High-frequency Jet Ventilation in Postoperative Respiratory Failure: Determinants of Oxygenation

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Twenty-four critically ill patients in postoperative respiratory failure received high-frequency jet ventilation (HFJV). In 15 patients (Group A) respiratory frequency was maintained at 100/min and three different inspiratory/expiratory time (I/E) ratios were used at random: 0.25, 0.43, and 0.67. In nine patients (Group B) I/E ratio was maintained at 0.43, and six respiratory frequencies were used at random: 100/min, 200/min, 300/min, 400/min, 500/min, and 600/min. HFJV significantly increased Pa_{O_2} in both groups. Mean airway pressure did not alter with respiratory frequency but increased with I/E ratio. In Group A patients, a significant relationship was found between improvement in Pa_{O_2} and the increase in mean airway pressure ($r = 0.897, P < 0.001$). Also, a very close relationship was observed between mean alveolar pressure and mean airway pressure ($r = 0.973, P < 0.001$), suggesting that a "PEEP effect" had been created at the alveolar level. The increase in mean airway pressure induced an increase in pulmonary volume. In Group A patients, the mean increase in pulmonary volume above apneic functional residual capacity was +362 ml for an I/E ratio of 0.25, +1,095 ml for an I/E ratio of 0.43, and +1,936 ml for an I/E ratio of 0.67. In Group B patients, the mean increase in pulmonary volume above apneic functional residual capacity did not alter significantly with respiratory frequency. For a given ventilatory setting, the greater the static respiratory compliance, the greater was the increase in pulmonary volume. These results suggest that mean airway pressure is a principal determinant of arterial oxygen tension during HFJV and that I/E ratio rather than respiratory frequency influences Pa_{O_2} during this type of ventilation. (Key words: Lung: airway pressure; compliance; respiratory failure. Oxygen: blood levels. Ventilation: high-frequency jet.)

IN PATIENTS with acute respiratory failure, conventional ventilation with positive end-expiratory pressure (PEEP) often produces improvement in pulmonary gas exchange. This beneficial effect has been related to an increase in functional residual capacity.[1] Nevertheless, barotrauma[2] and cardiovascular depression[3] often occur when levels of PEEP > 10 cmH_2O are used in conjunction with conventional ventilation.

Since 1967, certain investigators advocating high-frequency jet ventilation (HFJV) have proposed that with the use of very small lung volume displacements these risks could be reduced. In patients with acute respiratory failure, several advantages of HFJV have been claimed: less barotrauma and cardiovascular depression

as a result of lower tidal volume and decreased airway pressure,[4] improved gas exchange,[5,6] adequate alveolar ventilation in patients or animals with bronchopleural fistula,[7,8] and reflex suppression of discoordination between the patient and the ventilator.[9]

Most investigators have used respiratory frequencies ranging from 60–150 breaths/min and inspiratory/expiratory time (I/E) ratios ranging from 0.25–1. Up to now, no definite explanation exists as to why HFJV improves gas exchange in patients with acute respiratory failure. The variations in functional residual capacity and alveolar pressure induced by increasing respiratory frequency or I/E ratio are as yet unknown. The aim of this study was to clarify the determinants of arterial oxygenation during HFJV. The variations in blood gases, pulmonary volume, airway, and alveolar pressures induced either by increasing respiratory frequency or I/E ratio were studied in 24 patients with postoperative respiratory failure.

Methods

PATIENTS

Twenty-four acutely ill patients in the surgical intensive care unit (16 men and eight women, mean age 42 ± 16 yr) were selected according to the following criteria: postoperative respiratory failure defined as a $Pa_{O_2} < 250$ mmHg at Fi_{O_2} 1 under intermittent positive pressure ventilation (IPPV) with radiologic evidence of pulmonary involvement; absence of chronic obstructive lung disease; stable hemodynamic condition and no evident hypovolemia; and presence of an arterial catheter as an integral part of cardiovascular monitoring.

