



Efficacy and safety of intrapulmonary percussive ventilation superimposed on conventional ventilation in obese patients with compression atelectasis

Ryosuke Tsuruta MD*, Shunji Kasaoka MD,
Kiyoshi Okabayashi MD, Tsuyoshi Maekawa MD

Advanced Medical Emergency and Critical Care Center, Yamaguchi University Hospital, Ube, Yamaguchi 755-8505, Japan

Received 8 July 2005; revised 16 December 2005; accepted 28 March 2006

Keywords:

Intrapulmonary percussive ventilation;
Compression atelectasis;
Conventional ventilation

Abstract

Purpose: To investigate the efficacy and safety of intrapulmonary percussive ventilation (IPV) in obese patients, we assessed their respiratory and hemodynamic functions during IPV superimposed on conventional ventilation.

Materials and Methods: Ten obese patients with acute respiratory failure due to compression atelectasis who had not improved by conventional ventilation were treated with IPV. Hemodynamic parameters, ventilator settings, and intracranial pressure ($n = 1$) were recorded every hour. Arterial blood gas was analyzed every 3 hours. The efficacy and safety of IPV was assessed at the start of weaning.

Results: Before IPV, $\text{PaO}_2/\text{FIO}_2$ ratio remained low (189 ± 63 mm Hg), which significantly increased to 243 ± 67 mm Hg at 3 hours from the initiation of IPV ($P < .01$). Furthermore, it continuously increased to 280 ± 50 mm Hg at 24 hours ($P < .01$). Intrapulmonary percussive ventilation induced significant increase in dynamic compliance from control value of 30 ± 8 mL/cm H_2O at 0 hours to 35 ± 9 mL/cm H_2O at 12 hours ($P < .05$) and to 38 ± 8 mL/cm H_2O at 24 hours ($P < .01$). Heart rate and mean arterial pressure were not significantly changed during IPV. Improvement of compression atelectasis was confirmed by their chest computed tomographic scans. Adverse effects such as pneumothorax and intracranial hypertension were not seen.

Conclusions: These results demonstrated that IPV was effective and safe in improving compression atelectasis without adverse effects in obese patients.

© 2006 Elsevier Inc. All rights reserved.

1. Introduction

Compression atelectasis, which was consistently found by computed tomographic (CT) scans in normal anesthetized men, was described by Brismar et al [1]. They

demonstrated the high-density regions of lung CT in gravity-dependent lung. This finding has been reported in acute respiratory failure or acute respiratory distress syndrome (ARDS) [2,3] and the heterogeneity shown by CT scans in ARDS has been well known. They are often sedated and/or paralyzed, immobilized, or obese. Prone positioning has been reported to be effective in improving oxygenation in acute respiratory failure [4,5]. Likewise,

* Corresponding author. Tel.: +81 836 22 2656; fax: +81 836 22 2344.
E-mail address: ryosan-ygc@umin.ac.jp (R. Tsuruta).

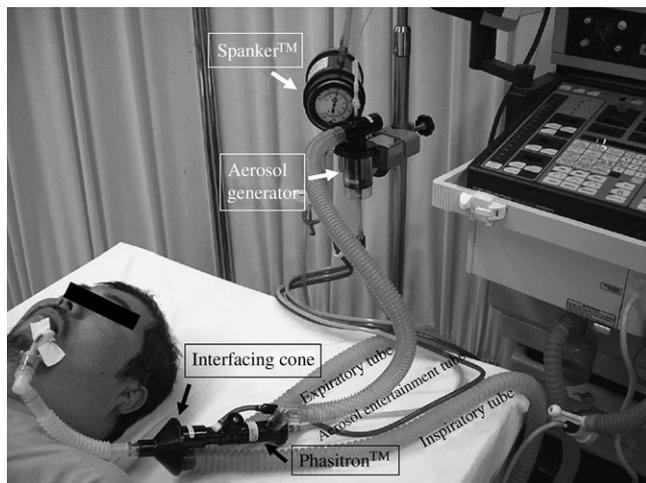


Fig. 1 Composition of an intrapulmonary percussive ventilator. Spanker has the dial to control the frequency rate, the monitor of airway pressure, and the monitor of driving pressure. Sterile water is dripping into the aerosol generator. Phasitron has a sliding Venturi body, which moves back and forth by inspiration jet stream from the orifice, serving as an opening/closing valve.

intrapulmonary percussive ventilation (IPV) has been reported to improve oxygenation in a small study of ARDS [6,7]. The aim of this study is to evaluate the efficacy and safety of superimposed IPV on conventional ventilation in adult obese patients with compression atelectasis in whom prone positioning is not easy to achieve.

