Factors Effecting the Outcome of Acute Respiratory Distress Syndrome in Pediatric Patients Treated with High Frequency Oscillatory Ventilation

SORASAK LOCHINDARAT, MD*,
PANIDA SRISAN, MD*,
PRAVIT JATANACHAI, MD*

Abstract

Objectives : To evaluate the survival rate and factors affecting the outcome of pediatric patients treated with high-frequency oscillatory ventilation (HFOV) for diffuse alveolar disease (DAD) compatible with acute respiratory distress syndrome (ARDS).

Method : A cohort study was conducted at the pediatric intensive care unit of Queen Siritkit National Institute of Child Health from 1st January 1999 to 31st December 2001. Children who suffered from DAD compatible with ARDS were enrolled. Inclusion criteria were $PaO_2/FiO_2 < 200$ and oxygenation index (OI) > 10. High-frequency oscillatory ventilator (3100A Sensor Medics Corp, Yorba Linda, Calif) was used applying high volume strategy of treatment. Patients were weaned to conventional ventilation (CV) once clinical improvement occurred. Demographic data, duration of CV mode before changing to HFOV, duration of HFOV, ventilator parameters and gas exchange variables from beginning and during the course of HFOV were recorded, so patient data could be compared between surviving and non-surviving groups.

Results : A total of 21 children were enrolled during the 3 year period. There were 4 patients with simultaneous air leak syndrome and a total of 10 male patients. The average age was 3.58 ± 3.9 years. There were 11 surviving patients (52.4%). Data of ventilator parameters and gas exchange variables after changing to HFOV for 4-6 hours for the two groups, FiO_2 was higher (0.99 ± 0.32 *vs* 0.84 ± 0.18; p = 0.02) and alveolar arterial oxygen gradient [$P(A\text{-}a)O_2$] was lower (448.5 ± 140.8 *vs* 562.7 ± 99.9 mmHg; p = 0.047) in the surviving group than in the non-surviving group. Concerning mean airway pressure (Paw), oxygenation index (OI), $P(A\text{-}a)O_2$ and PaO_2/FiO_2 at initiation and during the course of HFOV with comparison of the surviving and non-surviving groups: Paw and OI decreased in the surviving group and was significantly different at 36 and 24 hours respectively. $P(A\text{-}a)O_2$ was statistically significantly lower at 6 hours after HFOV initiation in the surviving group. PaO_2/FiO_2 was statistically significantly increased at 24 hours in the surviving group.

Conclusion : Implement of HFOV is useful in patients with DAD, ARDS and air leak syndrome from the initial phase of illness which fulfill criteria for decreasing ventilator induced lung injury and thus decrease the mortality rate from ARDS. Predisposing survival factor showing statistically

significant differences was lower Paw during CV before changing to HFOV, lower Paw at 36 hours, lower OI at 24 hours, lower $P(A\text{-}a)O_2$ at 6 hours and higher PaO_2/FiO_2 at 24 hours. These parameters are good indicators for the prognosis of ARDS for patients responding or not responding to HFOV.

Key word : acute respiratory distress syndrome, high-frequency oscillatory ventilation

LOCHINDARAT S, SRISAN P, JATANACHAI P
J Med Assoc Thai 2003; 86 (Suppl 3): S618-S627

* Division of Pediatric Pulmonology, Department of Pediatrics, Queen Sirikit National Institute of Child Health, Bangkok 10400, Thailand.

High-frequency ventilation (HFV) was introduced for pediatric respiratory failure in 1960. The US-FDA approved this rescue therapy in 1980. In Thailand, HFV has been used for ten years. HFV is divided into 4 types[1,2]: high-frequency positive pressure ventilation (HFPPV), high-frequency jet ventilation (HFJV), high-frequency flow interruption (HFFI) and high-frequency oscillation (HFO). The function of HFO is the displacement of the diaphragm or piston for the oscillating pressure. Mean airway pressure (Paw) can be set for the background oscillating pressure. The tidal volume (V_T) is 1-3 ml/kg which is less than the dead space volume. The oxygenation depends on FiO_2 and Paw. The ventilation depends on fresh gas flow and amplitude (magnitude of pressure oscillation) but reverses with frequency. The common instrument models for HFO are 3100 A (Sensor Medics Corp., Yorba Linda, Calif), SLE 2000 HFO (SLE Limited, Survey, UK) and Humming II (Senko Medical Instruments mfg Co, Tokyo, Japan). The major problem of patients with diffuse alveolar disease is ventilator induced lung injury. High V_T is an important cause of lung injury[3]. Change in lung volume is more important than change in airway pressure as a predisposing factor in lung injury. Now, the term volutrauma is used instead of barotrauma[4,5]. HFV is used for the problem of ventilator induced lung injury. The mechanism of HFV[6] is a high Paw, a high volume strategy to recruit alveoli in the low compliance lung in order to improve gas exchange. A low V_T, less than the dead space volume (V_D) in HFV, will decrease volutrauma but is decompensated by high frequency. The benefits of HFV are decrease of pressure swings to reduce barotrauma and change in flow patterns to improve ventilation-perfusion matching. Gas exchange mechanisms in HFOV[7,8] are convection, molecular diffusion, asymmetric airway velocity profile, pendulluft and cardiogenic oscillation.

