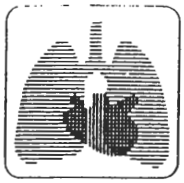

clinical investigations in critical care

High-frequency Percussive Ventilation Improves Oxygenation in Patients With ARDS*

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Study objectives: **To evaluate changes in respiratory and hemodynamic function of patients with ARDS and requiring high-frequency percussive ventilation (HFPV) after failure of conventional ventilation (CV).**
Design: **Retrospective case series.**
Setting: **Surgical ICU (SICU) and medical ICU (MICU) of an academic county facility.**
Measurements and results: **Thirty-two consecutive patients with ARDS (20 from SICU, 12 from MICU) who were unresponsive to at least 48 h of CV and were switched to HFPV were studied. Data on respiratory and hemodynamic parameters were collected during the 48 h preceding and the 48 h after institution of HFPV and compared. Between the period of CV and the period of HFPV, the ratio of Pao_2 to the fraction of inspired oxygen (FIo_2) increased ([mean ± SE] 130 ± 8 vs 172 ± 17; p = 0.027), peak inspiratory pressure (PIP) decreased (39.5 ± 1.7 vs 32.5 ± 1.9 mm Hg; p = 0.002), and mean airway pressure (MAP) increased (19.2 ± 1.2 vs 27.5 ± 1.4 mm Hg; p < 0.001). The rate of change of Pao_2/FIo_2 per hour was also significantly improved between the two periods. The same changes in Pao_2/FIo_2, PIP, and MAP were observed when the last value recorded while the patients were on CV was compared with the first value recorded after 1 h of HFPV. This improvement was sustained but not amplified during the hours of HFPV. The patterns of improvement in these three parameters were similar in SICU and MICU patients as well as in volume-control and pressure-control patients. There were no changes in hemodynamic parameters.**
Conclusion: **The HFPV improves oxygenation by increasing MAP and decreasing PIP. This improvement is achieved soon after institution of HFPV and is maintained without affecting hemodynamics.**

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Key words: ARDS; high-frequency percussive ventilation; mean airway pressure; oxygen consumption; oxygen delivery; Pao_2/fraction of inspired oxygen; peak inspiratory pressure

Abbreviations: CPAP = continuous positive airway pressure; CV = conventional ventilation; Do_2I = oxygen delivery index; FIo_2 = fraction of inspired oxygen; 1-h post = the first value after 1 h of HFPV; 48-h post = the last recorded value within the 48-h monitored period of HFPV; HFPV = high-frequency percussive ventilation; MAP = mean airway pressure; MBP = mean arterial BP; MICU = medical ICU; O_2ER = oxygen extraction ratio; PEEP = positive end-expiratory pressure; PIP = peak inspiratory pressure; Qs/Qt = intrapulmonary shunt fraction; Sao_2 = arterial oxygen saturation; SICU = surgical ICU; TE = expiratory time; TI = inspiratory time; VDR = volumetric diffusive respirator; Vo_2I = oxygen consumption index; VT = tidal volume

High-frequency ventilation has emerged during the last 10 years as a way of maintaining gas exchange while, it is hoped, limiting respirator-associated lung parenchymal injury.[1–9] This mode of ventilation is mainly administered by three types of equipment: (1) jet ventilators, (2) oscillators, and (3) flow interrupters.[2] Although high-frequency jet ventilation has been extensively tested in pediatric patients with encouraging results, the experience is limited with other types of high-frequency ventilation in this group.[10–13] Studies in adults have produced conflicting results.[3,14,15]

The latest such technique is high-frequency percussive ventilation (HFPV) delivered by the volumetric diffusive respirator (VDR).[1] In an attempt to

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combine the beneficial effects of high-frequency and conventional ventilation (CV), this device delivers small tidal volumes (VTs) at rapid rates by a reciprocating system. It delivers a series of high-frequency breaths with a shortened expiratory time (TE) to allow breath stacking. This is then interrupted to allow pressure reduction back to baseline. A unique feature of this technique is that exhalation is an active phenomenon. Additionally, the endotracheal tube cuff is partially deflated, allowing air to escape around it and avoiding the generation of potentially damaging intra-alveolar pressures.

