

Hemodynamic Effects of High-frequency Jet Ventilation in Patients With and Without Circulatory Shock

J. Fusciardi, M.D.,* J. J. Rouby, M.D.,† T. Barakat, M.D.,‡ H. Mal, M.D.,§ G. Godet, M.D.,¶ P. Viars, M.D.‡

Nineteen critically ill patients with acute respiratory failure were studied to compare the hemodynamic effects of continuous positive-pressure ventilation (CPPV) and high-frequency jet ventilation (HFJV) at comparable levels of alveolar ventilation. Patients were divided into three groups: Group 1 included seven patients without circulatory shock in whom mean airway pressure (\overline{Paw}) was slightly higher during CPPV than during HFJV (17.3 ± 3.0 vs. 13.0 ± 2.9 mmHg); Group 2 included six patients without circulatory shock in whom HFJV and CPPV were compared at the same level of \overline{Paw} (19.2 ± 5.0 mmHg); Group 3 included seven patients with circulatory shock in whom HFJV and CPPV were compared at the same level of \overline{Paw} (16.0 ± 3.9 mmHg). The following respiratory frequencies were used in HFJV: Group 1, 200 ± 76 beats/min; Group 2, 238 ± 103 beats/min; Group 3, 286 ± 149 beats/min. In all patients comparable levels of Pa_{CO_2} were obtained with CPPV and HFJV. In Group 1 patients, mean arterial pressure, cardiac index, and stroke index were significantly higher during HFJV. In Group 2 patients, no significant difference was found between HFJV and CPPV. In Group 3 patients, the following hemodynamic variables were significantly higher during HFJV: mean arterial pressure (71 ± 24 vs. 84 ± 23 mmHg), cardiac index (3.6 ± 1 vs. 4.1 ± 1.4 l · min⁻¹ · m⁻²), and oxygen delivery (403 ± 93 vs. 471 ± 124 ml · min⁻¹ · m⁻²). However, Pao_2 was significantly lower (210 ± 105 vs. 155 ± 99 mmHg), fractional inspired oxygen content [F_{I,O_2}] 1 and pulmonary shunt (Q_p/Q_t) was significantly higher (31 ± 12 vs. $36 \pm 11\%$) during HFJV. These results demonstrate that patients with circulatory shock and acute respiratory failure have a more favorable hemodynamic profile during HFJV than during CPPV at identical levels of \overline{Paw} . (Key words: Shock; effects of ventilation. Ventilation: continuous positive pressure breathing; failure; high-frequency.)

DURING EXPERIMENTAL studies on the carotid sinus reflex, Jonzon *et al.* in 1971 demonstrated that the spontaneous-respiration-synchronous blood-pressure-variations (vasomotor waves of the traube-Hering type) could be eliminated by ventilating the animals with high respiratory frequencies (f) and small tidal volumes (V_t).¹ Since that time, high-frequency ventilation has received considerable attention as an alternate mode of mechanical ventilatory support. One theoretical advantage claimed for this method of ventilation is improved cardiovascular

tolerance to sustained increase in intrathoracic pressure. However, several studies in normotensive experimental animals have suggested that hemodynamic function is similar when comparable levels of mean airway pressure (\overline{Paw}) are applied during high-frequency jet ventilation (HFJV) and during continuous positive-pressure ventilation (CPPV).^{2,3} Another study, performed in dogs with PEEP-induced functional hypovolemia, clearly demonstrated a better hemodynamic tolerance of HFJV over CPPV when a lower \overline{Paw} was applied during HFJV.⁴ Thus, it is generally believed that the hemodynamic advantage of HFJV over CPPV is directly related to the extent that \overline{Paw} is lowered. However, a recent experimental study has suggested that HFJV could improve hemodynamic tolerance of sustained increases in intrathoracic pressures, even when \overline{Paw} was equivalent during HFJV and CPPV.⁵ Because of these conflicting results in animals and the lack of any controlled study in humans, we undertook this prospective study to compare the hemodynamic effects of CPPV and HFJV in critically ill patients in acute respiratory failure, both with and without circulatory shock.

Methods

PATIENTS

Nineteen critically ill patients (14 men and five women), admitted in the surgical intensive care unit of La Pitié Hospital for acute respiratory failure, were prospectively studied. Patients were included in the study when they had at least three of the four following criteria of acute respiratory failure: 1) auscultatory evidence of pulmonary rales; 2) radiologic evidence of patchy bilateral alveolar infiltrates; 3) $Pa_{O_2} < 250$ mmHg during intermittent positive-pressure ventilation (IPPV) at a fractional inspired oxygen content (F_{I,O_2}) 1; and 4) static respiratory compliance (CT) < 70 ml · cmH₂O⁻¹. Patients with cardiogenic pulmonary edema, unilateral acute lung disease, chronic obstructive pulmonary disease, and asthma were excluded. Before the study, all patients were receiving large doses of fentanyl ($60 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{day}^{-1}$) to obtain good ventilatory coordination. During the study, they were paralyzed with pancuronium $0.1 \text{ mg} \cdot \text{kg}^{-1}$. Each patient had pulmonary arterial catheters and arterial catheters in place as part of their clinical care.

