Comparison of High-Frequency Oscillatory Ventilation and High-Frequency Jet Ventilation in Cats With Normal Lungs

Stephen J. Boros, MD, Mark C. Mammel, MD, J. Michael Coleman, MD, Phillip Horcher, MD, Margaret J. Gordon, RRT, and Dennis R. Bing, RRT

Summary. Four adult cats received alternating high-frequency oscillatory ventilation (HFOV) and high-frequency jet ventilation (HFJV) at equivalent proximal airway pressures. Physiologic measurements were made before and after each ventilator change. Proximal airway pressures were then adjusted as necessary to reestablish normal pH and P_{aco2} values. Aortic, pulmonary, arterial, and central venous pressures were monitored. Cardiac outputs were measured. Pulmonary and systemic vascular resistance, intrapulmonary shunt, and alveolar-arterial oxygen gradient were determined. Following the change from HFOV to HFJV at similar proximal airway pressures, HFJV always produced higher pH values (P < 0.0001), higher P_{aco2} values (P < 0.05), lower P_{aco2} values (P < 0.0001), as well as higher cardiac outputs (P < 0.01), lower pulmonary artery pressures (P < 0.001), and lower pulmonary vascular resistances (P < 0.001). Following the reciprocal crossover, from HFJV to HFOV, HFJV pH values were again higher (P < 0.001), and P_{aco2} values were again lower (P < 0.001). A comparison of HFOV and HFJV at similar pH and P_{aco2} values showed that HFOV consistently required higher peak inspiratory pressures (P < 0.001), higher mean airway pressure (P < 0.001), and higher pressure wave amplitudes (P < 0.001). Under the circumstances of this study, HFJV produced better gas exchange at lower proximal airway pressures. Pediatr Pulmonol. 1989; 7:35–41.

Key words: Proximal airway pressure; peak inspiratory pressure; mean airway pressure; P_{aco2}, P_{aco2}, pH, aortic, O_{2} gradient; aortic pressure; PAP; central venous pressure, pulmonary vascular resistance.

INTRODUCTION

The basic assumption most clinicians make about high-frequency ventilation (HFV) is that it produces comparable gas exchange using lower airway pressures than are possible with conventional mechanical ventilation (CMV). Lower airway pressures produce less lung distention and consequently should produce less barotrauma. In fact, the major clinical success of HFV has been the treatment of life-threatening pulmonary air leaks.

Today, the most common forms of HFV are high-frequency oscillatory ventilation (HFOV), high-frequency jet ventilation (HFJV), and high-frequency flow interruption (HFFI), a modified form of HFV. All have been studied extensively in animals and, occasionally, in humans. Most reports of HFJV and HFFI describe adequate oxygenation and ventilation using lower proximal airway pressures than CMV.\(^{1,2}\) Although fewer data are available for HFOV, all have successfully treated seemingly intractable pulmonary air leaks.\(^{3-6}\) Many studies have compared HFJV, HFFI, and HFOV to CMV. Few have compared the various forms of HFV to each other. Recently, Bell and coworkers compared HFOV, HFFI, and CMV in primates with hyaline membrane disease. In that study, HFOV generated higher airway pressures than either HFFI or CMV.\(^{7}\) These findings raised questions regarding the efficacy of HFOV. We address some of those questions in the following report. Our report compares the cardiopulmonary and airway pressure effects of alternating HFOV and HFJV in animals with normal lungs.

From the Infant Pulmonary Research Center, Children’s Hospital, St. Paul, and Department of Pediatrics, University of Minnesota, Minneapolis, Minnesota.

Received September 19, 1988; (revision) accepted for publication January 30, 1989.

This work was supported by grants from the Hugh J. Anderson Foundation and Mr. William Holcomb.


Address correspondence and reprint requests to Dr. S.J. Boros, Children’s Hospital, 345 North Smith Avenue, St. Paul, MN 55102.
MATERIALS AND METHODS

Four adult cats (mean weight 4.5 kg) were anesthetized with 30 mg/kg of ketamine, paralyzed with 0.1 mg/kg of pancuronium bromide, intubated with 4.0 triple-lumen "high-low jet" uncuffed neonatal endotracheal tubes (Mallinckrodt, Inc.), and ventilated with pressure-preset infant ventilators (BabyBird, 3M Corp.). Catheters were placed in the aortas and right atria through femoral vessels. Thermol dilution catheters (Edwards Laboratories) were placed in the pulmonary arteries through femoral or external jugular veins. Aortic, pulmonary artery, and central venous pressures were continuously recorded using standard transducers and recorders (Hewlett-Packard). Thermol dilution cardiac outputs (95020A Cardiac Output Computer, Edwards Laboratories) were determined in triplicate at regular intervals, as were central venous pressures, arterial and mixed venous blood gas values, and oxygen saturation values. Alveolar-arterial oxygen gradients, intrapulmonary shunts, and total pulmonary and systemic vascular resistances were calculated using standard formulas.

