

Effects of Expiratory Rib Cage Compression and/or Prone Position on Oxygenation and Ventilation in Mechanically Ventilated Rabbits with Induced Atelectasis

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INTRODUCTION: Expiratory rib cage compression is a chest physiotherapy technique known as “squeezing” technique in Japan. It has been claimed that rib cage compression effectively treats and/or prevents lung collapse, but no studies have been reported on rib cage compression focused on improving ventilation and/or oxygenation in subjects with collapsed lung. Therefore, we studied whether rib cage compression, with and without prone positioning, improves the ratio of P_{aO_2} to fraction of inspired oxygen (P_{aO_2}/F_{IO_2}), P_{aCO_2} , or dynamic compliance of the respiratory system. **METHODS:** We used anesthetized adult rabbits with induced atelectasis. An endotracheal tube and an 18-gauge catheter were placed into the airway via a tracheostoma, and pressure-controlled mechanical ventilation was used. To create atelectasis, artificial mucus was infused into the airway via the catheter. The rabbits were randomly assigned to one of 4 groups ($n = 10$ in each group): (1) supine without rib cage compression, (2) supine with rib cage compression, (3) prone without rib cage compression, and (4) prone with rib cage compression. Each rib cage compression session lasted for 5 min and was repeated 5 times every 30 min. After these interventions for 180 min all animals were placed in the supine position for 120 min. **RESULTS:** The prone-position groups had significantly higher P_{aO_2}/F_{IO_2} than the supine-position groups at 60 min after the beginning of the intervention, and at 60, 90, and 120 min after the end of the intervention ($p < 0.05$). Rib cage compression did not significantly affect P_{aO_2}/F_{IO_2} , P_{aO_2} , or dynamic compliance. **CONCLUSIONS:** It is unlikely that rib cage compression re-expands collapsed lung. Prone positioning improved oxygenation in rabbits with induced atelectasis. *Key words:* atelectasis, mucus, oxygenation, physical therapy, rib cage compression, prone position. [Respir Care 2003;48(8):754–762. © 2003 Daedalus Enterprises]

Introduction

Lung collapse is a common complication in mechanically ventilated patients. It can lead to impaired oxygenation¹ and to longer hospital and intensive care unit length of stay.² Expiratory rib cage compression is well known as the “squeezing” technique in Japan. Advocates for rib cage

compression claim that it is effective in the treatment and/or prevention of lung collapse and is safe for critically ill patients (compared to percussion or vibration).^{3,4} This rib cage compression technique consists of manual compression of the rib cage during the expiratory phase and release from the compression at the end of the expiration, in an attempt to remove pulmonary secretions, to facilitate active inspiration, and to improve alveolar ventilation.³

Despite there being few studies regarding its effects, rib cage compression is being used for mechanically ventilated patients in Japan. In 1958 Opie and Spalding⁵ reported on the effects of rib cage compression on the removal of pulmonary secretions. To date there have been no experimental studies on rib cage compression focused on improving ventilation and/or oxygenation in subjects with atelectasis.

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Furthermore, the effects of the prone position and induced atelectasis have not been studied, although previous studies demonstrated that prone positioning improved arterial oxygenation in patients with acute respiratory distress syndrome or acute lung injury, and in animals with oleic acid-induced acute lung injury.^{6,7} Prolonged supine positioning may result in accumulation of mucus in the dependent lung zone. Thus, prone positioning might help mobilize mucus from the dependent zone, thereby improving ventilation and/or oxygenation in an induced atelectasis model as well. In addition, the combination of prone positioning and rib cage compression may have synergistic effects.

To test the hypothesis that rib cage compression and/or prone position improves ventilation and oxygenation, we studied mechanically ventilated rabbits with atelectasis caused by artificial mucus infusion to the trachea. We chose a rabbit model because the ratio of chest wall compliance to lung compliance is similar in the human infant and rabbit.⁸ The improvement in ventilation and oxygenation for the collapsed lung was assessed by serial measurements of gas exchange and dynamic compliance of the respiratory system (C_{RS}). Gas exchange was assessed by the ratio of P_{aO_2} to fraction of inspired oxygen (P_{aO_2}/F_{IO_2}) and P_{aCO_2} .