In patients with ARDS, lung stiffness is a predictable and dangerous complication.[7] As the maintenance of adequate oxygenation in the face of impending barotrauma is at best a trade-off with CV, HFPV offers an attractive alternative for such patients. There are only a few reports evaluating the efficacy of HFPV in adult patients suffering from ARDS.[3,16,17] Even if some of these studies show improvement in respiratory function with HFPV, there are only speculations on mechanisms responsible for favorable outcome.

In this study, we analyzed our experience with HFPV in patients with ARDS. We evaluated the changes in respiratory and hemodynamic function after the patients were switched from CV to HFPV. We propose a mechanism by which these changes occur.

Materials and Methods

Materials

Patients who developed ARDS and were placed on HFPV from June 1994 to December 1995 were included in this study. ARDS was considered present if (1) the Pao_2/fraction of inspired oxygen (FIO_2) ratio was < 200, (2) bilateral diffuse lung infiltrates were present on a plain chest radiograph, and (3) wedge pressure was ≤ 18 mm Hg. Patients placed on HFPV before they were placed on CV for at least 48 h were excluded. Similarly, patients who had fewer than three values in respiratory and hemodynamic data during the 24 h preceding or after the institution of HFPV were excluded due to the inability to evaluate the acute changes associated with the new mode of ventilation.

In total, 32 patients met the inclusion criteria. Twenty were being cared for in the surgical ICU (SICU) and 12 in the medical ICU (MICU). All 20 surgical patients had severe injuries (12 penetrating, 8 blunt) as the reason for their admission. Nontrauma patients were managed during the period of the study by a different surgical team that was not familiar with the VDR device. The admitting diagnoses among the 12 medical patients included pneumonia (3), tuberculosis (2), meningitis (1), cirrhosis (3), pancreatitis (1), hypertension with renal failure (1), and malignancy with aspiration (1). The mean ± SE Pao_2/FIO_2 for this population before HFPV was 130 ± 8.

Mechanical Ventilation

In the absence of improvement in respiratory function, the mode of mechanical ventilation was converted to HFPV after patients remained on CV for a mean period of 4 days (range, 2 to 6 days). Volume- and pressure-control ventilation were delivered by one of two types of ventilators (Servo Ventilator 900C; Siemens Medical System; Iselin, NJ or 7200 Micropressor Ventilator; Puritan-Bennett; Wilmington, MA). Although there is no universal agreement on the classifications of high-frequency ventilators, the percussive ventilator that we used (Percussionaire; Bird Space Technologies; Sandpoint, ID) can be described as a time-cycled and pressure-limited ventilator with an additional piston (phasitron) mechanism positioned at the end of the endotracheal tube. The piston is driven by a high-pressure gas supply at a high-frequency rate of 200 to 900 beats/min, superimposed on a conventional inspiratory/expiratory pressure-controlled cycle that is set at a rate of 10 to 15 breaths/min. There are five control parameters: (1) peak inspiratory pressure (PIP), (2) inspiratory time (TI), (3) TE, (4) positive end-expiratory pressure (PEEP) and continuous positive airway pressure (CPAP), and (5) percussive frequency. The VT of both types of breaths is determined by the set PIP, the set TI, and the lung compliance. The high-frequency breath volume is also determined by the pressure difference between PEEP and CPAP. The PEEP and CPAP are used in combination. This is a unique feature of the VDR that allows progressive exhalation in subtidal volumes rather than rapid elimination of the entire exhaled volume. The endotracheal tube cuff is left partially deflated to allow for a continuous air leak through the trachea. Humidification during HFPV may be difficult, and failure to adequately humidify the large volumes of air passing through the circuit may result in erosion and desiccation of the mucosa, potentially leading to airway obstruction. A high-volume dual nebulizer is included in the ventilator circuit to deliver water at 30 mg/L of air to raise the humidity of circulating air to 100%.

Oxygenation is controlled by the FIO_2, PEEP/CPAP, PIP, TI, and frequency. Ventilation is governed by the relationship between TI and TE, PIP, and frequency. Typical starting settings are a high-frequency rate of 500 beats/min superimposed on a rate of 12 breaths/min, a peak pressure of two thirds the pressure used on the conventional ventilator, a TI/TE of 1:1, and a PEEP/CPAP of 10. Adjustments are made on the basis of values obtained by monitoring continuous arterial oxygen saturation (Sao_2) and arterial blood gas levels. The mean airway pressure (MAP) is measured by the ventilator and displayed continuously.

