High Frequency Chest Wall Compression in Cats with Normal Lungs

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ABSTRACT. Ten anesthetized, paralyzed adult cats were ventilated by high frequency chest wall compression (HFCWC) at 3, 5, 7, and 9 Hz by means of a single chamber cuff enclosing the thorax from the axillae to the xyphisternum. The effects of HFCWC in terms of gas exchange, end-expiratory lung volume, and respiratory system compliance were compared to conventional intermittent positive pressure ventilation (IPPV) (30 breaths/mn). HFCWC and IPPV were compared at three levels of matched end-expiratory airway pressure [continuous positive airway pressure (CPAP)/positive end-expiratory pressure (PEEP) of 0, 2, and 5 cm H₂O]. In the absence of CPAP, HFCWC resulted in a marked decrease (up to 50%) in end-expiratory lung volume with significantly lower PaO₂, lower compliance, and higher alveolar-arterial oxygen gradient than during IPPV. No differences in PaO₂ and a-ADO₂ were observed when HFCWC was combined to CPAP ≥ 2 cm H₂O. At frequencies below 9 Hz, PaCO₂ became significantly lower during HFCWC + CPAP than during IPPV. During HFCWC + 2 cm H₂O CPAP, lung volume was lower than during IPPV + 2 cm H₂O and similar to the volumes observed during IPPV + 0 positive end-expiratory pressure. Additional studies in six cats at HFCWC + 3 cm H₂O confirmed that CPAP > 2 cm H₂O more than adequately compensated the decrease in lung volume associated with HFCWC alone. Peak cuff pressures between 14 and 17 cm H₂O generated oscillatory tidal volumes between 4.5 and 2.1 ml/kg. The size of the oscillatory volume was significantly affected by increasing frequencies (decrease in tidal volume) and increasing levels of positive airway pressure (increase in tidal volume). We conclude that in cats with normal lungs, HFCWC can provide for normal gas exchange, provided that it is combined with low level CPAP in order to prevent the occurrence of airway closure associated with HFCWC alone. (*Pediatr Res* 21: 183-187, 1987)

Abbreviations

HFCWC, high frequency chest wall compression
HFO, high frequency oscillation
IPPV, intermittent positive pressure ventilation
CPAP, continuous positive airway pressure
VT, tidal volume
FRC, pulmonary functional residual capacity
VL, end-expiratory lung volume

Crs, respiratory system compliance
Paw, airway pressure
Pcuff, cuff chamber pressure
PaO₂, partial arterial oxygen pressure
PaCO₂, partial arterial carbon dioxide pressure
a-ADO₂, arterial-alveolar oxygen gradient
PEEP, positive end-expiratory pressure
He, helium
ABG, arterial blood gases

There are a number of modes of high frequency ventilation which provide adequate gas exchange with VT lower than the dead space. These include high frequency positive pressure ventilation, high frequency jet ventilation, and HFO, all of which are administered via the airway and thus require intubation. Recently, several investigators (1-5) have used oscillations around the chest wall (external HFO) rather than at the airway opening. Such a method may potentially avoid the use of an endotracheal tube and its associated complications (6, 7). In terms of gas exchange, external HFO was shown to be as efficient as the internal form (3).

External HFO has been produced by various means such as pneumatic compression in a plethysmograph (3, 4), high frequency negative pressure ventilation (5), and high frequency chest wall compression (2). The requirement for strict enclosure of most of the body may limit the feasibility of the first two methods in terms of its potential clinical use. Such might not be the case for high frequency chest wall compression (HFCWC) produced by means of pressure oscillations in an air filled cuff wrapped around the lower thorax. Zidulka *et al.* (2) showed that it was possible to ventilate, by HFCWC, paralyzed dogs with normal lungs, but at lung volumes moderately below FRC. The possibility that CPAP, combined to HFCWC, might counterbalance the deleterious effect on FRC was not addressed in that study.

This report presents the effects of HFCWC in another species whose chest wall compliance might be of greater similarity to that of a human newborn infant. The experiments were performed on paralyzed healthy cats in whom HFCWC at various frequencies, with or without CPAP, was compared to conventional positive pressure ventilation in terms of gas exchange, FRC, and lung compliance.

