

Combined High-frequency Ventilation (CHFV) in the Treatment of Acute Lung Injury-A Case Report

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Abstract

A 22-year-old man was admitted to a district general hospital with chest injuries, a ruptured spleen and limb fractures, sustained in a road traffic accident. After an emergency splenectomy, the patient developed unilateral pulmonary oedema with hypoxaemia which was resistant to both conventional controlled mechanical ventilation (CMV) and independent lung ventilation (ILV). He was transferred to a specialist cardiothoracic unit where high frequency jet ventilation (HFJV) also failed to achieve adequate oxygenation. Combined high frequency ventilation (CHFV), using high frequency pulses from a Bromsgrove Penlon Jet ventilator superimposed onto small tidal volumes from an Engstrom Erica improved oxygenation rapidly to allow decreases in inspired oxygen fraction (FiO_2), peak airway pressure (PA WP) and positive end expiratory pressure (PEEP). Progressive weaning from ventilatory support was then possible over five days. CHFV is a valuable technique in the treatment of acute catastrophic lung injury and needs wider recognition.

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Introduction

The role of ventilatory support in acute lung injury is supportive, whilst the damage to alveolar-capillary membranes resolves and alveolar stability is restored.¹ The optimum mode of support varies with individual patients, but none can reliably prevent progression of acute lung injury and high frequency ventilation (HFV) has been disappointing in this respect.² The conventional approach to artificial ventilation involves the use of tidal volume preset mandatory positive pressure ventilation in combination with positive end expiratory pressure (PEEP).³ The ideal level of PEEP remains controversial and this combination predisposes to high peak inspiratory airway pressure (PAWP) in the presence of a poorly compliant lung. A safe PAWP cannot be identified.^{4,5} A conventional tidal volume, when applied to the residual component of functional lung tissue in acute lung injury, may result in regional overdistension of the remaining functional alveoli.⁶ Because of these drawbacks in conventional modes of ventilation, there is a continuous search for more efficient means of respiratory support. These, in general, attempt to maintain mean airway pressure and lung volume, on which oxy-

genation depends,^{7,8} while maintaining cardiac output and avoiding high PAWP, to minimize ventilator induced pulmonary damage.

Compared to conventional controlled mechanical ventilation (CMV), the reduced PAWP resulting from HFV may be associated with less barotrauma⁹ and cardiac depression,¹⁰ but both methods can fail to provide satisfactory gas exchange in some patients with catastrophic lung injury. We describe such a patient, who improved dramatically when CMV was combined with HFV.

Case Report

A 22-year-old man was admitted to a district general hospital with multiple injuries after a road traffic accident. A chest drain was immediately inserted because of respiratory distress with multiple rib fractures and absent breath sounds over the right hemithorax. A subsequent chest radiograph (CXR) showed no residual pneumothorax and the lung fields appeared reasonably clear. Intravenous fluids were given to restore and maintain circulatory volume, whilst further assessment revealed intra-abdominal bleeding and long bone fractures. After initial resuscitation, a nasogastric tube

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was inserted and the patient was transferred to theatre for emergency laparotomy and immobilisation of the fractures.

Before induction of anaesthesia, the patient's oxygen saturation (SpO_2) was 85%, breathing an inspired oxygen fraction of 1.0 ($\text{FiO}_2 = 1.0$); after rapid sequence induction, intubation and controlled mechanical ventilation, SpO_2 improved to 95% ($\text{FiO}_2 = 0.8$). During surgery, a ruptured spleen was removed and the patient was given a total of 14 units donor blood, 4 units of fresh frozen plasma and crystalloids 2.5 L to maintain cardiovascular stability. At the end of surgery, the presence of pulmonary oedema was noticed with pink frothy secretions in the endotracheal tube, arterial hypotension, increased central venous pressure (cvp) and oxygen desaturation to below 90% ($\text{FiO}_2 = 1.0$, PaO_2 and $\text{PaCO}_2 = 6.5$ and 7.0 kPa, respectively). Airway inflation pressures were high thus likely exacerbating the haemodynamic compromise. Intravenous frusemide 120 mg was given, a glyceryl trinitrate patch 10 mg applied, and the patient's blood pressure and cardiac output were maintained with infusions of adrenaline $0.1 \mu\text{g kg}^{-1} \text{min}^{-1}$ and dopamine $3 \mu\text{g kg}^{-1} \text{min}^{-1}$. The possibility of cardiac tamponade was excluded by pericardiocentesis and a subsequent echocardiogram. A repeat chest radiograph showed that a "white-out" appearance had developed over the right hemithorax since admission (Fig. 1). The pulmonary oedema and hypoxaemia increased until a left medium size double-lumen endobronchial tube (Robertshaw) was inserted to institute independent lung ventilation (ILV); this confirmed the decreased compliance and copious secretions in the right lung. Oxygenation improved slightly but remained unsatisfactory (SpO_2 90%). At this time, 12 hours after the accident, the patient was transferred to a specialist cardiothoracic unit where extracorporeal membrane oxygenation (ECMO) could have been possible if further deterioration had occurred.