The diagnoses were multiple injuries (13), tetraplegia (3), cerebral hemorrhage (3), cyphoscoliosis (2), gastrointestinal cancer (1), renal transplantation (1), and osteosarcoma (1). The causes of respiratory failure were bacterial pneumoniae (9), acute respiratory distress syndrome (5), aspiration pneumonia (4), pulmonary contusion (3), and fat embolism (3). At the beginning of the study, the patients had a mean Pa_{O_2} of 198 ± 40 mmHg and a mean static respiratory compliance of 61 ± 21 ml/cmH_2O (Fi_{O_2} 1, IPPV). All these patients were sedated with large doses of morphine, as previously described.[10] Informed consent was obtained in all cases from the patient's relatives and authorization was given by the Clinical Investigation Committee of this institution.
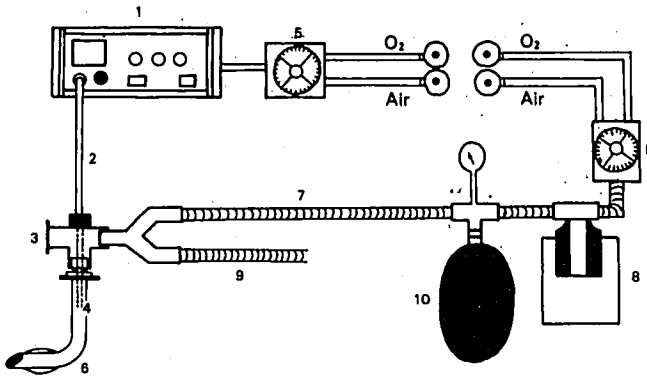
FIG. 1. Circuit for high-frequency jet ventilation. (1) Ventilator MK 800; (2) uncompliant Teflon® tube; (3) three-ways swivel adapter; (4) 14-gauge injector cannula; (5) air oxygen blender; (6) tracheostomy tube; (7) additional gases (30 1/min); (8) humidifier; (9) expiratory line; (10) anesthesia bag.

EQUIPMENT

HFJV was delivered by an IDC Ventilator model MK800 (Acutronic, Medical Systems AG Switzerland). As shown in figure 1, air and oxygen were supplied under a pressure of 30 psi, mixed with a blender and pulsed by the electronically controlled solenoid valve through an uncompliant Teflon® tube 0.7 cm in diameter and 65 cm in length, the pressure wave form being essentially square. This tube was connected to an injector cannula, 14 gauge in diameter and 4 cm in length, inserted into a three-way swivel adapter fixed to the endotracheal or the tracheostomy tube. Gas for entrainment was provided by an open anesthesia circuit connected to the three-way swivel adapter and delivering warmed and humidified gases (30 1/min) at the same Fi_{O_2} as the jet. Three different ventilatory parameters of the ventilator could be changed: respiratory frequency, I/E ratio, and driving pressure. The jet volume was measured directly by connecting the injector cannula to a calibrated water-sealed spirometer. For a frequency of 100/min and a driving pressure of 30 psi, the increase in I/E ratio resulted in an increase in the jet volume: 160 ml for an I/E ratio of 0.25, 240 ml for an I/E ratio of 0.43, and 320 ml for an I/E ratio of 0.67. For an I/E ratio of 0.43 and a driving pressure of 30 psi, the increase in respiratory frequency resulted in a decrease in the jet volume: 240 ml for a respiratory frequency of 100/min, 125 ml for 200/min, 85 ml for 300/min, 60 ml for 400/min, 50 ml for 500/min, and 35 ml for 600/min. During HFJV tidal volume results from the addition of the entrained volume and the jet volume. Because we were unable to measure the entrained volume, the tidal volume corresponding to the different ventilatory settings used during this study could not be reported.

Airway pressure was monitored continously with an air-filled 1.8-mm id polyethylene catheter advanced into the endotracheal tube 10 cm distal to the tip of the injector cannula and connected via the three-way swivel adapter to a calibrated quartz pressure transducer 1290 A Hewlett Packard. Mean airway pressure was obtained by electronic damping of the signal.

MEASUREMENT OF PULMONARY VOLUME ABOVE FUNCTIONAL RESIDUAL CAPACITY AND ALVEOLAR PRESSURE

During the study each patient received morphine 0.5 mg/kg and pancuronium 0.1 mg/kg. Pulmonary volume above apneic functional residual capacity (FRC) and alveolar pressure were measured in the following way: the ventilator was stopped and the uncompliant Teflon® tube and the expiratory circuit of the three-way swivel adapter were clamped simultaneously for a few seconds. This stopped all flow and allowed pressures throughout the lung to become equal at the alveolar pressure level. The expiratory tube then was connected to a calibrated water-sealed spirometer and pulmonary volume was measured by declamping the expiratory line. Five consecutive measurements were made to minimize error caused by clamping at different phases of the respiratory cycle. For a given ventilatory setting, the greatest difference between these measurements never exceeded 250 ml, *i.e.*, approximatively, the greatest gas volume delivered. The mean values of the five measurements then were considered to be a good approximation of the mean pulmonary volume above apneic functional residual capacity (ΔFRC) and of the mean alveolar pressure for a given ventilatory setting.

