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Early Clinical Experience with High-Frequency Oscillatory Ventilation for ARDS in Adult Burn Patients

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Lung protective ventilation strategies are recommended in acute respiratory distress syndrome to avoid ventilator associated lung injury, a recently characterized complication of mechanical ventilation. High-frequency oscillatory ventilation (HFOV) is an unconventional ventilation strategy which may achieve this goal. We reviewed our experience with HFOV in six severely burned patients with acute respiratory distress syndrome. The mean age (± SD) of the patients was 34 ± 13 years, and the mean TBSA burn was 52 ± 10%, with a mean full-thickness injury of 49 ± 12%. HFOV was initiated as "rescue therapy" in three patients with oxygenation failure (mean PaO_2/FIO_2 ratio of 71 ± 8 and mean oxygenation index [OI] of 42 ± 3) that was unresponsive to conventional ventilation (mean FIO_2, 1.0 ± 0; mean positive end expiratory pressure, 14.8 ± 2.8 cm H_2O; and mean inhaled nitric oxide, 20 ± 0 ppm). In the other three cases, HFOV was initiated "prophylactically" as a lung protective ventilation strategy in an attempt to prevent further respiratory deterioration. All six patients showed a rapid and substantial improvement in oxygenation after initiation of HFOV, with significant improvements in the PaO_2/FIO_2 and OI by 12 hours ($P = 0.02$). In four patients HFOV was also used during anesthesia and surgery, where a total of 10 procedures involving a mean excision and closure of 15 ± 7% TBSA burns was performed. Five of the six patients died, but none died because of oxygenation failure. In three patients death resulted from sepsis and multiple organ dysfunction syndrome; their mean PaO_2/FIO_2 was 107 ± 31 and their mean OI was 30 ± 11 immediately before death. Two patients with multiple organ dysfunction syndrome died after withdrawal of life support; their mean PaO_2/FIO_2 and OI were 178 ± 31 and 18 ± 2, respectively, at the time of this decision. Although HFOV had no impact on mortality, it played a useful role in the supportive management of burn patients with severe oxygenation failure unresponsive to conventional ventilation. Importantly, HFOV allowed surgery to proceed in patients who may have otherwise been too unstable to go to the operating room. As far as we are aware, this is the first report of the use of intraoperative HFOV in burn patients.
(J Burn Care Rehabil 2001;22:325–333)

Respiratory complications resulting from smoke inhalation and cutaneous burns continue to be a major cause of morbidity and mortality for thermally injured patients.[1–3] Chief among these complications is the acute respiratory distress syndrome (ARDS), which may arise as a result of direct lung injury from smoke inhalation,[4] or from an inflammatory process[5–7] initiated by infection, sepsis, or the products of burned tissue.[4]

An emerging concept in the management of ARDS is that conventional mechanical ventilation may itself cause a lung injury and adversely affect clinical outcome.[8–10] The injury in the ARDS lung is heterogeneous, and only selected zones remain compliant and capable of gas exchange.[11] Delivery of large tidal volumes with elevated inspiratory pressures may injure

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these functional zones through repetitive alveolar overdistention.[12-15] Alternatively, underinflation will allow chronic atelectasis to develop in these zones, and injury then occurs from repeated closure and reexpansion of the alveoli.[16] Hence, a "safe window" of ventilation exists between a danger zone of alveolar derecruitment and atelectasis at low airway pressures and tidal volumes, and a danger zone of alveolar overdistention at high airway pressures and tidal volumes.[16] A ventilation approach that attempts to correct atelectasis while avoiding overinflation seems to be beneficial in animal models[17] and in premature infants suffering from respiratory distress syndrome.[18] In adult humans with ARDS, current guidelines for mechanical ventilation reflect this understanding and incorporate lung protective ventilation strategies (LPVS).[19] The large ARDS network study,[20] a prospective, randomized, controlled trial of a simple LPVS, showed improved survival in the group treated with lower tidal volume (6 ml/kg) than in the group treated with a traditional tidal volume (12 ml/kg). Another strategy, which seems to affect outcome favorably, uses pressure-limited techniques such as pressure support or inverse ratio pressure control, elevated positive end expiratory pressure (PEEP), limited tidal volumes, and permissive hypercapnia.[21] A related LPVS which may achieve ventilation within the "safe window"[16] uses high-frequency oscillatory ventilation (HFOV) with small sub-dead space tidal volumes and shows great promise in improving gas exchange in adults who are failing conventional mechanical ventilation because of ARDS.[11,22,23,24]

