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Bias Flow Does Not Affect Ventilation During High Frequency Oscillatory Ventilation in a Pediatric Animal Model of Acute Lung Injury

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Abstract

Objective—During high frequency oscillatory ventilation (HFOV), bias flow is the continuous flow of gas responsible for replenishing oxygen and removing carbon dioxide (CO₂) from the patient circuit. Bias flow is usually set at 20 liters per minute (lpm), but many patients require neuromuscular blockade (NMB) at this flow rate. The need for NMB may be eliminated by increasing the bias flow rate, but CO₂ retention is a potential concern. We hypothesize that in a swine model of acute lung injury (ALI), increased bias flow rates will not affect CO₂ elimination.

Design—Prospective, randomized, experimental study.

Setting—Research laboratory at a university medical center.

Subjects—Sixteen juvenile swine.

Interventions—Sixteen juvenile swine (12–16.5 kg) were studied using a saline lavage model of ALI. During HFOV, each animal was ventilated with bias flows of 10, 20, 30, and 40 lpm in random sequence. For 10 animals, power was set at a constant level to maintain PaCO₂ 50–60 mmHg, and amplitude was allowed to vary. For the remaining 6 animals, amplitude was kept constant to maintain PaCO₂ within the same range, while power was adjusted as needed with changes in bias flow. Linear regression was used for data analysis.

Measurements and Main Results—Median overall PaCO₂ was 53 mmHg (range: 31–81 mmHg). Controlling for both power and amplitude, there was no statistically significant change in PaCO₂ as bias flow varied from 10 to 40 lpm.

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Conclusions—Changes in bias flow during HFOV did not affect ventilation. Further clinical investigation is ongoing in infants and children with ALI being managed with HFOV to assess the impact of alterations of bias flow on gas exchange, cardiopulmonary parameters, sedation requirements and other clinical outcomes.

Keywords

high frequency ventilation; bias flow; carbon dioxide elimination; mean airway pressure; acute lung injury; acute respiratory distress syndrome; oscillation; mechanical ventilation; pediatric; neonate

Introduction

High frequency oscillatory ventilation (HFOV) is a commonly employed mode of lung protective ventilation for patients with acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) (1-6). HFOV represents an ‘open lung’ approach to mechanical ventilation that utilizes tidal volumes less than physiologic deadspace and sustained mean airway pressures (mPaw) to maintain lung recruitment. Lung recruitment allows for improved alveolar gas exchange, and mPaw is the ventilator parameter that primarily determines oxygenation. Once recruitment is achieved using adequate mPaw, ventilation is then manipulated during HFOV by adjusting the amplitude and frequency of the oscillatory waveform. The rates of these high frequency oscillations are usually set in hertz (hz, 1 hz = 1 cycle/second), at approximately 300-900 breaths per minute, utilizing tidal volumes smaller than physiologic deadspace. This approach allows for a limitation of lung stretch and provides a number of other potential physiologic advantages when compared to conventional mechanical ventilation (CMV) including improved gas exchange, decreased lung injury, and prevention of atelectasis (3, 7-9).

In pediatrics, HFOV is achieved primarily using the SensorMedics 3100A ventilator (CareFusion, Palm Springs, CA). During mechanical ventilation with this device, bias flow provides fresh, humidified gas to the HFOV circuit to replenish oxygen and remove carbon dioxide. The recommended bias flow for the pediatric patient is 20 liters per minute (lpm), which falls in the middle of the available range (0-40 lpm). However, this recommended level of inspiratory flow may be inadequate to support a spontaneous respiratory drive and, thus, may contribute to patient discomfort, poorly coordinated respiratory effort, and significant fluctuations in mean airway pressure (10). These potential problems may necessitate increased pharmacologic sedation and neuromuscular blockade (11-14). Subsequently, neuromuscular blockade may contribute to critical illness myopathy and prolongation of the duration of mechanical ventilation (14-17).

In an attempt to minimize the use of neuromuscular blocking agents and improve patient comfort in the setting of HFOV, some clinicians increase the bias flow to 40 lpm. Higher bias flows may provide patients with more adequate inspiratory flow and lead to a subsequent decrease in work of breathing. While this approach is based on physiologic principles, it has never been systematically studied, and controversy exists regarding the potential impact of increased bias flow on ventilation. Increased bias flow has the potential to compromise ventilation by altering the entrainment of gas during oscillatory ventilation (18, 19) and/or changing the performance of the HFOV piston.