Data Collection and Statistical Analysis

Values for the following parameters were collected from a computerized ICU database: Pao_2/FIO_2, partial $Paco_2$, pH, PIP, MAP, Sao_2, mean arterial BP (MBP), oxygen delivery index (Do_2I), oxygen consumption index ($\dot{V}o_2I$), oxygen extraction ratio (O_2ER), and intrapulmonary shunt fraction (Qs/Qt). These data were inserted prospectively into the ICU-computer database and collected retrospectively for this study. Data collection for each patient covered a period of 48 h before and 48 h after HFPV application. All values were abstracted from the ICU database and entered into another database (Excel; Microsoft; Redmond, WA) for analysis.

Each monitored parameter was studied in three ways. The first way was to evaluate the difference between the 48-h CV value and the 48-h HFPV value for each parameter. Patients with three or more values taken at each time period were included in this analysis. The average values during the 48 h before and the 48 h after starting HFPV for each patient were derived and compared using the paired Student's *t* test. Significance was considered to be the $p < 0.05$ level. Differences between the average values of each parameter for MICU and SICU patients were examined to determine whether the patterns of ventilatory and hemodynamic changes were similar in the two groups. For the same reason, we

evaluated differences in averages between patients who received volume-control ventilation and patients who were given pressure-control ventilation during the 48 h before receiving HFPV. To determine if the differences before and after initiating HFPV were consistent between the two types of ICU patients (SICU or MICU) and between the type of CV (volume- or pressure-control), their interactions were analyzed using the repeated measures analysis of variance model, in which time was the repeated-measure variable and type of ICU patients or type of CV was the main-effect variable. For this purpose, the general linear-model procedure of the computer software (PROC GLM, Statistical Analysis System; SAS Institute; Cary, NC) was used.

The second way of analyzing the data was to examine the rates of change per hour of each parameter during the 48 h before and the 48 h after starting HFPV for each patient, and compare these values using the paired Student's t test and a significance level of $p < 0.05$. The third way was to examine the difference of each parameter among three time points: (1) the last value immediately before institution of HFPV; (2) the first value after 2 h of HFPV; and (3) the last value during the 48-h period of HFPV. Comparisons were made between each pair of two time points using the paired Student's t test. To accommodate multiple comparisons, the significance level was adjusted to $p < 0.005$. The general linear-model procedure was similarly performed to evaluate the differences over time, between patient types, between CV types, and their interactions.

Results

Comparison of mean values recorded over the 48 h of CV with mean values taken over the 48 h of HFPV (Table 1) revealed the following four differences: (1) PaO_2/FIO_2 was increased; (2) PIP was decreased; (3) MAP was increased; and (4) SaO_2 was increased. Of these four statistically significant differences, the first three were also deemed to be clinically significant; the elevation in SaO_2 was of questionable clinical significance. None of the hemodynamic parameters changed when the mode of ventilation was switched from CV to HFPV.

The rate of change per hour of each parameter is shown in Table 2. It is obvious that PaO_2/FIO_2 was deteriorating during CV and remained unchanged during HFPV. However, there was significant improvement between the values recorded on CV and those recorded after placement on HFPV. No other values achieved statistical significance.

When the patients were divided according to the type of CV received before HFPV in two groups (patients who received volume-control ventilation and patients who received pressure-control ventilation), the following two observations were made (Table 3): (1) there was significant improvement in $PaCO_2$, SaO_2, pH, PIP, and MAP among patients who were placed on HFPV after a period of volume-control ventilation; and (2) there was significant improvement in PaO_2/FIO_2, SaO_2, and MAP among patients who were placed on HFPV after pressure-control ventilation. The SICU and MICU patients were evaluated separately for all monitored values. In Table 4, the following two observations were made: (1) SICU patients demonstrated significant improvement in SaO_2, pH, PIP, and MAP after switching from CV to HFPV; and (2) MICU patients showed significant improvement in PaO_2/FIO_2, PIP, and MAP after converting to HFPV. None of the interactions between SICU or MICU patients or changes of respiratory or hemodynamic parameters from CV to HFPV time periods were statistically significant. This finding indicated that the patterns of change of these parameters in the two subgroups were similar. The same was found for the two subgroups defined by pressure or volume-control ventilation.