Methods

Animal Preparation and Measurements

The protocol was approved by our Institutional Animal Research Committee, and the care of the animals was in

accordance with guidelines for ethical animal research. Forty-one female Japanese white rabbits (3.4 ± 0.35 kg) were used (Fig. 1). A 24-gauge intravenous cannula was placed via an ear vein. The rabbits were anesthetized with intravenous injection of 75–150 mg sodium pentobarbital and were infused with lactated Ringer's solution (40 mL/h). The rabbits underwent tracheostomy under local anesthesia with 0.5–1.0 mL of 1.0% lidocaine solution and the trachea was intubated with a 3-mm inner-diameter endotracheal tube (ETT) (Blue Line, SIMS-Portex, Kent, United Kingdom). To instill artificial mucus into the airways an 18-gauge catheter was inserted outside of the ETT, via the tracheostoma. The tip of the catheter was inserted 7 cm below the thyroid cartilage. Both the ETT and the catheter in the trachea were secured by external ligation of the trachea, without allowing air leak. After the rabbits were paralyzed with 0.375 mg of pancuronium, pressure-controlled mechanical ventilation was started (using a Servo 900B, Siemens-Elcoma AB, Solna, Sweden) connected to a pressure relief valve (external pressure limiter #6–600, Lifecare, Lafayette, Colorado). The ventilator settings were: respiratory rate = 30 breath/min, inspiratory flow = 15 L/min, inspiratory time = 33% of the total breathing cycle, positive end-expiratory pressure (PEEP) = 5 cm H₂O, and $F_{IO_2} = 0.3$. Peak inspiratory pressure was adjusted to achieve P_{aCO_2} of 40 ± 5 mm Hg and was kept unchanged thereafter. Anesthesia and muscle paralysis were maintained by continuous infusion of sodium pentobarbital (20

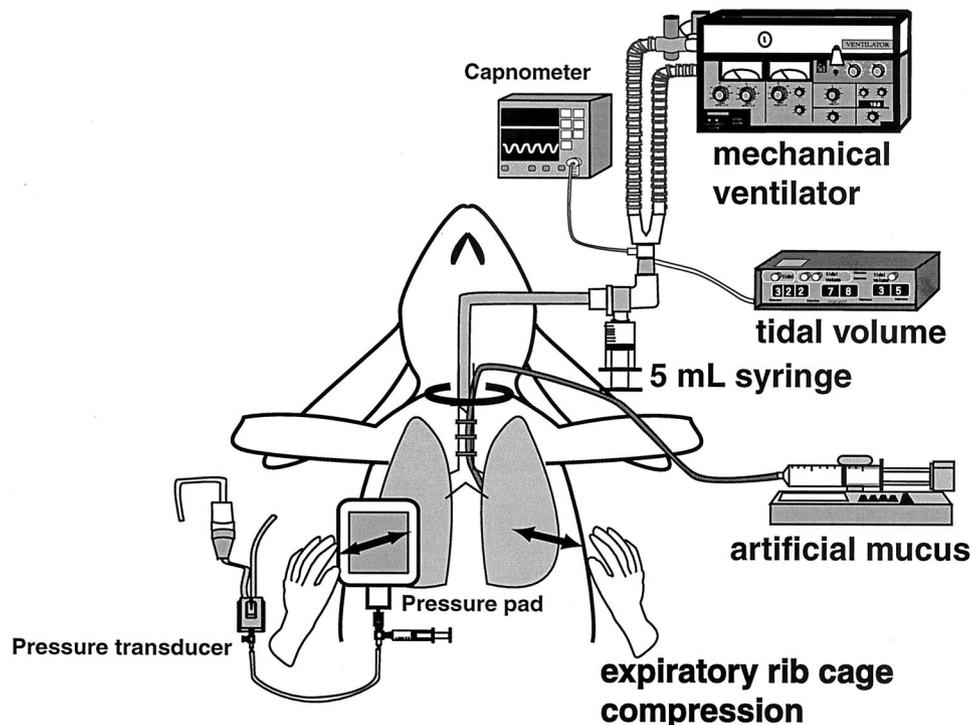


Fig. 1. Experimental setup.

mg/h) and pancuronium (1 mg/h) throughout the experiment. An 18-gauge vascular catheter was inserted into a femoral artery for continuous blood pressure monitoring and intermittent blood sampling. Arterial blood pressure and heart rate were measured continuously, as were expiratory tidal volume (V_T) (NVM-1, Bear Medical Systems, Riverside, California) and end-tidal carbon dioxide (Capnomac Ultima, Datex Instrumentarium, Helsinki, Finland). Rectal temperature was monitored, and an electric heat blanket was used to maintain the rectal temperature at 38–39° C.

To measure output mucus volume, a 5 mL syringe was attached in-line to the proximal end of the ETT (see Fig. 1). To measure the pressure applied to the rib cage by the operator, we made a hand pressure pad that was filled with 28 mL of distilled water and had a contact area of 17.5 cm². The pad was positioned between the operator's hand and the rabbit's rib cage, and was connected to a pressure transducer. Correlation between measured and applied pressure was excellent ($r^2 = 0.988$).

