Prophylactic Use of High-frequency Percussive Ventilation in Patients with Inhalation Injury

WILLIAM G. CIOFFI, JR., M.D., LORING W. RUE III, M.D., THERESA A. GRAVES, M.D., WILLIAM F. McMANUS, M.D., ARTHUR D. MASON, JR., M.D., and BASIL A. PRUITT, JR., M.D.

From the United States Army Institute of Surgical Research, Fort Sam Houston, San Antonio, Texas

Death and the incidence of pneumonia are significantly increased in burn patients with inhalation injury, despite application of conventional ventilatory support techniques. The effect of high-frequency percussive ventilation on mortality rate, incidence of pulmonary infection, and barotrauma were studied in 54 burn patients with documented inhalation injury admitted between March 1987 and September 1990 as compared to an historic cohort treated between 1980 and 1984. All patients satisfied clinical criteria for mechanical ventilation. High-frequency percussive ventilation was initiated within 24 hours of intubation. The patients' mean age and burn size were 32.2 years and 47.8%, respectively (ranges, 15 to 88 years; 0% to 90%). The mean number of ventilator days was 15.3 ± 16.7 (range, 1 to 150 days), with 26% of patients ventilated for more than 2 weeks. Fourteen patients (25.9%) developed pneumonia compared to an historic frequency of 45.8% ($p < 0.005$). Mortality rate was 18.5% (10 patients) with an expected historic number of deaths of 23 (95% confidence limits of 17 to 28 deaths). The documented improvement in survival rate and decrease in the incidence of pneumonia in patients treated with prophylactic high-frequency ventilation (HFV), as compared to a cohort of patients treated in the 7 years before the trial, indicates the importance of small airway patency in the pathogenesis of inhalation injury sequelae and supports further use and evaluation of HFV.

DURING THE PAST three decades, improvements in burn wound management, infection control, and metabolic support increased the survival of thermally injured patients. Inhalation injury, however, continues to be a significant comorbid factor in such patients, and its treatment has been improved little by the use of conventional means of pulmonary support. Bacterial pneumonia, which historically occurred in 38% of all patients with inhalation injury but in only 8% of those without such injury, continues to be the leading cause of morbidity and death. The combination of inhalation injury and pneumonia exert independent but additive effects on the age-related death attributable to burn size.[1]

Current treatment for inhalation injury is supportive and includes aggressive pulmonary toilet, mechanical ventilatory support when indicated, and aggressive treatment of pneumonia when diagnosed. In an ovine model, we showed that the major insult after smoke injury (as indexed by early postinjury ventilation/perfusion (VA/Q) mismatching and histopathologic findings) is the obstruction and collapse of small airways leading to distal atelectasis and subsequent pneumonia.[2] Experimental and clinical data suggest that high-frequency ventilation (HFV) may be beneficial in recruiting and stabilizing such collapsed diseased lung segments.[3-12] In addition some investigators reported improved clearance of secretions from the tracheobronchial tree with the use of HFV.[13] These observations support the hypothesis that HFV, by preventing alveolar collapse and improving secretion clearance, may be beneficial in patients with inhalation injury.

We previously reported a small cohort of 10 patients with inhalation injury requiring mechanical ventilatory support in whom the prophylactic use of high-frequency percussive ventilation (HFPV) appeared to reduce the incidence of pneumonia.[14] This report extends our observations to 54 patients in whom HFPV was used in a prophylactic manner in an attempt to decrease the incidence of pneumonia and improve survival rate.

Presented at the 102nd Annual Scientific Session of the Southern Surgical Association, Boca Raton, Florida, December 3-6, 1990.

The views of the authors do not purport to reflect the positions of the Department of the Army and the Department of Defense.

Address reprint requests to U.S. Army Institute of Surgical Research, Fort Sam Houston, San Antonio, TX 78234-5012.

Accepted for publication January 9, 1991.

