Intensive Care Med (1988) 14:359–363

Intensive Care Medicine

Original articles

High frequency jet ventilation (HFJV) has no better haemodynamic tolerance than controlled mechanical ventilation (CMV) in cardiogenic shock*

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Received: 20 May 1987; accepted: 17 August 1987

Abstract. Six patients with acute myocardial infarction (AMI) complicated by cardiogenic shock were studied in order to compare the haemodynamic tolerance of controlled mechanical ventilation (CMV) and high frequency jet ventilation (HFJV). The comparative analysis of the two techniques was performed with the same levels of PaO_2 (CMV: 101 ± 13 mmHg; HFJV: 104.2 ± 14 p = ns); and $PaCO_2$ (CMV: 37 ± 1.7; HFJV: 35.7 ± 1.4 p = ns). In this situation the values of mean airway pressure ($\bar{P}aw$) did not differ significantly (CMV: 13 ± 3 cm H_2O; HFJV: 12.6 ± 3.8 cm H_2O) and no statistically significant difference in haemodynamic values was observed. These results demonstrate that in patients with cardiogenic shock, there is no difference between HFJV and CMV in terms of haemodynamic tolerance. Because of the more difficult clinical management of HFJV, this technique does not seem indicated as ventilatory support in patients with cardiogenic shock states.

Key words: Cardiogenic shock – High Frequency Jet Ventilation – Mechanical Ventilation – Haemodynamic effects

In recent years, High Frequency Jet Ventilation (HFJV) has been introduced into clinical use as a ventilatory technique for various pathologic conditions. Despite its spreading application, the role of HFJV in the treatment of respiratory failure is still debated and uncertain, and this is also true for its efficacy in situations where hemodynamic failure is a prominent symptom.

In fact, in contrast with initial reports [2, 12, 15], there is no clear evidence that HFJV is superior to mechanical ventilation at conventional frequencies and tidal volumes with regard to effects on cardiovascular function. Many experimental and clinical studies have been devoted to this issue, with contrasting results, ranging from better tolerance at the same level of $\bar{P}aw$ in a dog model [4] or in clinical practice [13] to no significant differences in patients with respiratory failure [7]. In a recent randomized study [8], the authors compared HFJV and CPPV at the same $\bar{P}aw$ level in two groups of ARF patients one of which was complicated by circulatory shock of septic origin, they clearly demonstrated better hemodynamic tolerance to HFJV in the ARF-circulatory shock patients.

As yet, no randomized study has been made to evaluate hemodynamic tolerance of HFJV in subjects with cardiogenic shock. In patients with AMI and cardiogenic shock, the work of breathing is often increased because of the hypoxia – and acidosis – induced hyperventilation. In conditions of low cardiac output, perfusion of the respiratory muscles may not be sufficient to cope with the increased work: on the other hand, the respiratory muscle demand may represent an important fraction of the cardiac output, increasing peripheral hypoperfusion [1]. In both cases, the maintenance of the spontaneous breathing complicates the already compromised clinical condition; therefore, in these subjects it is usual to employ CMV associated with sedation and muscle paralysis. In this context, the interest of a ventilatory technique capable of both ensuring adequate alveolar gas exchange and causing the least hemodynamic embarassment is obvious. We therefore tested the efficacy of HFJV in a group of patients suffering from cardiogenic shock.

Patients and methods

Six patients (4 males, 2 females) with AMI complicated by cardiogenic shock entered our study (Table

* Presented in part as a communication to the Third Congress of the European Society of Intensive Care Medicine (Hamburg 1986).
† Deceased

Table 1

No.	Sex	Age	Location of AMI	Previous AMI	Outcome
1	♂	59	Postero septal	No	Died
2	♀	49	Anterior	Yes	Died
3	♂	71	Inferior	Yes	Died
4	♂	67	Anterior ext.	No	Died
5	♂	65	Postero septal	No	Survived
6	♀	64	Infero posterior	Yes	Died

1). All patients had a previous history of cardiovascular disease, in particular, three of them had suffered from an AMI in the past.

