High-Frequency Percussive Ventilation as a Salvage Modality in Adult Respiratory Distress Syndrome: A Preliminary Study

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Despite multiple advances in critical care patients with severe adult respiratory distress syndrome (ARDS) can exhaust the capability of conventional ventilation; this results in respiratory failure and death. High-frequency percussive ventilation (HFPV), which was initially utilized for salvage of burn patients with smoke inhalation injury refractory to conventional ventilation, has evolved as a standard of burn care. Based on our experience with HFPV in burn patients the burn team was consulted to provide salvage ventilation for non-burn surgical intensive care unit patients with refractory respiratory failure. Over a 14-month period ten patients with refractory ARDS from multiple causes were treated. Retrospective chart review was performed. Respiratory parameters were assessed before and 24 hours after initiation of HFPV. Mean values of fraction of inspired oxygen (Fio_2), pH, partial pressure of O_2 in arterial blood (Pao_2), partial pressure of CO_2 in arterial blood ($Paco_2$), HCO_3, oxygen saturation in arterial blood (Sao_2), Pao_2/Fio_2, and peak inspiratory pressure were compared. Significant improvement in oxygenation was reflected by increases in Sao_2, Pao_2, and the Pao_2/Fio_2 ratio in the first 24 hours of HFPV. No significant increase in peak inspiratory pressure was documented by conversion from conventional ventilation to HFPV. No hemodynamic changes directly associated with HFPV were noted. Seven of ten patients failing conventional ventilation survived to hospital discharge after salvage therapy with HFPV. We advocate further studies of HFPV in non-burn patients with ARDS both as salvage therapy and as replacement for conventional ventilation for the initial treatment for ARDS.

HIGH-FREQUENCY PERCUSSIVE ventilation (HFPV) is a pressure-limited and time-cycled ventilation technique based on the successive stacking of subtidal volumes during inspiration. The claimed advantages of HFPV include the ability to achieve oxygenation at lower mean airway pressures and the improved clearance of pulmonary secretions. Early experience with HFPV took place in the burn unit setting, and those initial clinical reports focused on its use as a salvage modality. Cioffi et al.[1] utilized HFPV in a group of five burn patients with smoke inhalation injury and progressive hypoxemia and/or carbon dioxide retention unresponsive to conventional ventilation techniques. In each case normocapnia or arterial saturation >90 per cent on a fraction inspired oxygen (Fio_2) of 0.6 or less was achieved with HFPV but not with conventional ventilation.[1] On this basis the prophylactic use of HFPV was advocated. A second study of 54 burn patients with inhalation injury in which HFPV was instituted within 24 hours of intubation demonstrated a significant decrease in incidence of pneumonia along with a significant increase in patient survival when compared with historical controls.[2] Subsequently the benefits of HFPV in burn patients with smoke inhalation injury have been confirmed in other studies,[3–5] and this modality is now considered the standard of care in this and other burn centers. Successful use of HFPV in other conditions including combined head injury, respiratory failure,[6, 7] and acute chest syndrome secondary to sickle cell anemia[8] has also been reported.

Over the last 14 months the adult burn team at the Medical University of South Carolina has been asked to provide HFPV consultation to patients in our adjacent surgery, trauma, and neurosurgical intensive care unit (STNICU). This report documents our initial experience in the salvage use of HFPV for non-burn patients failing conventional ventilation.

Presented at the Annual Meeting, Southeastern Surgical Congress, February 1–5, 2002, Nashville, Tennessee.

The authors have no financial interest in any commercial device herein described.

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Materials and Methods

Burn team consultation for respiratory management was requested by the trauma, general surgery, or transplant team for patients in respiratory failure unresponsive to conventional ventilation. All patients met the criteria for adult respiratory distress syndrome (ARDS) established by the American Thoracic Society and the European Society of Intensive Care Medicine with a partial pressure of oxygen (Po_2) to Fio_2 ratio of <200.[9] Chest radiographs documented diffuse bilateral pulmonary infiltrates in all of these patients. HFPV was provided by a commercially available ventilator (VDR-4, Percussionaire Corp, Sandpoint, ID). Conventional ventilation utilized multiple modalities including intermittent mechanical ventilation, pressure-supported ventilation, and pressure regulated volume control ventilation (PRVC) on a Servo 300 system (Siemens-Elema AB, Solna, Sweden). All HFPV management was supervised by the same surgical intensivist. Ancillary modalities including fiberoptic bronchoscopy, bronchodilators, and aerosolized heparin administration were utilized as indicated.[10]