METHODS

Ten adult mongrel cats 2.2 to 3.0 kg were anesthetized with intravenous ketamine HCl (30 mg/kg) and paralyzed with pancuronium bromide (200 µg/kg intravenous). These medications were repeated as necessary to maintain anesthesia and paralysis. A catheter with a Clarke PO₂ electrode on its tip (G.D. Searle, Bucks, England) was inserted through the femoral artery for

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continuous PaO_2 monitoring. However, full arterial blood gas measurements were periodically performed as outlined below. The catheter was maintained patent by continuous infusion of 5% dextrose in 0.2 heparinized saline at a rate of 3 ml/kg/h. The arterial catheter was connected to a pressure transducer (model P23db, Gould Statham Instruments Inc., Sunduan, Puerto Rico) and systemic arterial pressure and heart rate recorded. A rectal temperature probe was inserted and body temperature maintained between 37 and 38°C using a heating pad. In order to be able to perform measurements of FRC and respiratory system compliance, a tracheostomy was done on each animal and a tight fitting endotracheal tube was inserted and sutured in place to ensure a seal.

HFCWC was administered by means of a single chamber cuff (30 × 11 cm) placed around the thorax of the cat (Fig. 1). The cuff covered the chest between the axilla and the xiphisternum. The outer membrane of the cuff consisted of stiff 4 mm thick rubber and the inner membrane was a thin sheet of highly distensible "dental" rubber. Two plastic tubes (1 cm ID) were inserted, through the anterior part of the outer membrane, into the cuff chamber. One was attached to a high frequency diaphragm oscillator (Metrex Instruments Ltd., Brampton, Ontario, Canada). The second tube was connected to a Valdyne MP 45 differential pressure transducer with a flat frequency response to 40 Hz. The oscillator was set to produce stroke volume between 50 and 120 ml for the frequencies tested in this study (3, 5, 7, and 9 Hz). In order to have good apposition of the soft inner rubber membrane and the chest wall, the cuff was maintained partially inflated by a bias flow of air (2–6 liter/min) fed into the tube connecting the oscillator to the cuff. An adjustable small leak was inserted at the entry of the bias flow into the connecting tube so that the minimal pressure in the cuff never exceeded 2 cm H_2O . In those experiments where CPAP was combined to HFCWC, positive airway pressure was produced by flowing air (10 liter/min) through a T-connector attached to the endotracheal tube and through a large bore tube placed under water to the depth of the desired CPAP (Fig. 1).

IPPV was produced by a MV-10 (Bio-Medical, Stanford, CT) pediatric pressure and time cycled ventilator. Respiratory rate and inspiratory:expiratory ratio were kept at 30 breaths/min and 1:2, respectively. Peak inspiratory pressure was adjusted (range 5–9 cm H_2O) to allow for normocapnea. Positive and expiratory

pressure was set at 0, 2, or 5 cm H_2O to match CPAP used during HFCWC.

Thirty-min periods of HFCWC were alternated with equivalent sequences of IPPV matched for end expiratory airway pressures. The order in which the various HFCWC frequencies (3, 5, 7, and 9 Hz) at various CPAP levels (0, 2, and 5 cm H_2O) were studied was randomized. The stroke volume of the oscillator was kept constant for each set of experiments done with each animal. Pressures at the proximal end of the endotracheal tube and cuff chamber pressure (P_{cuff}) were monitored via pressure transducers (model MP 45 ± 50 cm H_2O , Valdyne, Northridge, CA) connected to a Hewlett Packard recorder (model 37404 A, Waltham, MA). That system had a flat frequency response to 40 Hz.

Arterial blood gases, VL, and respiratory system compliance were measured at the end of each ventilatory sequence. The alveolar arterial oxygen gradient was calculated by the following formula: $A-a\text{DO}_2 = (\text{PIO}_2 - \text{PaCO}_2/R) - \text{PaO}_2$, where PIO_2 is the partial pressure of inspired oxygen. R was assumed to be 0.8.