2. Methods

2.1. Subjects

We used a prospective trial of IPV in adult obese patients with compression atelectasis who failed to be treated by conventional ventilation. Between May 2002 and March

2003, 10 obese patients with acute respiratory failure, who were unresponsive to conventional ventilation for at least 12 hours, were superimposed with IPV (Spanker, Percussionaire; Bird Space Technologies, Sand Point, Idaho). The conventional ventilation here corresponded to synchronized intermittent mandatory ventilation (SIMV) and more than 5 cm H₂O of positive end-expiratory pressure (PEEP). The Fig. 1 demonstrated an endotracheal tube, an interfacing cone, inspiratory/expiratory tubes to ventilator, an aerosol generator, and Phasitron (Percussionaire; Bird Space Technologies, Sand Point, Idaho), which has a sliding Venturi body moving back and forth by inspiration jet stream from the orifice, serving as an opening/closing valve.

The obesity was defined as body mass index of more than 25 by the Japan Society for the Study of Obesity, and the acute respiratory failure was due to compression atelectasis confirmed by chest CT scans and/or chest portable radiographs [8]. The infiltrations induced by infection and drugs were excluded by the clinical courses, the laboratory data, and the comments of the radiologists. The characteristics of subjects are given in Table 1. Prone positioning was not performed for all patients because of their obesity. Two of them received abdominal aortic repair, and 1 patient received craniectomy. The study protocol was approved by the institutional review board of our university hospital. Informed consent from the family was obtained for all patients.

2.2. Mechanical ventilation

All patients were treated with conventional ventilators (7200; Puritan-Bennett, Overland, Kan, and Evita or Dräger Werk AG, Lübeck, Germany). The mode of mechanical ventilation was pressure-controlled SIMV or pressure-limited SIMV in the case of Evita. They were sedated with a continuous infusion of midazolam and butorphanol. When the patient's improvement in oxygenation was absent by more than 5 cm H₂O of PEEP and prone positioning was

Table 1 Characteristics of the patients

No.	Age (y)	Sex	Diagnosis	APACHE II	BMI (kg/m ²)	ICU (d) ^a	IPV (d)	Ventilator	PaO ₂ /FIO ₂ (mm Hg)	PEEP (cm H ₂ O)	PIP (cm H ₂ O)
1	66	F	Rupture of AAA	15	25	22 (8)	6	Bennett	173	12	30
2	51	M	Multiple trauma	26	28	43 (8)	2	Evita	275	6	25
3	71	F	SAH	30	30	18 (5)	4	Bennett	184	5	17
4	66	M	Impalement injury	9	26	13 (4)	3	Evita	237	7	23
5	28	M	Diabetic coma	30	41	17 (7)	4	Bennett	256	5	22
6	27	M	Drug overdose	20	27	37 (6)	6	Evita	108	7	28
7	50	M	Saddle embolism	31	27	66 (13)	4	Bennett	227	7	22
8	75	M	Sepsis	12	29	21 (4)	8	Evita	120	7	30
9	24	M	Drug overdose	17	37	3 (1)	2	Bennett	210	10	25
10	61	M	Rupture of AAA	18	37	21 (3)	9	Bennett	99	12	27
Mean	52			21	31	26 (6)	5		189	8	25
SD	19			8	6	18 (3)	2		63	3	4

PaO₂/FIO₂, PEEP and PIP were collected at the initiation of IPV. APACHE indicates Acute Physiologic and Chronic Health Evaluation; BMI, body mass index; PaO₂/FIO₂, PaO₂/FIO₂ before IPV; AAA; abdominal aortic aneurysm, SAH; subarachnoid hemorrhage.

^a Showed ICU days before IPV.