Objectives

A cohort study was carried out to evaluate the survival rate and factors affecting the outcome of pediatric patients treated with HFOV for diffuse alveolar disease (DAD) compatible with acute respiratory distress syndrome (ARDS) according to criteria of the American-European Consensus[9] in 1994.

SUBJECTS AND METHOD

Subjects were pediatric patients under 15 years of age with DAD and acute respiratory failure compatible with ARDS[9] who had PaO_2/FiO_2 less than 200 and an oxygenation index(OI) more than 10. Oxygenation index was calculated from the formula $OI = (FiO_2 \times Paw)/PaO_2 \times 100$. All patients were admitted to the pediatric intensive care unit (PICU) of Queen Sirikit National Institute of Child Health (QSNICH) in the 3 year study period between 1 January 1999 and 31 December 2001. Model 3100 A (Sensor Medics Corp, Yorba Linda, Calif) was used

for high-frequency oscillatory ventilation. Initial ventilator settings were:[10,11]

1) FiO_2 1.0, 2) Frequency 5-15 Hz, 3) Inspiratory time (Ti) 33 per cent, 4) Paw 4-8 cmH_2O above CV mode, 5) Bias gas flow $\geq$ 18 l/m, 6) Pressure amplitude (ΔP) adjusted until visible vibration of the chest wall or a $PaCO_2$ between 45-55 mmHg.

After changing to HFOV, the supportive treatment was the same as before. Patients could be sedated or paralysed by midazolam and vecuronium. If after initial settings, the patients still had persistent hypoxemia but the cardiac function was normal and the chest X-ray didn't show hyperinflation, the ventilator settings were changed by increasing Paw until the oxygen saturation was more than 90 per cent, then FiO_2 was slowly decreased to less than 0.6. Paw was not increased to the point that the chest X-ray showed hyperinflation. If Paw was increased to 45 cm H_2O and FiO_2 1.0 but the oxygen saturation was still less than 90 per cent, the patient was considered not responding to HFOV.

Pressure amplitude was adjusted until there was visible vibration of the chest wall and the level of $PaCO_2$ was between 45-55 mmHg with a pH > 7.25. If ΔP was already high, and $PaCO_2$ was still high, ventilator adjustments were made by decreasing frequency down to 3 Hz because low frequency can increase piston displacement thus increasing V_T. Air-leak syndrome usually occurs from high pressure settings in the CV mode. In this condition when one switches to HFOV, initial Paw can be set equal to the level in the CV mode or the lowest level to maintain optimum lung volume. FiO_2 can be increased and adjust to accepted value of oxygen saturation of 85 per cent.

After clinical improvement, weaning was done[12] by decreasing Paw to prevent lung overdistension which could cause barotrauma and hemodynamic disturbance. Paw was decreased by 1-2 cm H_2O each time by monitoring oxygen saturation and the chest X-ray. From blood gas, if the level of $PaCO_2$ decreased, ΔP was reduced without adjusting the frequency. When the setting was decreased to Paw $\leq$ 15 cm H_2O, $FiO_2 \leq 0.4$, chest X-ray improvement and no air leak syndrome, then the patients were switched to CV mode. The setting in CV mode was Paw $\leq$ 15 cm H_2O, $FiO_2 \leq 0.4$, IMV rate $\leq$ 30 times/minute and PIP $\leq$ 35 cm H_2O with oxygen saturation $\geq$ 90 per cent.

Demographic data, duration of CV mode before changing to HFOV, duration of HFOV, ventilator parameters and gas exchange variables from the beginning of HFOV and then at 6, 12, 24, 36, 48, 60 and 72 hours were recorded, so patient data could be compared between the surviving and non-surviving groups.

Statistical analysis

Fisher's exact test was used for categorized data and student *t*-test for continuous variables.

RESULTS

A total of 21 patients suffering from DAD with acute respiratory failure compatible with ARDS were enrolled during the 3 year study from 1 January 1999 to 31 December 2001. There were 4 patients (19%) with air leak syndrome. There were 10 males (47.6%) and the average age was (X $\pm$ SD) 3.58 $\pm$ 3.9 years. There were 11 surviving patients (52.4%). Table 1 shows the demographic data comparing the surviving and non-surviving groups. In the surviving group, there were 7 males compared with 3 in the non-surviving group; no statistical difference was found between the two groups. Young infants have small lung volume and are prone to respiratory failure. There was 1 surviving and 4 non-surviving infants with small lung volume; no statistical difference was found. Two patients in the surviving and non-surviving groups had normal nutritional status; no statistical difference was noted. There were a total of 10 immunocompromised hosts due to leukemia, lymphoma, neuroblastoma and AIDS; 6 were in the surviving and 4 in the non-surviving groups; no statistical difference was found.

Table 2 shows the duration of CV and HFOV in the two groups. Duration of CV before changing to HFOV in the surviving group was 73.3 $\pm$ 70.5 hours and in the non-surviving group it was 93.1 $\pm$ 79.7 hours; no statistical difference was found. Duration of HFOV in the surviving group was 213.5 $\pm$ 160.4 hours and in the non-surviving group it was 116.6 $\pm$ 59.2 hours; no statistical difference was found.