End expiratory VL was measured using a modification of the closed circuit He dilution technique (8). A 50-ml glass syringe was filled with a mixture containing approximately 10% He in air. The He concentration was determined prior to each VL measurement using a He catharometer (PulmoAnalyser, Godart, DeBilt, Utrecht, Holland). The VL measurement required clamping the endotracheal tube at end expiration, and with HFCWC we found it necessary to perform up to eight clampings in order to obtain end-expiration, which was defined as the most positive cuff pressure in the oscillation cycle. When the airway was clamped at a point in the respiratory cycle other than end-expiration, the clamp was immediately released and resealed 10 s later. When end-expiration was obtained, the oscillator was stopped and the He-containing syringe was attached to the airway and manually oscillated at approximately 2 Hz, with a stroke volume of about 10 ml for 30 s. The He concentration in the syringe was determined after 30 s of "rebreathing" and VL calculated. We found in previous studies that 15 s was sufficient when the procedure is conducted as described. We have used this method repeatedly and, using a lung model, have found it both reproducible and accurate (± 5%).

Total Crs was measured by stepwise inflation and deflation of the lungs from end-expiratory VL. Twenty-five ml of air was injected every 3–4 s until an airway pressure of 25 cm H_2O was achieved. Then, the air was withdrawn at similar volume and speed. Compliance was calculated as the slope of the linear portion of the deflation limb of the pressure volume curve immediately above VL.

VT was obtained during HFCWC by the reverse plethysmographic technique (3). A plexiglass chamber (plethysmograph) containing 18 liter of air and steel wool (to minimize adiabatic pressure change) was connected to the endotracheal tube by a 20 cm long (0.9 cm ID) Tygon tube and the pressure changes in the plethysmograph were recorded during HFCWC. The pressure changes generated in the plethysmograph by the oscillatory VT were less than 1 cm H_2O . The amplitude of the signal produced by a fixed stroke volume (22 ml) metal bellows oscillator connected to the plethysmograph via the Tygon tube was found to be reduced by less than 3% at 30 Hz compared to 1 Hz. The resonant frequency of the plethysmograph, the Valdyne MP 45 differential pressure transducer, and the connecting tubing was 117 Hz.

At a later stage, six additional cats (2.4–3.6 kg) were added to the present study. These animals were ventilated by only one rate of HFCWC (5 Hz) combined with one exclusive level of CPAP: 3 cm H_2O . Thirty-min periods of HFCWC were alternated with periods of IPPV at 0 and 3 cm H_2O PEEP. At the end of each different ventilatory sequence, ABG, VL, VT, and Crs were measured as previously described. On three of the animals cardiac output was measured during HFCWC and during IPPV both at 3 cm H_2O PEEP. A 5 Fr flow directed catheter was inserted and cardiac output was measured in triplicate using

Fig. 1. Schematic diagram of the apparatus used for the delivery of HFCWC + CPAP depicting the single bladder cuff placed around the thorax connected to the pressure transducer and the oscillator. A bias flow was introduced into the tubing connecting the oscillator to the cuff to maintain apposition of the cuff around the chest wall, and small adjustments were made using the adjustable leak.

the thermodilution technique (Cardiac output computer 601. Instrumental Laboratory Inc., Lexington, MA) with the rapid injection of 3 ml of O°C normal saline into the distal port of the pulmonary artery catheter.

Statistical analysis was performed by one-way analysis of variance. When statistical significance was present, a Dunnett's multiple comparison procedure (9) was performed. Statistical significance was established at $p < 0.05$. Data are presented as mean $\pm$ SD.

RESULTS

Figures 2 and 3 summarize the effect on PaCO_2 and PaO_2 of HFCWC, compared to IPPV, at the various frequencies and levels of end-expiratory airway pressure tested in the first 10 animals.

At 0 end-expiratory pressure, mean PaCO_2 was similar during IPPV and the various HFCWC frequencies. At frequencies below 9 Hz, the combination of HFCWC and CPAP resulted in lower PaCO_2 than during HFCWC alone ($p < 0.01$). There were no significant differences between the two levels of CPAP tested. The lowest mean PaCO_2 (19 ± 4 torr) was observed during HFCWC at 3 Hz and 5 cm H₂O CPAP. Except for the latter combination, there was no significant difference between HFCWC + CPAP and IPPV at matched PEEP. During HFCWC + CPAP, PaCO_2 was lower at 3 than at 9 Hz frequencies ($p < 0.01$).