To address these theoretical concerns of higher set bias flows, we developed a pediatric model of ALI in juvenile swine to assess the role of bias flow in gas exchange during HFOV. We hypothesized that increasing bias flow would not adversely affect carbon dioxide elimination.

Materials and Methods

Animal Preparation

The surgical procedures and animal care in this investigation were in compliance with the guidelines established by the National Institutes of Health and were approved by the Institutional Animal Care and Use Committee of Duke University Medical Center. Sixteen juvenile swine underwent premedication with intramuscular acepromazine (1.1 mg/kg) and ketamine (22 mg/kg). Following these injections, intravenous access was obtained, and a dose of thiopental sodium (5 mg/kg) was administered. Normal saline was then provided at 4 ml/kg/hr and continued throughout the study. General anesthesia and neuromuscular blockade were maintained throughout the study period with a continuous infusion of thiopental sodium (5-8 mg/kg/hr) and intermittent bolus dosing of pancuronium bromide (0.1 mg/kg/dose).

Each animal was orotracheally intubated with a 5.5 French cuffed endotracheal tube and then placed on a conventional mechanical ventilator in the pressure-regulated volume control mode (AVEA; CareFusion, Palm Springs, CA). Ventilator settings included: set tidal volume 7 ml/kg, positive end expiratory pressure (PEEP) 5 cm H₂O, inspiratory time 0.7 seconds, rate 20 breaths per minute, and fraction of inspired oxygen (FiO₂) 1.0. Following initiation of mechanical ventilation, the femoral artery was exposed for placement of a systemic pressure catheter. Blood was drawn at that time for arterial blood gas analysis. A large bore intravenous catheter was placed in the femoral vein for further administration of medications. Each animal then underwent median sternotomy for placement of an ultrasonic flow probe (Transonic Systems, Ithaca, NY) around the pulmonary artery at the level of the pulmonary outflow tract to provide continuous cardiac output monitoring. Following instrumentation, the chest wall was surgically closed.

Interventions and Measurements

ALI was created using a modification of the technique of Lachmann et al and Hillman et al (20, 21). Warm normal saline lavage was performed via the endotracheal tube using 10-15 ml/kg aliquots. Lavage was continued until ALI was created as defined by the presence of a PaO₂ / FiO₂ ratio of 150 for at least 30 minutes, an increase in pulmonary artery pressure by 50% of baseline, and a decrease in dynamic compliance of greater than 30%. Following development of ALI, blood gas parameters were re-confirmed after a 20 minute waiting period to ensure stability of the lung injury model. Following these 20 minutes, hemodynamic and respiratory parameters were recorded, and the animal was transitioned to HFOV (3100A; CareFusion, Palm Springs, CA) with a set FiO₂ of 1.0. A lung recruitment maneuver was then performed by increasing the mPaw to 30 cm H₂O for 30 seconds. Other ventilator settings included a frequency of 10 hertz (Hz), inspiratory time 33%, and mPaw set 5 cm H₂O above the mean pressure required during CMV. Frequency, inspiratory time, and mPaw were not manipulated after being initially set, but adjustments to the *limit* and *adjust* settings were allowed to maintain a stable mean airway pressure.

For the first ten animals, the *power* was set to maintain PaCO₂ 50-60 mmHg and was then maintained constant, while *amplitude* was allowed to vary. Bias flow was initially set at 20 lpm, and after 15 minutes on HFOV, repeat hemodynamic and respiratory measurements were performed. PaCO₂ at that time was required to be 50-60 mm Hg, or a one-time *power* adjustment was made to maintain PaCO₂ in this range.

For the final six animals, *amplitude* was set to maintain PaCO₂ 50-60 mmHg and then kept constant by making *power* adjustments. As with the first ten animals, bias flow was initially set at 20 lpm with repeat hemodynamic and respiratory measurements being performed after 15 minutes on HFOV. PaCO₂ at that time was required to be 50-60 mm Hg, or a one-time

amplitude adjustment was made to maintain PaCO₂ in this range. For all sixteen animals, all other ventilator settings and protocol procedures remained constant.

For each animal, bias flow of 10, 20, 30, and 40 lpm was then adjusted in random order and maintained at each flow rate for 15 minutes. Data were collected after each 15 minute interval. At the conclusion of the experiment, the animals were then returned to a bias flow of 20 lpm for final data collection to ensure stability of the model.