Model of Atelectasis

To create pulmonary atelectasis artificial mucus was infused through the catheter into the airways. The artificial mucus was made of 1.6% (weight/volume) polyethylene oxide powder⁹ (Polyox [average molecular weight 5,000,000], Aldrich Chemical, Milwaukee, Wisconsin) and 0.1% (weight/volume) methylene blue (methylene blue alkali, Chroma Gesellschaft Schmid, Stuttgart, Germany) in phosphate-buffered saline. Dynamic viscoelasticity of the solution was measured with a controlled shear rate rheometer, at a driving frequency of 1 rad/s.¹⁰ The loss modulus (G'') of the artificial mucus was 45.2 dyn/cm² and the storage modulus (G') was 28.8 dyn/cm². The artificial mucus infusion was continued at a rate of 0.2 mL/min for 10 min. If, at 10 min after the initiation of artificial mucus infusion, P_{aO_2}/F_{IO_2} was > 60% of that before initiation of artificial mucus infusion, an additional 0.5 mL of artificial mucus was injected. After a stabilization period of 30 min arterial blood gas (ABG) values, expiratory V_T , and peak inspiratory pressure were recorded, and these values were defined as baseline.

Protocol

Figure 2 illustrates the experimental protocol. Initially all the rabbits were placed in the supine position and ABG measurements (288 Blood Gas System, Ciba-Corning, Medfield, Massachusetts) were made to confirm normal oxygenation and ventilation. The 40 rabbits were randomly assigned to one of 4 groups ($n = 10$ in each group): (1) supine position with rib cage compression (Sp_{comp} group), (2) prone position with rib cage compression (Pr_{comp} group),

(3) supine position without rib cage compression (Sp group), and (4) prone position without rib cage compression (Pr group). After the 180-min intervention period all the rabbits were placed in the supine position for 120 min. ABGs and expiratory V_T were measured every 15 or 30 min. One rabbit was studied additionally to obtain esophageal, airway, and hand pressure waveforms during rib cage compression, while in the supine position, without artificial mucus infusion. After the experiments the rabbits were sacrificed with intravenous injection of 2 mL potassium chloride (2 mol/L) solution, and necropsy was done to determine the laterality (left or right bronchus) of the catheter in each animal.

Expiratory Rib Cage Compression

Manual bilateral expiratory rib cage compression was performed by a single operator (TU), who attempted to use consistent technique, applying the same force with each animal. The rib cage compression method we used was based on the standard technique for clinical use,³ in which the operator delivers bilateral squeeze gradually to the lower rib cage during expiration. Each rib cage compression was interrupted at the end of the expiratory phase to allow free inspiration. Special care was taken to ensure that compression was applied only during expiration.

In the Sp_{comp} and Pr_{comp} groups rib cage compression was applied to every breath for 5 min, and was first performed at 50 min after the baseline measurements. Thereafter, a 5-min rib cage compression session was repeated 5 times 20 min after the previous ABG measurement (see Fig. 2). Additionally, expiratory V_T was recorded during rib cage compression.

To more precisely examine oxygenation, P_{aCO_2} , and C_{RS} during rib cage compression, one rabbit in the Pr_{comp} group had ABG values and expiratory V_T measured every 1 min from 5 min before the beginning of rib cage compression to 5 min after the end of it, 4 times.

Histopathology Examination

To confirm the appropriateness of the modified atelectasis model, the lungs of 5 rabbits (2 from the Pr_{comp} group and 1 from each other group) were removed for histopathology examination. The lungs were inflated with air at 5 cm H₂O and then the trachea was clamped. After 200 mL of 10% buffered formalin was injected through the right ventricle, the lungs were excised from the rabbit. The lungs were soaked in 10% formalin for a week. The reddened (atelectatic) and nonreddened (nonatelectatic) areas were excised from the lungs as the specimens. They were paraffin embedded and sectioned at 4–6 μ m. After deparaffinization and dehydration the sections were stained with hematoxylin and eosin.