The diagnosis of AMI was made following standard examinations (EKG, serum enzymes) while cardiogenic shock was suspected in the presence of a reduction of MAP to values 30% less than the patient's normal level, oligoanuria, peripheral cyanosis, cold and clammy teguments.

Soon after admission, all patients were submitted to standard haemodynamic monitoring by means of cannulation of a radial artery and insertion of a balloon tipped, flow directed, pulmonary artery catheter (Deseret, USA) via a subclavian vein. Systemic and pulmonary blood pressures were measured with transducers (Deseret, USA) connected to a Sirecust 404 monitoring system (Siemens Elema, Sweden).

Intravascular pressures were related to atmospheric pressure, the level of the transducers being collimated with the midaxillary line. Cardiac output was measured by thermodilution with the injection of 10 ml of cold 5% glucose in water. The thermistor system was connected to a dedicated module of the Sirecust 404. Five consecutive measurements were made for cardiac output. Boundary data were discarded, the accepted value being the arithmetic mean of the remaining three determinations.

The patients were considered to be in cardiogenic shock when they presented a cardiac index (CI) $<2\,l\cdot min^{-1}\cdot m^{-2}$ and a pulmonary wedge pressure (PWP) ≥ 17 mmHg.

When spontaneously breathing, all patients had a $PaO_2 \leq 50$ mmHg at a $FiO_2 \geq 0.5$.

All patients were sedated with diazepam and fentanyl and paralyzed with pancuronium bromide. Drugs were infused continously during the whole period of the study in order to obtain satisfactory levels of sedation and paralysis.

Intubation was performed in all patients with Deane's endotracheal tubes (Mallinckrodt, FRG) of appropriate size.

In all patients, correction of metabolic acidosis was attempted by the infusion of sodium bicarbonate. Dopamine was infused at a rate ranging from 7 to $10\,\mu g\cdot kg^{-1}\cdot min^{-1}$. The rate of dopamine infusion was never modified during the course of the study.

Once intubated, the patients were connected at random either to a Servo Ventilator 900 C (Siemens Elema, Sweden) or to a Jet-Ventilator (Soxijet, Soxil, Italy), whose characteristics have been described previously [5] (Fig. 1).

The initial ventilatory parameters were: CMV: FiO_2 0.5, RR $14\,min^{-1}$, V_T $10\,ml\cdot kg^{-1}$, I/E 0.5, ZEEP, square wave, no pause time; HFJV: FiO_2 0.5, DP 1.5 bar, I/E 0.20, RR 2 Hz. With the exception of FiO_2, the above parameters were subsequently modified, when necessary, in order to obtain PaO_2 levels within normal limits. During HFJV the $PaCO_2$ adjustment was obtained by modifying the RR (never exceeding 2.5 Hz). PaO_2 was maintained within normal limits acting on the driving pressure or I:E ratio (DP never higher than 1.8 bar and I:E ratio never higher 0.3) as during CMV no PEEP device was applied to the secondary circuit of HFJV. Once the same levels of PaO_2 and $PaCO_2$ were achieved, the haemodynamic status was assessed and blood samples