Data Analysis

Multivariable linear regression was used to examine the relationship between bias flow and PaCO₂, partial pressure of arterial oxygen/fraction of inspired oxygen (P/F) ratio, cardiac output, and pH, controlling for both power and amplitude. In all regressions, we clustered data by animal providing conservative estimates of confidence intervals. All analyses were done using STATA 11 (College Station, TX), with $p < 0.05$ considered significant.

Results

Data were collected on sixteen juvenile swine (15.1 ± 1.3 kg). Baseline ventilatory parameters after transitioning to HFOV included median mPaw 22 cm H₂O (range: 20-25 cm H₂O), inspiratory time 33%, frequency 10 Hz, and FiO₂ 1.0. Median overall PaCO₂ was 53 torr (range: 31-81 torr), and median overall P/F ratio was 238 (range: 35-494). Median overall cardiac output was 1252 ml/min (range: 500-2081 ml/min) (Table 1).

Controlling for both power and amplitude, there were no statistically significant changes in PaCO₂ as bias flow varied between 10 and 40 lpm (Figure 1) ($p = 0.22$). We observed a trend towards a decrease in PaCO₂ of 0.14 mmHg for each 1 lpm increase in bias flow (95% CI; $-0.37, 0.09$). P/F ratio decreased from 269 (range: 43-482) to 208 (range: 35-491) as bias flow was increased from 10 to 40 lpm ($p = 0.03$). On regression analysis, we observed a decrease of 5 in P/F ratio for each 1 lpm increase in bias flow (95% CI; $-9.5, -0.04$). Mean airway pressure was 22 cm H₂O and did not change significantly as bias flow varied between 10 and 40 lpm. Cardiac output and pH also did not change significantly with variation in bias flow. Median gas exchange, ventilation, and cardiac output parameters at each of the four tested bias flows (10, 20, 30, and 40 lpm) are depicted in Table 2.

Discussion

In an animal model of pediatric ALI, we found no relationship between the set bias flow during HFOV and PaCO₂. In this model, increasing the bias flow from 10 to 40 lpm did not hinder carbon dioxide elimination but did change the P/F ratio. P/F ratio was statistically lower at 40 lpm than 10 lpm, despite an unchanged mean airway pressure. One concern raised by this finding is that the potential benefit of enhanced spontaneous breathing associated with increased bias flow during HFOV may be mitigated by an impairment of oxygenation related to this change, if, in fact, this unexpected finding can be replicated. While the clinical implications and importance of a change in oxygenation of this degree is unclear, this trend is a key area of emphasis for future investigation of the impact of bias flow on gas exchange during HFOV.

Another important finding in this investigation is that alteration of bias flow between 10 and 40 lpm did not significantly impact cardiac output. These data support the notion that bias flow may be increased during HFOV without untoward effects on ventilation or hemodynamics; however, oxygenation may be impacted, potentially necessitating a slight increase in mean airway pressure as a compensatory response.

The effect of bias flow on gas exchange during HFOV is an area in which data are lacking (18, 22). Bancalari et al demonstrated in a rabbit model that increases in bias flow at the level of the endotracheal tube enhanced carbon dioxide removal (22). Similarly, Solway et al demonstrated increased PaCO₂ removal in normal dog lungs with increasing bias flow (18). To our knowledge, no additional data exist regarding the effect of bias flow on either gas exchange or hemodynamics during HFOV. It should be noted that despite these two prior publications, many clinicians remain concerned that elevated bias flow impedes ventilation. This concern regarding CO₂ elimination is based on the potential impact of excessive levels of bias flow on the negative deflection of the ventilator piston during HFOV and the recognized association between piston position and CO₂ removal (23).

Despite these concerns, increased bias flow represents a mechanism to improve patient comfort with spontaneous respiratory efforts during HFOV (10). Even though the mechanisms of gas exchange in HFOV differ substantially from CMV, inspiratory flow has been shown to be a key variable in patient comfort and respiratory muscle unloading during CMV (24, 25). It is possible that increased levels of bias flow during HFOV may similarly facilitate patient comfort by meeting spontaneous patient peak inspiratory flow demands (10, 26, 27).

When bias flow is inadequate, however, patients may become tachypneic with an increased and erratic work of breathing and mean airway pressure may fluctuate resulting in decreased oxygenation. Thus, this inconsistent and ineffective breathing pattern may, in turn, negatively impact gas exchange during HFOV. Optimal bias flow, in contrast, may facilitate comfortable spontaneous breathing and maintain a more stable mean airway pressure. It is possible that this may lead to decreased requirements for sedation and neuromuscular blockade. (14-17).