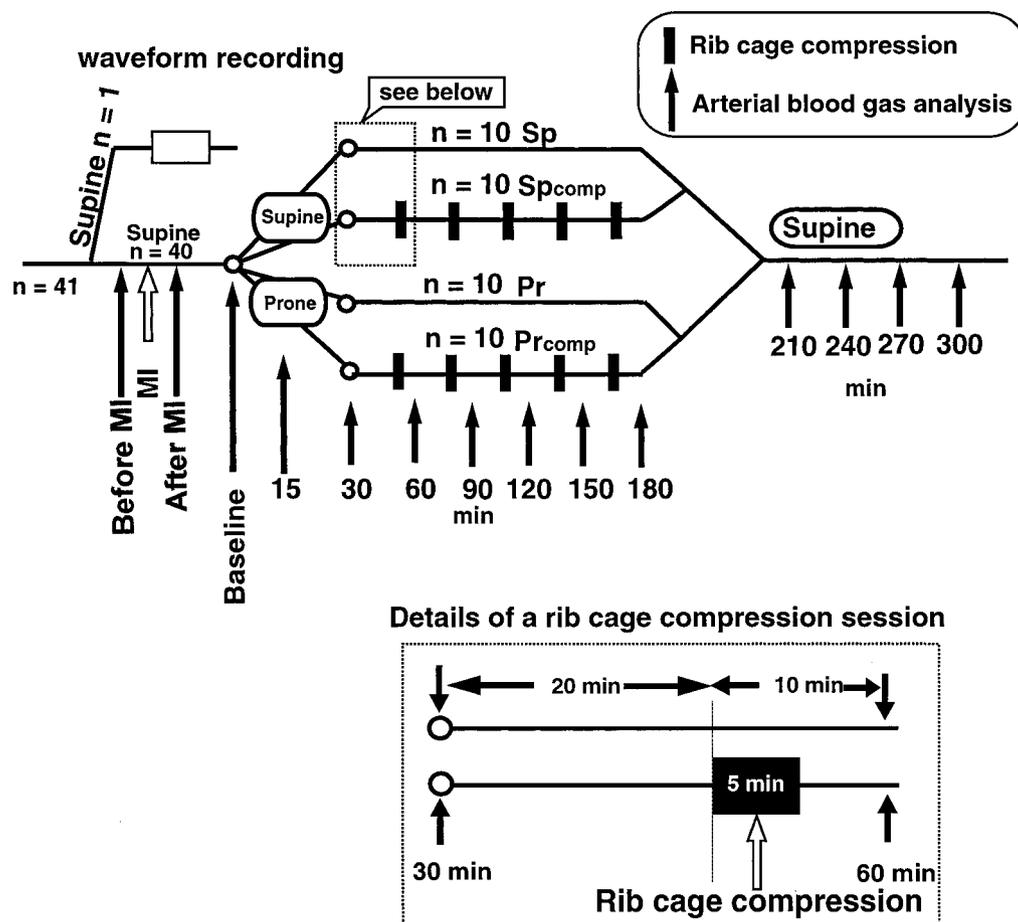


Fig. 2. Experimental protocol. Closed arrows represent periods of arterial blood gas (ABG) analysis. Small closed rectangles indicate expiratory rib cage compression for 5 min. Baseline was obtained 30 min after artificial mucus infusion (MI). Details of a rib cage compression session are shown in the box at the lower right. In the supine compression (Sp_{comp}) and prone compression (Pr_{comp}) groups the animals received expiratory rib cage compressions for 5 min from 20 min after the previous ABG measurement. ABG was measured 5 min after the end of rib cage compression.

Statistical Analysis

All values are reported as mean \pm SD unless otherwise specified. Group differences were determined via repeated-measures 2-way analysis of variance. The changes in physiologic values after the baseline period were analyzed by repeated-measures 1-way analysis of variance to compare the baseline values. Statistically significant differences were followed up by post-hoc analysis (Sheffe's multiple comparison test). In the rib cage compression groups V_T during rib cage compression was compared with that at 20 min before the beginning of the rib cage compression, using the paired t test, and each value was compared between the 2 body positions, using the unpaired t test. Differences between the groups with regard to the laterality of the airway catheter were determined via the chi-square test. Differences were considered statistically significant when $p < 0.05$. All statistical analysis

was performed with statistics software (StatView for Windows, Version 5.0, SAS Institute, Cary, North Carolina).

Results

Model of Atelectasis

In all rabbits the tip of the catheter inserted into the airway reached the main bronchus. In half of the rabbits the catheter tip was in the right bronchus. The lateralities of the catheters in each group were: Sp: 6 in the right bronchus, 4 in the left bronchus; Sp_{comp} : 4 right, 6 left; Pr: 6 right, 4 left; and Pr_{comp} : 4 right, 6 left. There were no significant differences in catheter laterality among the groups ($p = 0.66$). There were also no significant differences in the amount of artificial mucus infused: Sp 2.61 ± 1.44 mL; Sp_{comp} 2.78 ± 1.97 mL; Pr 2.25 ± 0.35 mL; and Pr_{comp} 2.15 ± 0.24 mL ($p = 0.64$). Histologically, most air

spaces were widely open, and tissue architecture was basically normal in the specimens that appeared macroscopically nonatelectatic. In the macroscopically atelectatic specimens air spaces were diffusely collapsed. In neither atelectatic nor nonatelectatic specimens was artificial mucus microscopically observed. In none of the rabbits was there any measurable mucus output from the ETT.