The purpose of this article is to report on our initial experience with HFOV in a series of severely injured burn patients with ARDS. HFOV was used either as "rescue therapy" in patients who were unresponsive to conventional ventilation, or as a LPVS in an attempt to prevent further respiratory deterioration. HFOV was also used during anesthesia and surgery in several patients. To our knowledge, this is the first report of the use of HFOV during burn surgery.

PATIENTS AND METHODS

The medical records of all patients treated with HFOV in our burn unit between January 1, 1999, and June 30, 2000, were reviewed. In each case the patient's age, sex, total body surface area burn size (TBSA), and full-thickness burn size were recorded. Inhalation injury was documented based on history, physical findings, and serial fiberoptic bronchoscopy in all cases. The diagnosis of ARDS was made using the American-European Consensus Conference criteria.[25] In-hospital mortality was recorded for each patient.

The ventilation parameters immediately before initiation of HFOV were noted. These included the mode of ventilation, the number of days on conventional ventilation, peak inspiratory pressure (cm H_2O), mean airway pressure (P_{mean} cm H_2O), positive end-expiratory pressure (PEEP cm H_2O), concentration of inhaled nitric oxide (iNO ppm), and the fractional inspired oxygen concentration (FIO_2). In all six patients, pharmacologic paralysis with vecuronium had been instituted during conventional ventilation. Neuromuscular blockade was continued in all cases after starting HFOV.

HFOV was delivered with a Sensormedics 3100B oscillatory ventilator (SenorMedics Critical Care Corporation Yorba Linda CA). Initial P_{mean} was set at 5 cm H_2O greater than the P_{mean} in the preceding conventional ventilation mode. Oscillatory frequency was set at 5 Hz, and amplitude was set for adequate chest wall vibration. During transition from conventional ventilation to HFOV, FIO_2 was set at 1.0. Subsequently, FIO_2 was progressively reduced to the minimum level at which arterial oxygen saturation (SaO_2) remained $\geq$ 92%. The arterial partial pressure of CO_2 ($PaCO_2$) was controlled by reduction of oscillatory frequency (to a minimum of 3 Hz), combined with increases in amplitude and partial deflation of the endotracheal tube cuff when necessary. The goal of these interventions was to maintain $PaCO_2 < 70$ mmHg, and arterial pH > 7.25. As a general rule, we attempted to reduce the P_{mean} only when the FIO_2 remained ≤ 0.4 for at least 12 hours. However, in our early experience, reductions in P_{mean} occasionally occurred before the FIO_2 had remained ≤ 0.4 for more than 12 hours. When P_{mean} reached 25 cm H_2O, transition back to conventional ventilation would be considered.

Ventilator parameters at 1, 3, 12, and 24 hours after initiation of HFOV were recorded and included the P_{mean}, the pressure amplitude of oscillation (ΔP), frequency of oscillation, FIO_2, and concentration of inhaled NO (iNO) if present.

The response to HFOV was determined by examining the arterial partial pressure of oxygen (PaO_2), the ratio of the PaO_2 to the FIO_2 (PaO_2/FIO_2), the oxygenation index (OI), where $OI = FIO_2 \times P_{mean} \times 100/PaO_2$, and the $PaCO_2$ both before and at intervals after institution of HFOV.

In our burn unit, conventional mechanical ventilation is performed with Puritan-Bennett 840 ventilators (Nellcor Puritan Bennett, Carlsbad, CA). Inhaled NO (Praxair, Mississauga, Ontario, Canada) when used, is available at 1000 ppm in N2 and is introduced into the inspiratory limb, downstream of the humidifier. The concentration is analyzed and

adjusted using an electrochemical analyzer (Pulminox, Tofield, Alberta, Canada) proximal to the "Y" on the inspiratory limb.

Routine burn management included fluid resuscitation according to the Parkland Formula, topical wound care with silver sulfadiazine, early enteral nutritional support, and early staged excision of deep partial-thickness and full-thickness burns. Wound closure was obtained either temporarily with human cryopreserved cadaveric allograft, or permanently with split-thickness autograft.