Table 3 compares data of ventilator parameters and gas exchange variables in CV before changing to HFOV in the two groups. Mean airway pressure was statistically significantly higher ($p = 0.02$) in the non-surviving group (17.6 $\pm$ 2.9 cm H_2O) than in the surviving group (14.9 $\pm$ 1.7 cm H_2O). There was no

Table 1. Demographic data of ARDS patients comparing the surviving (n = 11) and non-surviving groups (n = 10) showing number and per cent of patients.

	Surviving	%	Non-surviving	%	P-value
Sex					
Male	7	63.6	3	30	0.2
Female	4	36.4	7	70	
Age (yr)					
< 1	1	9.1	4	40	0.12*
1-5	6	54.5	6	60	
6-10	1	9.1	-	-	
11-15	3	27.3	-	-	
Nutritional status					
Normal	2	18.2	2	20	0.46**
1° malnutrition	6	54.5	7	40	
2° malnutrition	3	27.3	2	20	
3° malnutrition	-	-	2	20	
Immunological status					
Immunocompetent	5	45.5	6	60	0.41
Immunocompromised	6	54.5	4	40	

* Comparison of groups less than 1 year and more than 1 year
** Comparison of those with normal nutritional status and malnutrition

Table 2. Comparison of duration of CV and HFOV between the surviving (n = 11) and non-surviving groups (n = 10).

	Surviving	Non-surviving	P-value
Duration of CV before changing to HFOV (h)	73.3 ± 70.5	93.1 ± 79.7	0.56
Duration of HFOV (h)	213.5 ± 160.4	116.6 ± 59.2	0.10

Table 3. Comparing ventilator parameters and gas exchange variables during CV before changing to HFOV between the surviving (n = 11) and non-surviving groups (n = 10).

	Surviving	Non-surviving	P- value
FiO_2	0.95 ± 0.85	0.95 ± 0.1	0.91
PaO_2 (mmHg)	64.2 ± 16.7	66.2 ± 20.2	0.81
$PaCO_2$ (mmHg)	38.1 ± 9.6	48.4 ± 19.0	0.13
PaO_2/FiO_2	67.9 ± 17.0	70.5 ± 23.8	0.78
$P(A\text{-}a)O_2$ (mmHg)	562.2 ± 65.2	554.1 ± 76.0	0.80
OI	23.0 ± 5.7	27.4 ± 9.1	0.21
PIP (cm H_2O)	26.1 ± 3.4	28.6 ± 4.4	0.17
Paw (cm H_2O)	14.9 ± 1.7	17.6 ± 2.9	0.02*

P(A-a)O = alveolar arterial oxygen gradiant, OI = oxygenation index,
PIP = peak inspiratory pressure, Paw = mean airway pressure
* Statistically significant difference

statistically significant difference in other data. Table 4 compares the data of ventilator parameters and gas exchange variables after changing to HFOV for 4-6 hours for the two groups. FiO_2 was higher (0.99 ± 0.32 *vs* 0.84 ± 0.18; p = 0.02) and alveolar arterial oxygen gradient [$P(A\text{-}a)O_2$] lower (448.5 ± 140.8 *vs* 562.7 ± 99.9 mmHg; p = 0.047) in the surviving group than in the non-surviving group.

Table 4. Comparing ventilator parameters and gas exchange variables after changing to HFOV for 4-6 hours between the surviving (n = 11) and non-surviving groups (n = 10).

	Surviving	Non-surviving	p- value
FiO_2	0.99 ± 0.32	0.84 ± 0.18	0.02*
PaO_2 (mmHg)	83.1 ± 29.9	96.8 ± 39.5	0.38
$PaCO_2$ (mmHg)	34.3 ± 14.5	41.4 ± 11.4	0.4
PaO_2/FiO_2	84.8 ± 34.4	124.9 ± 71.2	0.12
$P(A\text{-}a)O_2$ (mmHg)	448.5 ± 140.8	562.7 ± 99.9	0.047*
OI	28.2 ± 12.1	23.3 ± 12.8	0.38
Paw (cm H_2O)	21.4 ± 2.6	22.2 ± 2.7	0.5
Frequency (HZ)	7.0 ± 0.8	7.1 ± 1.9	0.88
Amplitude (ΔP)	51.2 ± 8.5	55.4 ± 8.3	0.27