At this point, any beneficial effects on patient outcomes of increased bias flow rates can only be speculative, but our animal data support the use of this practice in regard to CO₂ elimination. We acknowledge that the ability to decrease sedation with increased bias flow settings is theoretical, and clinical data (which are in progress) are needed to test this hypothesis. It should be also stressed that the relationship between bias flow, sedation requirements, and spontaneous respiratory effort is beyond the scope of this pilot study, but these complex interactions do represent a target for future investigations.

This study adds to the limited body of literature regarding the impact of bias flow on gas exchange during HFOV and has important potential implications for HFOV management. These data support the concept that higher bias flow rates may be employed without adverse effects on ventilation. Thus, a strategy to attempt to improve patient comfort and encourage spontaneous breathing during HFOV by increasing bias flow may be possible without the risk of impaired ventilation or hemodynamics. However, an important caveat is that oxygenation may be negatively impacted by elevated bias flow rates independent of mean airway pressure. One could speculate that the potential benefits of improved patient comfort and need for less sedation/neuromuscular blockade with elevated bias flow would likely outweigh the potential need to minimally increase mean airway pressure to compensate for a possible negative impact on oxygenation; however, further investigation is needed in this area. If our findings are confirmed clinically, these data could impact the respiratory management, sedation practices, and potentially outcomes in pediatric patients managed with HFOV.

Conclusions

In an animal model of acute lung injury, adjustments in bias flow did not alter ventilation or cardiac output, but did negatively influence oxygenation. These results begin to answer the question of the impact of bias flow on gas exchange and hemodynamics during HFOV. Further investigation is warranted in the clinical arena to not only validate these findings but also to assess the effect of altering bias flow on clinical parameters including work of breathing, patient comfort, patient sedation requirements, length of ventilation, and long term outcomes.

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Figure 1.
Box Plot comparing pCO₂ versus set bias flow rate.

Table 1

Overall Ventilatory and Hemodynamic Parameters (n=16)

Parameter	Median	Range
PaCO ₂ (mmHg)	53	31-81
P/F ratio	238	35 - 494
Cardiac Output (ml/min)	1252	600 - 2081
Mean Airway Pressure (cm H ₂ O)	22	20 - 25
Amplitude	50	41 - 76
Power	6	4 - 10
pH	7.26	7.06 - 7.48
Hgb (mg/dL)	9	7 - 12
HCO ₃ (mmol/L)	24	14 - 31

Table 2

Ventilatory and Hemodynamic Parameters at Each Bias Flow (n = 16)

Bias Flow (lpm)	Parameter	Median	Range
10	PaCO ₂ (mmHg)	53	31-81
	PaO ₂ /FiO ₂ *	269	43-482
	Cardiac Output (ml/min)	1258	620-1871
	pH	7.31	7.08 - 7.48
	Mean Airway Pressure (cm H ₂ O)	22	20-24
	Amplitude	53.5	42-75
	Power	5.6	4.1-8.5
	Hgb (mg/dL)	9	7 - 12
	HCO ₃ (mmol/L)	24	15-30
20	PaCO ₂ (mmHg)	57	40 - 73
	PaO ₂ /FiO ₂ *	231	54 - 465
	Cardiac Output (ml/min)	1391	720 - 1961
	pH	7.26	7.06 - 7.44
	Mean Airway Pressure (cm H ₂ O)	22	20-25
	Amplitude	50	42 - 76
	Power	6	4.5 - 10
	Hgb (mg/dL)	9	7 - 12
	HCO ₃ (mmol/L)	24	15 - 29
30	PaCO ₂ (mmHg)	54	34 - 78
	PaO ₂ /FiO ₂ *	219	52 - 494
	Cardiac Output (ml/min)	1230	600 - 1870
	pH	7.27	7.08 - 7.48
	Mean Airway Pressure (cm H ₂ O)	22	20-25
	Amplitude	50	42 - 69
	Power	6	5 - 10
	Hgb (mg/dL)	9	8 - 12
	HCO ₃ (mmol/L)	24	15 - 29
40	PaCO ₂ (mmHg)	52	38 - 74
	PaO ₂ /FiO ₂ *	208	35 - 491
	Cardiac Output (ml/min)	1060	637 - 2081
	pH	7.25	7.08 - 7.45
	Mean Airway Pressure (cm H ₂ O)	22	20-25
	Amplitude	49	41 - 66
	Power	6	5 - 10
	Hgb (mg/dL)	9	8 - 12

Bias Flow (lpm)	Parameter	Median	Range
	HCO ₃ (mmol/L)	24	15 - 29

*
p < 0.05