Patients were divided into three groups. Group 1 included seven patients without circulatory shock, in which

* Assistant Professor of Anesthesiology.

† C. H. A., 1er grade, clinical Chief Critical Care Unit.

‡ Resident of Anesthesiology.

§ Professor and Chairman of Anesthesiology.

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Address reprint requests to Dr. Fusciardi: Département d'Anesthésie-Réanimation, Hôpital de la Pitie, 75651 Paris Cédex 13, France.

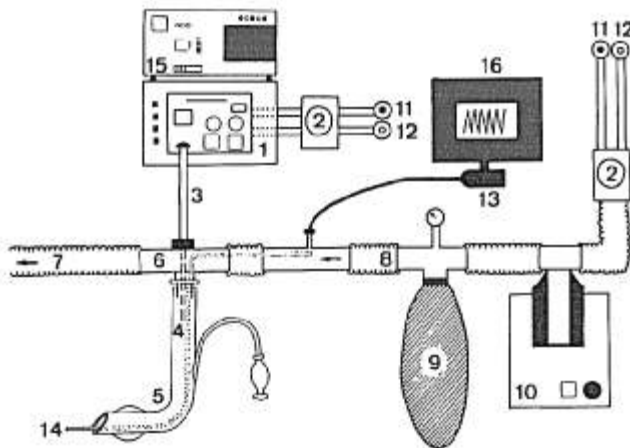


FIG. 1. Circuit for high-frequency jet ventilation. 1: ventilator VS 600 S. 2: blender. 3: connecting tube. 4: 14-gauge injector cannula. 5: tracheostomy tube. 6: three-way swivel adapter. 7: expiratory line (no PEEP valve). 8: additional gases ($30 \text{ l} \cdot \text{min}^{-1}$). 9: anesthesia bag. 10: humidifier. 11: O_2 source. 12: air source. 13: pressure transducer. 14: intratracheal catheter. 15: jet humidifier HH-812. 16: airway pressure.

HFJV and CPPV were compared at different levels of $\overline{\text{Paw}}$ —the causes of respiratory failure were: bilateral bacterial pneumonia (four patients), acute postoperative respiratory failure (two patients), and aspiration pneumonia (one patient). Group 2 included six patients without circulatory shock, in which HFJV and CPPV were compared at the same level of $\overline{\text{Paw}}$ —the causes of respiratory failure were: acute postoperative respiratory failure (three patients), bilateral bacterial pneumonia (one patient), fat embolism (one patient), and aspiration pneumonia (one patient). This last patient was studied twice and was included in Groups 1 and 2. Group 3 included seven patients with circulatory shock, in which HFJV and CPPV were compared at the same level of $\overline{\text{Paw}}$ —the causes of respiratory failure were: acute postoperative respiratory failure (three patients), amniotic embolism (one patient), acute pancreatitis (one patient), extensive pneumonia due to *Pneumocystis carinii* (one patient), and fat embolism (one patient).

In all cases, circulatory shock was related to sepsis and defined as a mean arterial pressure (MAP) ≤ 50 mmHg in the absence of any pharmacologic support. All patients were receiving dopamine ($6\text{--}33 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$); in two patients, dobutamine (6 and $10 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) was administered concurrently; and in one patient, epinephrine ($5 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$), dobutamine ($10 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$), and dopamine ($25 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) were administered concurrently.

Because all patients were receiving large doses of narcotic analgesics and were mechanically ventilated, informed consent was required from the patient's closest

relative. Authorization was given by the Clinical Investigation Committee of this institution.

In Groups 1 and 2, HFJV was administered only for the time of the study, and CPPV was used as prolonged ventilatory support throughout the course of respiratory insufficiency; only one patient in each of the two groups survived. In patients of Group 3, HFJV was used as prolonged ventilatory support throughout the course of respiratory insufficiency; two patients survived.

EQUIPMENT

HFJV was delivered by a ventilator model VS 600 S (Acutronic Medical Systems, AG, Switzerland). As shown in figure 1, air and oxygen were supplied under a pressure of 58 psi, mixed with a blender, and pulsed by an electronically controlled solenoid valve through a noncompliant connecting tube 0.7-cm in diameter and 120-cm in. This tube was connected to an injector cannula 1.8-mm ID and 4-cm in length, inserted into a three-way swivel adapter fixed to the tracheostomy or endotracheal tube. Gas for entrainment was provided by an open anesthesia circuit connected to the three-way swivel adapter that delivered warmed and humidified gases ($30 \text{ l} \cdot \text{min}^{-1}$) at the same FI_{O_2} as the jet. The third part of the three-way swivel adapter enabled exhalation at atmospheric pressure (no PEEP valve). Driving pressure, inspiratory: expiratory (I/E) ratio, and f could be changed independently. Airway pressure was continuously monitored with a 1.65 mm ID polyethylene catheter advanced into the trachea 10 cm distal to the tip of the injector cannula and connected to a calibrated quartz pressure transducer (1290 A, Hewlett-Packard). The catheter tubing and transducer were filled with air, and the entire system was calibrated to a frequency response of 5 Hz. $\overline{\text{Paw}}$ was obtained by electronic damping of the signal. Adequate re-warming and humidification of the gases delivered by the ventilator were provided using an Acutronic Jet Humidifier[®] HH-812 (Acutronic Medical Systems, AG, Switzerland). To achieve 100% relative humidity at 37°C , the water infusion rate ($\text{ml} \cdot \text{h}^{-1}$) was calculated by multiplying the minute volume of the jet gas ($\text{l} \cdot \text{min}^{-1}$) by 2.64.⁶