Peak Inspiratory Pressure

There were no significant differences in peak inspiratory pressure among the groups: Sp 17.8 ± 1.8 cm H₂O; Sp_{comp} 16.8 ± 2.5 cm H₂O; Pr 17.2 ± 1.9 cm H₂O; and Pr_{comp} 18.3 ± 1.2 cm H₂O ($p = 0.32$).

Oxygenation

The overall mean P_{aO₂}/F_{IO₂} (of all the rabbits) had decreased by 250.6 ± 74.9 mm Hg at 10 min after the end of artificial mucus infusion (Fig. 3A). At 60, 240, 270, and 300 min the Pr and Pr_{comp} groups had higher P_{aO₂}/F_{IO₂} than the Sp and Sp_{comp} groups. Rib cage compression did not significantly affect P_{aO₂}/F_{IO₂} throughout the experiment. None of the groups showed significant change in P_{aO₂}/F_{IO₂} from the baseline value (see Fig. 3A).

According to the data obtained from one rabbit during rib cage compression, P_{aO₂}/F_{IO₂} increased during rib cage compression but returned to the level before rib cage compression immediately after rib cage compression ended (Fig. 4).

Ventilation

The overall mean P_{aCO₂} had increased by 14.2 ± 9.2 mm Hg at 10 min after the end of artificial mucus infusion (see Fig. 3B). There were no significant P_{aCO₂} differences between the groups with and without rib cage compression ($p = 0.68$) nor between the supine and prone position groups ($p = 0.34$), throughout the experiment. No group showed significant P_{aCO₂} change from baseline throughout the experiment.

According to the data obtained from one rabbit during rib cage compression, P_{aCO₂} decreased during rib cage compression but returned to the level before rib cage compression immediately after rib cage compression ended (see Fig. 4).

Dynamic Compliance of the Respiratory System

The overall mean C_{RS} had decreased by 0.80 ± 0.44 mL/cm H₂O at 10 min after the end of artificial mucus infusion (see Fig. 3C). There were no significant C_{RS} differences between the groups that did and did not receive rib cage compression ($p = 0.74$) nor between the supine