MEASUREMENT OF STATIC RESPIRATORY COMPLIANCE

At the end of the study, static respiratory compliance was measured using a specially made 2-l syringe. To eliminate any degree of alveolar collapse, the patient's lungs were inflated for 10 s to a tracheal pressure of 30 cmH_2O prior to each set of measurements. Pressure was recorded from the airway, and the volume measured was the amount of displacement of the barrel of the syringe. Patients then were disconnected from the ventilator to allow functional residual capacity to be reached, and slow injections of oxygen were given with 2-s pauses at 50-ml steps. The pressure-volume curve on the inflation and deflation limb was recorded directly on an X-Y recorder 2,000 Omnigraphic Houston Instrument, and static respiratory compliance was calculated between 500 and 1,500 ml on the inflation limb. The normal value was considered to be around 100 ml/cmH_2O in ventilated patients without respiratory disease.[11]

TABLE 1. Respiratory and Hemodynamic Effects of Increasing I/E Ratio (n = 15)

	IPPV 15/min	HFJV 100/min		
I/E ratio	0.43	0.25	0.43	0.67
Pa_{O_2} mmHg	194 ± 40	218 ± 82	312 ± 98*·†	376 ± 86*·†
Pa_{CO_2} mmHg	31 ± 4.0	29 ± 8.1	26 ± 9.0*·†	24 ± 8.2*·†
Volume above FRC (ml)	—	362 ± 126	1,095 ± 433†	1,936 ± 705†
Mean alveolar pressure (cmH_2O)	—	10 ± 1.8	21 ± 6.4†	33 ± 8.2†
Mean airway pressure (cmH_2O)	8.2 ± 2.1	10 ± 2.9	20 ± 5.9*·†	34 ± 4.7*·†
Mean arterial pressure (mmHg)	104 ± 17	110 ± 19	112 ± 19	103 ± 24
Heart rate (beats/min)	110 ± 33	111 ± 35	119 ± 41	123 ± 31

* $P < 0.05$ if compared with control.

† $P < 0.05$ if compared with HFJV 0.25.

PROCEDURES

At control, each patient was under intermittent positive-pressure ventilation (IPPV): Bear I ventilator, tidal volume 15 ml/kg, f 15/min, I/E ratio 0.43, Fi_{O_2} 1. After a steady state of 15 min, arterial pressure, heart rate, mean airway pressure, and arterial blood gas were measured successively. The 24 patients then were ventilated with HFJV at Fi_{O_2} 1 (100% O_2 was used for both the gas delivered by the jet and gas entrainment) and divided into two groups. The control data of the two groups were not statistically different.

In Group A patients (n = 15), respiratory frequency was maintained at 100 breaths/min, and three I/E ratios were used at random: 0.25, 0.43 and 0.67. At each I/E ratio and after a steady state of 15 min, arterial pressure, heart rate, mean airway pressure, arterial blood gas, pulmonary volume above apneic FRC, and alveolar pressure were measured successively.

In Group B patients (n = 9), the I/E ratio was maintained at 0.43, and six respiratory frequencies were used at random: 100/min, 200/min, 300/min, 400/min, 500/min, and 600/min. At each frequency and after a steady state of 15 min, arterial pressure, heart rate, mean airway pressure, arterial blood gas, pulmonary volume above apneic FRC, and alveolar pressure were measured successively.

STATISTICAL ANALYSIS

All values of age, Pa_{O_2}, Pa_{CO_2}, mean pulmonary volume above apneic FRC, mean alveolar pressure, mean airway pressure, mean arterial pressure, heart rate, and static respiratory compliance were expressed as mean ± SD. Respiratory and hemodynamic variables during HFJV were compared with the control values obtained during intermittent positive-pressure ventilation, using Student's t test for paired samples; P values < 0.05 were regarded as significant. Relationships between mean airway pressure and Pa_{O_2}, between mean airway pressure and mean alveolar pressure, as well as between static respiratory compliance and increase in pulmonary

volume above FRC for a given I/E ratio, were analyzed with linear regressions by the least-squares method.