CPPV was delivered either by a Bear I[®] ventilator or an Ohmeda CPU I[®] ventilator. Gases delivered to the patient could be adequately warmed and humidified using a Bennett humidifier. Airway pressure was continuously monitored with a 1.65 mm ID polyethylene catheter advanced 10 cm into the trachea and connected to a calibrated quartz pressure transducer (1290-A Hewlett-Packard). $\overline{\text{Paw}}$ was obtained by electronic damping of the signal during three ventilatory cycles.

HEMODYNAMIC AND RESPIRATORY MEASUREMENTS

Systemic MAP, intravascular mean right atrial pressure (RAP), mean pulmonary arterial pressure (MPAP), and

pulmonary capillary wedge pressure (PCWP) were measured using a radial arterial cannula and a 7-F triple-lumen, flow-directed, balloon-tipped Swan-Ganz catheter connected to a calibrated quartz pressure transducer (1290 A Hewlett-Packard) positioned at the midaxillary line. Cardiac output was measured by serial determinations using the thermodilution technique and a bedside computer (15055 A Hewlett-Packard). Five serial injections of 10 ml of iced 5% dextrose were made during different moments of the ventilatory cycle in order to average the variations in cardiac output related to inspiratory and expiratory phases. Stroke index (SI), total systemic vascular resistance (SVR), and pulmonary vascular resistance (PVR) were calculated using the following formula:

$$\text{SVR units} \cdot \text{m}^{-2} = \frac{\text{MAP} - \text{RAP}}{\text{CI}}$$

$$\text{PVR units} \cdot \text{m}^{-2} = \frac{\text{MPAP} - \text{PCWP}}{\text{CI}}$$

Systemic and pulmonary arterial blood samples were drawn simultaneously within 1 min following the measurements of cardiac output. PaO_2 , $\text{P}\bar{\text{V}}\text{O}_2$, PaCO_2 , pH , hemoglobin concentration, and oxygen saturations (SaO_2 and $\text{S}\bar{\text{v}}\text{O}_2$) were measured with a Co-oxymeter® IL 182. Calculations with conventional formulas were used to derive the following: pulmonary shunt (Q_s/Q_t), arteriovenous oxygen content difference [$C(a - \bar{v})\text{O}_2$], oxygen consumption ($\dot{V}\text{O}_2$), and oxygen delivery (DO_2).

Prior to the beginning of the study, CT was measured using a specially made 2-1 syringe. Pressure was recorded from the airway using a Validyne® MP 45-I pressure transducer, and volume was measured from the displacement of the barrel of the syringe. Patients were disconnected from the ventilator to allow functional residual capacity (FRC) to be reached, and slow injections of O_2 were given with 2-s pauses at 100-ml steps. The pressure-volume curve on the inflation limb between 0 and 30 cmH_2O airway pressure was directly recorded using an X-Y recorder (2,000 Omnigraphic®, Houston Instruments). CT was considered as the slope of the curve between 500 and 1,500 ml. The opening pressure corresponding to the inflexion point on the inflation limb was determined.⁷

PROCEDURE

Each patient of each group received both CPPV and HFJV in random order. Group 1 comprised seven patients without circulatory shock. $\text{P}\bar{\text{a}}\text{w}$ during HFJV and PEEP during CPPV were adjusted just above the opening pressure, as indicated by the inflexion point of the pressure-