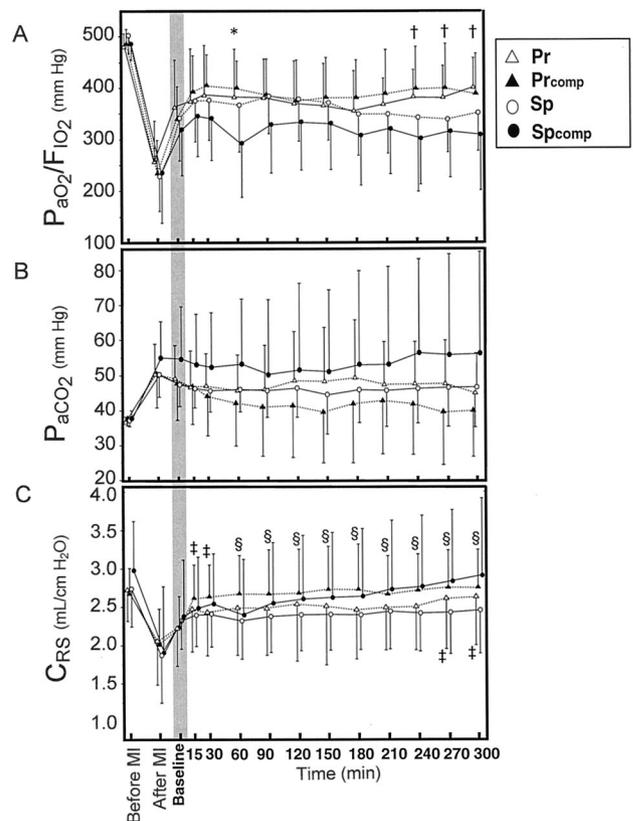


Fig. 3. Changes in (A) the ratio of P_{aO₂} to fraction of inspired oxygen (P_{aO₂}/F_{IO₂}), (B) P_{aCO₂}, and (C) compliance of the respiratory system (C_{RS}) in 4 groups (Pr = prone without expiratory rib cage compression; Pr_{comp} = prone with expiratory rib cage compression; Sp = supine without expiratory rib cage compression; Sp_{comp} = supine with expiratory rib cage compression). Shaded column indicates baseline. At 60, 240, 270, and 300 min the prone groups had higher P_{aO₂}/F_{IO₂} than the supine groups (* = $p < 0.05$, † = $p < 0.01$). There was no significant P_{aCO₂} difference between prone and supine position, nor between rib cage compression and no compression. There were no significant C_{RS} differences between prone and supine position, nor between rib cage compression and no compression. In the Pr_{comp} group C_{RS} from 15 to 300 min increased significantly above baseline. In the Pr group C_{RS} at 270 and 300 min was significantly higher than baseline (‡ = $p < 0.05$, § = $p < 0.01$ compared with baseline). MI = artificial mucus infusion.

and prone groups ($p = 0.65$), throughout the experiment. In the Pr_{comp} group, from 15 to 300 min, C_{RS} significantly increased above the baseline value ($p < 0.05$).

According to the data obtained from one rabbit during rib cage compression, C_{RS} decreased during rib cage compression but returned to the level before rib cage compression immediately after rib cage compression ended (see Fig. 4).

Tidal Volume

During rib cage compression V_T significantly increased above the value before rib cage compression ($p < 0.01$)

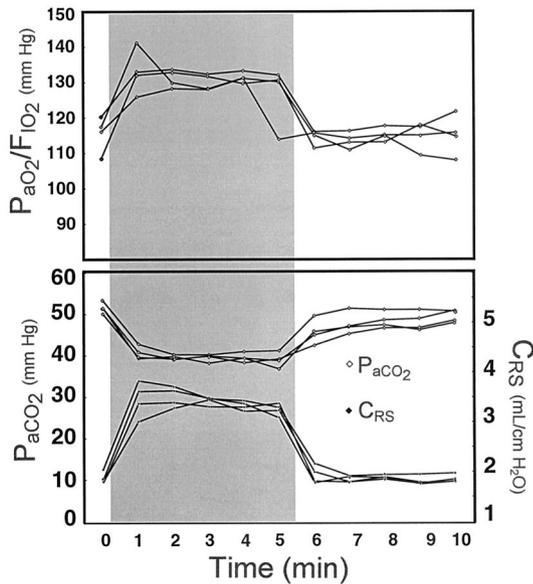


Fig. 4. Changes in the ratio of P_{aO_2} to fraction of inspired oxygen (P_{aO_2}/F_{IO_2}), P_{aCO_2} , and respiratory system compliance (C_{RS}), before, during, and after expiratory rib cage compression. The shaded column indicates the period of rib cage compression for 5 min. These data were obtained from 1 rabbit during 4 rib cage compression sessions. P_{aO_2}/F_{IO_2} was measured every 1 min for a total of 10 min.

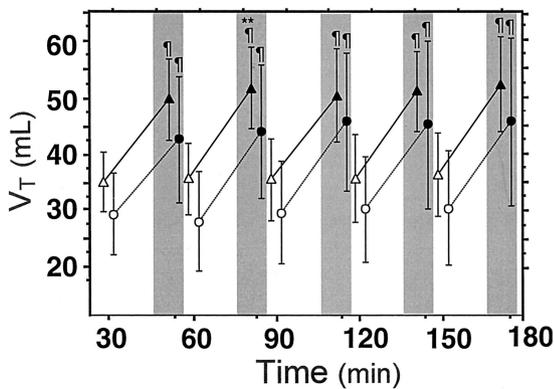


Fig. 5. Tidal volume (V_T) before (open symbols) and during (closed symbols) expiratory rib cage compression in the prone (Pr_{comp}) group (triangles) and supine (Sp_{comp}) group (circles). The shaded columns indicate periods of rib cage compression. ** = $p < 0.05$ compared with the Sp_{comp} group. ¶ = $p < 0.01$ compared with the value before rib cage compression.

(Fig. 5). There were no significant V_T differences between the supine and prone groups, except at 90 min, at which time V_T in the Pr_{comp} group was greater than that in the Sp_{comp} group.