Results

GROUP A

The respiratory effects of increasing I/E ratio are summarized in table 1. If compared with IPPV, HFJV induced an increase in Pa_{O_2}. A significant relationship between HFJV-induced increase in mean airway pressure and HFJV-induced improvement in Pa_{O_2} (fig. 2)
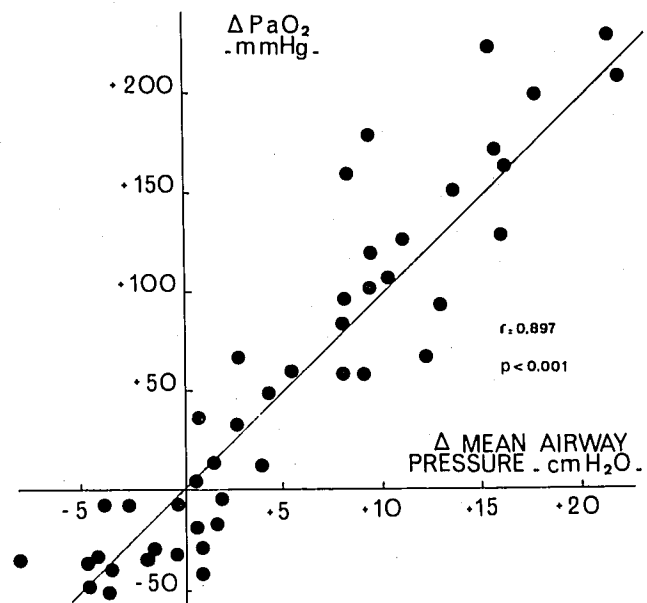
FIG. 2. Relationship between arterial oxygenation and mean airway pressure in the 15 patients of Group A. The increase in Pa_{O_2} (ΔPa_{O_2} = Pa_{O_2} at a given setting of HFJV minus Pa_{O_2} during IPPV) is plotted against the increase in mean airway pressure (Δ mean airway pressure = mean airway pressure at a given setting of HFJV minus mean airway pressure during IPPV). All measures are made at Fi_{O_2} 1. Changes in Pa_{O_2} and mean airway pressure are induced by increasing I/E ratio. For each patient, three different I/E ratio are used: 0.25, 0.43, and 0.67. The line of regression fits the equation y = 10(x) + 1.9 (where y = ΔPa_{O_2} and x = Δ mean airway pressure).
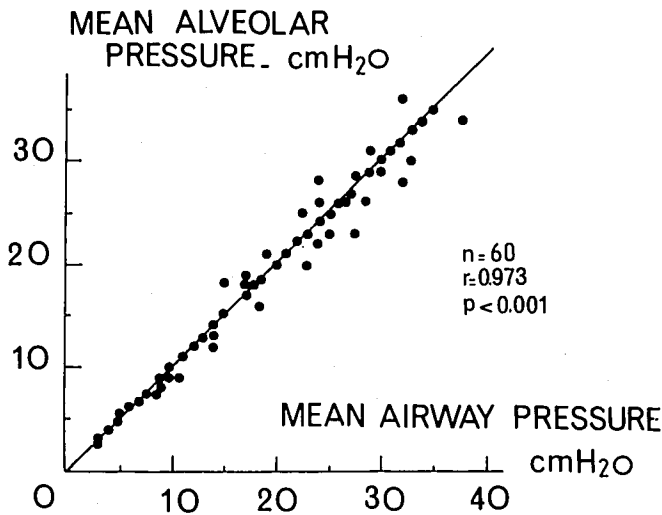
FIG. 3. Relationship between mean alveolar pressure and mean airway pressure in the 15 patients of Group A. Changes in pressures are induced by increasing I/E ratio. For each patient, three different I/E ratio are used: 0.25, 0.43, and 0.67. The dark line is the identity line.