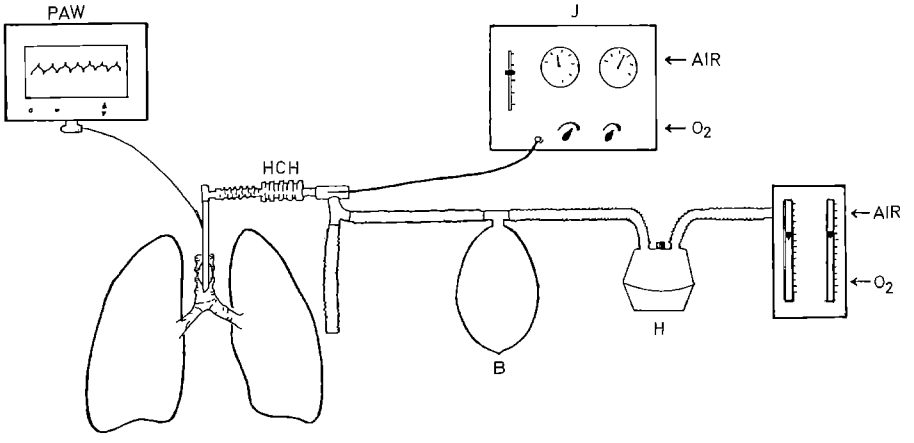

Fig. 1. PAW = Airway pressure; J = Jet ventilator; HCH = Hygroscopic condenser humidifier; B = Bag; H = Humidifier

Table 2. Comparative haemodynamic and respiratory data between CMV and HFJV (X ± SEM)

Parameter	CMV	HFJV	p
Mean arterial pressure – MAP (mmHg)	66 ± 6.43	67.7 ± 5.64	n.s.
Mean pulmonary artery pressure – $\overline{PAP}$ (mmHg)	31 ± 2.86	31 ± 2.56	n.s.
Pulmonary wedge pressure – PWP (mmHg)	18 ± 1.96	19.6 ± 2.71	n.s.
Right atrial pressure – RAP (cm H_2O)	14 ± 3.47	16.3 ± 3.39	n.s.
Heart rate – HR (beats·min^{-1})	103 ± 6.36	99.17 ± 5.5	n.s.
Cardiac index – CI (l·min^{-1}·m^{-2})	1.9 ± 0.25	2.1 ± 0.25	n.s.
Total pulmonary resistance – TpR (dynes·sec^{-1}·cm^{-5})	803 ± 103.6	708 ± 76	n.s.
Total peripheral resistance – TPR (dynes·sec^{-1}·cm^{-5})	1640 ± 95.5	1511 ± 94	n.s.
Mean airway pressure – $\bar{P}AW$ (cm H_2O)	13 ± 3	12.6 ± 3.8	n.s.
Arterial oxygen partial pressure – PaO_2 (mmHg)	101 ± 13.4	104.2 ± 14	n.s.
Arterial carbon dioxide partial pressure – $PaCO_2$ (mmHg)	37 ± 1.78	35.7 ± 1.4	n.s.
Arterial pH – pHa	7.37 ± 0.2	7.38 ± 0.2	n.s.
Mean venous oxygen partial pressure – $P\bar{v}O_2$ (mmHg)	30.6 ± 2.28	29.9 ± 1.97	n.s.
Mean venous oxygen saturation – $S\bar{v}O_2$ (%)	60.9 ± 4.1	58.5 ± 2	n.s.
Venous admixture – $\dot{Q}_{VA}/\dot{Q}_T$ (%)	20 ± 1	17.4 ± 0.1	n.s.
Arterio-venous oxygen difference – a-$\bar{v}O_2$D (ml %)	5.06 ± 0.5	5.52 ± 0.25	n.s.

were taken simultaneously from the radial and pulmonary arteries.

Contemporarily, $\bar{P}aw$ was measured with a 1.65 mm internal diameter non-compliant catheter connected to the dedicated channel of the Deane's endotracheal tube. The other end of the catheter was connected to a Gould Statham P50 Transducer and the monitoring system was filled with air. $\bar{P}aw$ was obtained by electronic damping of the signal.

Blood-gas analyses were performed using an ABL3 blood-gas machine (Radiometer Copenhagen, DK). O_2 saturation and Hb content were measured with an IL Oxymeter (Instrumentation Laboratory). Derived variables were obtained from standard equations.

Once the measurements were performed, each patient was submitted to the other form of respiratory support the parameters of which were modulated until gas exchange was similar to the previous values. At this moment, a second series of determinations was made, as described above.

The time between the two measurements was approximately 30 min.

Informed consent was obtained from the patients' legal guardians before commencing the study.