Methods

Patient Population

All adult patients admitted to the United States Army Institute of Surgical Research between March 1987 and September 1990 with a diagnosis of inhalation injury were eligible for enrollment in this study. Inhalation injury was confirmed in each patient by bronchoscopy and/or[133] Xenon ventilation–perfusion lung scan. The presence of carbonaceous debris beneath the true vocal cords, mucosal erythema, and ulceration were used to define moderate to severe inhalation injury. Patients with a positive[133] Xenon scan and negative bronchoscopy were determined to have mild inhalation injury. These criteria were established in our earlier review.[1] After meeting the entrance requirements listed in Table 1 and meeting the requirements for intubation and mechanical ventilatory support listed in Table 2, informed consent was obtained from each patient and HFPV initiated for pneumonia prophylaxis.

High-frequency Percussive Ventilation

Description of the high frequency percussive ventilator used in this study has been published.[14] Briefly, HFPV was delivered by a high-frequency pulse generator with gas from the high-frequency pulse generator delivered through a nongated sliding venturi to a standard endotracheal tube. The venturi entrains humidified gas from a fresh bias gas flow provided from the ventilator. The system combines serial high-frequency sub-dead space volume breaths with a variable inspiratory:expiratory (I:E) ratio. Periodic interruption of the high-frequency pulsatile flow is programmed to allow return of airway pressure to baseline continuous positive airway pressure (CPAP). The duration of the percussive phase and of the return to baseline phase are adjusted to manipulate oxygenation and CO_2 elimination. Peak airway pressure also can be varied independently to maintain CO_2 clearance. The frequency of sub-dead space breaths can range between 1.5 and 15 hertz. FIO_2 and PEEP are adjusted to maintain O_2 saturation greater than 90%.

All patients were placed initially on a conventional mechanical ventilator. In those patients intubated elsewhere, such support was of less than 24 hours duration, and all patients were converted to HFPV within 1 hour of admission. The patients intubated at our institution received conventional ventilation during admission processing but were converted to HFPV within 1 hour. After placing the patient on HFPV, standard ventilator settings were used as a baseline and then altered as indicated by arterial blood gas determinations, pulse oximetry, and end tidal CO_2 monitoring. The duration of the percussive phase was set at 2 seconds, with a rate of return to baseline approximately 2 less than the intermittant mandatory ventilation (IMV) setting required to maintain normal acid-base balance on conventional mechanical ventilation. Peak airway pressures were set at 5 cm H_2O less than those developed when a conventional volume-limited ventilator was set to deliver a tidal volume of 12 to 15 mL/kg. The FIO_2 and PEEP were maintained initially at the same levels as on conventional mechanical ventilation. The frequency of the sub-dead space tidal breaths was initially set at 10 hertz. After stabilization for approximately 30 minutes, arterial blood gas measurements were obtained and adjustments made as indicated. The goal of ventilator therapy was to maintain oxygenation and ventilation at the lowest possible peak airway pressure and fractional inspired oxygen concentration. Patients were weaned and extubated according to standard criteria.

TABLE 1. *Study Entrance Criteria*

Inhalation injury documented by bronchoscopy or Xenon lung scan
Clinical requirement for ventilatory support
Admission within 48 hours of injury
Older than 15 years

TABLE 2. *Requirements for Mechanical Ventilatory Support*

1. Respiratory rate > 35/min
2. Vital capacity < 15 mL/kg
3. Inspiratory force < 25 cm H_2O
4. PAO_2/FIO_2 < 200
5. PCO_2 > 50 mmHg
6. Vd/Vt > 0.6
7. Upper airway edema
8. PCO_2 < 50 mmHg but progressively increasing
9. Increased work of breathing

Diagnosis of Pneumonia

The diagnosis of pneumonia was based on standard criteria used in this institution for the past decade. Patients with sputum leukocytosis (more than 25 white blood cells per high-power field), lack of oropharyngeal contamination (less than 10 squamous cells per high-power field), a predominant organism on culture, and an infiltrate on chest roentgenograms were diagnosed as having pneumonia.