A retrospective chart review was undertaken. Data obtained included demographics, etiology of respiratory failure, best arterial blood gas and ventilator settings before institution of HFPV, and arterial blood gases with respective ventilator settings after the first 24 hours of HFPV. Outcome—death or survival to hospital discharge—was documented. Statistical analysis was undertaken with a standard software package (SPSS, Chicago, IL). Methodology included two-tailed Student's *t* test.

Results

Between November 2000 and January 2002 12 non-burn patients were treated with HFPV. One trauma patient and one liver transplant patient were started on HFPV as a prophylactic measure at onset of respiratory distress and were not included in this study of salvage ventilation. The remaining ten patients were started on HFPV as a salvage maneuver after failing all modes of conventional ventilation. Demographics of the study group were stratified (Table 1). The ten patients had a mean age of 36.6 years with a range of 17 to 63 years. There were seven males and three females. The initial diagnosis was traumatic injury in seven patients and end-stage liver disease in two patients. One morbidly obese patient developed respiratory complications after surgical correction of a peristomal hernia.

The initiating factor in the development of respiratory failure was known or presumed pulmonary or fat embolus in two patients, and they were diagnosed and treated on the basis of risk factors and clinical picture of sudden pulmonary deterioration. These patients were too unstable to be transported for definitive diagnostic studies. Five patients suffered initial pulmonary contusion that led to the development of pneumonia and subsequent fulminant ARDS. These patients and those with pulmonary embolism had the poorest oxygenation before HFPV and the most remarkable recovery later with HFPV. Finally, the two transplant patients both had nosocomial pneumonias.

The sixth patient was managed with HFPV for 2 days and then was started on extracorporeal membrane oxygenation (ECMO) by the referring surgical team. At the time of transfer to ECMO the arterial blood gas showed a Po_2 of 67 torr, a partial pressure of CO_2 (Pco_2) of 34 torr, a saturation of 93 per cent on a Fio_2 of 0.7, and a peak inspiratory pressure (PIP) of 44. Although oxygenation and ventilation were adequate the referring team was concerned over the need to raise Fio_2 by 5 per cent and PIP from 40 to 44 over a 48-hour course of HFPV. No adverse effects on hemodynamic parameters as a function of HFPV therapy were observed.

Arterial blood gas data for the study group after 24 hours of HFPV were compared with the last gas obtained on conventional ventilation (Table 2). Improved oxygenation at 24 hours was documented by improved arterial partial pressure of oxygen in seven cases, by lower Fio_2 in seven cases, and by improved Pao_2:Fio_2 ratios (P:F ratios) in eight patients. The P:F ratio was at least *tripled* after 24 hours of HFPV in seven cases.

There were four patients with severe hypoxia and Pao_2 levels less than or equal to 50 torr at the time institution of HFPV. After 24 hours of HFPV the average Pao_2 value for these patients was 145.5 torr. All four patients with initial PO_2 less than or equal to 50 torr survived and were discharged from the hospital.

Statistical analysis was performed on mean arterial blood gas and ventilator setting data (Table 3). All indices of oxygenation, including Po_2, Fio_2, oxygen saturation, and P:F ratio were significantly improved in the HFPV group. Indices of ventilation and acid-base balance were not significantly different. Levels of positive end-expiratory pressure (PEEP) during the conversion from conventional ventilation to HFPV were kept relatively constant, and no statistically significant alteration in PEEP was documented with change in the mode of ventilation. Improvement of oxygenation was achieved without statistically significant increases in PIP.