* Statistically significant difference

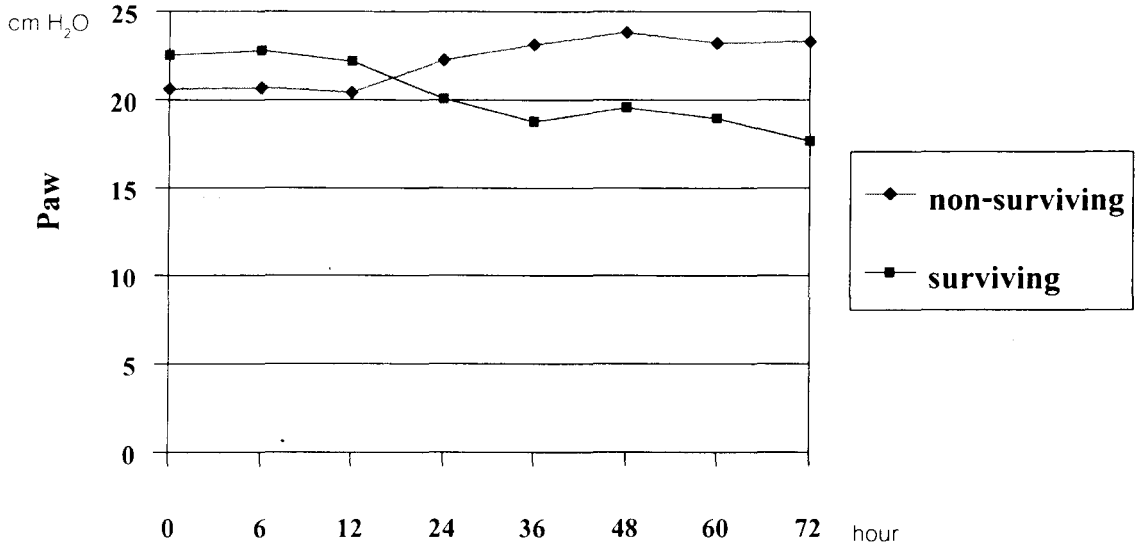

Fig. 1. Shows Paw and duration of HFOV in both groups.

Fig. 1-4 show mean airway pressure (Paw), oxygenation index (OI), alveolar arterial oxygen gradient [$P(A\text{-}a)O_2$] and PaO_2/FiO_2 at initiation of HFOV and then at 6, 12, 24, 36, 48, 60 and 72 hours with comparison of the surviving and non-surviving groups. Fig. 1 compares Paw which was not different initially. As time passed, Paw decreased in the surviving group and was significantly different at 36 hours (p = 0.038). At that time, the average Paw in the surviving group was 18.8 ± 4.7 cm H_2O and 23.1 ± 4.4 cm H_2O in the non-surviving group. Fig. 2 compares OI which initially showed no difference. As time passed, OI decreased in the surviving group and was significantly different at 24 hours (p = 0.012). At that time, the average OI of the surviving group was 15.5 ± 10.9 and 30.2 ± 13.5 in the non-surviving group. Fig. 3 compares $P(A\text{-}a)O_2$ between the two groups. Initially, $P(A\text{-}a)O_2$ of the surviving group was lower than in the non-surviving group and then statistically significantly lower at 6 hours (p = 0.047). At that time, the average $P(A\text{-}a)O_2$ of the surviving group was 448.5 ± 140.8 and 562.7 ± 99.9 in the non-surviving group. Fig. 4 compares PaO_2/FiO_2 between the two groups. Initially PaO_2/FiO_2 of the surviving group was higher

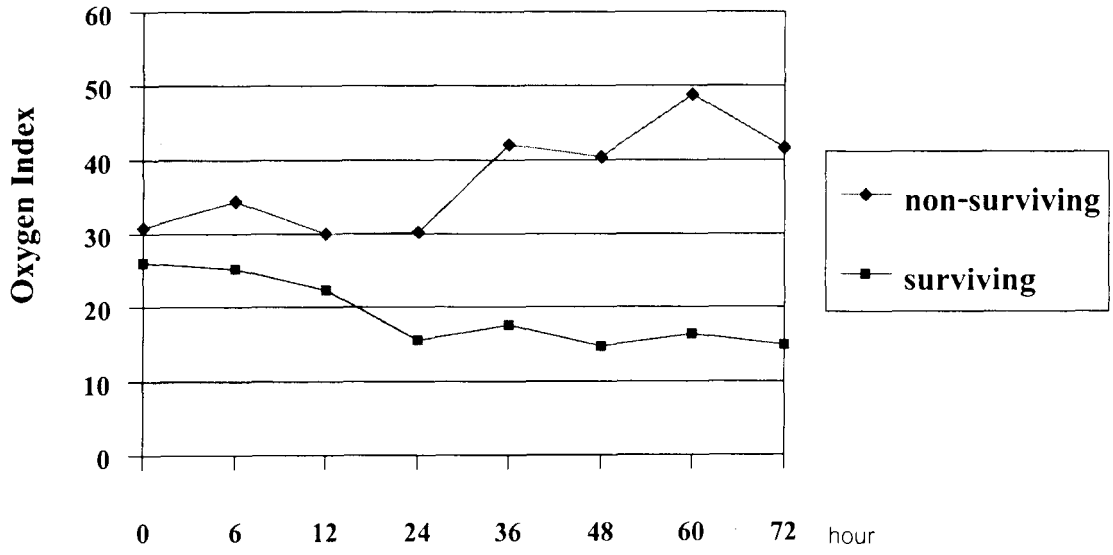

Fig. 2. Shows OI and duration of HFOV in both groups.

