

Effects of Expiratory Rib-Cage Compression on Oxygenation, Ventilation, and Airway-Secretion Removal in Patients Receiving Mechanical Ventilation

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BACKGROUND: Expiratory rib-cage compression, a chest physiotherapy technique, is well known as the “squeezing” technique in Japan. **OBJECTIVE:** To determine the effects of rib-cage compression on airway-secretion removal, oxygenation, and ventilation in patients receiving mechanical ventilation. **SETTING:** An intensive care unit of an emergency and critical care center at a tertiary-care teaching hospital in Tokyo, Japan. **METHODS:** Thirty-one intubated, mechanically ventilated patients in an intensive care unit were studied in a randomized, crossover trial. The patients received endotracheal suctioning with or without rib-cage compression, with a minimum 3-hour interval between the 2 interventions. Rib-cage compression was performed for 5 min before endotracheal suctioning. Arterial blood gas and respiratory mechanics were measured 5 min before endotracheal suctioning (baseline) and 25 min after suctioning. The 2 measurement periods were carried out on the same day. **RESULTS:** There were no significant differences in the ratio of arterial partial pressure of oxygen to fraction of inspired oxygen, P_{aCO_2} , or dynamic compliance of the respiratory system between the 2 periods (before and after endotracheal suctioning). Moreover, there were no significant differences in airway-secretion removal between the 2 periods. **CONCLUSIONS:** This study suggests that rib-cage compression prior to endotracheal suctioning does not improve airway-secretion removal, oxygenation, or ventilation after endotracheal suctioning in this unselected population of mechanically ventilated patients. *Key words:* mechanical ventilation, secretion clearance, critical care, physical therapy, rib-cage compression. [Respir Care 2005;50(11):1430–1437. © 2005 Daedalus Enterprises]

Introduction

A variety of chest physiotherapy techniques have been widely utilized with patients receiving mechanical venti-

lation. Expiratory rib-cage compression is well known as “squeezing” in Japan. This technique consists of manually compressing the rib cage during expiration and releasing the compression at the end of the expiration, with the objective of mobilizing and removing pulmonary secretions, facilitating active inspiration, and improving alveolar ventilation.^{1,2} It is advocated that rib-cage compression is effective in the treatment and/or prevention of lung collapse in mechanically ventilated patients with diverse pathophysiologies,^{1,3} but there are few studies regarding its efficacy. We previously reported that rib-cage compression was ineffective in improving oxygenation and ventilation in mechanically ventilated rabbits with atelectasis induced by artificial mucus infusion into the trachea.⁴ Moreover, using the same animal model, we found that rib-cage compression did not improve mucus clearance.⁵ To our knowledge, there are no published data concerning the effects of rib-cage compression in patients receiving mechanical ventilation, in terms of oxygenation, lung mechanics, or se-

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cretion clearance. Because the underlying conditions in patients receiving mechanical ventilation vary and differ from those in our animal model of induced atelectasis, it is necessary to clarify whether the effects of rib-cage compression are similar between our animal model and patients receiving mechanical ventilation.

In the present study we hypothesized that, as in our previous animal study, rib-cage compression would not improve oxygenation or ventilation or accelerate mucus clearance in mechanically ventilated patients. We measured oxygenation, ventilation, and lung mechanics of collapsed lungs with serial measurements of gas exchange and dynamic compliance of the respiratory system (C_{RS}). Gas exchange was assessed by the ratio of arterial partial pressure of oxygen to fraction of inspired oxygen (P_{aO_2}/F_{IO_2}) and P_{aCO_2} . Airway-secretion clearance was evaluated by weighing the sputum collected with suctioning.

Methods

Study Location and Patients

The study was carried out in the 8-bed intensive care unit of the emergency and critical care center at St Luke's International Hospital, Tokyo, Japan, a tertiary-care teaching hospital. Informed consent was obtained from each patient or next of kin, and the study was approved by the hospital's ethics committee. Patients who were likely to require continuous invasive mechanical ventilation for > 48 hours and an arterial cannula were considered eligible for the study. Exclusion criteria were rib fracture, presence of a chest tube, hemodynamic instability, and inadequate human resources to complete the study protocol with a given patient. No changes in individual ventilator settings were made for the purpose of the study, except when measurement of C_{RS} was needed, in which case we used mandatory ventilation during spontaneous breathing mode alone. Patients were excluded if their ventilator settings were changed during the study period. In the 2 periods (ie, expiratory rib-cage compression with endotracheal suctioning and endotracheal suctioning alone), all the interventions were delivered by the same operator.