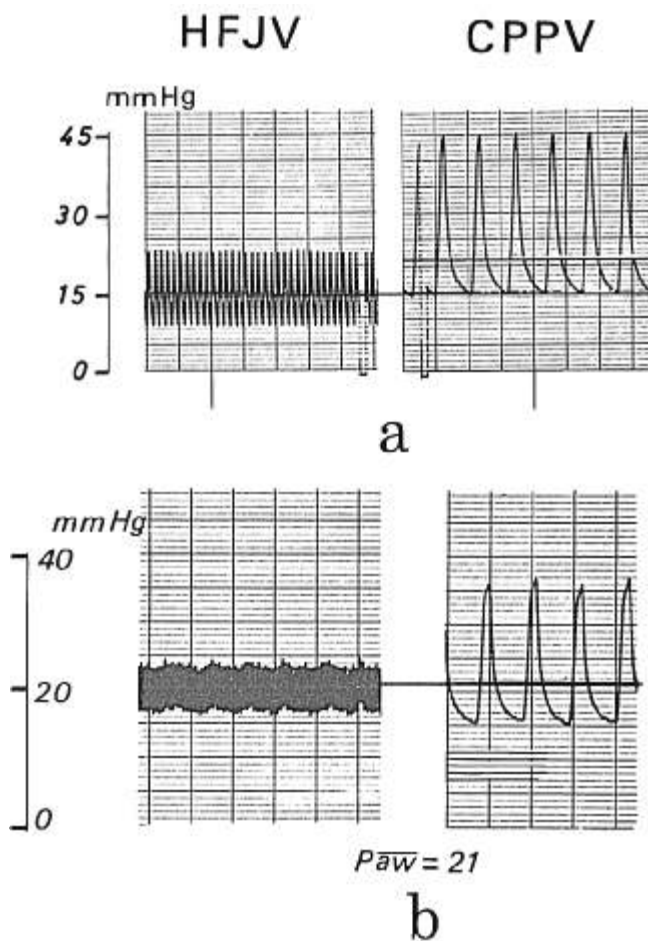


FIG. 2. Airway pressure recording during HFJV and CPPV in a patient with aspiration pneumonia who was studied twice and included in Groups 1 and 2. During the first study (Group 1), $\text{P}\bar{\text{a}}\text{w}$ during HFJV and PEEP during CPPV, were adjusted just above the opening pressure characterizing the inflexion point of the pressure-volume curve. As shown in *a*, $\text{P}\bar{\text{a}}\text{w}$ was consequently higher during CPPV (21 mmHg) than during HFJV (15 mmHg). During the second study (Group 2), $\text{P}\bar{\text{a}}\text{w}$ during HFJV and CPPV was adjusted just above the opening pressure as indicated by the inflexion point of the pressure-volume curve. Consequently, $\text{P}\bar{\text{a}}\text{w}$ was identical during HFJV and CPPV, as shown in *b*.

volume curve.⁷ Consequently, $\text{P}\bar{\text{a}}\text{w}$ was higher during CPPV than during HFJV, as shown in figure 2*a*.

Group 2 comprised six patients without circulatory shock, and Group 3 seven patients with circulatory shock. In these two groups, $\text{P}\bar{\text{a}}\text{w}$ was set just above the opening pressure characterizing the inflexion point of the pressure-volume curve, either during CPPV or during HFJV. Consequently, in a given patient, $\text{P}\bar{\text{a}}\text{w}$ was identical during CPPV and HFJV (fig. 2*b*).

In each patient, identical levels of PaCO_2 were obtained during HFJV and CPPV by modifying f .

During HFJV, the desired level of $\text{P}\bar{\text{a}}\text{w}$ was obtained

TABLE 1. Initial Status of Patients (mean \pm SD) under Intermittent Positive-Pressure Ventilation (FI_O₂ 1)

	Age (yr)	Pa _O ₂ (mmHg)	CT (ml · cm H ₂ O ⁻¹)	MAP* (mmHg)
Group 1 n = 7	49 \pm 17	154 \pm 66	64 \pm 19	100 \pm 12
Group 2 n = 6	49 \pm 24	124 \pm 37	48 \pm 22	101 \pm 17
Group 3 n = 7	49 \pm 16	108 \pm 54	55 \pm 19	39 \pm 10†

CT = respiratory compliance; MAP = mean arterial pressure.

* Without dopamine or dobutamine.

† $P < 0.05$ for Group 3 vs. Group 2 and Group 3 vs. Group 1.

by changing the I/E ratio and/or driving pressure, which are two of the determinants of \bar{P}_{aw} during HFJV.^{8,9} During CPPV, the desired level of \bar{P}_{aw} was obtained by changing the PEEP level, the I/E ratio remaining constant at 0.33.

In all patients, fluid volume expansion as well as increasing the infusion rate of exogenous catecholamines were prohibited throughout the study procedure.

During each mode of ventilation and after a steady state of 20 min at FI_O₂ 1, respiratory and hemodynamic parameters were determined. Data were expressed as mean values \pm SD.

STATISTICAL ANALYSIS

Initial clinical status of the three groups of patients were compared using Kruskal and Wallis' H test and Mann-Whitney's U test. Comparison of each parameter between CPPV and HFJV into the groups was made using Wilcoxon's test for paired data. Comparison of respiratory and hemodynamic data between groups was made using Kruskal and Wallis' H test and Mann-Whitney's U test.

Results

As shown in table 1, the three groups of patients were not significantly different in age, Pa_O₂ (IPPV, FI_O₂ 1), or CT. In contrast, patients in Group 3 had a significantly lower MAP than patients in Group 1 and 2.

In patients in group 1, comparative ventilatory settings and respiratory data are summarized in table 2. In this group, ventilatory settings were chosen to obtain a lower \bar{P}_{aw} during HFJV than during CPPV, and the difference between \bar{P}_{aw} and RAP was significantly higher during CPPV than during HFJV. Q_s/Q_t was significantly higher during HFJV. During both modes of ventilation, Pa_{CO}₂ remained identical. Comparative hemodynamic data are summarized in table 3. MAP and CI were significantly higher during HFJV than during CPPV. The increase in CI during HFJV was related to an increase in SI. All other hemodynamic variables were identical, whatever the mode of ventilatory support.