was observed. A significant relationship between mean airway pressure and mean alveolar pressure was noted (fig. 3). During HFJV, the increase in I/E ratio induced a significant increase in Pa_{O_2}, mean pulmonary volume above apneic FRC, mean alveolar pressure, and mean airway pressure. For each I/E ratio, there was a significant relationship between the increase in mean pulmonary volume above apneic FRC (ΔFRC) and the static respiratory compliance: for I/E ratio of 0.25, $r = 0.59$, $P < 0.05$; for I/E ratio of 0.43, $r = 0.85$, $P < 0.001$, (fig. 4); for I/E ratio of 0.67, $r = 0.83$, $P < 0.001$, (fig. 5). Arterial pressure and heart rate remained unchanged throughout the study.
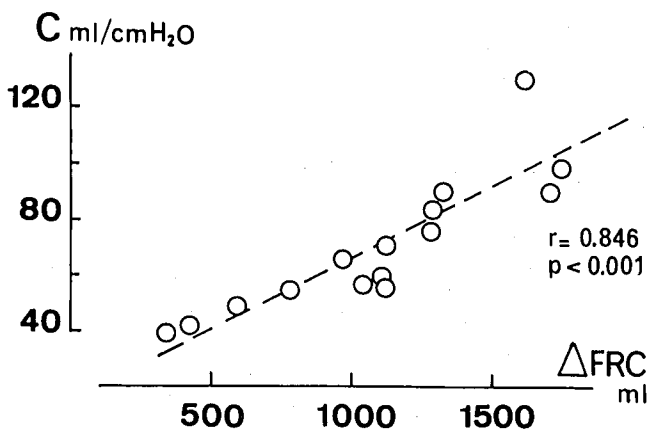

FIG. 4. Relationship between increase in pulmonary volume above apneic FRC (ΔFRC) and static respiratory compliance (C) for an I/E ratio of 0.43 in patients of Group A. The line of regression fits the equation $y = 0.031(x) + 11$ (where y = static respiratory compliance and x = increase in pulmonary volume above apneic FRC).

GROUP B

The respiratory effects of increasing respiratory frequency are summarized in table 2. Compared with IPPV, HFJV induced a significant rise in Pa_{O_2} and in mean pulmonary volume above apneic FRC. During HFJV, the increase in respiratory frequency did not induce any significant change in Pa_{O_2}, mean pulmonary volume above apneic FRC, mean alveolar pressure, and mean airway pressure. Pa_{CO_2} significantly increased, and most of the patients became hypercapnic at frequencies above 400/min. Arterial pressure and heart rate remained unchanged up to respiratory frequencies of 500/min and significantly increased at 600/min.

Discussion

These results demonstrate that HFJV can improve gas exchange in patients with postoperative respiratory failure. The data strongly suggest that HFJV increases arterial oxygenation via an increase in mean airway pressure (fig. 2). In this study we increased mean airway pressure by increasing I/E ratio.[12] The changes in respiratory frequency had no significant effect on mean airway pressure. When I/E ratio was increased, a very close relationship was found between mean airway pressure and mean alveolar pressure. In other words, during HFJV a "PEEP effect" was created at the alveolar level, and the difference between the mean pressure in the upper airways and the mean pressure at the alveolar level never exceeded 4 cmH_2O (fig. 3).

This increase in mean airway pressure resulted in an increase in pulmonary volume. During HFJV the patient's lungs did not expand and deflate continually but were kept at a new resting FRC. We measured the difference between this new FRC and the apneic FRC and we called it the mean pulmonary volume above apneic FRC. An increase in FRC has been described already in high-frequency positive-pressure ventilation, another type of high-frequency ventilation.[13,14] In our patients ventilated by HFJV, pulmonary distention resulted from the increase in mean airway pressure. Mean pulmonary volume above apneic FRC did not change with respiratory frequency and increased with I/E ratio. It must be emphasized that driving pressure remained constant throughout the trial. In our experience, any modification in driving pressure induces a variation in mean airway pressure and consequently in mean pulmonary volume above apneic FRC. HFJV-induced pulmonary distention was not only dependent on ventilatory settings but also on the patient's pulmonary condition: for a given I/E ratio, the increase in pulmonary volume above apneic FRC was related closely to the patient's static respiratory compliance. For an I/E ratio