The data obtained have been evaluated by means of the Student's t test for paired data. A $p < 0.5$ was considered significant.

Results

Table 2 shows the data obtained, expressed as the mean($\bar{X}$) of the "N" observations ± SEM.

With both types of ventilatory support, comparable gas exchange within normal limits could be achieved with slight modifications of the ventilatory parameters. For PaO_2 and $PaCO_2$ values not significantly different, similar levels of $\bar{P}aw$ were obtained with both CMV and HFJV. The comparative analysis of the hemodynamic variables did not show any statistically significant difference. In particular, CI showed a slight tendency towards higher values during HFJV in 5 out of 6 patients, but this was not sufficient to achieve statistical significance (Fig. 2).

Also $P\bar{v}O_2$ measurements were not different with both CMV and HFJV, confirming a similar pattern of peripheral perfusion during both forms of ventilation.

Discussion

The aim of this study was to compare the haemodynamic tolerance of HFJV and CMV in patients with cardiogenic shock. Our experimental protocol was structured to avoid the negative effects of hypoxia and hypercapnia on the myocardium by main-

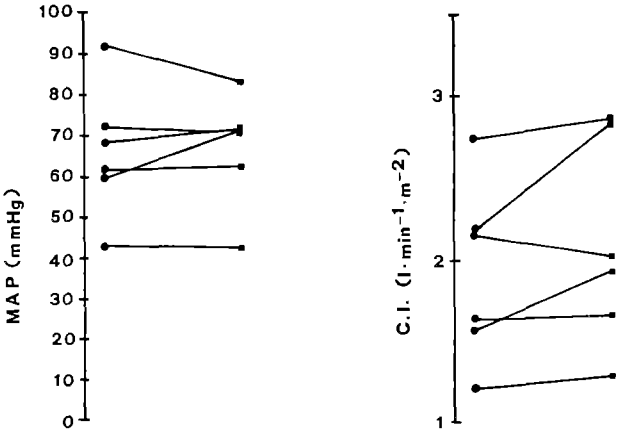

Fig. 2. Values of MAP and C.I. during CMV and HFJV at the same level of PaO_2, $PaCO_2$ and PAW. ● CMV; ■ HFJV

taining normal gas exchange with both types of ventilatory support. In these conditions of "security", easily achieved with CMV and HFJV, no difference was detected between the two forms of ventilation with regard to the haemodynamics. In particular, $\bar{P}aw$ was considered as a dependent variable, as the therapeutic goal was to obtain satisfactory levels of PaO_2 and $PaCO_2$, whereas an essential experimental feature was to induce an equivalent gas exchange with both types of mechanical ventilation. These two conditions having been satisfied, we observed similar values of $\bar{P}aw$, as well as comparable haemodynamic function.

Our finding need to be discussed further, since there is still controversy about the interactions between $\bar{P}aw$ and the haemodynamics when HFJV and CMV are compared.

In general, there is good agreement that HFJV has a better impact on the haemodynamic functions than CMV when a lower $\bar{P}aw$ is employed. This has been demonstrated in hypovolaemic animals [9] and in humans with ARF [7]. In the latter, however, $\dot{Q}s/\dot{Q}t$ increased and PaO_2 decreased during HFJV, these effects being attributable to a combination of capillary recruitment (increased CO) and loss of airspace (decreased FRC). This is hardly surprising, as, in HFJV, $\bar{P}aw$ is known to affect pulmonary volumes and cardiovascular function with features that are similar to those acting during CMV [10]. Furthermore, studies comparing CMV and HFJV at the same $\bar{P}aw$ level have shown no difference in gas exchange and haemodynamics in normal experimental animals [3] and in humans with ARF [8]. In sharp contrast, a recent experimental study has clearly demonstrated that in dogs rendered hypovolaemic by the application of high PEEP levels, HFJV had a better haemodynamic tolerance than CPPV at the same $\bar{P}aw$ levels [4], while in patients with ARF and circulatory shock, CPPV and HFJV at equivalent $\bar{P}aw$ values induced significantly different haemodynamic embarassment, with both CI and MAP higher during HFJV; concomittantly, PaO_2 decreased and $\dot{Q}s/\dot{Q}t$ increased, the net result being an increase in oxygen delivery [8].