Data Analysis

The incidence of pneumonia and death in the study patients was compared with predicted values based on two previous studies. The first predictor used relates burn size and age to death for all patients admitted to the Institute of Surgical Research between January 1980 and December 1986. The second predictor used as a basis for comparison relates burn size, age, the presence of inhalation injury and the occurrence of pneumonia to death

TABLE 3. *Burn Mortality Predictors*

$$\text{Predicted Mortality (PM)} = \frac{e^Y}{1 + e^Y}$$

I. Logistic equation relating burn size and age to mortality: 1980–1986

$$Y = -4.8216 + 0.10299\,(\text{PCTB}) - 0.18879\,(\text{Age}) + 0.50873\,(\text{Age}^2/100) - 0.27915\,(\text{Age}^3/10{,}000)$$

II. Logistic equation relating burn size, age, inhalation injury, and pneumonia to mortality: 1980–1984

$$Y = -3.4953 + 0.09589\,(\text{PCTB}) - 0.1988\,(\text{Age}) + 0.4478\,(\text{Age}^2/100) - 0.20314\,(\text{Age}^3/10{,}000) + 0.59056\,(\text{II}) + 0.92530\,(\text{PNeu})$$

PCTB, percentage of total body surface burned.
II = −1.0 if inhalation injury absent; +1.0 if inhalation injury present.
PNEU, −1.0 if pneumonia absent; +1.0 if pneumonia present.

in patients admitted between 1980 and 1984. The incidence of pneumonia in this latter patient population also was used for comparison purposes. Solution of the logistic equations listed in Table 3 provide the exponents for use in calculating the two values for predicted death.

Results

Patient Population

Fifty-four patients meeting the entrance criteria were enrolled in the study. Routine demographic data are included in Table 4. Ten patients died, for a mortality rate of 18.5%. The distribution of patients by burn size demonstrates that 50% of the patients had burns ranging between 30% and 60% of the body surface, which is the group of patients in whom inhalation injury has been reported to have its greatest impact on death (Fig. 1). Segregation of the patients by outcome revealed the expected differences between the two groups (Table 5); nonsurvivors were older and had larger burns and a greater incidence of pneumonia. Fifty-two of the fifty-four patients were diagnosed as having inhalation injury by bronchoscopy. The two patients with negative bronchoscopy but positive ^{133}Xenon scans developed severe adult respiratory distress syndrome (ARDS) in the first postburn week, necessitating mechanical ventilatory support.

TABLE 4. *Demographic Data*

Age	32.2 ± 1.8 (15–88)*
TBSB	47.8 ± 3.1 (0–90)
Sex	40 male, 14 female
Days on ventilator	15.3 ± 2.2 (1–150)
Bronchoscopy positive	96.3%
Incidence of pneumonia	25.9%
Mortality	10/54 (18.5%)

* X ± SEM (range).
TBSB, total body surface burn.

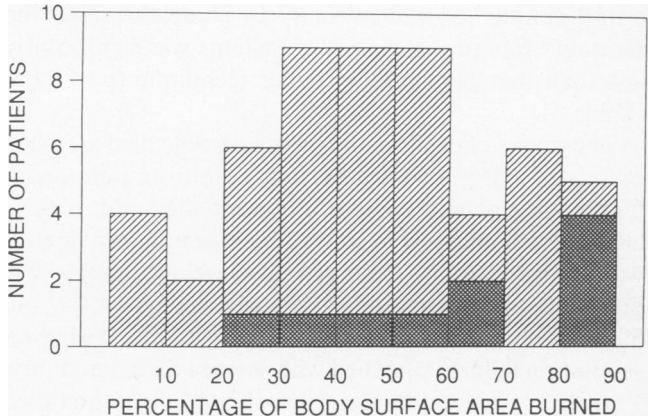

FIG. 1. Distribution of burn size for the 54 patients in the study. Of note is that 50% of the patient burn sizes are between 30% to 60% of the body surface area, which is the group of patients in whom inhalation injury exerts its greatest influence on mortality. Survivors are reported by the crossed bars, and nonsurvivors by the crosshatched bars.

Historically 45.8% of patients with positive bronchoscopy and 19.5% of patients with negative bronchoscopy but a positive ^{133}Xenon lung scan developed pneumonia. Based on that experience, 25 of the study patients would have been expected to develop pneumonia during hospitalization. Pneumonia was diagnosed in only 14 (26%) of the patients in this study, an incidence differing significantly from that of the comparison cohort ($p < 0.003$).