In Table 5, three values for each parameter were compared: (1) the last value on CV (pre-HFPV), (2) the first value after 1 h of HFPV (1-h post), and (3) the last recorded value within the 48-h monitored period of HFPV (48-h post). The PaO_2/FIO_2, SaO_2, pH, PIP, and MAP were significantly improved on comparison of the pre-HFPV values with either the 1-h post or the 48-h post values. Of these parame-

Table 1—*Respiratory and Hemodynamic Parameters During 48 h of CV and 48 h After Institution of HFPV in 32 Critically Ill Patients With ARDS

Parameters	CV	HFPV	CV − HFPV	p Value
PaO_2/FiO_2	130 ± 8	172 ± 17	+ 42 ± 18	0.027
$PaCO_2$, mm Hg	42.5 ± 1.7	39.4 ± 1.5	− 3.0 ± 1.5	0.056
SaO_2, %	95.1 ± 0.4	96.5 ± 0.5	+ 1.3 ± 0.4	0.002
pH	7.33 ± 0.02	7.37 ± 0.01	+ 0.04 ± 0.02	0.058
PIP, cm H_2O	39.5 ± 1.7	32.5 ± 1.9	− 7.0 ± 2.0	0.002
MAP, cm H_2O	19.2 ± 1.2	27.5 ± 1.4	+ 8.4 ± 1.5	< 0.001
Qs/Qt, %	37.7 ± 1.9	34.7 ± 2.4	− 3.0 ± 1.9	0.134
Do_2I, ml/min/m²	619 ± 44	639 ± 50	+ 20 ± 45	0.668
Vo_2I, ml/min/m²	164 ± 9	155 ± 7	− 9 ± 5	0.113
O_2ER, %	28.7 ± 1.9	26.2 ± 1.3	2.6 ± 1.9	0.206
MBP, mm Hg	82.4 ± 2.6	81.6 ± 2.5	0.8 ± 2.3	0.738

*Values given as mean ± SE, unless otherwise indicated.

Table 2—*Rates of Change Per Hour of Ventilatory and Hemodynamic Parameters During 48 h of CV and 48 h of HFPV in 32 Critically Ill Patients With ARDS*

Parameters	Rate of Change per Hour CV		Rate of Change per Hour HFPV		Difference Between Rates	
	Mean ± SE	p Value	Mean ± SE	p Value	Mean ± SE	p Value
PaO_2/FiO_2	−1.63 ± 0.57	0.008	+0.28 ± 0.57	0.623	+1.91 ± 0.82	0.028
$PaCO_2$, mm Hg	−0.06 ± 0.10	0.569	+0.09 ± 0.20	0.644	+0.03 ± 0.24	0.889
SaO_2, %	−0.06 ± 0.04	0.150	+0.01 ± 0.02	0.606	+0.07 ± 0.05	0.134
pH	−0.011 ± 0.13	0.935	−0.14 ± 0.22	0.543	−0.13 ± 0.27	0.644
PIP, cm H_2O	+0.14 ± 0.09	0.140	−0.16 ± 0.14	0.241	−0.31 ± 0.16	0.070
MAP, cm H_2O	+0.11 ± 0.06	0.062	−0.03 ± 0.03	0.372	−0.14 ± 0.07	0.045
Qs/Qt, %	+0.03 ± 0.22	0.906	−0.15 ± 0.08	0.074	−0.18 ± 0.25	0.479
Do_2I, mL/min/m²	+1.28 ± 1.45	0.391	−2.18 ± 2.36	0.367	−3.46 ± 2.59	0.199
$\dot{V}o_2I$, mL/min/m²	−0.01 ± 0.35	0.988	−0.29 ± 0.37	0.447	−0.28 ± 0.56	0.622
O_2ER, %	−0.20 ± 0.21	0.355	+0.04 ± 0.08	0.634	+0.23 ± 0.21	0.286
MBP, mm Hg	−0.53 ± 0.26	0.051	+0.02 ± 0.07	0.786	+0.55 ± 0.27	0.053

ters, the changes in PaO_2/FIO_2, PIP, and MAP were deemed to be clinically significant. There was no difference between the 1-h post and the 48-h post values. Eleven surgical and 8 medical patients died, for an overall mortality rate of 59%.