After a 60-minute ambulance journey, the patient was hypoxaemic, hypotensive, hypovolaemic and anuric with profuse pulmonary oedema secretions pouring from both sides of the double-lumen endobronchial tube. Colloidal solutions (4.5% albumin, 1 L and blood, 2 units) were given to restore arterial and venous blood pressures, but the hypoxaemia persisted ($\text{SpO}_2 = 70\%$, $\text{FiO}_2 = 1.0$, $\text{PEEP} = 10$ cm H_2O). The patient was sedated with intravenous propofol 50 to 100 mg h^{-1} and morphine 1 mg h^{-1} and atracurium 0.5 mg $\text{kg}^{-1} \text{h}^{-1}$ to maintain neuromuscular blockade. Very high airway pressures ($\text{PAWP} \approx 65$ to 70 cm H_2O) were needed to ventilate the lungs, but the position of the double-lumen tube appeared satisfactory. A pulmonary artery catheter was inserted and haemodynamic parameters were cardiac output 3.6 L min^{-1} , pulmonary artery wedge pressure of 10 mm Hg and systemic vascular resistance of 1100 dyne

cm^{-5} . High frequency jet ventilation (HFJV) was tried on the right lung, using a Bromsgrove Penlon ventilator* [rate (f) = 180 min^{-1} , driving pressure = 35 cm H_2O , inspiratory time = 35%, $\text{PEEP} = 10$ cm H_2O] whilst the left lung was ventilated conventionally using the Engstrom Erica (f = 16 min^{-1} , tidal volume, $V_t = 700$ ml, $\text{PEEP} = 0$). SpO_2 increased to 90% ($\text{FiO}_2 = 1.0$). The double-lumen tube was no longer considered necessary and was changed to a single lumen tube (Mallinckrodt Hi-lo jet) with HFJV being applied to both lungs; SpO_2 decreased to below 80% ($\text{FiO}_2 = 1.0$). Conventional CMV was tried again with varying flow rates, inspiratory/expiratory (I/E) ratios and levels of PEEP, but without an improvement in oxygenation. A combination of conventional CMV plus HFJV was then applied simultaneously, with a dramatic improvement in oxygenation and no adverse cardiovascular effects (ventilator settings: CMV-f = 18 min^{-1} , $V_t = 100$ ml, $\text{PEEP} = 10$ cm H_2O , I/E ratio = 2/1, $\text{FiO}_2 = 1.0$, and HFJV-f = 180 min^{-1} , driving pressure = 35 cm H_2O , inspiratory time = 35% of cycle, $\text{FiO}_2 = 1.0$). The patient's oxygenation continued to improve rapidly, allowing FiO_2 and PEEP to be progressively reduced to 0.7 and 5 cm H_2O respectively, within 1 hour ($\text{PaO}_2 = 15$ kPa, $\text{PaCO}_2 = 3.7$ kPa). During the same period, PAWP fell from over 65 to 40 cm H_2O , mean airway pressure remained unchanged at 16 cm H_2O and the patient began to produce urine again after stimulation with frusemide and mannitol. He developed a small air leak from the right lung and surgical emphysema over his face and trunk, but these problems resolved as it became possible to reduce the HFJV driving pressure.

