV. We believe that further investigation into comparison of different modes of HFV (i.e., HFOV, HFJV, and HFPV) in animals with higher OIs as well as the clinical utility of V_t measurement during HFV is warranted.

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