Actual Versus *Predicted Death*

Ten deaths occurred in this group of patients, for an observed mortality rate of 18.5%. To determine whether HFPV influenced outcome in this group of patients, we compared this observed mortality rate with two mortality predictions generated from patient data from this institution, as noted above. The first, based on burn size and age, related death in all patients admitted to this institution between January 1980 and December 1986, predicts the deaths of 19 patients (35%) in the study population, with a 95% confidence interval of 13 to 25 deaths. The second, based on burn size and age, related death in conjunction with the additive effects of inhalation injury and pneumonia and generated from patient data between January 1980 and December 1984, predicts 23 deaths (42.6%) with

TABLE 5. *Comparison of Survivors and Nonsurvivors*

	Survivors	Nonsurvivors	
Age (years)	29.6 ± 1.5*	43.3 ± 6.5	$p < 0.05$
TBSB	43.7 ± 3.2	65.3 ± 7.1	$p < 0.01$
Incidence of pneumonia	20.5%	50%	$p < 0.05$

* Mean ± SEM.
TBSB, total body surface burn.

a 95% confidence interval of 17 to 28 deaths. Thus the mortality rate in this cohort of patients was significantly less than that predicted by either technique ($p < 0.05$) (Table 6).

The causes of death in those patients who died are listed in Table 7. Of the 10 deaths, four were from pulmonary failure. One patient could not be ventilated and oxygenated and was changed to conventional ventilatory support with the same result. Three patients developed progressive pulmonary failure and died on postburn days 12, 43, and 50, respectively. Of the remaining 6 patients, 2 were resuscitation failures who died with severe inhalation injury, 1 patient extubated himself on postburn day 7 and died of cardiopulmonary arrest despite an emergency tracheostomy, and 1 patient was removed from the study by his attending surgeon. Two patients died from cerebrovascular accidents after they were extubated for 30 and 45 days, respectively.

Ventilator complications were rare. Two patients developed severe necrotizing tracheobronchitis. It could not be determined whether this was secondary to the ventilator or the disease process itself. Barotrauma occurred in three patients. Two developed significant subcutaneous emphysema and one patient developed bilateral pneumothoraces requiring tube thoracostomies.

Discussion

The combination of cutaneous thermal injury and inhalation injury results in a significantly higher mortality rate than that attributable to cutaneous thermal injury alone. This additive effect of inhalation injury on death is most apparent in patients in whom predicted mortality attributable to age and burn size ranges from 40% to 60%. Inhalation injury also results in a marked increase in the incidence of bacterial pneumonia. As previously stated, only 8.8% of patients with thermal injury but without inhalation injury develop pneumonia during their course of treatment. The presence of inhalation injury, whether diagnosed by bronchoscopy or ^{133}Xenon scan, historically resulted in a 38% incidence of pneumonia, and the combination of inhalation injury and pneumonia has an even more drastic effect on outcome, increasing the mortality rate by as much as 60%.[1]

TABLE 6. *Actual* Versus *Predicted Outcome*

Predictor	Predicted Deaths	95% CL	Observed
#1 (1980–1986)	19	13–25	10
#2 (1980–1984)*	23	17–28	10

* This predictor includes the impact that inhalation injury and pneumonia have on outcome.
CL, confidence level.

TABLE 7. *Cause of Death*

TBSB (%)	Age (years)	PBD	Cause of Death
90	32	01	Resuscitation failure
85	25	03	Resuscitation failure
59	40	07	Accidental extubation
36	59	40	Removed from study
47	29	50	SBE, CVA, 30 days after extubation
65	60	80	CVA, 45 days following extubation
89	25	12	Pulmonary failure
30	88	43	Pneumonia (*Staph. aureus*), pulmonary failure
64	49	01	Unable to ventilate
86	29	50	Pneumonia, Aspergillus wound infection

TBSB, total body surface burn; PBD, postburn day.