Esophageal and Airway Pressure

During rib cage compression esophageal pressure increased during the expiration phase (Fig. 6). Airway pres-

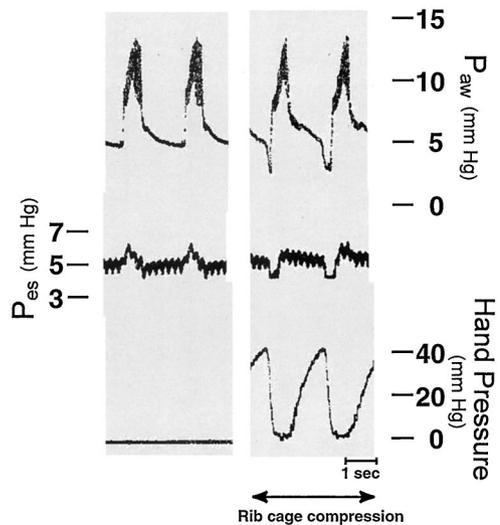


Fig. 6. Representative trace of airway pressure (P_{aw}), esophageal pressure (P_{es}), and hand pressure waveforms, before (left panel) and during (right panel) rib cage compression. Note that P_{aw} and P_{es} temporarily decreased when the hand pressure was released at end-expiration.

sure did not change remarkably during rib cage compression, although airway and esophageal pressure decreased temporarily when the hand pressure was released at end-expiration.

Discussion

The 2 major findings of this study are (1) rib cage compression did not improve oxygenation, ventilation, or C_{RS} , but (2) prone positioning improved oxygenation. P_{aO_2}/F_{IO_2} and C_{RS} increased during rib cage compression but these effects occurred only during rib cage compression. This suggests that rib cage compression did not offer sustained improvement of the ventilation of the atelectatic lung in our atelectasis model.

Effects of Rib Cage Compression on Oxygenation

The oxygenation improvement following rib cage compression was temporary. None of the animals that received rib cage compression showed sustained P_{aO_2}/F_{IO_2} improvement. This may be attributable to the fact that ABGs were obtained 5 min after the end of rib cage compression. The mechanism of temporary oxygenation improvement during rib cage compression may be the V_T increase (discussed further below).

Effects of Rib Cage Compression on Compliance

C_{RS} in the Pr_{comp} group increased significantly above baseline, but it is unlikely that that increase was due to rib

cage compression, because C_{RS} in the Pr_{comp} group was already greater than baseline at 15 and 30 min, at which time rib cage compression had not yet been started. It has been reported that in humans under general anesthesia there is not a close relationship between change in C_{RS} after an alveolar recruitment maneuver and amount of atelectasis.¹¹ In the present study, however, the collapsed lung units might not have been recruited, because rib cage compression had no effect on C_{RS} , P_{aO_2}/F_{IO_2} , or P_{aCO_2} . To recruit the collapsed lung it is necessary to remove the mucus plugs in the airways and increase transpulmonary pressure.¹² The increase in expiratory flow is important for mucus removal. To generate and increase expiratory flow in the collapsed lung, re-expansion of the alveoli may be necessary. However, the re-expansion of the collapsed lung usually requires high pressure.¹³ We used a viscous fluid to create atelectasis, and the high viscosity may increase the re-opening pressure. Moreover, in none of the rabbits was there mucus output from the ETT. Accordingly, rib cage compression might fail to remove the artificial mucus and to re-expand the collapsed lung because of the artificial mucus' relatively high viscosity.¹⁴

In contrast to our results, Goldsmith and Saunders¹⁵ reported a case in which a mother helped her son, who had a high cervical spinal cord injury, move pulmonary secretions up by lower chest compression. It appears that the presence of gas in peripheral airways enabled mucus removal by rib cage compression in that patient. Presumably, it is possible to remove mucus by rib cage compression in incomplete airway obstruction.

Effects of Rib Cage Compression on Tidal Volume

In this study V_T increased during rib cage compression. Similar findings have been reported in humans.¹⁶ There are 2 possible mechanisms of V_T increase. First, rib cage compression may decrease end-expiratory lung volume. It was reported that the rapid thoracic compression technique decreased end-expiratory lung volume during the expiratory phase in preterm infants.¹⁷ Furthermore, the ratio of chest wall compliance to lung compliance in rabbits is much higher than that in human adults,¹⁸ so it seems quite probable that rib cage compression is much more effective in decreasing end-expiratory lung volume in rabbits than in human adults. Second, the increase in elastic recoil pressure of the rib cage caused by release from rib cage compression might decrease pleural and airway pressure. In the present study, because the rabbits were ventilated in a pressure-controlled mode, it is possible that the V_T increased because of the increase in the elastic recoil pressure of the rib cage. In fact, airway and esophageal pressure decreased temporarily when the hand pressure was released at end-expiration. This finding indicates that rib cage compression caused the increase in the elastic recoil

pressure of the rib cage. However, allowing for the difference in chest wall compliance between rabbits and humans,¹⁸ it remains to be determined whether rib cage compression can cause an increase in the elastic recoil pressure of the rib cage in humans.