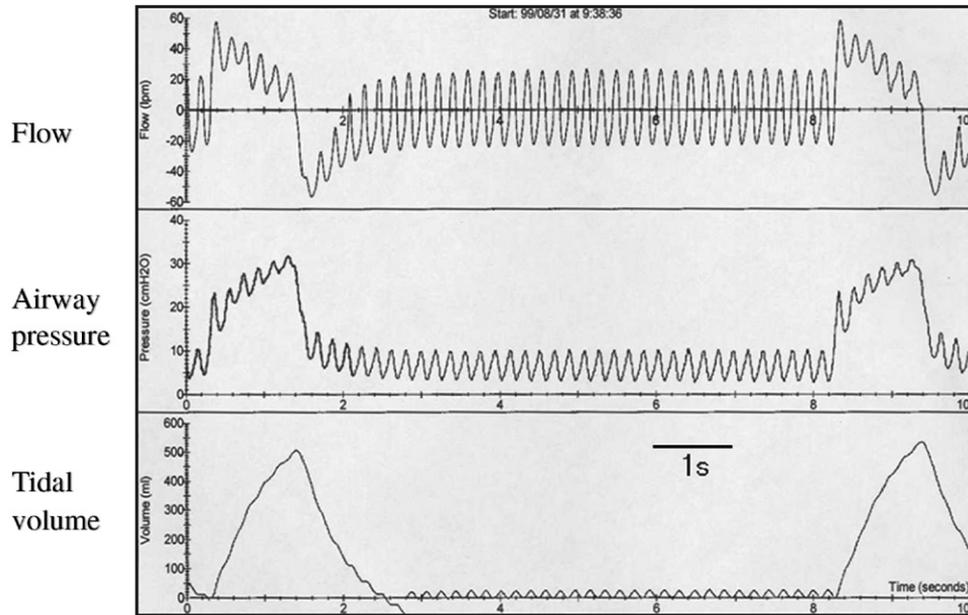


Fig. 2 Ventilatory mechanical curves produced by an intrapulmonary percussive ventilator superimposed on a conventional ventilator. The flow sensor was located in the ventilator circuit. The waveforms were recorded by Ven Trak 1550 (Novamatrix Medical Systems Inc, Wallingford, Conn). Flow waveform is symmetrical across the baseline, which means the input volume is equal to output volume. There are 5 waves in 1 second (therefore 300 beats per minute). The third waveform shows tidal volume.