Continuous variables are reported as mean ± SD. Paired observations were compared with Student's two-tailed *t*-test, with significance set at $P < 0.05$. No correction for repeated comparisons was performed. Statistical calculations were executed in Excel® 97 (Microsoft® Corp., Redmon, WA).

RESULTS

Between January 1, 1999, and June 30, 2000, eight patients were treated with HFOV. One patient, admitted with a diagnosis of toxic epidermal necrolysis syndrome, and another, who was part of a prospective study on HFOV, were excluded, leaving a study population of six patients whose characteristics are shown in Table 1. The mean Acute Physiology and Chronic Health Evaluation II score of the group was 16 ± 9.

Initiation of HFOV

The decision to use HFOV was made on clinical grounds and not as part of an established research protocol. In three cases HFOV was initiated to "rescue" the patient from oxygenation failure, unresponsive to conventional ventilation. In three cases HFOV was started "prophylactically" as an LPVS in an attempt to limit further respiratory deterioration. The "rescue" patients differed from the "prophylactic" patients mainly with respect to the preHFOV FIO_2, PEEP, use of inhaled NO, and OI, reflecting early institution of HFOV under conditions of less intense conventional mechanical ventilation in the latter group (Table 2).

Ventilator Parameters on HFOV

Table 3 shows the ventilator parameters at 1, 3, 12, and 24 hours after initiation of HFOV. In general there was a steady decline in the FIO_2 over the first 24 hours. The concentration of iNO dropped dramatically at 1 hour, and iNO was not required beyond 3 hours in any of the patients.

Early Response to HFOV

The mean PaO_2, PaO_2/FIO_2, OI, and $PaCO_2$ before and at 1, 3, 12, and 24 hours after initiation of HFOV are shown in Table 4. There was an immediate increase in the PaO_2/FIO_2 ratio, which became significant by 12 hours ($P = 0.02$). Similarly, the OI dropped progressively, becoming significantly less than the preHFOV OI at 12 hours ($P = 0.02$).

Intraoperative HFOV

Four patients (patients 2, 4, 5, and 6) underwent anesthesia and surgery while being treated with HFOV. A total of 10 procedures involving a mean excision and closure of 15 ± 7%TBSA burns were performed. In the case of patient 2, HFOV was started specifically to create a window of opportunity to allow surgery to proceed, which had been judged impossible because of profound respiratory deterioration on conventional ventilation. This patient had sepsis, and although burns were almost completely debrided and closed, had persisting unexcised burns totaling 9% TBSA, which were believed to be a possible source of sepsis. In patients 4, 5, and 6, surgery proceeded subsequent to and independent of the decision to start HFOV. Because of the need for electrical power and two independent gases (oxygen and pressurized room air), HFOV cannot be maintained during patient transfer to the operating room. There-

Table 1. Patient characteristics, including age, gender, % total area burn size and full-thickness burn size (% TBSA/% Full), presence of smoke inhalation injury, diagnosis of acute respiratory distress syndrome (ARDS), and outcome

Patient	Age (yr)	Sex	%TBSA/%Full	Inhalation	ARDS	Outcome
1	37	Female	54/45	No	Yes	Died
2	25	Female	35/30	No	Yes	Survived
3	28	Male	60/60	Yes	Yes	Died
4	54	Male	47/42	Yes	Yes	Died
5	42	Male	60/60	Yes	Yes	Died
6	19	Male	55/55	Yes	Yes	Died

Table 2. Ventilation parameters immediately before initiation of high-frequency oscillatory ventilation for "rescue" and "prophylactic" indications, including the mode, inspiratory to expiratory ratio (I:E), days on conventional ventilation (days), peak inspiratory pressure (PIP cmH_2O), mean airway pressure (P_{mean} cm H_2O), fraction of inspired oxygen (FiO_2), positive and expiratory pressure (PEEP cm H_2O), concentration of inhaled nitric oxide (iNO ppm), and the oxygenation index (OI)