Study Design

The study was prospective, controlled, randomized, and of a crossover design. Using an envelope method, patients were randomly allocated to receive either (1) expiratory rib-cage compression prior to endotracheal suctioning in the first period, followed by endotracheal suctioning without rib-cage compression in the second period or (2) endotracheal suctioning without rib-cage compression in the first period, followed by expiratory rib-cage compression

prior to endotracheal suctioning in the second period (Fig. 1).

Measurements

P_{aO_2} and P_{aCO_2} were measured with a blood gas analyzer (860 Blood Gas System, Ciba Corning Diagnostics, East Walpole, Massachusetts). Total dynamic respiratory compliance was calculated in individual ventilator settings, except for pressure-support ventilation (PSV). Peak inspiratory pressure and tidal volume (V_T) were recorded using a ventilator monitor (model 7200 [Puritan Bennett, Pleasanton, California] or Wave VM200 [Newport Medical Instruments, Newport Beach, California] or Adult Star [Infrasonics, San Diego, California]). When PSV without mandatory breaths was used, several mandatory breaths (10 mL/kg) were temporarily added by the ventilator, and peak inspiratory pressure and V_T were recorded. Dynamic C_{RS} was calculated as $V_T/(\text{peak inspiratory pressure minus positive end-expiratory pressure})$. Airway secretions collected via endotracheal suctioning were weighed per the methods of a previous study.⁶ An in-line closed-suctioning system (TrachCare, Kimberly-Clark/Ballard Medical Products, Draper, Utah) was used with all patients. Sputum was collected in a pre-weighed sputum trap attached to the suction catheter. Either 2 mL or 5 mL of sterile saline was flushed through the suctioning tubing into the trap to clear any secretions in the catheter. The weight of the aspirated secretions was calculated by subtracting the weight of the trap and either the 2 mL or 5 mL of sterile saline from the total weight of the trap and secretions obtained.

Protocol

The patients were positioned so that the most affected lung region, as determined from a chest radiograph (atelectasis and/or infiltration), and/or crackles or rhonchi on auscultation, was uppermost. Radiograph interpretations were made by radiologists independent of the study. Body position was decided by an experienced nurse (YK, TU, YY, or YF) before the first measurement period. Expiratory rib-cage compression and endotracheal suctioning were performed by one of 4 nurses, who were trained. The patients were placed in the same position during each measurement period, and had the 2 measurement periods on the same day, allowing a minimum 3-hour interval. Baseline measurements of arterial blood gas and respiratory mechanics were made 5 min before endotracheal suctioning in both periods. Expiratory rib-cage compression was applied for 5 min between baseline measurement and endotracheal suctioning. In both measurement periods, the collected airway secretions were weighed as described above. Post-intervention measurements of arterial blood gases and respiratory mechanics were made 25 min after

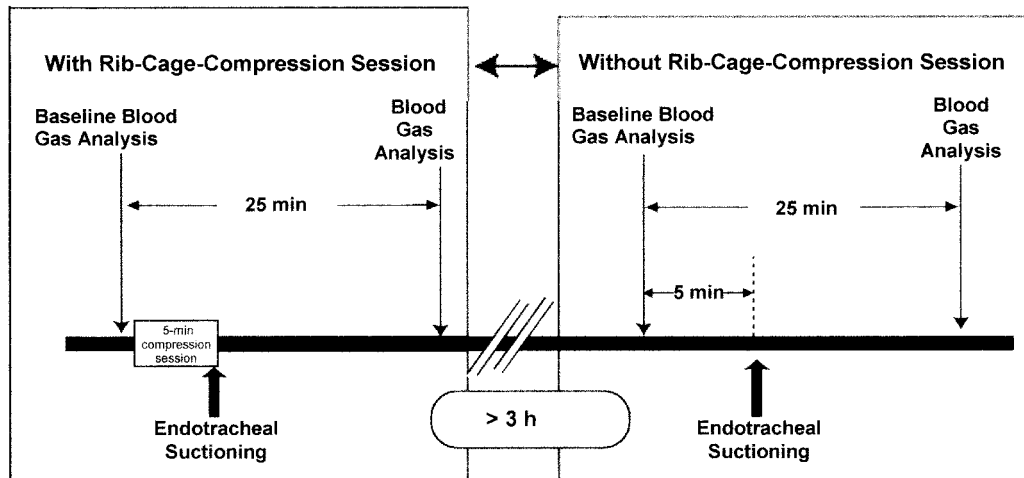


Fig. 1. Study protocol. Each patient underwent endotracheal suctioning, either with or without expiratory rib-cage compression, in random order.

