

Pneumomediastinum due to High-frequency Jet Ventilation in a Near-drowned Infant

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High-frequency jet ventilation was initially developed by Klain et al.¹ and has attracted considerable recent interest as a promising new alternative method of mechanical ventilation. However, several authors have suggested that oxygenation by high-frequency ventilation depends on the mean airway pressure level in the same way as does conventional ventilation in the involved lung^{2,3}. Furthermore, Kolton et al. demonstrated that in rabbits with surfactant removed by saline lavage, the optimal ventilation with high frequency oscillation is necessarily superimposed on the periodic lung inflations, causing an increase in mean airway pressures and hightend risk of barotrauma⁴. However, the combined use of high frequency ventilation (HFV) with intermittent positive pressure ventilation (IPPV) was reported to clinically improve oxygenation in acute lung disease with a lower mean airway pressure than that of conventional ventilation⁵. We thus applied high-frequency jet ventilation (HFJV) with IPPV to a near-drowned infant, who had been submerged in sewage in a lavatory tank, and did not

repond to conventional ventilation with PEEP. Three hours after the start of HFJV with IPPV, this respiratory mode caused a critical pneumomediastinum in spite of the lowered airway pressure. In the present report, the mechanism of this pneumomediastinum is discussed.

Case Report

A 15-month-old girl accidentally fell into a lavatory tank and was submerged for approximately 5 min. When rescued by emergency medical technicians, physical examination revealed the cessation of both spontaneous respiration and heart beat. She was transported to a hospital near her house under the resuscitation, where heart beat, spontaneous respiration and light reflex of the pupils was restored after positive pressure ventilation with oxygen and intracardiac administration of epinephrine. Heart beat was restored about 18 min after the accident. Five hours after the accident, she was transferred to our intensive care unit for the management of acute respiratory failure and CNS-oriented therapy.

On admission, she was in a deep coma and showed a generalized cyanosis, hyperthermia (39.2°C rectal temperature), tachycardia (230 bpm, without arrhythmias), tachypnea (60/min, effortive) and hypertension (120/80 mmHg). Moist rales were audible through-

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out both lungs. Bloody sputums smelling of feces drained from the nasotracheal tube. A chest X-ray obtained on admission demonstrated a marked infiltration in both lung fields (fig. 1). We instituted conventional ventilation using the Bourns-BP 200 (California, USA), a pressure-preset type of ventilator with ventilator setting of 40 breaths/min, total gas flow 9 L/min, peak inspiratory pressure 40 cmH₂O, and inspired oxygen fraction (F_IO₂) 1.0 with positive end-expiratory pressure (PEEP) of 10 cmH₂O. This respiratory mode, however, did not improve the arterial oxygenation. An arterial blood gas analysis (ABG) revealed extremely large alveolar-arterial differences of oxygen tension and a severe metabolic acidosis; F_IO₂ 1.0, pH 7.224, PaCO₂ 32.1 mmHg, PaO₂ 63.2 mmHg, hemoglobin saturation 86.6%, and base excess -13.7 mEq/L. In spite of continuous positive pressure ventilation (CPPV) at F_IO₂ 1.0 and a frequent tracheal lavage, severe hypoxia did not improve even 1 hour after the institution of CPPV. We therefore decided to apply a high-frequency-jet ventilation (HFJV) to improve oxygenation. HFJV, using a HFOJ ventilator (Mera Corp. Tokyo, JAPAN) at a frequency of 5 Hz, a driving pressure of 0.8 kg/cm² and an F_IO₂ of 0.8, was superimposed on IPPV via a jet injector at the oral end of endotracheal tube. Total gas flow of the pressure-preset ventilator was reduced from 9 to 4 L/min in order to prevent acute hyperinflation with attendant risk of barotrauma. Immediately after the employment of HFJV with IPPV, the ABG showed a dramatic improvement; F_IO₂ 0.8, pH 7.543, PaCO₂ 23.2 mmHg, PaO₂ 436.8 mmHg, hemoglobin saturation 99.8% and base excess -0.4 mEq/L. This respiratory mode, however, caused circulatory depressions. To counteract the hemodynamic deterioration, intravascular volume loading using colloid solution and the administration of an inotropic agent (dopamine 3 μg/kg/min) was performed. The ventilatory settings were also changed; the peak inspiratory pressure from 40 to 30 cmH₂O, total flow from 4 to 3 L/min, the driving pressure of jet ventilator from 0.8 to 0.6 kg/cm² and

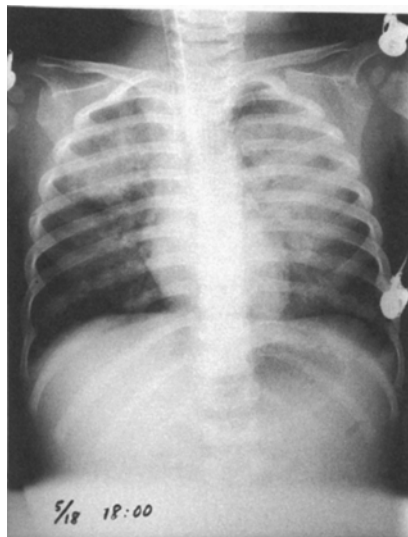


Fig. 1. A chest X-ray taken on admission shows a marked infiltration in both lung fields.