Fig. 2. Comparison of PaCO_2 during IPPV and HFCWC at the different frequencies and levels of positive end-expiratory pressure tested (mean $\pm$ SD).

Fig. 3. Comparison of PaO_2 during IPPV and HFCWC at the different frequencies and levels of positive end-expiratory pressure tested (mean $\pm$ SD).

Fig. 4. Comparison of end-expiratory lung volume during HFCWC (left) and IPPV (right) at various levels of end-expiratory pressure (mean $\pm$ SD).

Mean PaO_2 was lower during HFCWC than during IPPV at 0 end-expiratory pressure. This difference was statistically significant at frequencies above 3 Hz ($p < 0.05$). The combination of HFCWC and CPAP, at both levels tested, resulted in higher PaO_2 than during HFCWC alone ($p < 0.01$) and no differences were then observed between HFCWC and IPPV at matched PEEP. With CPAP, PaO_2 was least improved during HFCWC at 9 Hz frequency and the difference between 3 and 9 Hz was statistically significant ($p < 0.05$).

Only in the absence of PEEP, was A-aDO₂ significantly smaller during IPPV ($8.5 \pm$ torr) than during HFCWC at frequencies above 3 Hz ($p < 0.01$). The widest gradient was observed at 9 Hz (31 ± 18 torr).

There were significant differences between VL (Fig. 4) during HFCWC and IPPV at 0 and 2 cm H₂O end-expiratory airway pressure ($p < 0.01$), with the lowest VL obtained on HFCWC + 0 PEEP (14.6 ± 8 ml/kg). During IPPV + PEEP 2 cm H₂O mean VL was 35 ± 6 ml/kg, while HFCWC + PEEP 2 cm H₂O and IPPV + 0 PEEP yielded an intermediate value for VL (23 ± 13 and 27 ± 10 ml/kg, respectively). There were no differences between the respective HFCWC frequencies although there was a trend for VL to be larger at 3 Hz than at the other rates during HFCWC + 0 PEEP (17 ± 8 ml/kg).

Crs (Table 1) was lower during HFCWC than during IPPV at 0 and 2 cm H₂O end-expiratory airway pressure ($p < 0.01$). There were no differences between the various HFCWC frequencies. Specific compliance (compliance per unit of FRC) was similar during IPPV and HFCWC when PEEP was used.

Oscillatory tidal volumes, measured at the tracheal tube level, are shown in Figure 5. There was a significant reduction in VT with increasing frequencies from an average of 3.6 ± 0.9 ml/kg at 3 Hz to 2.4 ± 0.8 ml/kg at 9 Hz ($p < 0.01$). For all frequencies, the efficiency of HFCWC in inducing VT improved with increasing level of CPAP. This effect was largest at 3 Hz (2.9 ± 0.6 versus 4.5 ± 0.5 ml/kg at 0 and 5 cm H₂O CPAP, respectively), and minimal at 9 Hz (2.1 ± 0.5 versus 2.7 ± 1.0 ml/kg at 0 and 5 cm H₂O CPAP, respectively).

The mean peak cuff pressure during HFCWC ranged from 14 ± 3 cm H₂O at 3 Hz to 17 ± 3 cm H₂O at 9 Hz, while mean peak to trough cuff pressures ranged from 13 ± 3 and 15.5 ± 3 cm H₂O, respectively. These differences between the two extreme rates were statistically significant ($p < 0.01$).

Mean arterial blood pressure and heart rate were similar during HFCWC and IPPV. Mean systolic blood pressure was lower when end-expiratory pressure was above zero (139 ± 20 mm Hg

Table 1. Comparison of Crs and CRS/FRC during HFCWC and IPPV at various levels of end-expiratory airway pressure

PEEP (cm H ₂ O)	CRS (ml/cm H ₂ O)			CRS/FRC (liter/cm H ₂ O/liter FRC)		
	0	2	5	0	2	5
HFCWC	7.3 ± 1.2*	7.1 ± 0.8*	8.3 ± 1.2	0.65 ± 0.35*	0.33 ± 0.07	0.22 ± 0.05
IPPV	8.5 ± 1.9	9.0 ± 2.2	8.3 ± 1.8	0.36 ± 0.12	0.27 ± 0.08	0.21 ± 0.14

* $p < 0.001$.