endotracheal suctioning, with the same body position as decided before the first measurement.

Endotracheal Suctioning

Endotracheal suctioning was carried out with a 14 or 16 French in-line suction catheter, by experienced intensive-care nurses, according to the institutional standards. Hypoxygenation and hyperinflation were not performed before or after endotracheal suctioning. Patients did not receive endotracheal suctioning within the 1 hour before each intervention. Sputum was collected in a sputum trap connected to the suction catheter, and was measured as described above.

Rib-Cage Compression

Manual expiratory rib-cage compression was performed by any of the 4 trained nurses, who attempted to use consistent technique, applying the same force. The method of rib-cage compression in the present study was in accordance with the standard technique for clinical use.¹ Briefly, the operator uses both hands to gradually squeeze the rib cage during expiration (Fig. 2). The operator attempted to give rib-cage compression over the part of the rib cage that included the most affected lung region, from the end of inspiration to the end of expiration. Every rib-cage compression was interrupted at the end of each expiratory phase, to allow free inspiration, with both spontaneously breathing and mechanically ventilated subjects. Special care was taken to ensure that the compression was applied only during expiration. In patients undergoing volume-controlled mode, rib-cage compression was performed ev-

ery 2 breaths. The operator attempted to synchronize compression rate with the patient's individual respiratory rate.

Statistical Analysis

We estimated the sample size to detect a 60-mm Hg P_{aO_2}/F_{IO_2} difference between the 2 post-intervention data, yielding a power of 80%. We used an α -error of 0.05 (two-tailed) and normal distribution of differences of 80 mm Hg. We estimated the normal distribution of difference in a pilot study of the present study ($n = 10$). On the basis of these assumptions, we estimated that about 30 patients were needed. All physiologic values except for the quantity of secretions removed were analyzed using the paired t test. Because of the non-normal distribution of the quantity of secretions removed, we used the Wilcoxon matched pairs test to analyze it. All statistical analyses were performed with commercially available software (JMP 4J, SAS Institute, Cary, North Carolina).

Results

Patient Enrollment

During the period from October 2001 to September 2003, 975 patients were admitted to our intensive care unit (Fig. 3). One-hundred fifty-eight patients received mechanical ventilation and arterial cannulation for > 48 hours. Of those 158 patients, 14 were excluded because they had chest tube and/or rib fracture, 2 patients were excluded because of hemodynamic instability, and 111 patients were excluded because of inadequate human resources. Thus, 31 patients were studied. Table 1 shows patient character-



Fig. 2. Application of manual expiratory rib-cage compression to the lower lung region.

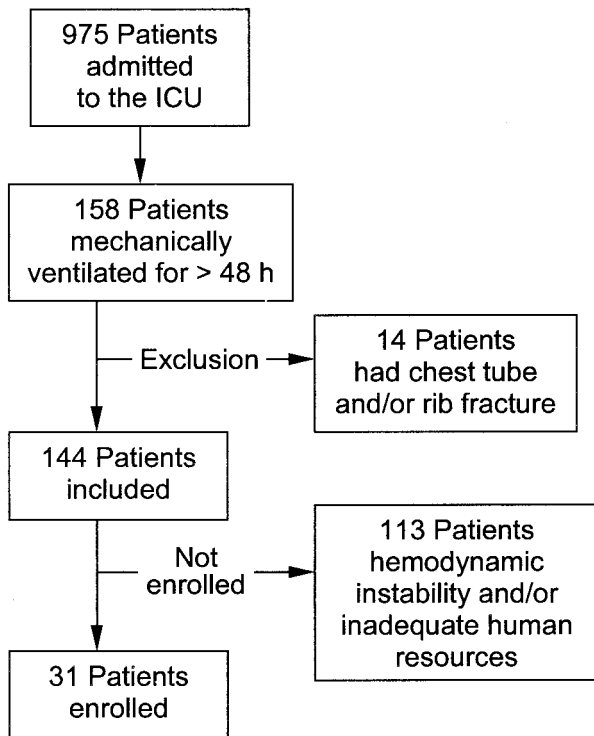


Fig. 3. Patient enrollment. ICU = intensive care unit.