Our jet ventilator, the Bunnell Life Pulse (Bunnell Life Systems, Inc.), is a pressure-preset, time-cycled ventilator attached to a separate jet injector. The system is connected in parallel to a conventional infant ventilator and patient circuit. The jet ventilator provides HFV. The conventional ventilator controls positive-end expiratory pressure (PEEP) and provides fresh humidified gas for entrainment. A solid-state, air-filled, pressure transducer (Micro-Switch Division, Honeywell, Corps.) measures peak inspiratory pressure (PIP) at the endotracheal tube tip. Peak inspiratory pressures are limited to preset values by a servo-control microprocessor that adjusts the machine's driving pressure to maintain the desired PIP. PIP can be varied from 5 to 80 cm H₂O. Frequencies can be varied between 240 and 990 cycles/min (cpm). Inspiratory times can be varied between 0.02 and 0.25 seconds. The jet injector and airway pressure transducer are housed in a separate component located near the endotracheal tube. The injector-transducer unit is connected to two ports of the triple lumen endotracheal tube by two 5 cm lengths of high-pressure PVC tubing. Gas enters the airway through the endotracheal tube's injector port 7 cm proximal to the endotracheal tube tip. Airway pressures are measured through a monitoring port in the endotracheal tube tip. Gas is heated and humidified by a servo-controlled, heated cartridge system.

Our oscillator was the Gould #4800 HFOV (Gould, Inc.). This machine produces oscillations via a low-compliant, diaphragmatically sealed electromagnetic piston. A 76 cm-long, 25 mm inner diameter (ID) mesh-reinforced PVC tube connects the oscillator source to the endotracheal tube. A side port on the endotracheal tube adaptor attaches to an air exit or air return line. Fresh gas bias-flow from a standard blender-flow meter enters the circuit via a T-connector 8 cm proximal to the air exit port. Positive end-expiratory pressure is adjusted by changing the bias-flow or by adjusting a venturi device on the air exit line. A minimum bias-flow of 5 L/min is always maintained. Gas is heated and humidified by a standard humidifier (Bird Humidifier-Controller, 3M Corp.) prior to reaching the patient circuit. Oscillatory frequencies can be varied between 300 and 3,000 cpm. I:E ratios can be set anywhere from 1:3 to 3:1. Pressure wave amplitudes or ΔP (PIP minus PEEP) are changed by changing the bias-flow rate and/or percent power. Percent power describes the relative thrust of the electromagnetic piston in terms of its electromotive driving force. Airway pressures are measured at the endotracheal tube tip through the monitoring port of the previously described triple-lumen endotracheal tube.

To achieve consistency in airway pressure measurements, HFOV and HFJV pressures were both measured by the internal transducer monitoring system of the Bunnell Life Pulse. The frequency response of the endotracheal tube and Microswitch transducer was flat to more than 15 Hertz. The frequency response characteristics of the triple-lumen endotracheal tube have been previously described.

All animals were first stabilized with CMV. Mean CMV settings were FiO₂, 1.0 ± 0.5 cm H₂O; frequency, 29.8 ± 0.5 cpm; I:E ratio, 1:3; PEEP, 2.0 ± 1.0 cm H₂O; PIP, 7.9 ± 1 cm H₂O. They then received either HFOV or HFJV.

Initial HFV settings were HFJV, FiO₂, 1.0; frequency, 400 cpm; I:E ratio, 1:3; PEEP, 2–6 cm H₂O; PIP was set to produce a mean airway pressure (Paw) similar to CMV, then adjusted to maintain PaCO₂ values less than 40 mm Hg; HFOV, FiO₂, 1.0; frequency, 900 cpm; I:E ratio, 1:1; PEEP, 2–6 cm H₂O; bias-flow rate 5 L/min; PIP was set to produce a Paw similar to CMV, then adjusted to maintain PaCO₂ values less than 40 mm Hg.