Two days after the accident it was possible to wean the patient from CHFV to CMV, without increasing FiO_2 , PEEP or inflation pressures to dangerous levels. The patient was then taken to the operating theatre for internal fixation of fractures to the left femur and right humerus. However, during surgery the FiO_2 needed to be increased to 1.0 (with $\text{PEEP} = 5$ cm H_2O) to maintain satisfactory oxygenation. When the patient returned to the intensive care unit (ICU), CHFV was resumed on previous settings and gas exchange improved once again. The FiO_2 , PEEP and driving pressure were progressively decreased over the next 48 hours until HFJV was withdrawn. Ventilatory support was needed for a further 24 hours, using synchronized intermittent mandatory ventilation (SIMV), with pressure support and PEEP, until it was considered safe to remove the endotracheal tube. On the seventh day after his accident, the patient was transferred to a general orthopaedic ward and has subsequently made a full recovery.

Discussion

The term "combined high-frequency ventilation" (CHFV) was first used in 1983, to describe the combination of high-frequency positive-pressure (60



Fig. 1a. Chest radiograph on admission to the Cardiothoracic unit

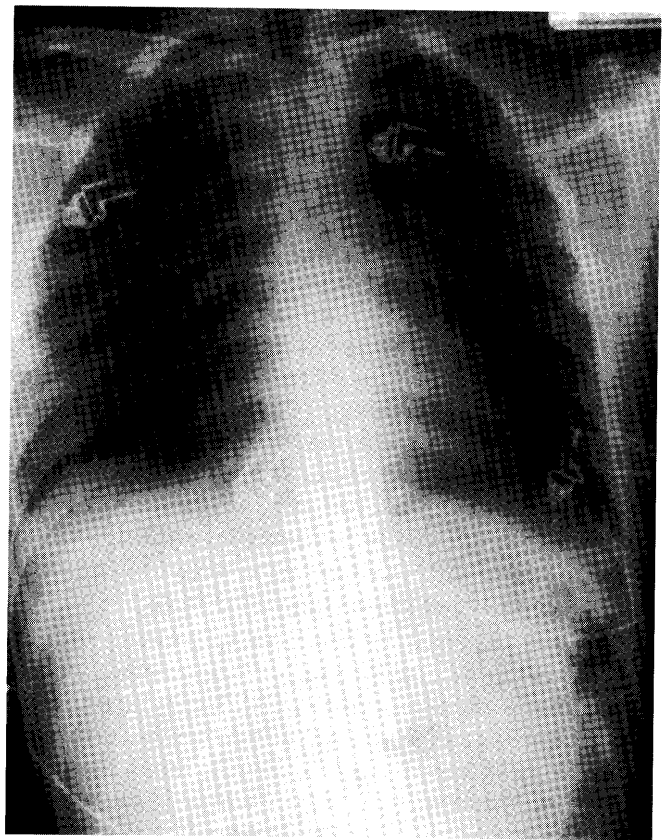


Fig. 1b. Chest radiograph prior to discharge to the general ward.

breaths min^{-1}) and high-frequency oscillation (HF03000 breaths min^{-1}) in the management of 7 patients with severe adult respiratory distress syndrome (ARDS).¹² Since then, the term CHFV has been applied more widely to include combinations of conventional controlled mechanical ventilation (CMV) with either high-frequency jet ventilation (HFJV)¹³ or HFO.¹⁴⁻¹⁶ Recurrent findings in these reports include improved oxygenation, reduced airway pressures and maintained cardiac output when compared to CMV alone. However, prospective, controlled and randomized studies are extremely difficult to perform in critically ill patients; one such multicentre study was initiated,¹⁷ but was abandoned because of the difficulty in recruiting suitable patients (personal communication: Siemens-Elma E B, Solna, Sweden). In a prospective study which was not randomized, CHFV resulted in the survival of 8 out of 35 patients (23%) who were clinically and physiologically indistinguishable from a group of 65 non-survivors treated with CMV alone.¹⁵ In surviving CHFV patients, the decrease in PAWP permitted a sustained, or increased cardiac output with a rise in the oxygen supply demand ratio. In a prospective study of patients with catastrophic lung disease admitted to Scandinavia over a 4-year period (1983 to 1987), 105 patients who were unresponsive to conventional ventilation were studied.¹⁶ Patients were initially ventilated on inverse ratio ventilation (IRV) and