an F_IO₂ from 0.8 to 0.4 (table 1). Thereafter, hemodynamic parameters improved and urinary output also increased. The ABG (F_IO₂ 0.4) on the changed respiratory mode was pH 7.546, PaCO₂ 24.0 mmHg, PaO₂ 232.2 mmHg, hemoglobin saturation 99.7% and base excess -0.4 mEq/L. Three hours after the start of HFJV with IPPV, despite a reduction in airway pressure, circulatory depression abruptly reappeared. A chest X-ray obtained on this event revealed hyperinflation of both lungs and a marked pneumomediastinum (fig. 2). Asthmatic wheezing was not detected and ABG did not show any aggravation; the ABG (F_IO₂ 0.4) was pH 7.541, PaCO₂ 21.0 mmHg, PaO₂ 200.1 mmHg and base excess -4.3 mEq/L. Thus, we ruled out an asthmatic attack as the cause of this pneumomediastinum. In order to treat the pneumomediastinum, several respiratory settings were tried and an appropriate setting was found as follows; HFJV at a frequency of 5 Hz, a driving pressure of 0.5 kg/cm², an F_IO₂ of 0.45 and IPPV at a peak inspiratory pressure of 22 cmH₂O, at a rate of 20 breaths/min, total gas flow 0L/min. This combined respiratory mode provided a "staircase" profile of inspiration during the closure of exhalation valve in the Bourns BP-

Table 1. Time course of arterial blood gas analysis on 1st hospital day

Respiratory Mode	IPPV with PEEP (on admission)	HFJV with IPPV (1 hr after admission)	HFJV with IPPV (PN developed)	HFJV with IPPV (PN disappeared)
pH	7.224	7.543	7.541	7.380
PaCO ₂ (mmHg)	32.1	23.2	21.0	36.5
PaO ₂ (mmHg)	63.2	436.8	200.1	102.3
B.E. (mEq/L)	-13.7	-0.4	-4.3	-3.0
PIP (cmH ₂ O)	40	40	30	20
R.R. (breaths/min)	40	40	30	20
T.F. (l/min)	9	4	3	0
PEEP (cmH ₂ O)	10	0	0	0
D.P. (kg/cm ²)		0.8	0.6	0.5
Frequency (Hz)		5	5	5
FI _{O₂}	1.0	0.8	0.4	0.45

PN; Pneumomediastinum, PIP; setting level of peak inspiratory pressure of Bourns BP-200, R.R; respiratory rate of Bourns BP-200, T.F; total flow of Bourns BP-200, D.P; driving pressure of HFOJ ventilator.

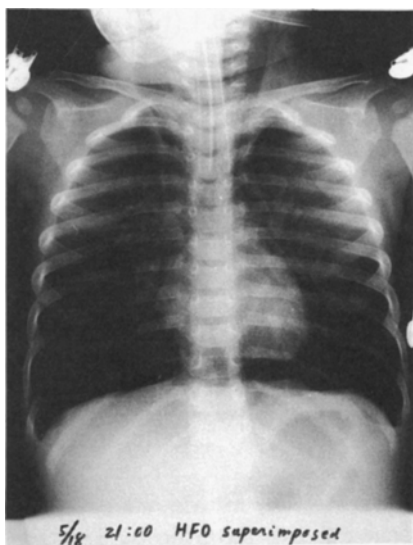


Fig. 2. A chest X-ray obtained 3 hours after the institution of HFJV with IPPV demonstrates bilateral lung hyperinflation and a pneumomediastinum.

200 ventilator. Heart beat gradually dropped (from 180 to 120 bpm), mean blood pressure increased (from 50 to 90 mmHg). The ABG on this setting was pH 7.380, PaCO₂ 36.5 mmHg, PaO₂ 102.3 mmHg, hemoglobin saturation 97.7% and base excess -3.0 mEq/L.

She was extubated on the 17th day of hospitalization and recovered her smile on the 27th day. Seven months after the accident, although there remained involuntary movements, she was generally cheerful and leading a full and meaningful life appropriate for a child of her age.

Discussion

Rouby et al.⁷ indicated that an HFJV-induced "PEEP effect" is greater in patients with a normal or elevated time constant of the total respiratory system than in patients with a low time constant⁶. Therefore, it is reasonable that in patients with high airway resistance or high lung compliance, the risk of barotrauma may increase during HFJV. Simon et al. demonstrated also that during high frequency ventilation, the underestimation of the difference between mean airway pressure and mean alveolar pressure results from differences between inspiratory and expiratory impedances, depending on airway geometry, compliance, and lung volume⁷. It is probable that the airway resistance of our patient was high due to aspirated substances, presumably hyperosmotic, from the lavatory tank. Thus, we cannot rule out the possibility that pneumomediastinum

occurred in this patient is partly due to a high airway resistance. However, there was evidence that pneumomediastinum did not occur immediately after the start of HFJV with IPPV. Furthermore, the auscultation during the pneumomediastinum did not indicate any expiratory prolongation and the ABG did not show any retension of arterial CO₂ level (PaCO₂, 21.0 mmHg). Thus, we believe it is unlikely that high airway resistance caused this pneumomediastinum during HFJV with IPPV. It is conceivable that lung compliance when pneumomediastinum occurred may have improved: arterial oxygenation did not deteriorate even though the peak inspiratory pressure was reduced from 40 to 30 cmH₂O, and the driving pressure of the jet ventilator from 0.8 to 0.6 kg/cm². Thus, we speculate that pneumomediastinum occurred in this patient may be mainly due to improved lung compliance, impairment to which was improved by eliminating aspirated substances through HFJV with IPPV. Therefore, it is necessary to give attention to the potential development of barotrauma especially when HFJV with IPPV is employed in an infant with initially low lung compliance which later improves.

We have reported the clinical course of an infant with severe hypoxia and deep coma after nearly drowning in a lavatory tank. Although we applied HFJV with IPPV while controlling hemodynamic parameters safely for 3 hours, this ventilatory mode elicited a marked abrupt pneumomediastinum in spite of a reduction in airway pressure. Therefore, we urge physicians to beware of the onset of barotrauma especially when HFJV with IPPV is applied to an infant with low lung compliance that later improves.

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