istics and ventilator settings. Twenty-four patients were ventilated with volume-controlled synchronized intermittent mandatory ventilation. None of the patients had deleterious effects (eg, drop in arterial oxygen saturation,

hemodynamic instability, tachypnea, or barotrauma) related to rib-cage compression.

P_{aO_2}/F_{IO_2}

The mean baseline P_{aO_2}/F_{IO_2} values for the 2 periods were similar (Table 2 and Fig. 4). There were no significant P_{aO_2}/F_{IO_2} differences between the 2 post-intervention periods. Additionally, there were no significant P_{aO_2}/F_{IO_2} differences between before and after the interventions in both periods.

Compliance

Mean baseline C_{RS} values in the 2 periods were similar (see Table 2 and Fig. 4). There were no significant C_{RS} differences between the 2 post-intervention periods. Additionally, there were no significant C_{RS} differences between before and after the interventions in both periods.

P_{aCO_2}

Mean baseline P_{aCO_2} values in the 2 periods were similar (see Table 2 and Fig. 4). There were no significant differences in P_{aCO_2} between the 2 post-intervention periods. Additionally, there were no significant P_{aCO_2} differences between before and after the interventions in both periods.

Table 1. Characteristics of the Study Patients*

Age (mean ± SD y)	56.7 ± 17.6
Gender (number and % male)	24 (87)
Simplified Acute Physiology Score (mean ± SD)	59.4 ± 10.7
Duration of mechanical ventilation from intubation to the study enrollment (median and range d)	5 (2–27)
Duration of mechanical ventilation from the study enrollment to completion of weaning (median and range d)	11 (5–41)
Tracheostomy (number and %)	4 (13)
<u>Diagnosis at intensive-care unit admission (number and %)</u>	
Intracerebral hemorrhage	6 (19.4)
Cardiac arrest	6 (19.4)
Pneumonia	4 (12.9)
Cerebral infarction	4 (12.9)
Aneurysmal subarachnoid hemorrhage	3 (9.7)
Sepsis	3 (9.7)
Head injury	2 (6.5)
Others	3 (9.7)
<u>Chest radiograph findings (number and %)</u>	
Atelectasis	13 (42)
Infiltration	13 (42)
Other	5 (16)
<u>Mechanical ventilation mode (number and %)</u>	
Assist-control ventilation	1 (3)
Synchronized intermittent mandatory ventilation	28 (90)
Pressure-support ventilation	2 (6)
Pressure-control ventilation (number and %)	4 (13)
Fraction of inspired oxygen (median and range)	0.3 (0.21–0.7)
Positive end-expiratory pressure (median and range cm H ₂ O)	5 (0–15)

*n = 31

Weight of Aspirated Sputum

There were no significant differences in the weight of the collected sputum in the 2 periods (Fig. 5).

Discussion

The present study shows that expiratory rib-cage compression had no beneficial effects on oxygenation, ventilation, or sputum clearance in this unselected population of mechanically ventilated patients.

To the best of our knowledge, this is the first clinical study to examine the effects of rib-cage compression on gas exchange, pulmonary mechanics, and secretion clearance with patients receiving mechanical ventilation.

Oxygenation and Ventilation

There were no significant P_{aO₂}/F_{IO₂} differences between the 2 periods. This finding suggests that expiratory rib-cage compression has no beneficial effect on oxygenation in mechanically ventilated patients. This is in agreement with our previous study with mechanically ventilated rabbits with induced atelectasis.⁴ The mechanisms for the development of atelectasis and impaired mucociliary clearance appear to be multifactorial in critically ill patients, who are unable to move or to give effective coughs by themselves.