Discussion

This study demonstrates an improvement in respiratory function of trauma and medical patients with ARDS after institution of HFPV, with no adverse effects in circulatory function. The benefit in oxygenation was shown when the mean values and the rates of hourly change of the PaO_2/FIO_2 ratio on CV were compared with those after initiation of HFPV. The main mechanism responsible for these results seems to be the ability of the percussive ventilator to reduce the PIP while, at the same time, increase the mean airway pressure. The HFPV provides for the delivery of accumulative/diminishing subtidal stroke volumes until time interruption or a prescheduled plateau occurs. Therefore, instead of literally pushing the pulmonary structure out of the way (during mechanical VT delivery by an uninterrupted intrapulmonary pressure/flow gradient), time is allowed for lung volume increase (pulmonary conformance) by fracturing the inspiratory flow gradient through pulsation of the proximal-distal inspiratory pressure/flow gradient. Breaking up the inspiratory pressure gradient with the pulsed (subtidal) delivery of VT provides a more uniform intrapulmonary gas exchange that requires a lower lung volume for an equivalent blood gas interface. The net result is improved gas exchange, with an associated reduction in the potential for barotrauma due to a lower mechanically induced airway pressure during tidal delivery.

Although Emerson[18] conceived of high-frequency oscillation in 1952 and Butler et al [19] clinically validated his concept in 1980, the development of

Table 3—***Respiratory and Hemodynamic Parameters During 48 h of CV and 48 h of HFPV in 32 Patients With ARDS, Stratified by Type of CV****

Parameters	Volume Control (n = 12)			Pressure Control (n = 20)		
	CV	HFPV	p Value	CV	HFPV	p Value
PaO_2/FiO_2	146 ± 16	176 ± 24	0.352	123 ± 9	170 ± 22	0.051
$PaCO_2$, mm Hg	42.5 ± 2.7	37.6 ± 2.6	0.037	42.4 ± 2.2	40.5 ± 1.9	0.367
SaO_2, %	95.8 ± 0.5	97.3 ± 0.4	0.004	94.7 ± 0.5	95.9 ± 0.7	0.050
pH	7.31 ± 0.04	7.40 ± 0.02	0.014	7.34 ± 0.02	7.34 ± 0.01	0.836
PIP, cm H_2O	38.3 ± 3.2	27.1 ± 2.4	0.004	40.2 ± 2.0	35.7 ± 2.6	0.098
MAP, cm H_2O	15.0 ± 1.7	24.5 ± 2.0	0.001	21.7 ± 1.4	29.3 ± 1.9	0.001
Qs/Qt, %	36.1 ± 4.3	34.7 ± 4.6	0.587	38.6 ± 2.0	34.6 ± 2.9	0.175
Do_2I, mL/min/m²	609 ± 82	666 ± 67	0.368	625 ± 53	623 ± 71	0.971
$\dot{V}o_2I$, mL/min/m²	159 ± 11	157 ± 10	0.811	166 ± 12	154 ± 9	0.112
O_2ER, %	30.3 ± 4.6	24.7 ± 1.9	0.272	27.8 ± 1.5	27.0 ± 1.8	0.610
MBP, mm Hg	87.2 ± 5.6	81.6 ± 4.1	0.117	79.5 ± 2.5	81.6 ± 3.3	0.491

*Values given as mean ± SE, unless otherwise indicated

Table 4—*Respiratory and Hemodynamic Parameters During 48 h of CV and 48 h of HFPV in 32 Patients With ARDS, Stratified by Type of ICU**