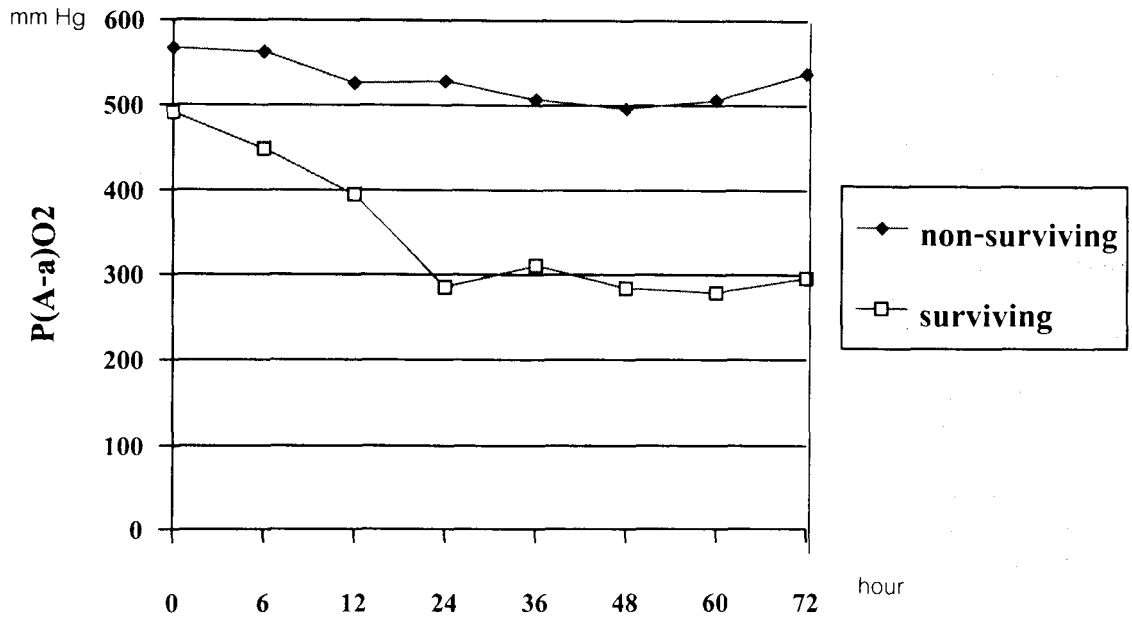

Fig. 3. Shows $P(A\text{-}a)O_2$ and duration of HFOV in both groups.

than in the non-survival group and then statistically significantly higher at 24 hours ($p = 0.023$). At that time, the average PaO_2/FiO_2 of the surviving group was 191.2 ± 127.1 and 86.2 ± 38.8 in the non-surviving group.

DISCUSSION

High frequency oscillatory ventilation has been used in infants since 1989. The HIFI study group (13) reported the results of HFOV in 673 newborn infants with respiratory distress syndrome (RDS). They

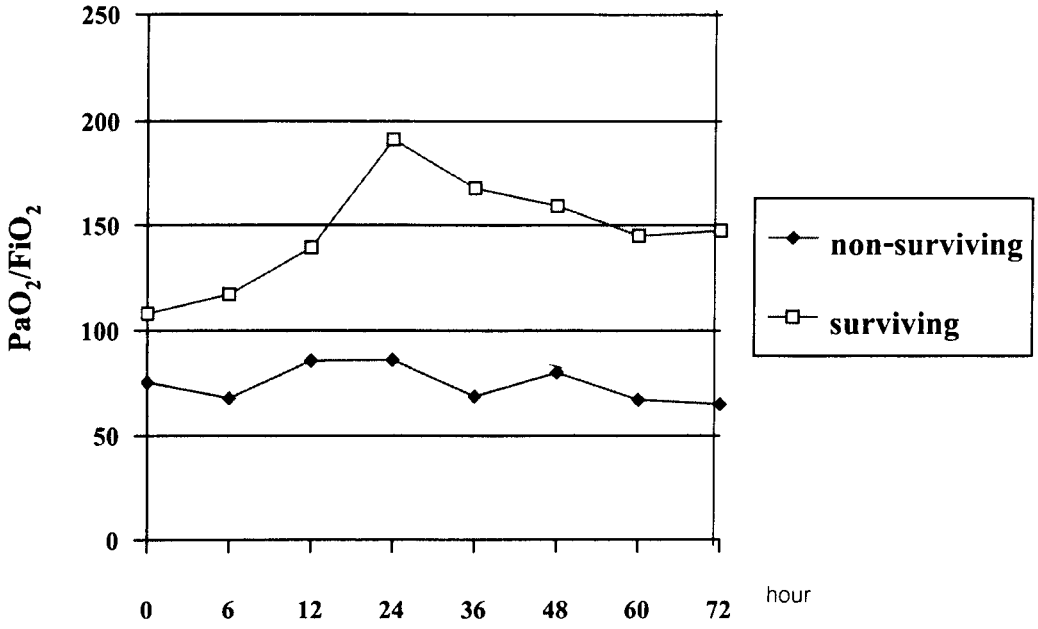

Fig. 4. Shows PaO_2/FiO_2 and duration of HFOV in both groups.

found that HFOV could not reduce the incidence of bronchopulmonary dysplasia (BPD), but increased the incidence of pneumoperitonium, intraventricular hemorrhage and periventricular leukomalacia. In 1993, a multicenter study of HFOV in preterm infants with RDS was conducted in Japan[14]. It found benefits of HFOV in improving oxygenation while not increasing complication of air leak syndrome, intraventricular hemorrage or periventricular leukomalacia as compared to the CV group. Subsequent studies support the benefits of HFOV in decreasing air leak syndrome and BPD in RDS[14]. Re-analysis of the HIFI study found that the level of Paw in the HFOV mode was equal to Paw in the CV mode[14]. In a following study, a high volume strategy was used to recruit the atelectatic alveoli and Paw was higher than the closing pressure[14]. The benefits of the high volume strategy are uniform lung expansion, improved pulmonary mechanics and gas exchange and prevention of lung injury[15,16]. In the present study, the authors also used a high volume strategy by setting the initial Paw level at 4-8 cm HO_2 higher than in the CV mode.