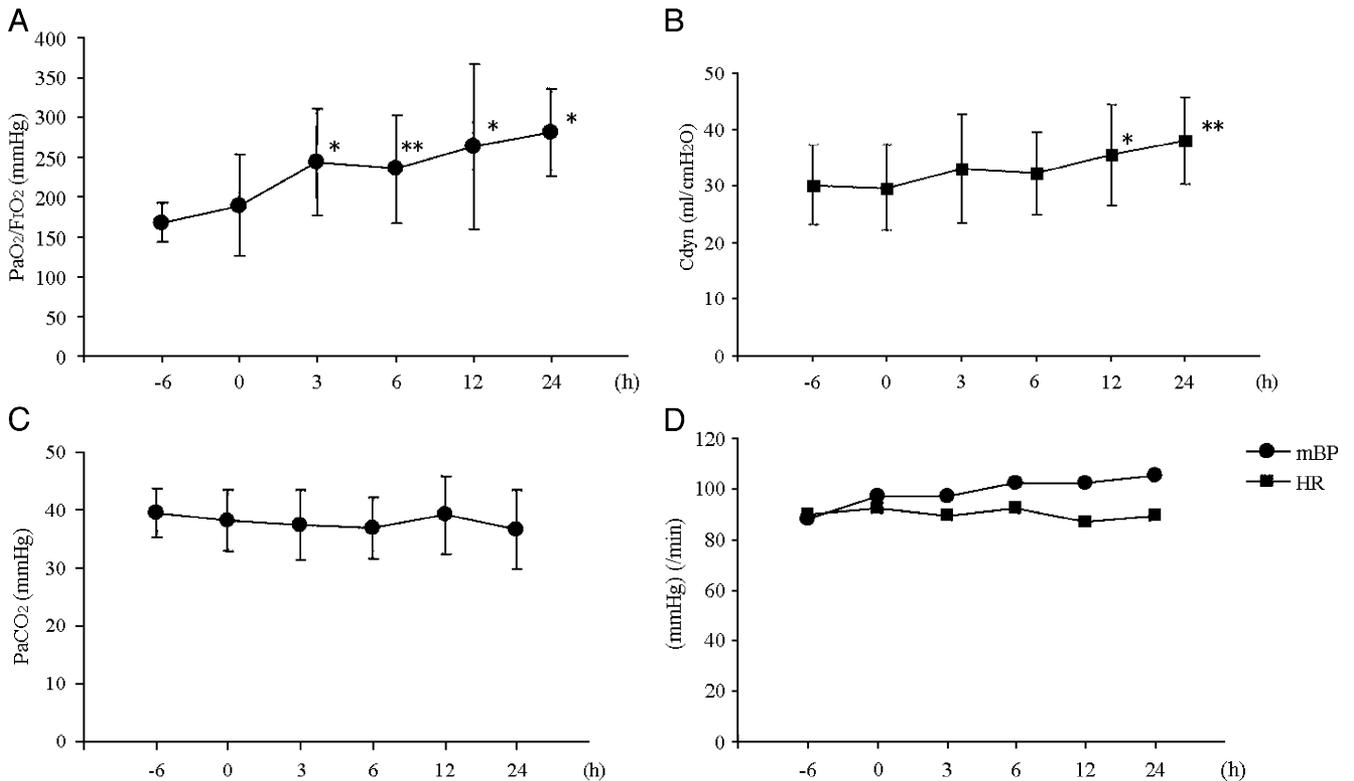


Fig. 3 Effects of IPV on respiratory and hemodynamic parameters. A, Changes in the PaO₂/F_iO₂ before and during IPV. Before IPV, the PaO₂/F_iO₂ ratio remained low, but it significantly increased from 3 to 24 hours from the initiation of IPV (**P* < .01 vs 0 hours, ***P* < .05 vs 0 hours). B, Changes in the dynamic compliance before and during IPV. Intrapulmonary percussive ventilation induced significant increases in dynamic compliance at 12 (**P* < .05 vs 0 hours) and at 24 hours (***P* < .01 vs 0 hours). C, Changes in the PaCO₂ before and during IPV. PaCO₂ did not change significantly. D, Hemodynamic changes before and during IPV. Heart rate (closed square) and mean arterial blood pressure (closed circle) did not change during IPV.

impossible because of abdominal aortic repair, craniectomy, or obesity, IPV was superimposed on the same conventional ventilator. The initial setting of the ventilators were shown in Table 1, and the levels of PEEP and peak inspiratory pressure (PIP) did not change during IPV. The setting of IPV was a high-frequency rate of 300 beats per minute (Fig. 2), which was controlled by the dial turned to “EASY” (the highest frequency). The driving pressure was 138 kPa (20 psi), and oxygen was initially used as driving gas. Therefore, inhaled oxygen was both driving gas and adjusted oxygen through the ventilator. When oxygenation in the patient improved, oxygen was converted to air. Inhaled oxygen percentage was measured by the oxy-monitor (A-100; Yamato Sanki, Osaka, Japan). The IPV was terminated at the start of weaning.

2.3. Data collection

Hemodynamic parameters (heart rate and mean arterial pressure), ventilator settings (tidal volume, PIP, PEEP, respiratory rate), and intracranial pressure ($n = 1$) were recorded every hour. While the ventilator settings were checked, IPV was interrupted. Arterial blood gases (PaO_2 , PaCO_2 , and pH) were analyzed every 3 hours. At 6 hours before and 0, 3, 6, 12, and 24 hours after the start of IPV, $\text{PaO}_2/\text{FIO}_2$ ratio and dynamic compliance [tidal volume/(PIP – PEEP)] were calculated. Chest anteroposterior portable radiographs were obtained everyday, and chest CT scans were obtained as needed by the physicians.

2.4. Statistics analysis

Data are represented as mean \pm SD. A 1-way analysis of variance for repeated measures was used, with significance between time points determined by Dunnett test. Significance was accepted at $P < .05$.