In the present study, not all the patients had radiographically visible atelectasis, but, because microatelectasis is usually undetectable by conventional chest radiograph, we think it is likely that many of the patients had atelectasis.⁷ If that is true, rib-cage compression seemed to be ineffective in reexpanding collapsed alveoli. One possible explanation is that rib-cage compression did not generate elastic

Table 2. Changes in P_{aO₂}/F_{IO₂}, P_{aCO₂}, and C_{RS} Between Baseline and 25 Minutes After Endotracheal Suctioning

		Baseline (mean ± SD)	25 min After Endotracheal Suctioning (mean ± SD)	p†
P _{aO₂} /F _{IO₂} (mm Hg)	Control	297.2 ± 81.1	301.9 ± 82.0	0.48
	Rib-cage compression	300.3 ± 79.0	300.2 ± 86.7	0.98
	p‡	0.88	0.94	
P _{aCO₂} (mm Hg)	Control	39.6 ± 6.1	39.3 ± 6.8	0.31
	Rib-cage compression	38.9 ± 6.6	38.6 ± 7.4	0.52
	p‡	0.66	0.69	
C _{RS} (L/cm H ₂ O)	Control	39.6 ± 12.7	41 ± 14.1	0.10
	Rib-cage compression	39.7 ± 12.8	39.8 ± 14.4	0.93
	p‡	0.99	0.73	

†Paired t test between baseline and 25 min after endotracheal suctioning

‡Paired t test between control and rib-cage compression

P_{aO₂}/F_{IO₂} = ratio of partial pressure of oxygen to fraction of inspired oxygen