Table 1 shows the demographic data of surviving and non-surviving patients. Sex, age, nutritional status and immunological status were not statistically different in these patients. Arnold et al in 2000 [17], found that an immunocompromised host was a risk factor for death with an ODD ratio of 5.28 (1.52, 18.33). Table 2 compares duration of CV and HFOV between groups. The duration of CV before changing to HFOV was not statistically different in the groups but the duration of HFOV in the non-surviving group (116.6 $\pm$ 59.2 hours) was shorter than in the surviving group (213.5 $\pm$ 160.4 hours). This is because almost all non-surviving patients died on HFOV before being switched back to CV. Of 12 patients that were switched back to CV, eleven patients survived and one patient died from sepsis. CV was used for a long period before changing to HFOV which could cause severe ventilator associated lung injury. In 1994, Arnold et al [11] found that patients treated with HFOV from the beginning had a 6 per cent mortality rate, and the group of patients started with CV and then switched to HFOV had a 42 per cent mortality rate. In 2000, Fedora et al [18], found that patients switched to HFOV within 24 hours had a 58.8 per cent mortality rate, but patients switched to HFOV after 24 hours had a 81.5 per cent mortality rate.

Table 3 compares ventilator parameters and gas exchange variables during CV before changing to HFOV in the surviving and non-surviving groups. Ventilator parameters including FiO_2 and PIP did not

show any statistical difference, but Paw in the non-surviving group (17.6 ± 2.9 cm H_2O) was statistically significantly higher (p = 0.02) than in the surviving group (14.9 ± 1.7 cm H_2O). High Paw meant a high ventilator setting which predisposed to ventilator associated lung injury. Gas exchange variables included PaO_2, $PaCO_2$ and severity of respiratory failure indices, such as PaO_2/FiO_2, $P(A\text{-}a)O_2$ and OI did not show any statistically significant differences. Table 4 is similar to Table 3, but compares variables after changing to HFOV for 4-6 hours. FiO_2 in the surviving group (0.99 ± 0.32) was statistically significantly higher (p = 0.02) than in the non-surviving group (0.84 ± 0.18). FiO_2 may have been decreased in non-surviving group too fast. $P(A\text{-}a)O_2$ in the non-surviving group (562.7 ± 99.9 mm H_2O) was statistically significantly higher (p = 0.047) than in the surviving group (448.5 ± 140.8 mm H_2O), which meant that the non-surviving group may have had a more severe pulmonary pathology.

Fig. 1 shows Paw and duration of HFOV in both groups. In the surviving group, Paw decreased slowly with a statistically significant difference from the non-surviving group at 72 hours. Arnold et al[11, 17] also found that Paw gradually decreased when switched to HFOV with a statistically significant difference at 72 hours. Fig. 2 shows OI and duration of HFOV in both groups. In the surviving group, OI decreased slowly showing a statistically significant difference from the non-surviving group at 24 hours. Arnold et al[10,11,17] also found that OI was statistically significant decreased at 24 hours in surviving patients after they were switched to HFOV. Fig. 3 shows $P(A\text{-}a)O_2$ and duration of HFOV in both groups. $P(A\text{-}a)O_2$ was statistically significantly lower at 6 hours after HFOV initiation in the surviving group as compared to the non-surviving group. In 1993, Rosenberg et al[19] also found a statistically significantly decreased $P(A\text{-}a)O_2$ in the surviving group at 24 hours after being switched to HFOV. Fig. 4 shows PaO_2/FiO_2 and duration of HFOV in both groups. The value of PaO_2/FiO_2 in the non-surviving group was not changed while on HFOV but was statistically significantly increased at 24 hours in the surviving group. In 1996, Sarnaik et al[20] also found that the surviving group had a statistically significantly increased PaO_2/FiO_2 after 6 hours of HFOV. Median PaO_2/FiO_2 increased from a baseline of 63 mmHg to 71 mmHg 6 hours after beginning HFOV.

SUMMARY

Implementation of HFOV is useful in patients with DAD, ARDS and air leak syndrome from the initial phase of illness due to its potential of decreasing ventilator induced lung injury and thus the mortality rate from ARDS. The survival rate in the present study was 52.4 per cent. Factors in the present study including sex, age, nutritional status, immunological status, duration of CV before changing to HFOV, ventilator parameters such as : FiO_2, PIP during CV before changing to HFOV and gas exchange variables included PaO_2, $PaCO_2$, PaO_2/FiO_2, $P(A\text{-}a)O_2$ and OI during CV before changing to HFOV did not show statistically significant differences between the groups. Predisposing survival factors showing statistically significant differences were lower Paw during CV before changing to HFOV, lower Paw at 36 hours, lower OI at 24 hours, lower $P(A\text{-}a)O_2$ at 6 hours and higher PaO_2/FiO_2 at 24 hours. These parameters are good indicators for prognosis of ARDS for patients responding or not responding to HFOV. In non-responsive patients, another management strategy can be employed.