Discussion

This study is a retrospective review of a limited number of patients in whom HFPV was emergently

TABLE 1. *Demographics and Outcome of Study Population*

Patient No.	Age Years	Sex	Diagnosis	Etiology of Respiratory Failure	Outcome
1	23	M	Multiple gunshot wounds	DVT and probable PE	Discharge to home
2	23	F	Motor vehicle crash	Pulmonary contusion	Died
3	51	F	Peristomal hernia/sleep apnea	Pulmonary edema	Discharged to rehabilitation
4	41	M	Motor vehicle crash/quadraplegia	Pulm contusion and pneumonia	Discharged to rehabilitation
5	23	M	Motor vehicle crash, multiple fractures	Pulm contusion	Discharged to home
6	27	M	Motor vehicle crash, femur fractures	Probable fat embolus	Discharged to rehabilitation
7	17	F	Motor vehicle crash, multiple fractures	Pulmonary contusion and pneumonia	Discharged to home
8	39	M	Motor vehicle crash, rib and long bone fractures	Pulmonary contusion and pneumonia	Died
9	59	M	End-stage liver disease/liver transplant	*Staphylococcus* pneumonia	Died
10	63	M	End-stage liver disease/liver transplant	*Enterobacter* pneumonia	Discharged to rehabilitation

DVT, deep venous thrombosis; PE, pulmonary embolism.

instituted as salvage therapy in severe ARDS. For this reason no definitive conclusions can be drawn. Nevertheless some salient points regarding this mode of ventilation merit discussion.

The hallmark of ARDS is hypoxemia. This can be treated by increasing Fio_2; however, high inspired concentration of oxygen may produce free radicals that lead to tissue oxidant-induced injury and may reduce surfactant turnover resulting in compromise of lung compliance.[11] PEEP has become integral in maximizing oxygenation. PEEP adds positive pressure at the end of expiration to distend collapsing alveoli. This recruitment results in increased functioning lung volume and improved oxygenation.[12] Although PEEP has been very valuable in treating ARDS it can contribute to lung injury and cardiovascular compromise. HFPV utilizes a traditionally safe level of PEEP while improving oxygenation at lower airway pressure.

In this series emphasis was placed on improvement in oxygenation. Oxygenation rather than ventilation is the immediate concern. In other studies[13, 14] and in our clinical experience HFPV appears equally facile in managing insufficiency of ventilation. The ability to program continuously variable inspiratory:expiratory ratios of both the sinusoidal or low-frequency rate and the subtidal or high-frequency rate gives this modality unusual flexibility. The high-frequency rate can also be set to enhance oxygenation, to maximize

TABLE 2. *Arterial Blood Gas Data*

Patient No.	Mode	pH	Pco_2	Po_2	HCO_3^-	SaO_2	Fio_2	PIP	P:F Ratio
1	CV	7.09	85	37	24	51	1.0	48	37
	HFPV	7.33	49	149	25	99	1.0	56	149
2	CV	7.33	58	76	30	94	1.0	45	76
	HFPV	7.34	61	71	32	94	1.0	52	71
3	CV	7.33	63	84	32	96	1.0	53	84
	HFPV	7.53	42	210	34	99	0.8	54	263
4	CV	7.39	43	64	25	94	1.0	35	64
	HFPV	7.48	39	146	29	100	0.4	30	365
5	CV	7.60	29	41	32	83	1.0	53	41
	HFPV	7.48	36	105	26	98	0.7	46	150
6	CV	7.39	47	50	28	87	1.0	40	50
	HFPV	7.47	34	186	24	100	0.65	40	286
7	CV	7.38	42	41	23	77	1.0	40	41
	HFPV	7.46	37	142	25	100	0.7	48	203
8	CV	7.26	69	145	28	99	1.0	54	145
	HFPV	7.33	54	128	27	98	0.8	54	160
9	CV	7.38	35	220	20	100	1.0	31	220
	HFPV	7.31	38	120	18	98	1.0	40	120
10	CV	7.41	44	89	27	100	0.7	40	63
	HFPV	7.40	43	139	27	99	0.45	32	309

SaO_2, oxygen saturation in arterial blood; CV, conventional ventilation.

TABLE 3. *Comparison of Means*

	FiO_2	pH	Pco_2	Po_2	HCO_3	SaO_2	P:F	PIP	PEEP
CV	0.97 ± 0.03	7.35 ± 0.04	51.5 ± 5.4	84.7 ± 18.2	27.9 ± 1.2	86.7 ± 5.1	82.1 ± 18.3	43.6 ± 2.7	13.7 ± 3.2
HFPV	0.75 ± 0.07	7.41 ± 0.03	43.6 ± 2.9	139.6 ± 12.3	27.6 ± 1.5	98.5 ± 0.6	207.6 ± 29.7	45.2 ± 2.9	14.5 ± 4.2
P	0.009	NS	NS	0.02	NS	0.02	0.002	NS	NS

Data are presented as mean ± standard error of the mean. Sao_2, O_2 saturation in arterial blood; NS, not significant.

ventilation, or to establish a compromise between the two.