C_{RS} = dynamic compliance of the respiratory system

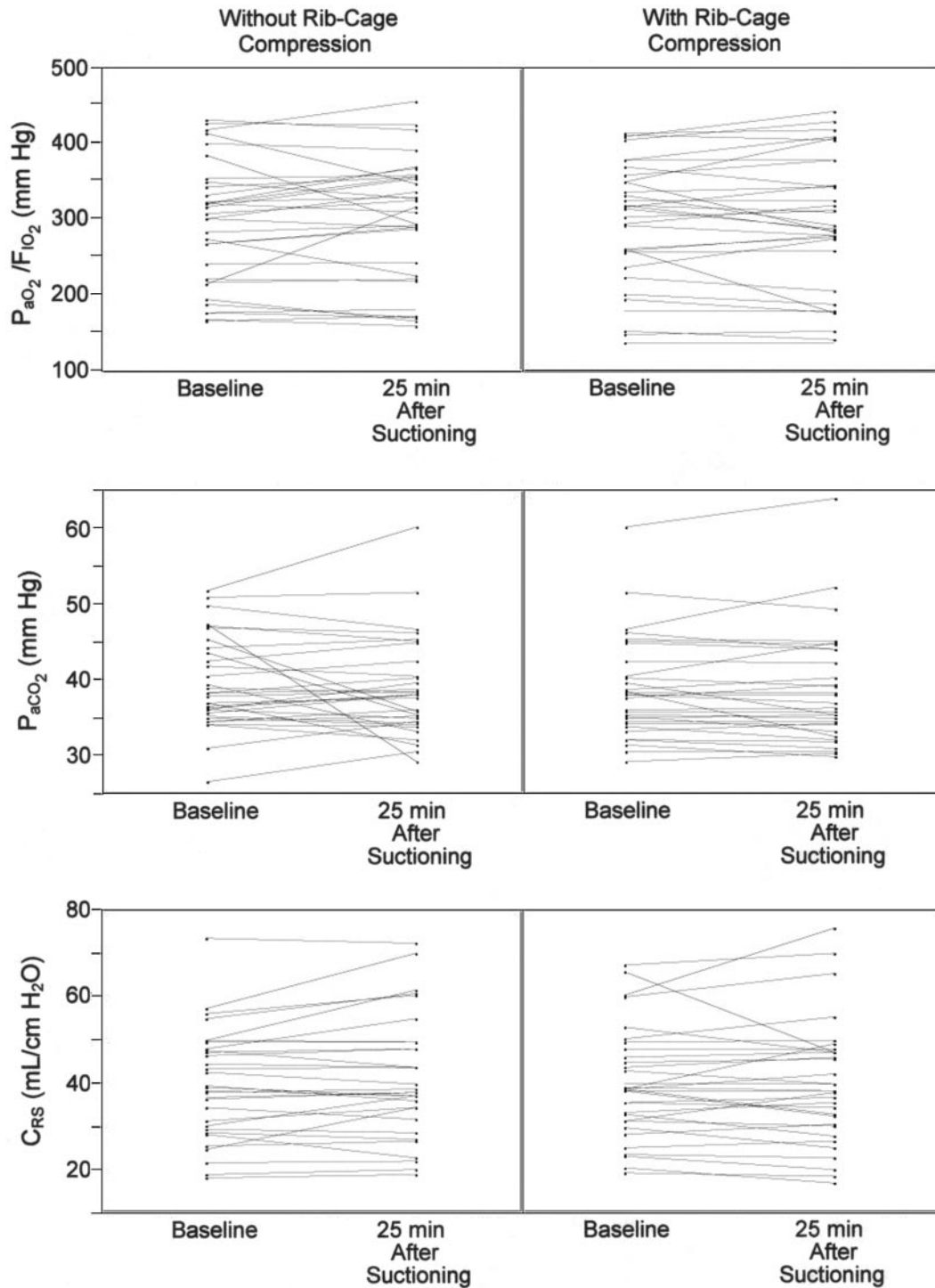


Fig. 4. Individual values for ratio of arterial partial pressure of oxygen to fraction of inspired oxygen (P_{aO_2}/F_{iO_2}), P_{aCO_2} , and dynamic compliance of the respiratory system (C_{RS}) before and 25 min after endotracheal suctioning, with or without rib-cage compression.

recoil pressure enough to reexpand collapsed alveoli. However, it is claimed that the elastic recoil pressure of the rib cage in the beginning of inspiration immediately after rib-cage compression is able to expand collapsed alveoli.¹ Rothen et al⁸ reported that 40 cm H₂O of airway pressure

and time were needed to reexpand collapsed alveoli in patients under general anesthesia. Based on our results, it is unlikely that the elastic recoil pressure of the rib cage in early inspiration immediately after rib-cage compression is enough to expand collapsed alveoli.

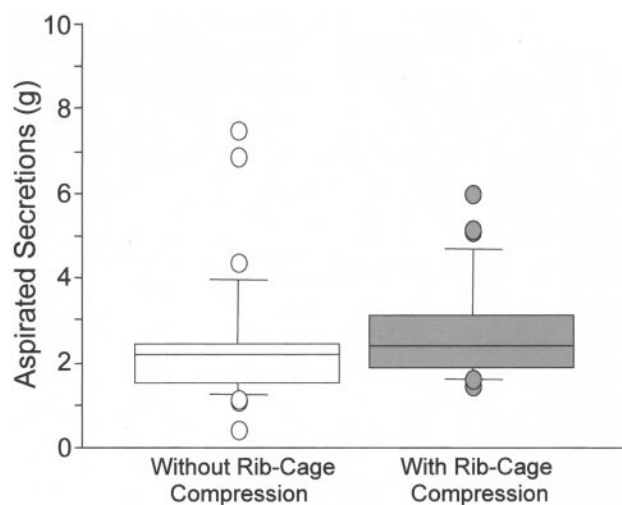


Fig. 5. Weight of collected airway secretions collected with and without expiratory rib-cage compression. The horizontal lines inside the boxes represent the medians. The boxes encompass the 25th through 75th percentile, and the error bars indicate the 10th and 90th percentiles. The circles represent outliers.

Expiratory rib-cage compression may induce lung collapse by further reducing lung volume at end-expiration. It was previously shown that expiratory rib-cage compression causes important deleterious effects on oxygenation and C_{RS} in mechanically ventilated rabbits with induced atelectasis.⁵ It is possible that expiratory rib-cage compression reduces end-expiratory lung volume, thereby causing further deleterious effects on previously noncollapsed lung.

Our previous study showed that P_{aO_2}/F_{IO_2} and V_T increased only during rib-cage compression but returned to the pre-compression level immediately after rib-cage compression was discontinued.⁴ Herala and Gislason⁹ reported that transcutaneously measured P_{CO_2} decreased during expiratory rib-cage compression, but transcutaneously measured P_{CO_2} returned to the pre-compression level after 20 min in 11 of 15 patients with chronic obstructive airway disease. Although these studies showed that rib-cage compression temporarily improved P_{aO_2}/F_{IO_2} , C_{RS} , and P_{aCO_2} , we could not demonstrate that expiratory rib-cage compression increases P_{aO_2} in the present study. This might be related to the differences in ventilation methods and timing of blood gas analysis. In our animal studies, the animals were ventilated with pressure-controlled ventilation, during which end-expiratory lung volume was probably reduced by rib-cage compression, but subsequent inspiratory lung volume was increased according to the preset pressure limit. This is a possible mechanism of V_T increase during rib-cage compression in our previous animal study.