In patients in Group 2, ventilatory settings were adjusted to obtain the same \bar{P}_{aw} during HFJV and CPPV, and the differences between \bar{P}_{aw} and RAP were identical in both methods. The I/E ratio was significantly higher during HFJV (table 2). There was no significant difference in any of the other respiratory or hemodynamic variables measured during either CPPV or HFJV (tables 2 and 3).

In patients in Group 3, ventilatory settings were adjusted to obtain the same \bar{P}_{aw} during HFJV and CPPV, and the difference between \bar{P}_{aw} and RAP was not statistically different in both methods. The I/E ratio was significantly higher during HFJV. Pa_{CO}₂ was comparable in both modes of ventilation, whereas a significant decrease in Pa_O₂ and a significant increase in Q_s/Q_t were observed during HFJV (table 2). MAP, CI, and \dot{V}_{O_2} were significantly higher during HFJV (table 3). The increase in MAP was observed in six of the individuals, whereas the increase

TABLE 2. Comparative Respiratory Data Between CPPV and HFJV in the Three Groups

Ventilatory mode	Group 1		Group 2		Group 3	
	CPPV	HFJV	CPPV	HFJV	CPPV	HFJV
Frequency (beats/min)	16 \pm 1	200 \pm 76	16 \pm 2	238 \pm 103	16 \pm 2	286 \pm 149
I/E ratio	0.33	0.52 \pm 0.40	0.33	0.70 \pm 0.25†	0.33	0.61 \pm 0.21†
Driving pressure (psi)	—	27 \pm 9	—	34 \pm 7	—	35 \pm 6
PEEP (mmHg)	11.0 \pm 5.0	—	12.0 \pm 4.0	—	10.5 \pm 2.7	—
\bar{P}_{aw} (mmHg)	17.3 \pm 3.0	13.0 \pm 2.9†	19.2 \pm 5.0	19.2 \pm 5.0‡	16.0 \pm 3.9	16.0 \pm 3.9
V_t (ml · kg ⁻¹)	10.3 \pm 2.5	NM*	10.2 \pm 3.0	NM	10.5 \pm 2.9	NM
Pa _{CO} ₂ (mmHg)	38.5 \pm 6.0	39.5 \pm 6.0	39.5 \pm 1.9	39.5 \pm 1.9	37.8 \pm 4.3	37.4 \pm 4.5
Pa _O ₂ (mmHg)	306 \pm 89	215 \pm 113	435 \pm 63‡	410 \pm 63‡	210 \pm 105§	155 \pm 99†§
Q_s/Q_t (%)	21 \pm 5	26 \pm 5†	17 \pm 4	19 \pm 6	31 \pm 12§	36 \pm 11†§
\bar{P}_{aw} - RAP (mmHg)	6.0 \pm 4.9	2.2 \pm 3.1†	3.5 \pm 5.6	3.7 \pm 4.5	4.6 \pm 8.6	3.3 \pm 8.1

CPPV = continuous positive-pressure ventilation; HFJV = high-frequency jet ventilation; I/E = inspiratory/expiratory; \bar{P}_{aw} = mean airway pressure; V_t = tidal volume; Q_s/Q_t = pulmonary shunt; RAP = right atrial pressure.

* NM = not measured.

† $P < 0.05$ HFJV vs. CPPV in each group.

‡ $P < 0.05$ Group 2 vs. Group 1

§ $P < 0.05$ Group 3 vs. Group 2 } comparisons between groups.

TABLE 3. Comparative Hemodynamic Data Between CPPV and HFJV in the Three Groups (mean ± SD)

Ventilatory mode	Group 1		Group 2		Group 3	
	CPPV	HFJV	CPPV	HFJV	CPPV	HFJV
MAP (mmHg)	85 ± 5	96 ± 7*	94 ± 11	98 ± 28	71 ± 24	84 ± 23*
HR (beats/min)	112 ± 4	112 ± 4	113 ± 15	112 ± 18	118 ± 19	122 ± 23
CI (l · min ⁻¹ · m ⁻²)	3.6 ± 0.9	4.1 ± 0.9*	4.2 ± 1.1	4.3 ± 0.8	3.6 ± 1.1	4.1 ± 1.4*
SI (ml · m ⁻²)	32 ± 8	37 ± 7*	37 ± 7	38 ± 5	31 ± 10	34 ± 12
C(a - \bar{v})O ₂ (vol · 100 ml ⁻¹)	4.5 ± 0.9	4.2 ± 1.1	3.9 ± 1.1	3.8 ± 1.3	4.1 ± 1.8	3.7 ± 1.8
\dot{V} O ₂ (ml · min ⁻¹ · m ⁻²)	160 ± 37	167 ± 41	162 ± 54	164 ± 62	129 ± 26	131 ± 23
D _O ₂ (ml · min ⁻¹ · m ⁻²)	528 ± 161	581 ± 172	524 ± 134	521 ± 132	403 ± 93	471 ± 124*
RAP (mmHg)	11.3 ± 1.0	12.4 ± 2.0	15.6 ± 4.0	15.6 ± 4.0	11.4 ± 5.0	12.7 ± 5.0
MPAP (mmHg)	27 ± 2	28 ± 3	31 ± 6	34 ± 7	26 ± 6	30 ± 7*
PCWP (mmHg)	15 ± 1	15 ± 2	17 ± 6	19 ± 10	12 ± 4	13 ± 4
SVR units · m ⁻²	22 ± 3	21 ± 3	20 ± 8	19 ± 5	20 ± 15	20 ± 13
PVR units · m ⁻²	3.2 ± 0.4	3.1 ± 0.4	2.9 ± 0.8	2.5 ± 1.1	4.3 ± 2.5	4.5 ± 2.2