of 0.43 used by most investigators, the mean increase in pulmonary volume above apneic FRC was 1,095 ml in Group A patients but in individuals it ranged from 320 ml to 1740 ml, according to the initial level of static respiratory compliance: the greater the static respiratory compliance, the greater HFJV-induced pulmonary distention (fig. 4). From this, it is evident that indiscriminate use of high I/E ratio and elevated driving pressure may induce pulmonary overdistention in patients with normal lungs. Consequently, a low I/E ratio should be used when HFJV is indicated for open-chest surgery,[15] diagnostic bronchoscopy,[16] cardiopulmonary resuscitation, or difficult intubation.[17] For the same reasons, HFJV appears to be dangerous in patients with chronic obstructive pulmonary disease. Most of these patients have a marked increase in static respiratory compliance, and even a low I/E ratio may induce pulmonary distention. HFJV, using an I/E ratio of 0.25 and a driving pressure of 30 psi, would increase lung volume to 1,034 ml in a patient with chronic obstructive lung disease and a static respiratory compliance of 150 ml/cmH2O. This would correspond to a mean airway pressure of 7 cmH_2O. It is fundamental to note that the same increase in lung volume could be obtained in a patient with acute respiratory failure and a static respiratory compliance of 44 ml/cmH2O, by using an I/E ratio of 0.67 and a driving pressure of 30 psi (fig. 5). In this case, it would correspond to a mean airway pressure of 24 cmH_2O. This could be beneficial to the patient, because mean airway pressure and not PEEP alone has been regarded by several investigators as the major determinant of arterial oxygenation during acute respiratory failure.[18-20] Our study demonstrates that HFJV improves Pa_{O_2} in postoperative respiratory failure by provoking a "PEEP effect" at the alveolar level. In a previous study, we demonstrated that an increase in mean airway pressure markedly could decrease venous admixture and improve Pa_{O_2} in patients with acute respiratory failure treated by HFJV.‡ A significant decrease in cardiac index also was found, suggesting that capillary unrecruitment could account for the improvement in arterial oxygenation. In this study we found that HFJV could increase the pulmonary volume, which suggests an alveolar recruitment. Finally several mechanisms are probably involved in the HFJV-induced improvement in arterial oxygenation. Our results demonstrate that mean airway pressure is one of the main factors determining oxygenation during HFJV. HFJV should not be undertaken in patients with respiratory failure without continuous monitoring of mean airway

‡ Fusciardi J, Rouby JJ, Bourgain JL, Viars P: High frequency jet ventilation: Respiratory and hemodynamic effects of increments in inspiratory/expiratory ratio. Unpublished data.

TABLE 2. Respiratory and Hemodynamic Effects of Increasing Frequency (n = 9)

	IPPV	HFJV						
	15/min	100/min	200/min	300/min	400/min	500/min	600/min
Respiratory rate	15/min						
Pa$_{O_2}$ (mmHg)	204 ± 41	325 ± 134*	357 ±114*	353 ± 84*	367 ± 88*	373 ± 99*	375 ± 94*
Pa$_{CO_2}$ (mmHg)	29 ± 3.8	22 ± 4.4*	30 ± 9.2†	36 ± 11.8†	44 ± 14.0*†	44 ± 12.4*†	53 ± 7.7*†
Volume above FRC (ml)	—	1,228 ± 684*	1,267 ± 626*	1,278 ± 546*	1,354 ± 422*	1,478 ± 600*	1,563 ±753*
Mean alveolar pressure (cmH₂O)	—	18.4 ± 6.4	20.6 ± 6.9	21.1 ± 6.8	21.2 ± 4.5	23.6 ± 8.6	25.2 ± 9.3
Mean airway pressure (cmH₂O)	11.0 ± 4.8	19.0 ± 6.4*	20.7 ± 6.9*	20.9 ± 6.9*	22.2 ± 5.7*	24.0 ± 7.8	24.2 ± 8.4*
Mean arterial pressure (mmHg)	95 ± 19	100 ± 23	102 ± 21	110 ± 30	108 ± 27	121 ± 32	123 ± 20*†
Heart rate (beats/min)	112 ± 27	113 ± 28	109 ± 30	110 ± 27	112 ± 28	119 ± 33	128 ± 25*†

* P < 0.05 if compared with IPPV.