ACKNOWLEDGEMENTS

The authors wish to thank the PICU staff, Dr Surapee Ruangsuwan (Director of Queen Sirikit National Institute of Child Health) and Dr Wiboon Karnjanapattanakul for the statistical analysis.

(Received for publication on June 24, 2003)

REFERENCES

1. Gerstmann DR, Clark RH, deLemos RA. High frequency ventilation: Issues of strategy. Clin Perinatol 1991; 18: 563-80.
2. Clark RH. High frequency ventilation. J Pediatr 1994; 124: 661-70.
3. Parker JC, Hernandez LA, Peevy KJ. Mechanisms of ventilator-induced lung injury. Crit Care Med 1993; 21: 131-43.
4. Hernandez LA, Peevy KJ, Moise RA, Parker JC. Chest wall restriction limits high airway pressure-induced lung injury in young rabbits. J Appl Physiol 1989; 66: 2364-8.
5. Dreyfus D, Saumon G. Role of tidal volume, FRC and end-inspiratory volumes in development of pulmonary edema following mechanical ventilation. Am Rev Respir Dis 1993; 148: 1194-203.
6. Doctor A, Arnold J. Mechanical support of acute lung injury: Options for strategic ventilation. New Horizons 1999; 157: 294-323.
7. Kallas HJ. Non-cenventional respiratory support modalities applicable in the older child. High frequency ventilation and liquid ventilation. Crit Care Clin 1998; 14: 655-83.
8. Slutsky AS. Nonconventional methods of ventilation. Am Rev Respir Dis 1988; 138: 175.
9. Bernard GR, Artigas A, Brigham KL. The American-European consensus conference on ARDS. Am J Respir Crit Care Med 1994; 149: 818-24.
10. Arnold JH, Troug RD, Thompson JE. High-frequency oscillatory ventilation in pediatric respiratory failure. Crit Care Med 1993; 21: 272-8.
11. Arnold JH, Hanson JH, Toto-Figuero LO. Prespective, randomized comparison of high-frequency oscillatory ventilation and conventional ventilation in padiatric respiratory failure. Crit Care Med 1994; 22: 1530-9.
12. Grenier B, Thompson J. High-frequency oscillatory ventilation in pediatric patients. Respir Care Clin N Am 1996; 2: 545-57.
13. HIFI Study Group. High-frequency oscillatory ventilation compared with conventional mechanical ventilation in the treatment of respiralory failure in preterm infants. N Engl J Med 1989; 320: 88-93.
14. Ogawa Y, Miyasaka K. Kawano T. A multicenter randomized trial of high frequency oscillatory ventilation as compared with conventional mechanical ventilation in preterm infants with respiratory failure. Early Hum Dev 1993; 32: 1-10.
15. Hamilton PP, Onayemi A, Smyth JA. Comparison of conventional and high-frequency ventilation: Oxygenation and lung pathology. J Appl Physiol 1983; 55: 131-8.
16. McCulloch PR, Forkert PG, Froese AB. Lung volume maintenance prevents lung injury during high frequency oscillatory ventilation in surfactant-deficient rabbits. An Rev Respir Dis 1988; 137: 1185-92.
17. Arnold JH, Anas NG, Luckett P. High frequency oscillatory ventilation for acute pediatric respiratory failure : A multicenter experience. Crit Care Med 2000; 28: 3913-9.
18. Fedora M, Klimovic M, Seda M. Effect of early intervention of high-frequency oscillatory ventilation on the outcome in pediatric acute respiratory distress syndrome. Bratist Lek Listy 2000; 101: 8-13.
19. Rosenberg RB, Broner CW, Peters KJ. High-frequency ventilation for acute pediatric respiratory failure. Chest 1993; 104: 1216-21.
20. Sarnaik AP, Meert KL, Pappas MD. Predicting outcome in children with severe acute respiratory failure treated with high-frequency ventilation. Crit Care Med 1996; 24: 1396-402.

ปัจจัยที่มีผลต่อการรักษาผู้ป่วยเด็ก ARDS โดยเครื่องช่วยหายใจชนิดความถี่สูง

สรศักดิ์ โล่ห์จินดารัตน์, พบ,*
พนิดา ศรีสันต์, พบ, ประวิทย์ เจตนชัย, พบ**

วัตถุประสงค์ : เพื่อประเมินปัจจัยที่มีผลต่อการรักษาและอัตราการรอดชีวิตในผู้ป่วยเด็กที่ป่วยเป็น diffuse alveolar disease (DAD) เข้าได้กับภาวะ acute respiratory distress syndrome (ARDS) โดยเครื่องช่วยหายใจชนิดความถี่สูง (high-frequency oscillatory ventilation, HFOV)