	Patient	Mode	I:E	Days	PIP	P_{mean}	FiO_2	PEEP	iNO	OI
Rescue	1	PCV	1.5:1	6	43	28	1.0	12	20	41.2
	2	PCV	2:1	10	44	31	1.0	17.5	20	39.8
	5	PCV	1:1.3	3	40	29	1.0	15	20	45.3
Mean ± SD				6 ± 3.5	42 ± 2.1	29 ± 1.5	1.0 ± 0	14.8 ± 2.8	20 ± 0	42 ± 2.9
Prophylactic	3	PCV	1:1	6	48	30	0.6	15	0	26.9
	4	PCV	1:1.5	2	41	24	0.7	12.5	0	22.4
	6	PCV	1:1	2	39	28	0.7	10	0	27
Mean ± SD				3 ± 2.3	43 ± 4.7	27 ± 3.1	0.7 ± 0.1	12.5 ± 2.5	0 ± 0	25 ± 2.6

Table 3. Ventilation parameters at 1, 3, 12, and 24 hours after initiation of high-frequency oscillatory ventilation, including mean airway pressure (P_{mean}), amplitude of oscillation (ΔP), frequency of oscillation, the FiO_2, and the concentration of inhaled nitric oxide (iNO)

	1 hr	3 hr	12 hr	24 hr
P_{mean} (cm H_2O)	37 ± 5.2	37 ± 5.6	36 ± 2.9	33 ± 5.7
ΔP (cm H_2O)	76 ± 8.8	77 ± 8.4	77 ± 6.7	81 ± 8.7
Frequency (Hz)	4.8 ± 0.7	4.6 ± 0.9	4.2 ± 1.1	4.1 ± 1.0
FiO_2	0.71 ± 0.3	0.59 ± 0.1	0.61 ± 0.3	0.51 ± 0.1
iNO (ppm)	0.7 ± 1.6	0 ± 0	0 ± 0	0 ± 0

Figures are mean ± SD.

Table 4. PaO_2, PaO_2/FiO_2 ratio, oxygenation index (OI), and $PaCO_2$ immediately before institution of high-frequency oscillatory ventilation (PreHFOV), and at 1, 3, 12, and 24 hours after initiation of HFOV

	PreHFOV	1 hr	3 hr	12 hr	24 hr
PaO_2 (mmHg)	73 ± 6.2	135 ± 78.2	97 ± 38.7	117 ± 40	93 ± 18
PaO_2/FiO_2	92 ± 21.2	170 ± 54.7	159 ± 47.6	227 ± 43.4*	192 ± 28
OI	32 ± 9.1	23 ± 8.9	24 ± 6.7	15.1 ± 4.1†	15.6 ± 3
$PaCO_2$ (mmHg)	52 ± 12.5	63 ± 5.4	69 ± 15.5	60 ± 8.5	64 ± 11.1

Figures are mean ± SD.

* $P = 0.02$ compared with PreHFOV.

† $P = 0.02$ compared with PreHFOV.

fore, during transfer, patients were manually ventilated with a Laerdal bag with the PEEP valve set at 20 cm H_2O, and with the FIO_2 set at 1.0. A separate team simultaneously moved the Sensormedics 3100B ventilator to the operating room to allow prompt resumption of HFOV when the patient arrived. Typically, slight derecruitment with a drop in the SaO_2 occurred during transfer, but in all cases the pretransfer SaO_2 was reestablished within minutes of resuming HFOV. No cardiovascular or respiratory complications occurred intraoperatively in any of the cases.

Outcome

The mean duration of HFOV was 6.1 days (range, 2 hours to 12 days). Five of the patients in this study died. However, none of the deaths were solely attributable to oxygenation failure. In three cases (patients 1, 3, and 6) death was caused by sepsis and multiple organ dysfunction syndrome (MODS). In these patients the mean PaO_2/FIO_2 ratio was 107 ± 31 and the mean OI was 30 ± 11 immediately before death. In the other two deaths (patients 4 and 5) both pa-

tients had sepsis and MODS, and care was withdrawn at the request of family members. The mean PaO_2/FIO_2 ratio and OI were 178 ± 31 and 18 ± 2, respectively, at the time of this decision. All patients who died were on HFOV at the time of death or at the time of the decision to withdraw care. Patient 2, who was one of the "rescue" cases, remained on HFOV for 7 days and was the lone survivor. HFOV settings at the resumption of conventional ventilation were FIO_2, 0.45; oscillatory frequency, 4.5 Hz; P_{mean}, 25 cm H_2O; and ΔP, 65 cm H_2O. The OI on these settings was 12. Figures 1 and 2 show the changes in the OI across time for the "rescue" and "prophylactic" patients, respectively. In general patients treated with HFOV for rescue showed a progressive improvement in the OI, whereas those placed on OI prophylactically tended to show a variable response, with no substantial improvement in the OI over time.