Fig. 5. Oscillatory tidal volume during HFCWC. Effects of different frequencies and different levels of CPAP (mean + SD).

Table 2. Comparison of HFCWC (5 Hz) + 3 cm H₂O CPAP and IPPV + 3 cm H₂O PEEP

	PaCO ₂ (torr)	PaO ₂ (torr)	A-aDO ₂ (torr)	VL* (ml/kg)	CRS (ml/cm H ₂ O)
HFCWC	20 ± 2	100 ± 12	19 ± 6	35 ± 9.5	7.7 ± 1.6
IPPV	37 ± 5	91 ± 7	11 ± 7	42 ± 9.5	9.1 ± 1.9

* $p < 0.005$.

at 0 PEEP versus 130 ± 20 and 126 ± 23 mm Hg at 2 and 5 cm H₂O, respectively; $p < 0.01$).

Data from the six cats comparing IPPV + PEEP to HFCWC (5 Hz) + CPAP at a similar end-expiratory pressure are presented in Table 2. PaCO₂ decreased significantly with HFCWC ($p < 0.005$), the lowest PaCO₂ achieved was 11.7 torr. There were significant differences between VL during IPPV + PEEP 3 cm H₂O (42.4 ± 9.5 ml/kg), HFCWC + CPAP 3 cm H₂O (35 ± 9.6 ml/kg), and IPPV + 0 PEEP (30.4 ± 7.7 ml/kg) ($p < 0.001$).

Cardiac output was measured in only three cats. There were no differences between the mean cardiac output on HFCWC (5 Hz) + 3 cm H₂O CPAP and IPPV + 3 cm H₂O PEEP in those animals (267 ± 93 versus 288 ± 64 ml/min, respectively).

DISCUSSION

This study confirms the results of Zidulka *et al.* (2) who demonstrated that it is possible to ventilate anesthetized paralyzed animals with normal lungs by high frequency chest wall compression. In addition, we demonstrated that lung volume can be maintained by the application of 2–3 cm H₂O CPAP.

There are a number of possible explanations why we were able to achieve adequate gas exchange with cuff pressures six to seven times lower than in the study of Zidulka *et al.* (2). In the latter study, in dogs, peak cuff pressures up to 230 cm H₂O were required to generate oscillatory tidal volume which, per unit of body weight, were of equal magnitude to those measured in our

experiments. One factor may be due to the differences in species with possible less attenuation of the pressure from the chest wall to the lung in cats. The differences in chest wall compliance between cats (3.9 mg/kg) and dogs (2.5 ml/kg) (10) are likely to be too small to account for the marked discrepancy between cuff pressure requirement between the two species. A second factor may be the use of a different technique regarding the type of cuff and the manner in which the cuff was apposed to the chest. By designing a single chamber cuff enclosing the entire lower thorax, with an outer membrane of stiff, poorly compliant rubber and thin distensible rubber on the inner surface, we were able to achieve good apposition with the bias flow, without constricting the chest wall. Also, there was no bulging of the outer membrane, a difficulty encountered by Zidulka *et al.* (2), who used a double blood pressure cuff positioned on either side of the rib cage, with an outer membrane made of canvas. This outward bulge, which allowed for a large portion of the pumped-in volume not to be used efficiently toward squeezing the chest wall, as well as the relatively poor compliance of their cuff, were the most likely causes for the higher cuff pressure needed by Zidulka *et al.* (2) to achieve adequate displacement of the chest wall. It appeared that increasing peak cuff pressure up to a certain point (usually 25 cm H₂O) was associated with adequate gas exchange, whereas above this pressure there was a deterioration in gas exchange.