were changed to CHFV if IRV failed to provide a significant improvement. In 30 out of 38 patients who needed CHFV, FiO_2 could be reduced to a median of 0.6 and PAWP to a median value of 50 cm H_2O . However, only 12 patients survived more than 3 weeks and only 4 were discharged. Berner et al¹³ reported on the use of CHFV in 6 children unresponsive to CMV; there was sustained increase in PaO_2 , accompanied by a reduction in PAWP and PEEP. Persistent improvement on CHFV allowed resumption of CMV and survival in 4 patients, but 2 children died of multiple organ failure despite maintenance of satisfactory gas exchange on CHFV. These reports illustrate the negative bias which is introduced in the evaluation of CHFV as this mode of ventilation is often only introduced as a last resort, in patients with catastrophic lung disease unresponsive to conventional ventilation,¹⁵ inclusive of pressure controlled-inverse ratio ventilation (PC-IRV).

In CHFV, the superimposed HFV pulses are thought to improve gas distribution by enhanced convection and use of frequency-dependent pendelluft effects, while the basal breaths provide sufficient pressure to open and stabilize airways.^{15,18} HFJV is associated with a degree of auto-PEEP effect¹⁹ which is probably one of the essential mechanisms by which it improves oxygenation. This is difficult to quantify clinically, but may explain why CHFV allows a decrease in "external" PEEP.²⁰ The im-

provement in gas exchange at lower airway pressures may reduce the complications from barotrauma and cardiovascular disturbances. It is logical to postulate that the wider application of CHFV might reduce the morbidity which is associated with long-term positive pressure ventilation in intensive care units.^{4,6} In the present case, the severity of the underlying lung injury has been questioned by other observers because the patient only required artificial ventilation for five days. An alternative explanation for this rapid recovery could be that early CHFV prevented further lung injury from barotrauma. Re-expansion pulmonary oedema may have played a role in the aetiology of acute lung injury in this case,²¹ although in these cases improvement is more rapid following conventional therapy. Although massive aspiration of gastric contents may have contributed to the lung injury, this was considered unlikely as there was an absence of pulmonary sepsis requiring antibiotics and chest aspirates were pink and frothy.

CHFV is known to be beneficial in patients with increased extravascular lung water²² and this was a notable feature in the case reported. CHFV may also be used as a prognostic tool in severe ARDS, where survivors are more likely to improve within 24 to 36 hours than non-survivors.¹⁵ More recently, CHFV was used successfully to treat a pregnant asthmatic with atypical pneumonia when conventional ventilation had failed; this was in spite of relative contraindications to this technique in a patient with prolonged respiratory time constants at risk of gas trapping.^{23,24} We suggest that CHFV is worth a carefully monitored trial in any patient with unacceptable hypoxaemia at an acceptable level of plateau airway pressure, after attempting volume or pressure-cycled ventilatory modes, with or without inverse-ratio ventilation, or postural manoeuvres.²⁵ To commence CHFV, the ventilatory settings need to be tailored for each individual because of differences in pathology. We recommend gradually superimposing HFV or HFO onto CMV, whilst observing the patient's airway pressure and SpO₂; it will be necessary to reduce the conventional inspired volume (or inspiratory time) in order to increase the driving pressure from the high frequency ventilator, without an increase in airway pressure. If the patient's airway pressure and oxygenation are satisfactory, then time can be taken to optimize all other settings; the monitors and alarms which are built into conventional ventilators allow safe and easy change in variables such as tidal volume, minute ventilation, I/E ratios and PEEP.²⁶ It is important to emphasize that HFV, being an infrequently used ventilatory modality can be potentially hazardous to the occasional and inexperienced user.

The value of CHFV was realised in this seemingly hopeless case by a process of trial and error. A wider recognition of the value of this technique may have

avoided a hazardous inter-hospital transfer. Equipment described in the literature have mostly been "home-made", such as in this case. Versions of the Siemens CHFV unit for use with the Servo ventilator, have been developed to support research.^{15,16,22,23} Further studies are required to clarify the early use of CHFV in the management of acute lung injury, however available evidence suggests it is a valuable alternative when CMV and HFV are ineffective following acute catastrophic lung injury. The use of CHFV in unilateral lung injury avoids the potentially deleterious effect of volutrauma on the uninjured lung when conventional modes of ventilation are used. In this case, we were able to avoid the use of ECMO and its possible complications.

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