3. Results

3.1. Effects on oxygenation and ventilation

$\text{PaO}_2/\text{FIO}_2$ ratio was demonstrated at –6, 0, 3, 6, 12, and 24 hours after the start of IPV (Fig. 3A). Before IPV, $\text{PaO}_2/\text{FIO}_2$ ratio remained low (189 ± 63 mm Hg). It significantly increased to 243 ± 67 mm Hg at 3 hours from the initiation of IPV ($P < .01$). Furthermore, it continuously increased to 280 ± 55 mm Hg at 24 hours ($P < .01$). Fig. 3B demonstrates the changes in the dynamic compliance. IPV induced significant increases in the dynamic compliance from 30 ± 8 mL/cm H_2O at the beginning to 35 ± 9 ($P < .05$) and 38 ± 8 mL/cm H_2O ($P < .01$) at 12 and 24 hours after the treatment, respectively. However, PaCO_2 did not significantly change (Fig. 3C).

3.2. No hemodynamic changes

Heart rate and mean arterial pressure did not significantly change with IPV (Fig. 3D).

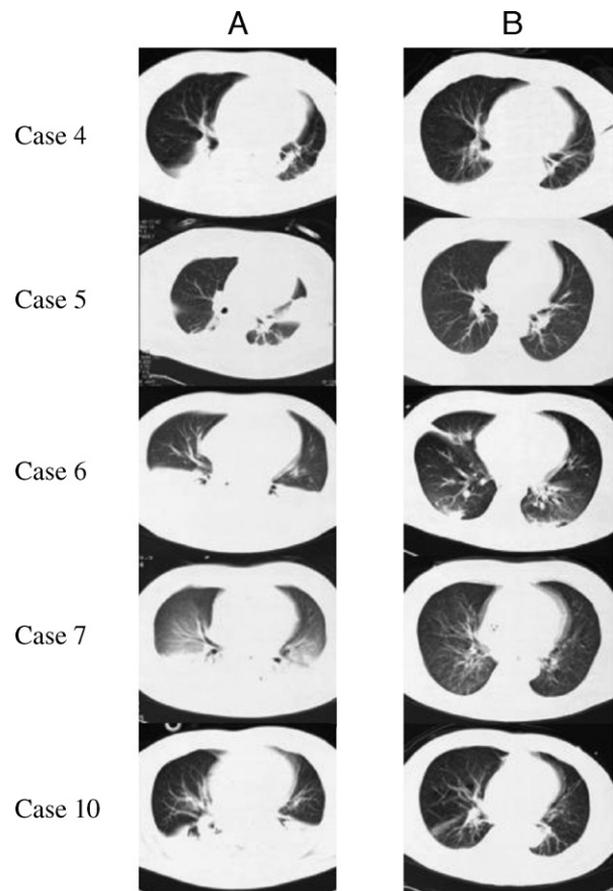


Fig. 4 Chest CT scans before (A) and after (B) IPV. The chest CT scans in 5 cases demonstrated compression atelectasis (A) and absorption of dorsal atelectasis by IPV (B).

3.3. Improvement of compression atelectasis

Improvement of compression atelectasis was recognized by chest CT scans in 7 patients and by chest portable radiographs in 10 patients. Absorption of dorsal atelectasis was demonstrated by chest CT scans in 5 cases (Fig. 4).

3.4. No adverse events

Adverse effects such as pneumothorax and intracranial hypertension were not seen during IPV. The intracranial pressure in case 3 showed 11, 11, 8, 9, and 13 mm Hg at –6, 0, 3, 6, 12, and 24 hours. All patients were successfully weaned from the mechanical ventilators and survived.

4. Discussion

Prone position ventilation is a safe and efficient technique to improve oxygenation [4,5]. This is explained by recruiting the collapsed dependent dorsal pulmonary regions, thereby improving ventilation-perfusion relationships and decreasing venous admixture. This maneuver is relatively simple and useful to improve oxygenation, but prone positioning cannot be practically performed in

patients with abdominal aortic repair, craniectomy or severe obesity. Therefore, we hypothesize that IPV might be more suitable to such patients instead of prone positioning.

This study shows that IPV is effective in improving oxygenation ($\text{PaO}_2/\text{FIO}_2$ ratio) and dynamic compliance in obese patients with acute respiratory failure due to compression atelectasis. Although IPV has been reported to improve oxygenation in ARDS [6,7] and in smoke inhalation [9], patients were switched from the conventional ventilators to IPV. In our study, the efficacy of IPV superimposed on the conventional ventilation was demonstrated in obese patients. Because the improvement of compression atelectasis is recognized by chest CT scans and/or portable radiographs, the mechanisms are considered as a lung opening effect and a better alveolar recruitment by IPV.