MAP = mean arterial pressure; HR = heart rate; CI = cardiac index; SI = stroke index; C(a - \bar{v})O₂ = arteriovenous oxygen content difference; \dot{V} O₂ = oxygen consumption; D_O₂ = oxygen delivery; MPAP = mean pulmonary artery pressure; PCWP = pulmonary capillary

wedge pressure; SVR = systemic vascular resistance; PVR = pulmonary vascular resistance. See table 2 for additional abbreviations.

* *P* < 0.05 HFJV vs. CPPV in each group.

in CI was observed in all patients (fig. 3). D_O₂ increased during HFJV in all patients despite a higher Q_s/Q_t in each individual (fig. 4). A slight but statistically significant increase in MPAP was observed during HFJV, whereas all other hemodynamic variables remained in the same range during both modes of ventilation.

Discussion

This study demonstrates that when identical levels of \bar{P}_{aw} are applied to patients with acute respiratory failure and circulatory shock, systemic perfusion improves with HFJV compared with perfusion during CPPV. MAP and CI were both significantly higher during HFJV in Group 3, although \bar{P}_{aw} was kept constant and alveolar ventilation was controlled (fig. 3).

Several mechanisms may be invoked to explain these results. Improvement in hemodynamic variables during HFJV may have been secondary to inequality of lung inflation in HFJV and CPPV. In other words, the increase in mean lung volume above FRC may have been lower during HFJV when compared with CPPV, although identical levels of \bar{P}_{aw} were applied. This seems unlikely for several reasons. All patients with a past history of chronic obstructive pulmonary disease or asthma were excluded. Each individual in Group 3 had marked alterations in pulmonary mechanics related to acute respiratory failure, and we previously demonstrated that in case of "stiff lungs," the tracheo-alveolar pressure gradient was minimal during HFJV.^{8,10} Moreover, in case of inadvertent gas trapping due to increased airway resistance—a variable that was not measured in this study—the increase in mean lung volume above FRC would most likely have been higher during HFJV than during CPPV, leading to

improved hemodynamic condition during CPPV. The opposite result was found in this study. If one compares the difference between \bar{P}_{aw} and RAP, which is a good approximation of mean transpulmonary pressure in patients receiving positive-pressure ventilation, identical values were found during HFJV and CPPV. This suggests

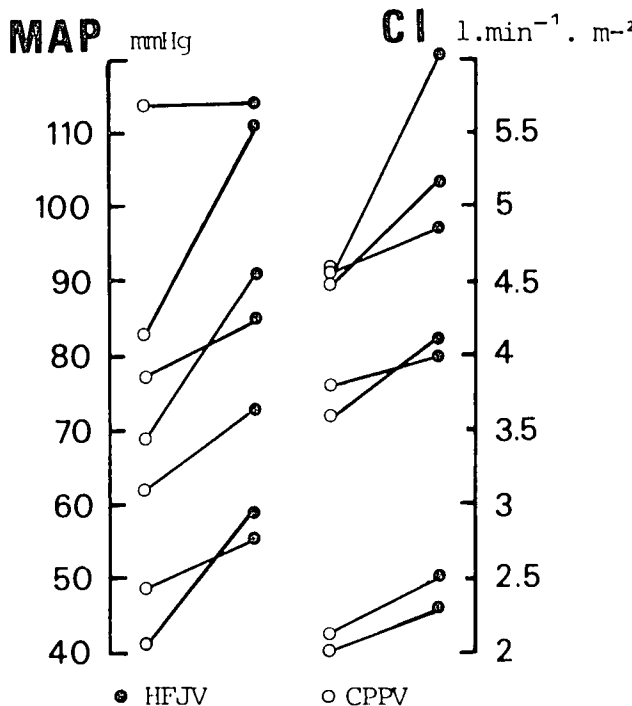


FIG. 3. Comparative values of MAP and CI during HFJV and CPPV in each patient of Group 3. All patients had acute respiratory failure associated with circulatory shock, and HFJV was compared with CPPV at the same level of P_{aCO_2} and P_{aw} .