DISCUSSION

High-frequency ventilation is defined as ventilation with sub-dead space tidal volumes at frequencies greater than 60 breaths per minute (1 Hz). Gas exchange occurs through bulk flow, Taylor dispersion, molecular diffusion, and nonconvective mixing, rather than by convection as occurs during conventional ventilation.[26] Available modes of high-frequency ventilation include high-frequency jet ventilation, high-frequency percussive ventilation (HFPV), and high-frequency oscillatory ventilation (HFOV). Compared with conventional ventilation, high-frequency ventilation achieves adequate alveolar gas exchange at lower peak and mean airway pressures and with less circulatory embarrassment and barotrauma.[27] Additionally, delivery of asymmetric high-frequency breaths may improve clearance of secretions.[28–30] Finally, with the recent interest in LPVS, it has been recognized that high-frequency oscillatory ventilation may be an effective way to ventilate the lungs within the "safe window" between atelectasis and overdistention.[11,16]

HFPV uses a high-frequency pulse generator to deliver rapid (1.5–15 Hz) sub-dead space volume breaths to a standard endotracheal tube. The pulsatile flow is periodically interrupted, which allows airway pressure to return to baseline continuous positive airway pressure. Oxygenation and ventilation are manipulated by altering the ratio between the percussive phase and the baseline continuous positive airway pressure phase. Cioffi et al[28] found that the incidence of pneumonia and the mortality rate were significantly reduced when HFPV was used prophylactically in burn patients with smoke inhalation injury, compared with historical controls where conventional ventilation was used. However, a comparison of gas exchange between HFPV and conventional ventilation was not reported. In a study of pediatric burn

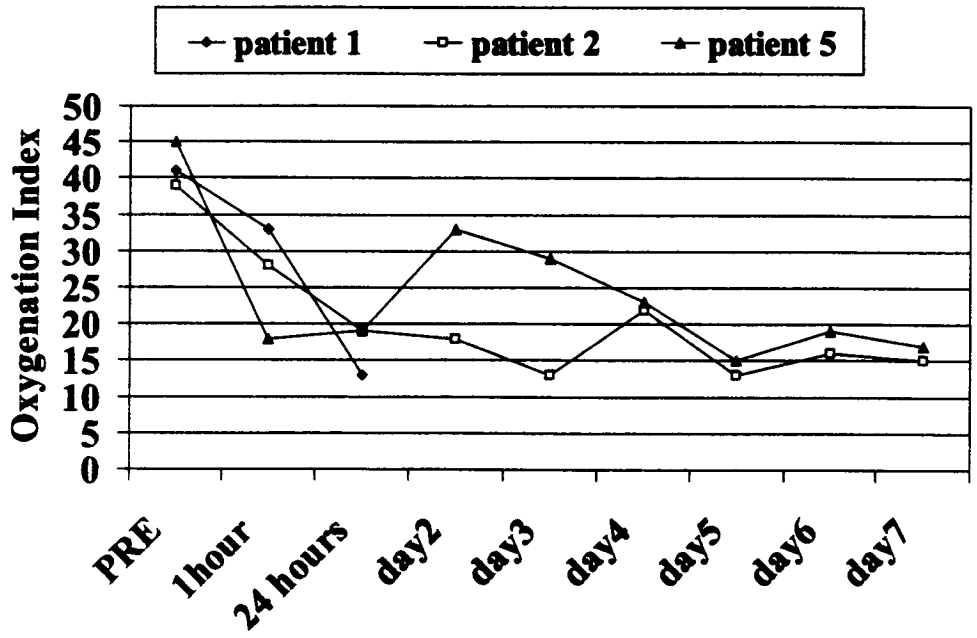

Figure 1. Daily changes in the oxygenation index in the "rescue patients" before (PRE) and after initiation of high-frequency oscillatory ventilation.