วิธีการศึกษา : รูปแบบงานวิจัยเป็นแบบ cohort study ของผู้ป่วยเด็กในตึกผู้ป่วยภาวะวิกฤติ ของสถาบันสุขภาพเด็กแห่งชาติมหาราชินี ระยะเวลาการศึกษาตั้งแต่ 1 มกราคม 2542 ถึง 31 ธันวาคม 2544 ผู้ป่วยเด็กที่มีการหายใจล้มเหลวจาก DAD ที่เข้าได้กับภาวะ ARDS โดยมีค่า PaO_2/FiO_2 < 200 และ oxygenation index (OI) > 10 เครื่องช่วยหายใจชนิดที่ใช้คือ high-frequency oscillatory ventilator (3100A Sensor Medics Corp, Yorba Linda, Calif) โดยใช้หลักการของ high volume strategy เมื่อผู้ป่วยมีอาการดีขึ้นจะเปลี่ยนกลับมาใช้เครื่องช่วยหายใจชนิดธรรมดา ข้อมูลทั่วไปของผู้ป่วย, ระยะเวลาที่ใช้เครื่องช่วยหายใจชนิดธรรมดาก่อนเปลี่ยนมาใช้ HFOV, ระยะเวลาที่ใช้ HFOV, ข้อมูล ventilator parameters และ gas exchange variables ตั้งแต่เริ่มใช้ HFOV จะได้รับการบันทึกไว้และนำมาเปรียบเทียบระหว่างกลุ่มที่รอดชีวิตและกลุ่มที่เสียชีวิต

ผลการศึกษา : มีผู้ป่วยที่เข้าร่วมการศึกษาในช่วงระยะเวลา 3 ปี จำนวน 21 ราย ในจำนวนนี้มี 4 ราย ที่มีปัญหา air leak syndrome ร่วมด้วย เป็นเพศชาย 10 คน อายุเฉลี่ย 3.58 $\pm$ 3.9 ปี ผู้ป่วยที่รอดชีวิตจำนวน 11 คน (52.4%) ข้อมูลทางด้าน ventilator parameters และ gas exchange variables หลังจากเปลี่ยนมาใช้ HFOV ได้นาน 4-6 ชั่วโมง ในทั้ง 2 กลุ่ม พบว่าในกลุ่มที่รอดชีวิตมีค่า FiO_2 ที่สูงกว่า (0.99 $\pm$ 0.32 *vs* 0.84 $\pm$ 0.18; p = 0.02) และค่า alveolar arterial oxygenation gradient [$P(A-a)O_2$] ที่ต่ำกว่า (448.5+140.8 *vs* 562.7+99.9 มม ปรอท; p = 0.047) เมื่อเปรียบเทียบกับกลุ่มที่เสียชีวิต เมื่อเปรียบเทียบค่า mean airway pressure (Paw), OI, $P(A-a)O_2$ และ PaO_2/FiO_2 ในระหว่างที่ใช้ HFOV ในทั้ง 2 กลุ่ม พบว่าค่า Paw และ OI ลดลงในกลุ่มที่รอดชีวิตอย่างมีนัยสำคัญทางสถิติที่ 36 และ 24 ชั่วโมงตามลำดับ สำหรับค่า PaO_2/FiO_2 ในกลุ่มที่รอดชีวิตต่ำกว่าอย่างมีนัยสำคัญทางสถิติที่ 6 ชั่วโมง และค่า PaO_2/FiO_2 ในกลุ่มที่รอดชีวิตสูงกว่าอย่างมีนัยสำคัญทางสถิติที่ 24 ชั่วโมง

สรุป : การใช้เครื่องช่วยหายใจชนิดความถี่สูง (HFOV) ในผู้ป่วยเด็กที่มีการหายใจล้มเหลวจาก DAD, ARDS และ air leak syndrome ตั้งแต่ในระยะแรกของโรคเมื่อมีข้อบ่งชี้ จะสามารถช่วยลดภาวะ ventilator induced lung injury และลดอัตราการเสียชีวิต ปัจจัยที่มีผลต่อการรอดชีวิตอย่างมีนัยสำคัญทางสถิติ คือค่าของ Paw ที่ต่ำกว่าในช่วงการใช้เครื่องช่วยหายใจชนิดธรรมดา, ค่า Paw ต่ำกว่าที่ 36 ชั่วโมง, ค่า OI ที่ต่ำกว่าที่ 24 ชั่วโมง, ค่า $P(A-a)O_2$ ที่ต่ำกว่าที่ 6 ชั่วโมง และค่า PaO_2/FiO_2 ที่สูงกว่าที่ 24 ชั่วโมงหลังเปลี่ยนมาใช้ HFOV ค่าต่าง ๆ เหล่านี้เป็นตัวชี้วัดที่ดีถึงพยากรณ์ของผู้ป่วย ARDS ที่ตอบสนองต่อ HFOV

คำสำคัญ : acute respiratory distress syndrome, เครื่องช่วยหายใจชนิดความถี่สูง

สรศักดิ์ โล่ห์จินดารัตน์, พนิดา ศรีสันต์, ประวิทย์ เจตนชัย
จดหมายเหตุทางแพทย์ ฯ 2546; 86 (ฉบับพิเศษ 3): S618-S627

* หน่วยระบบหายใจ, กลุ่มงานกุมารเวชกรรม, สถาบันสุขภาพเด็กแห่งชาติมหาราชินี, กรุงเทพ ฯ 10400