Previous animal data convincingly supports the concept of *reduced lung injury using IPV* [10]. In their study, bronchoalveolar lavage (BAL) elastase was significantly elevated in conventionally ventilated baboons on day 1. In contrast, BAL elastase was never elevated in the IPV group. Because the elevation of elastase is considered as neutrophil activation and tissue injury, this indicates that IPV reduces lung injury. However, BAL was not performed in our study, and the mechanism of IPV from the aspect of chemical mediators was unknown.

The patients were monitored before and during superimposition of IPV. No significant compromise in heart rate or mean arterial blood pressure was observed. Furthermore, intracranial pressure in a patient was not elevated. Adverse effects such as pneumothorax were not seen during IPV in any patients. All patients were successfully weaned from the mechanical ventilators and survived.

Intrapulmonary percussive ventilation initiation was determined by intensive care unit [ICU] physicians when patients were unresponsive to conventional ventilation (SIMV and $\text{PEEP} \geq 5$ cm H_2O) for at least 12 hours. The definition of conventional ventilation depends on the empirical mode of ventilators and does not include recruiting maneuvers and high PEEP (>15 cm H_2O). This resulted in several PEEP levels from 5 to 12 (8 ± 3) cm H_2O among the patients. For example, in case 3, the PEEP level was 5 cm H_2O because high PEEP elevated intracranial pressure. Recently, the clinical study of IPV switched from conventional ventilation in head-injured patients with ARDS has been reported [11]. Intrapulmonary percussive ventilation produced a significant improvement in oxygenation with a concomitant reduction in intracranial pressure. Our results might show that superimposition of

IPV on the conventional ventilation has a similar effect as the increase of PEEP.

In the present study, there was no control group treated by conventional ventilation, but the additional effect of IPV on conventional ventilators could be demonstrated.

5. Conclusion

We have demonstrated that IPV is effective and safe in adult obese patients with acute respiratory failure due to compression atelectasis.

Acknowledgments

The authors thank Mr Norimichi Matsuyama for his technical assistance.

References

- [1] Brismar B, Hedenstierna G, Lundquist H, et al. Pulmonary densities during anesthesia with muscular relaxation. A proposal of atelectasis. *Anesthesiology* 1985;62:422-8.
- [2] Gattinoni L, Pelosi P, Vitale G, et al. Body position changes redistribute lung computed-tomographic density in patients with acute respiratory failure. *Anesthesiology* 1991;74:15-23.
- [3] Pelosi P, D'andrea L, Vitale G, et al. Vertical gradient of regional lung inflation in adult respiratory distress syndrome. *Am J Respir Crit Care Med* 1994;149:8-13.
- [4] Jolliet P, Bulpa P, Chevrolet JC. Effects of the prone position on gas exchange and hemodynamics in severe acute respiratory distress syndrome. *Crit Care Med* 1998;26:1977-85.
- [5] Pelosi P, Tubiolo D, Mascheroni D, et al. Effects of the prone position on respiratory mechanics and gas exchange during acute lung injury. *Am J Respir Crit Care Med* 1998;157:387-93.
- [6] Paulsen SM, Killyon GW, Barillo DJ. High-frequency percussive ventilation as a salvage modality in adult respiratory distress syndrome: a preliminary study. *Am Surg* 2002;10:852-6.
- [7] Velmahos GC, Chan LS, Tatevossian R, et al. High-frequency percussive ventilation improves oxygenation in patients with AEDS. *Chest* 1999;116:440-6.
- [8] Gattinoni L, Caironi P, Pelosi P, et al. What has computed tomography taught us about the acute respiratory distress syndrome? *Am J Respir Crit Care Med* 2001;164:1701-11.
- [9] Reper P, Wibaux O, Van Laeke P, et al. High frequency percussive ventilation and conventional ventilation after smoke inhalation: a randomised study. *Burns* 2002;28:503-8.
- [10] Cioffi WG, deLemos RA, Coalson JJ, et al. Decreased pulmonary damage in primates with inhalation injury treated with high-frequency ventilation. *Ann Surg* 1993;218:328-37.
- [11] Salim A, Miller K, Dangleben D, et al. High-frequency percussive ventilation: an alternative mode of ventilation for head-injured patients with adult respiratory distress syndrome. *J Trauma* 2004;57:542-6.