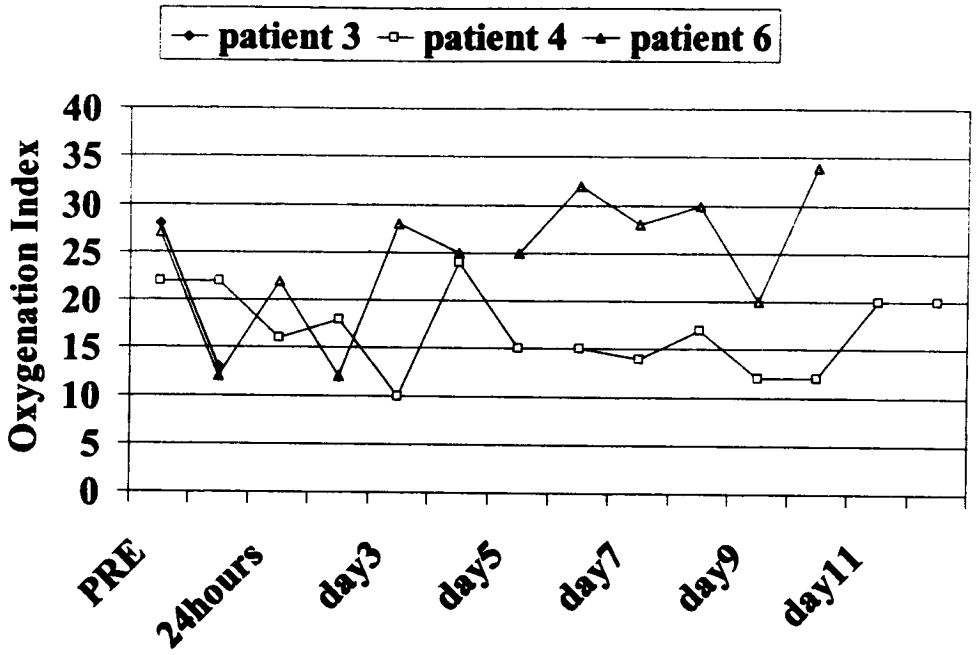

Figure 2. Daily changes in the oxygenation index in the "prophylactic" patients before (PRE) and after the initiation of high-frequency oscillatory ventilation.

patients with inhalation injuries, Cortiella et al[31] demonstrated fewer ventilator days, lower peak inspiratory pressures, significantly less pneumonia, and higher PaO_2/FIO_2 ratios when HFPV was used, compared with historical controls who had received conventional ventilation. HFPV also seems to be a useful salvage technique in burn patients with acute respiratory failure who are failing conventional ventilation. Both Cioffi et al[32] and Reper et al[33] demonstrated improvement in PaO_2/FIO_2 when patients were switched from conventional ventilation to HFPV. Similarly, Rodeberg et al[34] reported improved gas exchange at lower peak airway pressures by using HFPV delivered by the volumetric diffusive respirator, as compared with conventional ventilation in thermally injured children with respiratory failure.

HFOV is a unique form of high-frequency ventilation in which an oscillatory diaphragm produces both active inspiration and active expiration of small subdead space tidal volumes at frequencies up to 15 Hz.[22] Continuous airflow (bias flow) of 20 to 40 l/minute creates a ***sustained*** P_{mean} which helps to recruit and retain open alveoli. Thus, manipulation of the P_{mean} directly affects oxygenation. The amplitude pressure (Δ P) is centered on the P_{mean} and oscillates above and below the P_{mean} with each brief breath. Thus, the P_{mean} is only exceeded by $\Delta P/2$ at the peak of each short inspiratory phase. However, because of resistance in the endotracheal tube, oscillator tubing, and the airway, it is estimated that only 10% of the $\Delta P/2$ (the "effective $\Delta P/2$") is transmitted to the alveoli.[22] Therefore, during HFOV alveolar recruitment is achieved while only highly transient and relatively small peaks (10% of $\Delta P/2$) above the P_{mean} occur, as opposed to conventional ventilation, which features higher peak pressures of greater duration.[22] In this way, HFOV avoids the zone of alveolar overdistention that may occur at high airway pressures and high tidal volumes. Also, during HFOV expiration is active, and therefore, CO_2 elimination is manipulated by adjusting the oscillatory frequency and the ΔP.