One of the major concerns of HFCWC is the associated reduction in lung volume with the application of positive pressure to the chest wall. This decrease in lung volume was observed by Zidulka *et al.* (2) in their study on dogs in which they showed, using a pneumotachograph, that end-expiration with HFCWC occurred at 80–100 ml below the paralyzed FRC (zero airway pressure), producing an estimated fall of 10% in FRC. Some differences in end-expiratory volume can be expected when comparing IPPV, where end-expiratory volume is achieved at the resting position of the respiratory system, and HFCWC where end-expiratory volume at end compression of the chest wall is below the same resting position. Nevertheless the differences should be equal to the oscillatory VT, *i.e.* approximately 3 ml/kg. In our study we observed that lung volume, determined by the He dilution technique, decreased up to 50% once HFCWC without CPAP was applied to cats with normal lungs. This marked decrease in FRC, resulting in airway closure, could have been due to constriction of the chest wall by our cuff system. In this study, we used a variable stroke volume pump so that we were able to alter peak cuff pressure without increasing the bias flow, thus lessening the risk of constricting the chest wall. In preliminary experiments, we measured the pressure load applied to the chest wall with a wafer transducer (11) situated below the cuff. When apposition of the cuff was produced by the bias flow and the variable leak in such a manner that minimal cuff pressure did not exceed 2 cm H₂O (as performed in our experiments), no pressure load was measured at the chest surface at the end of the outward chest wall recoil. On the other hand, inadvertent constriction (similar in a reverse way to "inadvertent" PEEP) (12) can occur if the passive outward recoil of the chest wall requires more time to be completed than its active inward displacement. The observation made by Zidulka *et al.* (2) that peak expiratory flow at the mouth were significantly greater than peak inspiratory flow, illustrates the probable different time constant for inward (active) and outward (passive) displacement of the chest during HFCWC. As observed in our study, this will result in FRC to be

lowest for shortest deflation time (*i.e.* at 9 Hz) and to be least affected with the more prolonged times (*i.e.* at 3 Hz). As opposed to dogs, a greater degree of "inadvertent" constriction is also to be expected in cats who, because of their more compliant rib cage, are likely to have, during HFCWC, proportionally larger inward excursion of the chest wall. The cuff inflation: deflation time ratio was 1:1 and could not be changed as the oscillator we used generated a sinusoidal pressure waveform. We were therefore unable to assess the possibility that longer deflation than inflation time might lessen the decrease in FRC associated to HFCWC. In the present study we have shown that VL during HFCWC + CPAP > 2 cm H₂O is greater than VL at zero end-expiratory on IPPV, indicating that the combination of HFCWC + CPAP more than adequately compensated the decrease in VL associated with HFCWC alone. Also, HFCWC + CPAP, for similar cuff pressures, generated larger oscillatory VT than during HFCWC which indicates that the chest-lung outward recoil was of larger amplitude with the former combination. This "facilitated" outward recoil counteracting the tendency for "inadvertent" chest constriction is the likely mechanism by which CPAP preserves FRC during HFCWC.

Once that airway closure was prevented by the use of CPAP, there were no significant differences in the levels of PaO₂ and PaCO₂ reached by HFCWC at 3, 5, or 7 Hz. On the other hand, for a similar increase in VL and compliance, there was no significant improvement in PaCO₂ and PaO₂ when HFCWC at 9 Hz was combined with CPAP. This could possibly be because, at the latter rate, cuff deflation (inspiration) time becomes too short to allow the generation of VT of sufficient size. Similarly, Zidulka *et al.* (2) observed that HFCWC above 8 Hz failed to produce normoventilation in paralyzed dogs. The decreased ability to ventilate by HFCWC at those highest rates could also be related to the damping of the oscillatory energy from the chest wall to the alveoli. It has been calculated that in cats (13), the wave amplitude from chest wall to alveoli would be attenuated by 20% at 5 Hz and 50% at 10 Hz. Possibly this attenuation, which might differ for each species and between normal and sick lungs, could be a major factor in the ability to ventilate by external HFO.