Model of Atelectasis

We used the method described by Kim et al⁹ for preparing the artificial mucus. In comparison with the tracheal secretions in intubated dogs,¹⁹ the elasticity of the artificial mucus we used was lower, although the viscosity was higher. Allowing for the decrease in C_{RS} after the artificial mucus infusion and the smaller airway caliber of the rabbits, it appears that the viscoelasticity of the artificial mucus in the present study was comparable to that of previous studies. The amounts of infused artificial mucus (2–3 mL) were comparable among the 4 groups. The amount of methylene blue was less than that used for therapeutic purposes.²⁰ Histologically, the alveoli were collapsed in the macroscopically atelectatic areas, although the artificial mucus was not detected. It is likely that the artificial mucus plugged the larger airways and absorption atelectasis ensued.²¹ Accordingly, this atelectasis model seemed to be appropriate to examine the effects of rib cage compression.

Effects of the Prone Position on Oxygenation

Various mechanisms for oxygenation improvement from prone positioning have been suggested: (1) a redistribution of perfusion away from previously dependent regions,⁷ (2) more uniform pleural pressure,²² (3) change in regional diaphragm motion,²³ and (4) that the prone position minimizes compression of the lungs by the heart.²⁴

In this study oxygenation improved after the animals were turned from supine to prone position. Since the artificial mucus was infused in the supine position, it is likely that lung collapse occurred mostly in dorsal lung regions. Consequently, we speculate that the oxygenation improvement was caused by improved ventilation-perfusion matching and/or the removal of secretions from dorsal lung regions after the rabbits were placed in the prone position. Additionally, we found sustained improvement in oxygenation after the rabbits were turned supine, so it is likely that the alveolar recruitment of dorsal lung regions was one of the mechanisms of oxygenation improvement. To our knowledge no previous studies have shown that prone position improves oxygenation in an induced atelectasis model.

Effects of the Prone Position on Compliance

It is unlikely that C_{RS} increased because of prone positioning, although C_{RS} in the Pr_{comp} group increased sig-

nificantly above baseline. In this study there were no significant C_{RS} differences between the prone and supine groups, but C_{RS} in the Pr group was significantly higher at 270 and 300 min than at baseline.

Studies with humans^{6,25} have found that prone position did not affect C_{RS} , although one study found the opposite.²⁶

One possible cause of lower C_{RS} in the prone position is that the movement of the ventral rib cage is impeded by lying on the bed, because ventilation depends on the ventral rib cage rather than the dorsal rib cage.²⁷ In the present study the rabbits were placed in the prone position with the thorax and the abdomen released, using positioning rolls under the upper part of the chest wall and the bilateral thorax to allow free movement of the thorax and the abdomen. A study using pigs²⁸ found no significant differences in C_{RS} between the animals lying directly on the table and those with positioning rolls to allow free movement of the abdomen. In view of the differences in chest wall mechanics among different species, further study seems to be required.

Study Limitations and Clinical Relevance

There are several limitations to the present study. First, the differences between this controlled laboratory experiment and the clinical approach used with humans may be important. Endotracheal suctioning was not employed in the present study, although it is routinely carried out in patients undergoing mechanical ventilation via ETT. Clinically, withholding endotracheal suctioning for hours is exceptional. It appears that recruiting collapsed lung requires both removing the mucus plugs and increasing transpulmonary pressure. If rib cage compression can move the mucus toward the central airway, it might be possible to then remove the mucus with endotracheal suctioning, thereby improving oxygenation and ventilation. Further study regarding the interaction between rib cage compression and the endotracheal suctioning is required.

Second, we should consider the anatomic and physiologic differences among species. We should be cautious in attempting to extrapolate the present findings to humans, since it is likely that the effects of rib cage compression on oxygenation and respiratory mechanics depend on chest wall mechanics.

Third, although it is unclear whether morphology and function of collateral ventilation differ between humans and adult rabbits, we should consider differences in morphology and function of collateral ventilation among animal species.²⁹

Fourth, it is unclear whether the method we used to measure mucus output volume was appropriate, since measurable mucus output was not obtained. These differences might be important when there is airway obstruction.

Finally, the possibility of type II error was not completely excluded in the present study.

Conclusions

Our results suggest that rib cage compression does not improve oxygenation and ventilation in mechanically ventilated atelectatic rabbits. Thus it is unlikely that rib cage compression re-expands collapsed lung. Similar to previous studies in humans and animals with acute respiratory distress syndrome or acute lung injury we found that prone positioning improved oxygenation in rabbits with induced atelectasis.

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