Parameters	Surgical ICU Patients (n = 20)			Medical ICU Patients (n = 12)		
	CV	HFPV	p Value	CV	HFPV	p Value
Pao_2/Fio_2	145 ± 10	187 ± 23	0.141	106 ± 11	147 ± 22	0.040
$Paco_2$, mm Hg	42.9 ± 1.8	38.9 ± 1.9	0.052	41.7 ± 3.4	40.3 ± 2.9	0.605
Sao_2, %	96.1 ± 0.4	97.5 ± 0.4	0.008	93.5 ± 0.4	94.7 ± 0.8	0.120
pH	7.31 ± 0.02	7.37 ± 0.01	0.022	7.36 ± 0.01	7.36 ± 0.03	0.998
PIP, cm H_2O	40.3 ± 2.6	33.2 ± 2.2	0.017	38.1 ± 1.7	31.3 ± 3.8	0.051
MAP, cm H_2O	18.7 ± 1.4	27.6 ± 1.8	< 0.001	19.9 ± 2.2	27.4 ± 2.4	0.019
Qs/Qt, %	33.9 ± 1.4	31.4 ± 2.4	0.293	48.3 ± 3.1	43.8 ± 4.1	0.315
Do_2I, mL/min/m²	592 ± 44	596 ± 30	0.913	696 ± 115	759 ± 174	0.689
Vo_2I, mL/min/m²	165 ± 9	158 ± 7	0.181	159 ± 24	147 ± 18	0.500
O_2ER, %	30.0 ± 2.2	27.1 ± 1.0	0.283	25.2 ± 3.6	23.6 ± 4.4	0.471
MBP, mm Hg	83.7 ± 3.7	80.9 ± 3.1	0.384	80.2 ± 3.7	82.8 ± 4.4	0.421

*Values given as mean ± SE, unless otherwise indicated.

HFPV by the VDR is a relatively new and underexplored topic. In the limited published experience with HFPV in neonates, it is suggested that this method may improve oxygenation and CO_2 elimination in premature infants with hyaline membrane disease.[20] Gallagher et al[21] was the first to report the use of HFPV in adults. They studied six patients with ARDS and increased ventilatory requirements on CV, and demonstrated a dramatic improvement in Pao_2 levels, a slight fall in $Paco_2$ levels, and no change in cardiac output. Hurst et al[3] evaluated patients who developed ARDS after trauma. Hypoxemic patients showed significant improvement in Pao_2 and pulmonary shunt after treatment with HFPV. Similarly, hypercarbic patients had improved CO_2 elimination with a lower level of CPAP. There was no improvement in, or detriment to, cardiac output. An additional advantage of HFPV was shown by the same group in head-injured patients.[22] The decrease in PIP and CPAP caused by HFPV reflected lower intracranial pressure values. In the only large-scale study that exists in the literature,[14] 100 patients who entered a SICU and were thought to be at risk of developing respiratory failure were randomized to receive either HFPV or CV. Patients were treated to the same therapeutic endpoints (pH, > 7.35; $Paco_2$, 35 to 45 mm Hg; Pao_2/Fio_2, > 225). Although patients on HFPV reached the therapeutic endpoints at a lower level of pulmonary pressures, there was no significant difference in mortality, ICU days, hospital days, and incidence of barotrauma.

The favorable gas exchange profile offered by HFPV results in immediate improvement in oxygenation, as shown in our study. This improvement is consistent over different types of CV (volume-control or pressure-control) or patient diseases (medical or surgical). Pressure-control ventilation is usually reserved for the most sick patients. In these patients, conversion to HFPV improved dramatically their oxygenation. Patients who were initially maintained at volume-control CV had generally higher Pao_2/Fio_2 ratios than pressure-control patients; this may

Table 5—*Respiratory and Hemodynamic Parameters of 32 Critically Injured Patients With ARDS at Three Time Points**

Parameters	Pre-HFPV	1-h Post	48-h Post	Pre- vs 1-h Post	Pre- vs 48-h Post	1-h Post vs 48-h Post
Pao_2/Fio_2	111 ± 14	163 ± 18	193 ± 21	0.006	< 0.001	0.296
$Paco_2$, mm Hg	43.8 ± 1.8	38.1 ± 2.0	40.9 ± 1.3	0.005	0.129	0.044
Sao_2, %	95.2 ± 0.5	96.8 ± 0.6	96.6 ± 0.6	0.045	0.017	0.976
pH	7.32 ± 0.02	7.37 ± 0.02	7.36 ± 0.01	0.010	0.034	0.482
PIP, cm H_2O	42.4 ± 1.9	33.2 ± 2.1	32.5 ± 2.5	< 0.001	< 0.001	0.560
MAP, cm H_2O	21.0 ± 1.5	28.0 ± 1.5	27.0 ± 1.7	0.001	0.003	0.293
Qs/Qt, %	41.7 ± 1.9	34.8 ± 2.3	33.6 ± 2.4	0.118	0.112	0.801
Do_2I, mL/min/m²	624 ± 47	587 ± 50	630 ± 48	0.228	0.479	0.413
Vo_2I, mL/min/m²	155 ± 9	153 ± 8	150 ± 8	0.211	0.369	0.852
O_2ER, %	26.4 ± 1.6	28.0 ± 1.7	25.3 ± 1.7	0.722	0.805	0.535
MBP, mm Hg	76.2 ± 3.4	84.0 ± 4.1	79.2 ± 4.1	0.050	0.700	0.269