Ventilation of patients with ARDS within the "safe zone"—above the zone of atelectasis and derecruitment but below the zone of alveolar overdistention—is now recognized as an important principle in avoiding ventilator-associated lung injury.[11,15,16,35,36] The fundamental difference between HFOV and HFPV is that a constant mean airway pressure is maintained during HFOV, whereas during HFPV mean airway pressure transiently drops when the pulsatile gas flow is interrupted. Thus, although both methods of high-frequency ventilation avoid the zone of alveolar overdistention, only HFOV offers the theoretical advantage of avoiding transient excursions into the zone of alveolar derecruitment and atelectasis. The benefits of avoiding ventilation outside of the "safe zone" have been shown by Simma et al[37] in the surfactant-deficient rabbit lung

model. Use of HFOV resulted in significantly better oxygenation, improved lung mechanics, and less lung parenchyma damage than with other forms of high-frequency ventilation, which allowed either more atelectasis (combined high-frequency ventilation), or which allowed more alveolar overdistention (high-frequency positive pressure ventilation), than HFOV. In the clinical arena, HFOV and HFPV have not been directly compared, making it impossible to say whether one modality offers clinically relevant benefits over the other. It is important to note in the reports on HFPV by Cioffi et al[32] and Reper et al[33] that although all of their patients had respiratory failure, it is unclear whether all had ARDS, because criteria for ARDS were neither defined nor reported. Hence, it is also impossible to make any comparison between the results obtained by these investigators and those obtained in our study of patients with consensus criteria-defined ARDS. Thus, although HFOV is distinctly different from HFPV and although it offers both theoretical and in vitro[37] benefits over other forms of high-frequency ventilation, its actual role in the management of ARDS in burn patients remains unknown. This role will only be defined through prospective evaluation.

The prospective study by Fort et al[11] is the only study that has evaluated HFOV in adults with ARDS. HFOV was used to rescue patients who were failing conventional inverse ratio ventilation (PaO_2/FIO_2, 66 ± 19; OI, 48 ± 15). In contrast to findings by Carlon et al[27] with high-frequency jet ventilation, HFOV produced significant improvements in gas exchange and reductions in FIO_2 requirements. The improvement in gas exchange was believed to be on the basis of high-frequency oscillation and an alveolar recruitment strategy that attempted to obtain an optimal lung volume through tight control of the P_{mean}. A smaller study,[23] involving five patients with refractory lung dysfunction (PaO_2/FIO_2 52.2 ± 47.3) despite maximal conventional ventilation, found a significant increase in the PaO_2/FIO_2 and a decrease in the mean airway pressures after institution of HFOV.

Initiation of HFOV resulted in substantial and immediate improvement in oxygenation in all of the patients in our study. In particular, the "mean airway pressure cost of oxygenation,"[11] as reflected by the oxygenation index, was reduced in each case. In other words, HFOV allowed us to achieve better oxygenation at lower mean airway pressures and, most importantly, with lower peak airway pressures, with decreased intensity of mechanical ventilation. Although all of the patients experienced a dramatic reduction in the OI, the response was variable beyond 24 hours. Paradoxically, the "rescue" patients demonstrated a progressive improvement in the OI over time, whereas the "prophylactic" patients did not. It is difficult to explain this finding, and the small sample size may be the simplest answer. However, it is worth noting that only 33% of the "rescue" patients had sustained an inhalation injury, compared with 100% of the "prophylactic" patients. This raises the question of whether inhalation injury affects the response to HFOV. Another consideration is that the delay in starting HFOV in the prophylactic patients may have adversely affected their response. Although the mean (± SD) delay in this group was only 3 ± 2.3 days (Table 2), this is considerably longer than in other studies of "prophylactic" high-frequency ventilation, such as in the study by Cioffi et al[28] where patients were placed on HFPV within 1 hour of admission to the burn unit. The delay in our patients was probably related to inexperience in using HFOV in a prophylactic fashion. Additionally, there are no evidence-based guidelines that indicate the optimal time to initiate HFOV. Clearly, the next step will be to evaluate HFOV prospectively as an immediate lung protective ventilation strategy and to compare this with conventional lung protective strategies.