In severe respiratory failure the mistaken desire to obtain a "perfect" arterial blood gas may lead to the use of higher-than-required airway pressures or oxygen percentage. Elevated PIP or Fio_2 may then cause further pulmonary damage. Our current practice is to utilize permissive hypercapnia as long as acid-base balance is adequate and to accept oxygen saturation levels of 90 per cent or above. Tidal volumes of this pressure-based ventilation mode change literally with each breath and are not measured. When utilizing a ventilation mode where tidal volume is adjustable restriction of tidal volumes to 4 to 6 mg/kg is associated with improved outcome.[15]

The success of this ventilatory mode is directly proportional to the enthusiasm of the respiratory therapists and surgical housestaff involved. If either are unconvinced of the utility of HFPV or are intimidated by the ventilator, then application of HFPV will be an exercise in frustration for the attending intensivist. Many of the ventilator settings such as Fio_2, PEEP, and PIP are managed in the same manner as with conventional pressure-control ventilation. The sinusoidal (low-frequency) rate cannot be dialed in directly but requires adjustment of both inspiratory and expiratory times. Bedside experience is required to gain a comfort level and an appreciation of the versatility of the VDR-4 ventilator.

One previous study examined the efficacy of HFPV as a primary ventilation therapy in the care of surgical intensive care unit patients.[16] The trial showed no significant reduction in mortality, surgical intensive care unit days, hospital days, incidence of barotrauma, number of blood gases, or cardiovascular interventions over conventional modes of ventilation. The study was performed in the late 1980s before the commercial availability of the VDR system and at a time when extensive clinical experience was lacking. It and other early studies of high-frequency ventilation have recently been criticized by Herridge et al.,[17] who point out that such studies were clinically underpowered and that "knowledge of how to optimally implement this ventilatory strategy was not appreciated until fairly recently."[17] The authors were unable to conduct a metasummary of current published data and recommended a multicenter prospective trial. Based upon our initial success with HFPV for the salvage of ARDS patients refractory to conventional ventilation we agree that the time for such a trial has arrived. Furthermore we advocate a wider role for this ventilation technique in the SICU population on a case-by-case basis until such data are available.

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DISCUSSION

MARTIN A. CROCE, M.D. (Memphis, TN): Could the authors please expand on the addition of the "percussive" component to high-frequency ventilation? Why does that improve the gas exchange?

Years ago there was some enthusiasm for high-frequency jet ventilation and oscillators in treating patients with ARDS. One of the main problems was the desiccation of the airway mucosa and the development of concretions, not secretions. How do the authors address this problem?

Finally, what type of conventional ventilation was used prior to salvage therapy? Several investigators have suggested that failure of either pressure control or volume control ventilation may be due to inadequate levels of PEEP. Personally I ascribe to the Linus Pauling theory of PEEP—if a little is good, a lot is better. Could the authors expand on the role of PEEP with high-frequency percussive ventilation?

The authors have presented a provocative series of patients who improved with unconventional therapy and should be commended.

S.M. PAULSEN, M.D. (Closing Discussion) The "percussive" component of high-frequency ventilation is theoretical and is thought to cause agitation of oxygen molecules thus increasing diffusion into alveoli with improvement in oxygenation and gas exchange. With regard to mucosal desiccation the development of concretions is indeed true. In fact it's probably one of the drawbacks to this form of ventilation. It does require respiratory therapy involvement keeping the airways clear and carefully monitoring the patient. Further the ICU physicians managing these patients should be prepared to bronchoscope the patients frequently because there will be trouble with mucous plugging.

Finally, what type of conventional ventilation was used prior to salvage therapy? The patients would start on volume control ventilation and then move to pressure-controlled ventilation prior to being maximized on the form of conventional ventilation. The levels of PEEP were usually increased to about 15 cm of water, and at that point, if the primary team felt increasing PEEP was no longer beneficial then percussive ventilation was instituted. We were able to provide better oxygenation at the same airway pressure or better than those with increased PEEP.