Thus, in the face of failing cardiovascular function, HFJV seems to exert lesser impact on haemodynamics, due to factors independent of $\bar{P}aw$ levels.

A possible explanation for this is given on the basis of the fact that tidal volumes in HFJV are much less than during CMV. This latter could impair cardiovascular function via reflexes originating from stretch receptors in the lungs, activated by the cyclic hyperinflations of CMV causing vasodilation, bradycardia and inotropic impairment [6, 11].

In the patients studied by Fusciardi et al., this effect could have been magnified by the presence of parenchymal ARF. In this condition, the lungs are dishomogeneously injured. During mechanical ventilation, the distribution of the inspired gas is preferentially directed towards the "healthy" alveoli, and this is true for both CMV and HFJV [9]. During the latter, however, the alveolar volume oscillates around a mean value, which with the former cyclic volume variations are enhanced in the alveoli with more favourable time constants. In these circumstances, the stimulation of the parenchymal stretch receptors might be supramaximal, as the magnitude of the response is known to be proportional to the degree of alveolar inflation [11].

Nonetheless, this mechanism alone does not seem to be sufficient to induce major haemodynamic embarassment as in patients with ARF not complicated by circulatory shock, no significant difference could be detected between CMV and HFJV at the same $\bar{P}aw$ levels [8].

In a group of ARDS patients a higher circulatory impairment during HFJV was reported [9]: however in this series, $\bar{P}aw$ levels were significantly higher during HFJV, and the patients were not in shock.

Failing cardiovascular function therefore seems to be necessary to obtain better haemodynamic tolerance of HFJV. The question then arises as to why our patients did not react favourably to HFJV. Two factors might explain our results: (1) the absence of parenchymal ARF and, (2) the etiology of the shock state.

In effect, our patients were free from evident abnormalities of the mechanical properties of the lungs; therefore, a more even distribution of inspired gas during CMV might have avoided alveolar hyperinflation and, thus the pathophysiologic sequence starting with the stimulation of lung stretch receptors.

Concerning the etiology of the shock condition, in the group of animals studied by Chiaranda et al., this was a combination of hypovolaemia (decreased venous return) and cardiac failure (decreased biventricular compliance by the PEEP-induced increase of lung volumes), while in the patients of Fusciardi et al., hypotension was secondary to a septic condition and peripheral perfusion could be maintained only with large doses of inotropic drugs. In both cases, the cyclic hyperinsufflation induced by CMV could have caused further deterioration of the cardiac performance synchronously with mechanical inspiration.

In our patients, on the contrary, gross overdistension of the lungs was avoided by the selection of the ventilatory parameters, both in CMV (ZEEP, standard Vt and I/E) and in HFJV (relatively "low" frequencies, low DP and I/E). Moreover, owing to the 'pure' cardiogenic nature of the shock, a moderately increased intrathoracic pressure could have improved left ventricular ejection fraction by decreasing aortic im-

pedance, this effect being dependent on $\bar{P}aw$, no matter how it is generated [10]. In this sence, our results are in accordance with those of Räsänen, who, in a recent paper, demonstrated equivalent haemodynamic patterns when CMV, IMV and HFV were adjusted to deliver the same $\bar{P}aw$ [11].

We did not apply PEEP during CMV; thus we did not use jet frequencies higher than 2 Hz in order to prevent a gross auto-PEEP effect.

We conclude that:

1. in patients with cardiogenic shock, there is no difference between HFJV and CMV in terms of haemodynamic tolerance when the two types of mechanical ventilation are modulated to give equivalent gas exchange;
2. consequently because of the more difficult clinical management of HFJV, it is not indicated as ventilatory support in patients with cardiogenic shock.

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