The degree by which FRC will be reduced during HFCWC is likely to be inversely related to the compliance of the chest wall. In dogs and in healthy human adults (14), for whom chest wall compliance is lower than in cats, HFCWC resulted in minor decrease in FRC (10 and 5%, respectively). Normal gas exchange was obtained with isolated HFCWC. On the other hand, in species with a more compliant rib cage (*i.e.* cats, rabbits, and newborn infants), HFCWC requires combination with moderate amount of CPAP to prevent a major decrease in FRC and airway closure. This has been recently confirmed by Piquet *et al.* (15) who demonstrated that rabbits subjected to high-frequency body surface oscillation became hypoxemic unless external HFO was combined with 5 cm H₂O CPAP.

HFCWC can provide for normoventilation with VT close to or below dead space volume similar to other techniques of HFO

(16). In spontaneously breathing human adults, the superimposition of HFCWC resulted in a 40% decrease in spontaneous minute ventilation without changes in PaCO₂ (17). This enhanced gas mixing produced by HFCWC might also increase effective alveolar ventilation during IPPV. If this is so, the combination of HFCWC and IPPV and PEEP could allow for positive pressure ventilation of lesser intensity and decrease risks of barotrauma.

Further pathologic studies, such as in the surfactant depleted lungs, are necessary to determine the ultimate role of HFCWC.

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REFERENCES

1. Ward HE, Power JHT, Nicholas TE 1983 High-frequency oscillations via the pleural surface: an alternative mode of ventilation? *J Appl Physiol* 54:427-433
2. Zidulka A, Gross D, Minami H, Vartian V, Chang HK 1983 Ventilation by high-frequency chest wall compression in dogs with normal lungs. *Am Rev Respir Dis* 127:709-713
3. Harf A, Bertrand C, Chang HK 1984 Ventilation by high-frequency oscillation of thorax or at trachea in rats. *J Appl Physiol* 56:155-160
4. Ward HE, Armengol J, Jones RL 1985 Ventilation by high frequency oscillation in cats. *J Appl Physiol* 58:1390-1399
5. Eyal FG, Hayek Z, Armengol J, Jones RL 1986 Comparison of high frequency negative pressure oscillation with conventional mechanical ventilation in normal and saline-lavaged cats. *Crit Care Med* 14:724-729
6. Joshi VV, Mandavia SG, Stern L, Wigglesworth FW 1972 Acute lesions induced by endotracheal intubation. Occurrence in the upper respiratory tract of newborn infants with respiratory distress syndrome. *Am J Dis Child* 124:646-649
7. Phelps DL, Loew DA, Oh W 1974 The laryngo-tracheal function of surviving infants following prolonged oro-tracheal intubation during the neonatal period. *Clin Res* 22:242
8. Meneely GR, Kaltreider NL 1941 Use of Helium for the determination of pulmonary capacity. *Proc Soc Exper Biol Med* 46:266
9. Winer BJ 1971 *Statistical Principles in Experimental Design*. McGraw-Hill, New York, pp 201-204
10. Spells KE 1969/70 Comparative studies in lung mechanics based on a survey of literature data. *Respir Physiol* 8:37-57
11. Hoppin FG Jr, Green ID, Mead J 1969 Distribution of pleural surface pressure in dogs. *J Appl Physiol* 27:863-873
12. Peppe PE, Marine JJ 1982 Occult positive end expiratory pressure in mechanical ventilation in patients with airflow obstruction. The auto PEEP effect. *Am J Res Dis* 126:166-170
13. Brody AW, DuBois AB, Nissel OI 1956 Natural frequency, damping factor and inertance of the chest-lung system in cats. *Am J Physiol* 186:142-146
14. Harf A, Zidulka A, Chang HK 1985 Nitrogen washout during tidal breathing with superimposed high frequency chest wall oscillation. *Am Rev Respir Dis* 132:350-353
15. Piquet J, Isabey D, Chang KH, Harf A 1985 Stable normocapnia during high-frequency body surface oscillation in rabbits. *Am Rev Respir Dis* 132:104-108
16. Drazen JM, Kamm RD, Slutsky AS 1984 High-frequency ventilation *Physiol Rev* 64:505-543
17. Calverley PMA, Chang HK, Vartian V, Zidulka A 1986 High frequency chest wall oscillation assistance to ventilation in spontaneously breathing subjects. *Chest* 89:218-223