All four animals were ventilated at these settings for 1 hour. Ventilators were then crossed-over at hourly intervals as follows:

Animals 1 and 3:
HFJV—HFV—HFJV—HFV—HFJV;
Animals 2 and 4:
HFOV—HFJV—HFOV—HFJV—HFOV

Similar Paw, PEEP, and PIP were maintained during each cross-over. Complete physiologic measurements were made 15, 30, and 60 minutes after each ventilator change. After the initial 15-minute measurement, PIP was adjusted as necessary to reestablish normal pH and PaCO₂ values. The new ventilator pressure settings were recorded. A 45-minute stabilization period was then allowed before the next ventilator change. The 30-minute
and 60-minute measurements were made during this stabilization period. After the experiment, the animals were killed with intravenous pentobarbital.

Statistical Methods

We analyzed the data in two stages. The first analysis looked at the cardiopulmonary effects of changing ventilator systems at constant airway pressures. It compared the measurements immediately before and 15 minutes after each ventilator change. The second analysis examined both the cardiopulmonary and airway pressure effects of changing ventilator systems at a similar arterial pH and PaCO₂ values. Here we compared the measurements immediately before and 30 minutes after each ventilator change. The latter measurements (30 minutes) were preceded by adjustments in airway pressure to reestablish normal arterial pH and PaCO₂ values. There were 16 data sets comparing HFJV and HFOV at similar airway pressures and eight data sets comparing HFJV and HFOV at similar pH and PaCO₂ values. These data were compared using two-tailed, paired, Student t-tests.

RESULTS

CMV vs. HFOV and HFJV at Similar Airway Pressures

Figure 1 outlines the arterial blood gas and airway pressure values before and after the change from CMV to HFJV and HFOV for the four animals. The change from CMV to HFJV produced higher pH and lower PaCO₂ values. PaO₂ values remained relatively stable. Mean airway pressures and wave pressure amplitudes (∆P) were similar. Following the change from CMV to HFOV, pH values decreased and PaCO₂ increased. PaO₂ values decreased somewhat. Again, mean airway pressures and ∆P were similar.
HFOV vs. HFJV at Similar Airway Pressures

Figure 2 shows effects of changing from HFJV to HFOV at similar mean airway pressures and ΔP on arterial blood gases. Following this change, pH values decreased (P < 0.0001), PaO₂ values decreased (P < 0.05), and PaCO₂ values increased (P < 0.0001).

Figure 3 demonstrates that, following this change, pulmonary artery pressure increased (P < 0.001), pulmonary vascular resistance increased (P < 0.01), and cardiac output decreased (P < 0.01). These cardiovascular changes were likely caused by the severe hypercarbia and acidosis experienced during HFOV.

Figure 4 shows the effects of the change from HFOV to HFJV at similar mean airway pressures and ΔP. Following this change, HFJV pH values were higher (P < 0.001) and PaCO₂ values were lower (P < 0.001). PaO₂ values, pulmonary artery pressure, pulmonary vascular resistance, and cardiac output values were relatively unaffected (Fig. 5).

HFOV vs. HFJV at Similar pH and PaCO₂ Values

Figure 6 compares the HFOV and HFJV airway pressures required to produce similar arterial pH and PaCO₂ values. HFOV required higher PIP (P < 0.001), higher ΔP (P < 0.001), and higher Paw (P < 0.0001) to establish similar pH and PaCO₂. Pulmonary artery pressure, pulmonary vascular resistance, cardiac output and PaO₂ values were not significantly affected by the airway pressure manipulations.

DISCUSSION

The most common forms of HFV in clinical use today are HFOV, HFJV, and HFFI. High-frequency oscillators employ reciprocating mechanisms, usually piston pumps or vibrating diaphragms, to produce airway oscillations. Downstream from the oscillator source, a continuous transverse gas flow (bias-flow) delivers fresh gas into the airways. Tidal volume is determined by the amplitude of the oscillation and the bias-flow rate. Delivered tidal volumes are usually smaller than anatomical dead space. High-frequency jet ventilators deliver small, high-velocity pulses of gas directly into the airways through a valved injector. The volume of each pulse is determined by the gas flow, gas velocity, the endotracheal tube size, and the time the injector valve is open (inspiratory time). Delivered tidal volumes are larger than the volume of the jet itself because of ambient gases entrained by the high-velocity jet pulse. High-frequency flow interrupters are modified jet ventilators with injectors set further back from the airways and endotracheal tube. Other than producing lower airway velocities and somewhat higher
HFOV and HFJV in Normal Cats

Page dimensions: 612.0x789.0
[Image 0x0 to 612x789]

Fig. 4. Changes in blood gases (\(\text{PaO}_2\), \(\text{PaCO}_2\), pH) when changing from HFOV to HFJV at similar airway pressures (Paw, \(\Delta P\)).