In contrast to our previous study, the patients in the present study were ventilated with several modes, mainly

volume-control mode with PSV. During volume-controlled ventilation, rib-cage compression may reduce end-expiratory lung volume. Accordingly, subsequent end-inspiratory lung volume might also be reduced. Therefore, in the present study, with patients undergoing volume-controlled ventilation we applied compressions every 2 breaths, to avoid progressive lung-volume reduction. In addition, it is possible that we missed temporary P_{aO_2} increases immediately after rib-cage compression because of delay in blood gas analysis, since this phenomenon was observed in our previous animal study.⁴

Little is known about the interaction between rib-cage compression and the mode of mechanical ventilation, especially PSV. The patients in the present study were ventilated mainly with synchronized intermittent mandatory ventilation combined with PSV. The release of a rib-cage compression usually triggers PSV. In the present study, however, we could not examine the interaction between rib-cage compression and PSV, because it was difficult to evaluate respiratory mechanics and blood gas during rib-cage compression in the clinical setting.

Airway-Secretion Clearance

In the present study, rib-cage compression did not improve mucus clearance. To our knowledge, there has been no study to examine the effects of rib-cage compression on mucus clearance in a clinical setting. In our animal study we showed that rib-cage compression did not improve mucus clearance in mechanically ventilated rabbits with induced experimental atelectasis.⁵ The findings of the present study seem to be in agreement with those of our previous study.⁵ In general, forced expiration, which may increase expiratory flow rate, is likely to propel airway secretions. But there are few published data on the effect of rib-cage compression on peak expiratory flow rate in mechanically ventilated adult patients. In intubated patients, during manual ventilation, rib-cage compression increased peak expiratory flow from 73.3 L/min to 103.9 L/min.¹⁰ Thus, presumably, in the present study, peak expiratory flow increased during rib-cage compression, although the patients were mechanically ventilated. However, mucus clearance was not improved. It is advocated that chest physical therapy aimed at improving mucus clearance should be confined to patients who have excessive airway secretions.¹¹ A recent review mentioned that the increase in sputum secretion associated with chest physical therapy is generally small.¹² In the present study, none of the patients had excessive fluid secretion (eg, as in cystic fibrosis or bronchiolitis), and patients with a variety of respiratory dysfunctions were included. Therefore, the present study suggests that it is difficult to achieve a statistically significant improvement in the amount of aspirated airway mucus with rib-cage compression.

Limitations

First, in the present study, although 144 patients were considered eligible, only 31 patients were studied, so we should be cautious to examine whether our sample suffered selection bias. Since the main reason for exclusion was inadequate human resources, and patient selection was not made intentionally, we think selection bias is unlikely, although we cannot rule it out completely. Second, the patients' respiratory pathophysiologies that required mechanical ventilation were not uniform. It was practically impossible to standardize patients' clinical conditions in the present study. That lack of uniformity in our study population was probably inclined to cause negative results. One must be cautious in attempting to generalize our findings to all mechanically ventilated patients. If we could have selected patients with similar respiratory pathophysiologies and identical mechanical ventilation settings, such as patients with acute lobar atelectasis receiving PSV alone, the results might have been different. In addition, we were unable to carry out subgroup analysis according to diagnosis or mechanical ventilation settings, because of the small sample size. Therefore, the interactions between the effects of rib-cage compression and diagnosis or mechanical-ventilation settings should be elucidated in the future.

In the present study our goal was to investigate short-term physiologic effects of rib-cage compression. The effects on longer-term outcomes, such as duration of mechanical ventilation and intensive-care-unit stay remain to be evaluated further.

Conclusion

Rib-cage compression did not improve oxygenation, ventilation, C_{RS} , or secretion clearance in this unselected population of mechanically ventilated patients. Consequently, routine use of rib-cage compression in mechanically ventilated patients is not recommended.

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