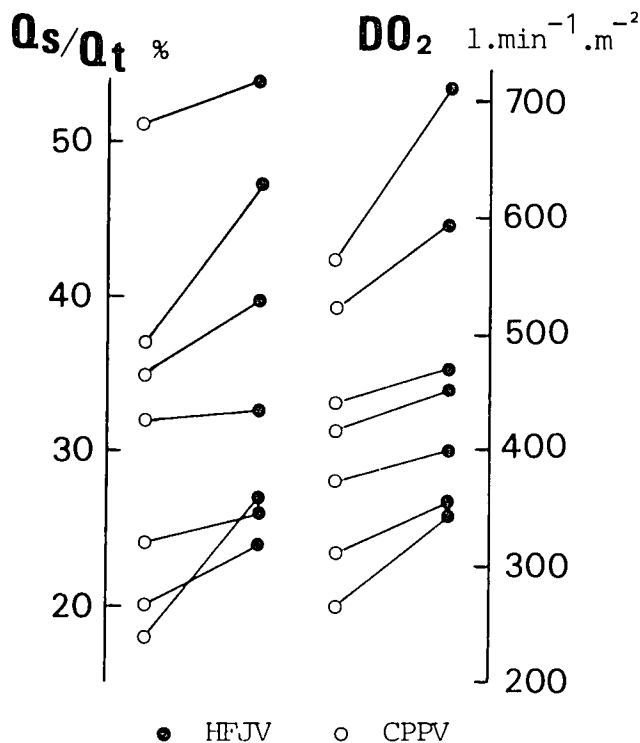


FIG. 4. Comparative values of Q_s/Q_t and DO_2 during HFJV and CPPV in each patient of Group 3. All patients had acute respiratory failure associated with circulatory shock, and HFJV was compared with CPPV at the same levels of P_{aw} and P_{aCO_2} .

that the differences in mean lung volume above apneic FRC were minimal between CPPV and HFJV in patients in Group 3 and cannot account for the improved hemodynamic condition observed during HFJV.

A second explanation for our findings could be that cardiovascular reflex responses originating in the lungs were modified by HFJV. It is well established that positive-pressure lung distention may induce a significant decrease in MAP, not only as a result of mechanical compression, but also as a result of a mechanoreflex-mediated arterial vasodilation.¹¹ This reflex originates in the lungs, where low-threshold stretch receptors stimulated by lung inflation provoke a reflex decrease in blood pressure as a result of systemic vessel dilation, bradycardia, and negative inotropic effect. The magnitude of this reflex arterial vasodilation is proportional to the volume of gas insufflated in the lungs.¹¹ It is also well demonstrated that this cardiac-depressor influence is antagonized by functional arterial baroreceptors¹² and that surpanormal V_t can significantly reduce baroreflex activity.¹³ Because V_t is much smaller during HFJV than during CPPV,¹⁰ two mechanisms could theoretically lead to higher arterial pressure during HFJV: an HFJV-induced decrease in the lung inflation-vasodepressor reflex; and/or an HFJV-induced increase in baroreflex sensitivity. In a recent experimental study in which

P_{aw} was identical during HFJV and CPPV, Chiaranda *et al.* demonstrated a significant improvement in arterial pressure and CI during HFJV when high PEEP levels were applied to dogs with normal lungs.⁵ The authors hypothesized that lung volume changes during each respiratory cycle may have contributed to differences in cardiovascular function. Our results support these data. Another study, by Schreuder *et al.*, goes along these lines by demonstrating in pigs that at comparable levels of PEEP, CI was significantly lower when large V_t were administered.¹⁴ The authors concluded that the cardiovascular depressor reflex elicited by lung stretch is shifted to a higher level of PEEP when ventilating with smaller V_t . Although attractive, these explanations still remain hypothetical and require further confirmation by measuring and comparing baroreflex sensitivity and the lung inflation-vasodepressor reflex during HFJV and CPPV.

In contrast with patients in Group 3, patients in Groups 1 and 2 were free of circulatory shock. A hemodynamic advantage for HFJV could be evidenced in patients in Group 1 only, who had a slightly higher P_{aw} during CPPV than during HFJV. In this group, the difference between P_{aw} and RAP was significantly higher during CPPV (6.0 ± 4.9 mmHg) than during HFJV (2.2 ± 3.1 mmHg), suggesting that the increase in mean lung volume above FRC was higher during CPPV than during HFJV. With HFJV, a significant increase in venous return was observed: when compared with CPPV, SI and CI were significantly higher. Because SVR remained unchanged, MAP was significantly higher. Similar hemodynamic results were found by Otto *et al.* when high PEEP levels were applied to animals with normal lungs, using either CPPV or HFJV.⁴ The authors concluded that the hemodynamic advantage of HFJV was directly related to the extent that P_{aw} was lower with HFJV than with CPPV. This assumption is confirmed by the results observed in Group 2. When identical levels of P_{aw} were applied to normotensive patients during CPPV and HFJV, no difference could be evidenced in any of the hemodynamic parameters measured. On the other hand, it had been clearly demonstrated that HFJV could induce dramatic hemodynamic impairment when extremely high levels of P_{aw} were applied to critically ill patients with acute respiratory failure.¹⁵