Ideally the optimal treatment of any disease should reverse the pathophysiologic process without causing further injury. When inhalation injury is severe enough to require conventional mechanical ventilatory support, such an outcome is not achieved. The pathophysiologic response to inhalation injury includes extensive tracheobronchial injury, which results in sloughing of the mucosal lining of the respiratory tract and leads to obstruction of small- and moderate-sized airways. In addition, the mucociliary transport mechanism is impaired, resulting in impaired clearance of secretions and the sloughed debris. Distal airway obstruction results in atelectasis and, in conjunction with the disruption of the endothelial and epithelial integrity of the alveolus, produces foci for the development of bacterial overgrowth and subsequent pneumonia. The combination of atelectasis, pneumonia, and airway obstruction produces significant derangement of ventilation–perfusion relationships.

Conventional mechanical ventilatory support does not reverse these processes, is not characterized by improved clearance of secretions, and may actually compound the existing injury.[15] Conventional volume-limited ventilation in patients with inhalation injury normally is instituted at a tidal volume of 12 to 15 mL/kg. With such a ventilatory setting, peak inspiratory pressures often are elevated during the resuscitative and fluid mobilization phase of care. Recently Tsuno[16] reported adverse pulmonary effects of volume-limited mechanical ventilation when peak inspiratory pressures exceed 30 cm of water in paralyzed, anesthetized healthy sheep. Animals ventilated with an FIO_2 of 40% and a tidal volume of 10 mL/kg, with peak inspiratory pressure less than 18 cm of water, showed no measurable deleterious changes in lung function or histopathology after 48 hours of support. Animals ventilated with larger tidal volumes, resulting in peak inspiratory pressures greater than 30 cm H_2O, demonstrated progressive deterioration in static lung compliance, functional residual capacity, and arterial blood gases. Severe pul-

monary atelectasis, increased wet lung weight, and an increase in the minimum surface tension of saline lung lavage fluid were noted at autopsy. These data indicate that even in normal healthy lungs, prolonged elevation of inspiratory pressures may result in injury.

If pneumonia develops after resuscitation, the requirement for increased inspired oxygen concentrations to achieve normoxia may result in increased pulmonary damage when infection is present. Coalson et al.[17] recently reported a synergistic effect of hyperoxia and infection resulting in significant pulmonary dysfunction and damage. In a primate model, the combination of 80% O_2 and Pseudomonas pneumonia was as injurious as 100% oxygen during an 11-day period, while 80% O_2 or pneumonia alone resulted in minimal dysfunction.

The reported beneficial effects of HFV (ventilator frequency greater than 60 breaths/minute and tidal volumes of less than anatomic dead space) include lower peak airway pressures than those generated by conventional ventilation, positive endotracheal pressure throughout the ventilatory cycle, increased functional reserve capacity, and more efficient pulmonary gas distribution.[18] Unfortunately each of the advantages claimed for specific high-frequency ventilators has been refuted in various reports.[9,10,12] If, however, a form of HFV could achieve some of these advantages and maintain oxygenation and CO_2 clearance at lower inspiratory pressures and fractional inspired concentrations of oxygen, it might be possible to provide ventilatory support and avoid the deleterious side effects of conventional support.

In evaluating clinical reports of HFV, the physician must recognize that there are several types of high-frequency ventilators, all with different characteristics and different potentially adverse effects. Furthermore one must differentiate between prophylactic use of the ventilator, as in this study, and therapeutic or salvage use of the high-frequency device for patients in whom conventional mechanical ventilatory support has failed. Many reports documented the effectiveness of short-term salvage use of HFV in patients with ARDS.[7,8] Our own previously reported experience demonstrated that the ventilator used in these studies could oxygenate and ventilate patients at lower airway pressures and inspired oxygen concentrations, but all the patients died despite improved pulmonary performance.[14] Other reports also failed to identify a survival advantage with the use of HFV as a salvage mode of ventilatory support.

In this study we used HFPV prophylactically in an attempt to avoid the adverse effects of mechanical ventilatory support while reversing or minimizing some of the pathophysiologic changes that occur after inhalation injury. Our data indicate that, as compared to a recent historic cohort, the use of HFPV resulted in a significant decrease in the incidence of pneumonia and a decrease in the number of deaths.