*Values given as mean ± SE, unless otherwise indicated.

be the reason that the mean ratio, although elevated after HFPV, was not improved significantly. This absence of statistical significance could also be a result of the limited number of patients available for analysis.

Another interesting observation derived from this study is based on the comparison of selected values among patients. It is obvious that the conversion to HFPV rapidly improved the PaO_2/FIO_2 ratio, increased the MAP, and decreased the PIP as shown by the comparison of the last value recorded before HFPV with the first value after initiation of HFPV. This improvement was sustained over the next 2 days, as shown by the significant difference between the last value in the three parameters before HFPV and the last value in the 48-h period after conversion to HFPV. However, it seems that the maximal effect of HFPV is achieved rapidly and further improvement is hard to document on the basis of the current number of patients; this is shown by the absence of a significant difference between the first and the last value of these three parameters during the monitored period of HFPV.

The combination of continuous pneumatic percussions with the partially deflated cuff allows for a dramatic mobilization of secretions and clearance of lung infiltrates. The effect of delivering high-frequency percussions on the clearance of secretions may be analogous to that of highly effective chest physical therapy (which is rarely possible in the setting of critically ill patients). It seems that there is no adverse effect on circulatory function. As most studies have only compared cardiac outputs, we decided to incorporate in our analysis parameters that reflect the circulatory function but are more specific for oxygen transport and tissue perfusion information. Oxygen delivery, consumption, and extraction ratio were not affected by the conversion to HFPV. Similarly, the mean arterial pressure remained unchanged.

The results of this nonrandomized study must be approached with caution. The course of the disease if the patients had been left on CV is not known. The rate of change per hour of different respiratory parameters demonstrated ongoing deterioration before HFPV. Although it is highly unlikely that this course could have been reversed without HFPV, this possibility has not been tested and therefore cannot be excluded. The absence of a control group managed without HFPV does not allow conclusions on outcome differences. This is the reason we elected to report only the short-term ventilatory and hemodynamic changes that we believed might have been directly associated with the change in mechanical ventilation strategies. Conclusions derived from analysis of recordings of these parameters for longer periods of time would be obscured by the multiple variables that interfere with the progression of the disease in such critically ill patients. Similarly, attempts to correlate these data with clinical outcome would be senseless in the absence of a control group. It is appropriate that we reserved this method of ventilation for only very sick patients. The mortality rate of 59% attests to this necessity. As with every mode of therapy, HFPV may be more useful when used before full-blown organ failure is established. Identification of patients at risk of ARDS before frank clinical manifestation of the disease may result in appropriate selection of subgroups most likely to benefit by this mode of ventilation. The diverse nature of disease profiles of the patients included in this study diminishes our ability to generalize from our data. Although we documented significant improvements in the respiratory function of all subgroups examined, we have not yet clearly identified the ideal patients for implementation of this method. We believe that trauma patients constitute a fairly uniform group with little likelihood of previous respiratory disease that could confound the clinical picture. For this reason, we are planning to evaluate the efficacy of HFPV with a prospective randomized study in critically injured patients.

In summary, it appears that in selected patients, HFPV can improve oxygenation with reduced peak airway pressures, potentially reducing the risk of barotrauma. This improvement can be achieved without elevations of CO_2 or deterioration of hemodynamic parameter values. The improved clearance of pulmonary secretions may reduce the risk of intrathoracic infection. Further studies are justified to define the population that can be maximally benefited by this method.

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