It must be pointed out that the improved hemodynamic condition observed during HFJV in Groups 1 and 3 was associated with a significant deterioration in arterial oxygenation. In all patients of these groups, Q_s/Q_t increased and P_{aO_2} decreased when CPPV was switched to HFJV. This appears to be a nonspecific effect resulting from capillary recruitment secondary to an HFJV-induced increase in CT. This phenomenon has been extensively described in patients with acute respiratory failure receiving plasma volume expansion or dopamine.^{16,17} In the current study, however, this deleterious effect was not important enough

to negate completely the HFJV-induced beneficial hemodynamic effect. Because all values of Pa_{O_2} were on the horizontal part of the hemoglobin dissociation curve, oxygen delivery was influenced only by cardiac output. Consequently, in each patient in Group 3, oxygen delivery was significantly higher during HFJV than during CPPV (fig. 4). This explains why all patients in this group were thereafter ventilated with HFJV, which could represent an attractive alternative to CPPV in critically ill patients with acute respiratory failure and circulatory shock.

Finally, the following conclusions can be drawn from this study: 1) In patients with acute respiratory failure without circulatory shock, the hemodynamic advantage of HFJV is directly related to the extent that \bar{P}_{aw} is lower with HFJV than during CPPV. When identical levels of \bar{P}_{aw} were applied during both modes, we failed to demonstrate that HFJV leads to a different hemodynamic profile than CPPV; and 2) In patients with acute respiratory failure and circulatory shock, HFJV induces a lesser degree of hemodynamic impairment than CPPV, even when identical levels of \bar{P}_{aw} are applied during both modes. Further investigations are required to explain the underlying mechanisms of this phenomenon.

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References

1. Jonzon A, Oberg PA, Sedin G, Sjostrand U: High-frequency positive pressure ventilation by endotracheal insufflation. *Acta Anaesthesiol Scand (suppl)* 43:3-43, 1971
2. Carlon GC, Ray C, Miodownik S, Howland WS, Guy Y, Groeger JS: Physiologic implications of high-frequency jet ventilation techniques. *Crit Care Med* 11:508-514, 1983
3. Shuptrine JR, Auffant RA, Gal TJ: Cerebral and cardiopulmonary responses to high-frequency jet ventilation and conventional mechanical ventilation in a model of brain and lung injury. *Anesth Analg* 63:1065-1070, 1984
4. Otto CW, Quan SF, Conahan TJ, Calkins JM, Waterson CK, Hameroff SR: Hemodynamic effects of high-frequency jet ventilation. *Anesth Analg* 62:298-304, 1983
5. Chiaranda M, Rubini A, Fiore G, Giron G, Carlon GC: Hemodynamic effects of continuous positive pressure ventilation and high-frequency jet ventilation with positive end-expiratory pressure in normal dogs. *Crit Care Med* 12:750-754, 1984
6. Doyle HJ, Napolitano AE, Lippman HR, Cooper KR, Duncan JS, Eakins K, Glauser FL: Different humidification systems for high-frequency jet ventilation. *Crit Care Med* 12:815-819, 1984
7. Matamis D, Lemaire F, Harf A, Brun-Buisson C, Ansquer JC, Atlan G: Total respiratory pressure-volume curves in the adult respiratory distress syndrome. *Chest* 86:58-66, 1984
8. Rouby JJ, Fuscuardi J, Bourgain JL, Viars P: High-frequency jet ventilation in postoperative respiratory failure: Determinants of Oxygenation. *ANESTHESIOLOGY* 59:281-287, 1983
9. Benhamou D, Ecoffey C, Rouby JJ, Fuscuardi J, Viars P: Impact of changes in operating pressure during high-frequency jet ventilation. *Anesth Analg* 63:19-24, 1984
10. Rouby JJ, Simonneau G, Benhamou D, Sartene R, Sardnal F, Deriaz H, Duroux P, Viars P: Factors influencing pulmonary volumes and CO_2 elimination during high-frequency jet ventilation. *ANESTHESIOLOGY* 63:473-482, 1985
11. Shepherd JT: The lungs as receptor sites for cardiovascular regulation. *Circulation* 63:1-10, 1981
12. Eckberg DL, Orshan CR: Respiratory and baroreceptor reflex interactions in man. *J Clin Invest* 59:780-785, 1977
13. Stinnett HO: Altered cardiovascular reflex responses during positive pressure breathing. *Fed Proc* 40:2182-2187, 1981
14. Schreuder JJ, Jansen JRC, Versprille A: Contribution of lung stretch depressor reflex to nonlinear fall in cardiac output during PEEP. *J Appl Physiol* 56:1578-1582, 1984
15. Fuscuardi J, Rouby JJ, Benhamou D, Viars P: Hemodynamic consequences of increasing mean airway pressure during high-frequency jet ventilation. *Chest* 86:30-34, 1984
16. Cheney FW, Colley PS: The effect of cardiac output on arterial blood oxygenation. *ANESTHESIOLOGY* 52:496-503, 1980
17. Jardin F, Eveleigh MC, Gurdjian F, Delille F, Margairaz A: Venous admixture in human septic shock: Comparative effects of blood volume expansion, dopamine infusion and isoproterenol infusion on mismatching of ventilation and pulmonary blood flow in peritonitis. *Circulation* 60:155-159, 1979