There are several problems inherent to the use of historic controls. The development of more sensitive diagnostic techniques resulting in the diagnosis of less severe injury could favorably bias the results of recent studies, although the diagnostic modalities and criteria have remained constant since 1976. It is generally accepted that during the past three decades, survival of all patients with thermal injury has improved. Even so the effects of inhalation injury and pneumonia on outcome have remained refractory to standard treatment, as indicated by the mortality predictor used at this institution. Furthermore the predictors used in this study introduce some bias against finding an improvement in outcome in the current study population as compared to the populations on which the predictors were based. The predictor that account for the effects of both burn size and age as well as pneumonia and inhalation injury on death was based on all patients with inhalation injury admitted during the years 1980 to 1984, regardless of whether they required mechanical ventilatory support. The present study population includes only the sickest patients with the most significant injuries, all requiring ventilatory support. Demonstration of a survival advantage in this group of patients compared to a group that included patients with less severe injury supports the hypothesis that HFPV has a significant, beneficial effect. In short it seems reasonable to assign a major portion of the decrease in incidence of pneumonia and improvement in outcome of the study patients to the ventilatory support used.

Only two other published studies in the literature evaluated the prophylactic use of HFV in patients requiring ventilatory support as prophylaxis against ARDS. In 1986 Carlon[18] reported a study of 309 patients who were randomized to high-frequency jet ventilation or conventional ventilatory support. All patients who were admitted to the intensive care unit and who were at risk for the development of pulmonary failure were entered into the study. The use of high-frequency jet ventilation resulted in lower peak airway pressures but did not decrease the 4% incidence of barotrauma or improve the overall outcome as compared to conventional support. In 1990 Hurst et al.[19] reported a study of 113 patients at risk for the development of ARDS who were randomized to receive ventilatory support with HFPV or conventional mechanical ventilation before the onset of ARDS. Changes in ventilator settings were made to achieve the same therapeutic endpoints in both groups of patients. There was no difference in the percentage of patients who developed ARDS in either group. In the patients who developed ARDS, HFV achieved therapeutic endpoints at lower peak airway pressures, lower positive end expiratory pressures,

and an increased inspiratory time as compared to the conventional group. There was, however, no difference in the incidence of barotrauma or outcome in those patients. Both of these studies involved heterogeneous patient populations, in which the etiology of respiratory failure was diverse, usually a consequence of a systemic insult that resulted in diffuse parenchymal disease and dysfunction. This type of insult is quite distinct from that seen after smoke inhalation in both humans and animal models, in which edema resolves rapidly after resuscitation and repair of the airway mucosa typically occurs within 14 to 21 days.

The exact mechanism by which HFPV achieved the results reported in this study is not known. We hypothesize that the ability to maintain ventilation and oxygenation at lower peak airway pressures and inspired oxygen concentrations may decrease the iatrogenic injury that occurs with conventional mechanical ventilatory support. Extrapolation of the data reported by Tsuno[16] to humans would indicate that ventilation at lower peak airway pressures offers significant advantage, especially in lungs that have already been injured. In addition several studies now suggest that asymmetric high-frequency breaths improve clearance of secretions, results that have been obtained with high-frequency jet ventilators and high-frequency oscillators, both *in vitro* and *in vivo*.[13,20-22] Our clinical experience supports this finding. Patients with severe inhalation injury treated prophylactically with high-frequency percussive ventilation typically are found, by bronchoscopic examination, to have large deposits of secretions at the tip of the endotracheal tube. After removal of these secretions, the main stem bronchi and distal airways often are patent and free of pathologic secretions. The documented improvement in survival and the decrease in the incidence of pneumonia in patients treated with prophylactic HFPV, as compared to the recent historic cohort, indicate the importance of maintaining small airway patency in reducing the sequela of inhalation injury. The beneficial effects reported here and the paucity of ventilator complications support continued use and further evaluation of HFV in patients with inhalation injury.