Although HFOV did not have any effect on survival, the mortality rate of 83% may be misleading, because all of the deaths were attributable to sepsis and MODS. In two of the deaths, where care was withdrawn (patients 4 and 5), respiratory function was surprisingly good despite failure of other organ systems. In the other three deaths respiratory failure was not a major contributing factor relative to the hemodynamic instability and MODS that existed in these patients at the time of death.

Intraoperative use of HFOV has not been reported in burn patients. With increasing experience, we have become confident in the use of HFOV as a mode of intraoperative ventilation. In at least one case HFOV allowed us to proceed with burn excision, which otherwise would have been impossible with the patient failing on maximal conventional ventilation. Transfer of the patient between the burn unit and the operating room was the most difficult part of the process. The transfer requires careful planning and good coordination and communication among the anesthesiologist, surgeon, and respiratory therapist. Our operating room is located within the burn unit, which simplifies the logistics of patient transfer considerably. Two teams are essential: one in advance to move the oscillator and set it up, and a second following immediately behind with the patient. Although derecruitment and desaturation uniformly occurred during transfer, this was reversed rapidly when HFOV was resumed. Occasionally, a transient increase in the P_{mean} was required as a recruitment maneuver to re-

establish the pretransfer SaO_2. Overheating of the ventilator and excessive airway temperatures have been reported with the use of HFOV, because of the power requirements and heating of the electromagnetic coil.[22] Although we did not encounter this problem, we purposefully kept the operating room cooler than usual, out of concern for this possibility. The mechanical vibrations of the patient caused by the ventilation did not pose a problem for the surgical team.

The patients in this study did not experience any major complications related to HFOV. Inspissation of tracheal secretions and tracheitis are recognized problems during HFOV,[11] but these did not occur in any of our patients. Adequate humidification was confirmed by noting condensation in the endotracheal tube and inspiratory tubing. Tracheal suctioning was performed with an in-line suction catheter (Trach Care, Ballard Medical Products, Draper, UT). There were no pneumothoraces. None of the patients developed hypotension or hemodynamic instability after initiation of HFOV. In general, extreme hypercapnia was not a problem. In most of the cases a rising $PaCO_2$ was easily controlled by increasing the stroke volume by reducing the oscillatory frequency (the opposite of conventional ventilation), and by increasing the ΔP. In two patients these maneuvers failed to control a rising $PaCO_2$. In one patient (case 4) the $PaCO_2$ rose to 90 mmHg (pH 7.13) on the second day of HFOV. The $PaCO_2$ was reduced to 61 mmHg (pH 7.3) over six hours by creating an intentional endotracheal tube cuff leak. The cuff was subsequently reinflated with no further extreme hypercapnia. In the second patient (case 6) the $PaCO_2$ rose to 113 mmHg (pH 7.09) on day 6 of HFOV. This patient had massive edema of the head and neck and an intentional cuff leak had no effect. However, insertion of a 6.0 nasotracheal tube into the supraglottic pharynx as a vent, combined with an intentional cuff leak in the existing oral endotracheal tube, brought the $PaCO_2$ down to 76 mmHg (pH 7.31). Deflation of the endotracheal tube cuff creates an additional route for passive escape of CO_2 and augments active CO_2 removal that occurs as a result of the backward movement of the oscillating diaphragm.

In summary, we have found HFOV to be an extremely useful component in the management of burn patients with ARDS. The most useful role for HFOV was in the salvage of patients facing refractory oxygenation failure despite maximal conventional ventilatory strategies. In these cases a major improvement in oxygenation was obtained at less airway pressure cost, and this response tended to be sustained over their subsequent course on HFOV. In patients who had not yet reached failure or maximal conventional ventilation, early improvement in oxygenation occurred but was not sustained. We cannot explain this finding, but the contribution of inhalation injury in these patients cannot be overlooked, and we hope to examine this relationship more closely in the future. Although the mortality rate of patients on HFOV was high, none of the deaths were attributable to respiratory failure alone, and HFOV at least improved the management of one of many failing organ systems. Finally, we have had considerable success with intraoperative HFOV, and we believe that this capability has allowed us to proceed with burn excision in patients who would otherwise have been considered too unstable to go to the operating room on conventional ventilation. This study now lays the groundwork for a prospective study of HFOV, which we are about to undertake.

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