† P < 0.05 if compared with HFJV 100/min.
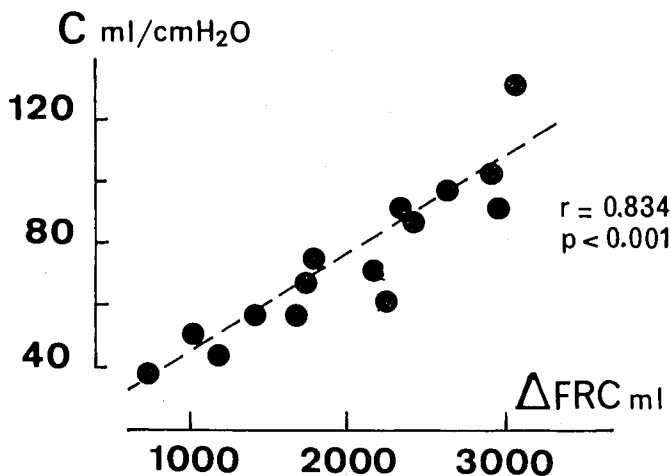
C ml/cmH2O

FIG. 5. Relationship between increase in pulmonary volume above apneic FRC (ΔFRC) and static respiratory compliance (C) for an I/E ratio of 0.67 in patients of Group A. The line of regression fits the equation $y = 0.049(x) + 18$ (where y = static respiratory compliance and x = increase in pulmonary volume above apneic FRC).

pressure. The choice of driving pressure and I/E ratio is important in order to obtain the mean airway pressure that improves arterial oxygenation according to standard criteria.[21,22] It is clear too that the addition of a PEEP valve on the expiratory circuit is unnecessary to obtain an increase in functional residual capacity during HFJV. It could even lead to pulmonary overdistention in patients with compliant lungs.

For a given patient, the level of Pa_{CO_2} was related to respiratory frequency and I/E ratio: the higher the respiratory frequency and the lower the I/E ratio, the higher was the Pa_{CO_2} level obtained. All patients but one had respiratory acidosis, tachycardia, and hypertension at a respiratory frequency of 600/min (table 2). In contrast, all but three had severe hypocarbia with a respiratory frequency of 100/min and an I/E ratio above 0.25 (table 1). Previous studies[23,24] concerning high-frequency oscillation, demonstrated that the rate of elimination of carbon dioxide was related to the product of respiratory frequency and tidal volume below 200/min and to the tidal volume alone at higher respiratory frequencies. Because we were unable to accurately measure tidal volume, we can only hypothesize as to the exact mechanisms involving CO_2 elimination during HFJV. Our results suggest that CO_2 clearance also depended on tidal volume, because the volume delivered by the ventilator decreased when respiratory frequency was increased.

Finally, the hemodynamic consequences of HFJV should be discussed. Improved hemodynamic tolerance has been claimed but never demonstrated in the case of HFJV. Most authors have added a PEEP valve on the

expiratory line and have compared continuous positive pressure ventilation and HFJV $\pm$ PEEP at different levels of mean airway pressure or Pa_{CO_2}.[5,25] Our study clearly shows that different settings of HFJV can induce considerable modifications in pulmonary volume, intrathoracic pressures, and Pa_{CO_2}. Therefore, comparative hemodynamic studies should be undertaken between conventional ventilation with PEEP and HFJV at the same level of mean pulmonary volume above apneic FRC and Pa_{CO_2}. In another study, we demonstrated in patients with compliant lungs that HFJV-induced increase in mean airway pressure could induce a marked decrease in cardiac output.§ On the other hand, our patients tolerated sustained increases in lung volume and alveolar pressure with no resulting hypotension or tachycardia: none of our patients became hypotensive, when we used an I/E ratio of 0.67, which increased lung volume above apneic FRC to 1936 ml and mean alveolar pressure to 33 cmH2O. It is likely that such an increase in intrathoracic pressures during conventional ventilation with PEEP would have provoked arterial hypotension. Nevertheless, further extrapolation to other hemodynamic variables is unwarranted, because cardiac output and cardiac filling pressures were not measured in this study.

In conclusion, HFJV improves Pa_{O_2} in hypoxemic patients with postoperative respiratory failure by increasing pulmonary volume above apneic FRC. This PEEP effect at the alveolar level can be monitored easily by recording mean airway pressure. The level of mean airway pressure increases with I/E ratio and driving pressure and depends little on respiratory frequency. Hemodynamic consequences of sustained increases in intrathoracic pressure as reflected by the measurement of arterial pressure and heart rate seem to be acceptable during HFJV.

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