References

1. Shirani KZ, Pruitt BA Jr, Mason AD Jr. The influence of inhalation injury and pneumonia on burn mortality. Ann Surg 1987; 205: 82–87.
2. Shimazu T, Yukioka T, Hubbard G, et al. Frequency of VA/Q rates following smoke inhalation. USAISR Annual Research Progress Report 1985.
3. Bland Rd, Kim MH, Light MJ, et al. High frequency mechanical ventilation in severe hyaline membrane disease. Crit Care Med 1980; 8:275–280.
4. Brichant JF, Rouby JJ, Viars P. Intermittent positive pressure ventilation with either positive end expiratory pressure or high frequency jet ventilation (HFJV), or HFJV alone in human acute respiratory failure. Anesth Analg 1986; 65:1135–1142.
5. Butler WJ, Bohn DJ, Bryan AC, et al. Ventilation by high frequency oscillation in humans. Anesth Analg 1980; 59:577–584.
6. El-Baz N, Faber LP, Doolas A. Combined high frequency ventilation for management of terminal respiratory failure: a new technique. Anesth Analg 1983; 62:39–49.
7. Hurst JM, Branson RD, DeHaven CB. The role of high frequency ventilation in post-traumatic respiratory insufficiency. J Trauma 1987; 27:236–242.
8. Hurst JM, DeHaven CB. Adult respiratory distress syndrome: improved oxygenation during high frequency jet ventilation/continuous positive airway pressure. Surgery 1984; 96:764–769.
9. Jibelian G, Lachmann B. Gas exchange during conventional and high frequency pulse ventilation in the surfactant deficient lung: influence of positive and expiratory pressure. Crit Care Med 1984; 12:769–773.
10. Kaiser KG, Davies NJ, Rodriguez R, et al. Efficacy of high frequency ventilation in presence of extensive ventilation perfusion mismatch. J Appl Physiol 1985; 58:996–1004.
11. Kolton MK, Cattran CB, Kent G, et al. Oxygenation during high frequency ventilation compared with conventional mechanical ventilation in two models of lung injury. Anesth Analg 1982; 61: 323–332.
12. Kumar BS, Beney K, Jastremski M, et al. High frequency jet ventilation versus conventional ventilation after surfactant displacement in dogs. Crit Care Med 1984; 12:738–741.
13. Freitag L, Long WM, Kim CS, Wanner A. Removal of excessive bronchial secretions by asymmetric high-frequency oscillations. J Appl Physiol 1989; 67(2):614–619.
14. Cioffi WG, Graves TA, McManus WF, Pruitt BA Jr. High-frequency percussive ventilation in patients with inhalation injury. J Trauma 1989; 29(3):350–354.
15. Mammel MC, Boros SJ. Airway damage and mechanical ventilation: a review and commentary. Pediatr Pulm 1987; 3:443–447.
16. Tsuno K, Prato P, Kolobow T. Acute lung injury from mechanical ventilation at moderately high airway pressures. J Appl Physiol 1990; 69(3):956–961.
17. Coalson JJ, King RJ, Winter VT, et al. O_2—and pneumonia-induced lung injury I. Pathological and morphometric studies. J Appl Physiol 1989; 67(1):346–356.
18. Carlon GC, Howland WS, Ray C, et al. High-frequency jet ventilation—a prospective randomized evaluation. Chest 1983; 84(5): 551–559.
19. Hurst JM, Branson RD, Davis K Jr, et al. Comparison of conventional mechanical ventilation and high-frequency ventilation. Ann Surg 1990; 211(4):486–491.
20. Hachenberg T, Wendt M, Deitmer T, Lawin P. Viscoelasticity of tracheobronchial secretions in high-frequency ventilation. Crit Care Med 1987; 15(2):95–98.
21. Freitag L, Kim CS, Long WM, et al. Mobilization of mucus by airway oscillations. Acta Anaesthesiol Scand 1989; 33(Suppl 90): 93–101.
22. Thangathurai D, Holm AP, Mikhail M, et al. HFV in management of a patient with severe bronchorrhea. Resp Mgmt 1988; Jan/Feb:31–33.

DISCUSSIONS

DR. HARVEY SUGERMAN (Richmond, Virginia): In this study, high-frequency percussive ventilation from 1987 through 1990 was associated with a significant decrease in, one, the incidence of pneumonia and, two, the mortality rate when compared, as mentioned, to historical controls for inhalational injury from 1980 through 1984 and mortality rate from 1980 through 1986.