Tidal volumes, HFFI and HFJV are mechanically very similar. 12

In 1984, Bell et al. compared HFOV, HFFI, and CMV in premature baboons with HMD. Initially, the animals receiving HFOV required higher airway pressures than those receiving either HFFI or CMV. 7 After 10 hours, airway pressures gradually equalized. In 1987, DeLemos et al. examined HFOV and CMV in baboons with hyaline membrane disease. Again, those receiving HFOV initially required higher airway pressures and, over time, airway pressures equalized. By 20 hours, HFOV airway pressures were less than CMV. 13

Fig. 5. Changes in cardiac output (C.O.), pulmonary artery pressure (PAP), and pulmonary vascular resistance (PVR) when changing from HFOV to HFJV at similar airway pressures (Paw, \(\Delta P\)).

Unlike Bell et al., our purpose was to compare only HFOV and HFJV. However, we too, noted major differences in ventilator performance following the transition from CMV to HFJV and CMV to HFOV. At similar airway pressures, HFJV always produced better gas exchange than CMV. Our HFOV system, similar in design to that used by Bell et al., consistently produced worse gas exchange than CMV.

Later, when we compared HFJV and HFOV, HFJV always produced better gas exchange using lower airway pressures. We measured airway pressures in the midtrachea. Tracheal pressures may or may not reflect pressures in the distal airways. Allen et al. showed that, depending on the size of the lung and the resonant frequency of the airways, alveolar pressures can be higher, equal to, or lower than tracheal pressures. 14 We did not measure distal airway pressures. Our purpose was to compare HFOV and HFJV as they are used in clinical practice. We focused only on proximal airway pressures because, currently, they are the standard clinical airway pressure measurements.

We suspect that at least some of the airway pressure and gas exchange differences we observed between the two HFV systems are the results of some basic differences in ventilator design.

The point at which fresh gas entered the two HFV systems may have been a factor. During HFJV, fresh gas entered the airways 7 cm upstream from the endotracheal tube tip. During HFOV, fresh gas bias-flow entered the system 8 cm upstream from the endotracheal tube adapter, 10–12 cm further upstream than during HFJV. Rossing et al. noted that during HFOV, CO\(_2\) transport improved dramatically when the entry point for fresh gas was moved from the proximal to the carinal end of the endotracheal tube. These investigators also demonstrated that endotracheal tubes accounted for more than 30% of the total resistance to gas transport. 15 Davy et al. made similar observations during HFJV. 16 We suspect that the relatively distal HFOV fresh gas entry point, at least in
part, accounted for the higher airway pressures observed during HFOV.

The distance between a ventilator’s energy source and the subject’s airway may have played a role. Our HFJV energy source, the injector valve, was located approximately 12 cm from its gas entry port within the endotracheal tube. Our HFOV energy source, the electromagnetic piston, was located approximately 80 cm from the endotracheal tube. The more than sixfold difference in the distance between the airway and energy source may also account for some of the differences we observed in ventilator efficiency. Studies examining the effects of different energy source-airway distances are now underway.

Differences in the mechanics of exhalation may be important. During HFJV and during CMV, exhalation is passive. Gas exiting the airways is driven by the elastic recoil of the lungs and chest wall. During HFOV, exhalation is active. Gas moves into, then out of, the airways as a piston or diaphragm moves back and forth. This action produces pressure changes with a sine wave configuration. HFOV airway pressures are lower, often negative, during exhalation, accelerating expiratory gas flow. Buncalari et al. recently observed that at similar rates and airway pressures, HFJV consistently produced larger tidal volumes and more trapped gas than HFOV. Increased gas trapping and, consequently, larger lung volumes may account for some of the improvements in gas exchange we observed during HFJV.

This study and the work of Buncalari et al. clearly demonstrate that high-frequency jet ventilators and high-frequency oscillators are very different machines. Seemingly subtle differences in design may produce major differences in function. Clinical and laboratory observations made regarding one system may or may not apply to the other.

ACKNOWLEDGMENTS

We wish to thank John Connett, Ph.D., of the Department of Biometry at the University of Minnesota, for his assistance in statistical analysis.

REFERENCES

13. DeLemos RA, Coalson JM, Gerstmann DR, Null DM, Ackerman NB, Escobedo MB, Robotham JL, Kuehl TJ. Ventilatory man-


