

Effects of Expiratory Rib Cage Compression Combined With Endotracheal Suctioning on Gas Exchange in Mechanically Ventilated Rabbits With Induced Atelectasis

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INTRODUCTION: In Japan, expiratory rib cage compression (a chest physiotherapy technique) is frequently used with mechanically ventilated patients. It has not been determined whether rib cage compression combined with endotracheal suctioning improves oxygenation, ventilation, and mucus clearance. We evaluated the effects of rib cage compression with and without endotracheal suctioning on P_{aO_2} , P_{aCO_2} , dynamic compliance of the respiratory system (C_{RS}), and mucus clearance in rabbits with induced atelectasis. **METHODS:** Anesthetized adult rabbits had an 18-gauge catheter placed into the airway, together with a tracheal tube via tracheostoma, and were mechanically ventilated. To create atelectasis, artificial mucus was infused into the airway via the catheter. Each rabbit was randomly assigned to one of 4 groups ($n = 7$ in each): (1) control, (2) received endotracheal suctioning alone, (3) received rib cage compression alone, and (4) received both rib cage compression and endotracheal suctioning. After these interventions, for 30 min, each animal was placed supine without intervention for 120 min. **RESULTS:** In the groups that received rib cage compression, oxygenation, ventilation, and C_{RS} were significantly worse than the groups that did not receive rib cage compression ($p < 0.05$). Endotracheal suctioning with and without rib cage compression did not improve oxygenation, C_{RS} , or mucus clearance. There were no significant differences in the weight of aspirated artificial mucus between the groups, with or without rib cage compression. **CONCLUSIONS:** In mechanically ventilated rabbits that had induced atelectasis, neither rib cage compression alone nor rib cage compression combined with endotracheal suctioning improved oxygenation, ventilation, C_{RS} , or mucus clearance. Alveolar and airway collapse was probably exacerbated by rib cage compression. **Key words:** atelectasis, suctioning, physical therapy, mucus, oxygenation, ventilation. [Respir Care 2004;49(8):896–901. © 2004 Daedalus Enterprises]

Introduction

A variety of chest physical therapy techniques, including expiratory rib cage compression, are widely used with mechanically ventilated patients to prevent and/or to treat atelectasis. Expiratory rib cage compression, which is well known as “squeezing” in Japan, involves manual compression

of the rib cage during expiration and compression-release at the end of the expiration, in an attempt to mobilize pulmonary secretions, to facilitate active inspiration, and to improve alveolar ventilation.¹ It is widely believed that rib cage compression effectively treats and/or prevents lung collapse and that it is safe for critically ill patients, compared to percussion or vibration,^{1,2} but there are few published studies

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regarding its effects. We reported previously that rib cage compression alone does not improve oxygenation or ventilation with mechanically ventilated rabbits that have induced atelectasis.³ Whereas endotracheal suctioning is a common procedure with intubated patients receiving chest physical therapy, endotracheal suctioning was not em-

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ployed over multiple hours of the experiment in our previous study,³ so we could not assess the effects of the combination of rib cage compression and suctioning on mucus clearance, ventilation, and oxygenation.

In the present study we hypothesized that rib cage compression combined with endotracheal suctioning would improve oxygenation and ventilation by accelerating mucus clearance in mechanically ventilated rabbits with induced atelectasis. Oxygenation, ventilation, and lung mechanics were assessed by serial measurements of gas exchange and dynamic compliance of the respiratory system (C_{RS}). Gas exchange was assessed by P_{aO_2} and P_{aCO_2} .

Methods

Animal Preparation and Measurements

The protocol was approved by our institution's animal research committee, and the care of the animals was in accordance with guidelines for ethical animal research. Twenty-eight female Japanese white rabbits (2.8 ± 0.24 kg) were used. A 24-gauge intravenous cannula was placed via an ear vein. The rabbits were anesthetized with intravenous injection of 75–150 mg of sodium pentobarbital and were infused with lactated Ringer's solution (28 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, England). To instill artificial mucus into the airways, an 18-gauge catheter was inserted into the ETT via the side port of an elbow. The tip of the catheter was advanced to 1.5 cm beyond the tip of the ETT. After the animals were paralyzed with 0.375 mg of pancuronium, volume-controlled mechanical ventilation was started (using a Servo 900B, Siemens-Elema, Solna, Sweden) connected to a pressure-relief valve (external pressure limiter #6–600, LifeCare, Lafayette, Colorado). Ventilator settings were: fraction of inspired oxygen (F_{IO_2}) 1.0, respiratory rate 30 breath/min, inspiratory time 33% of the breathing cycle. To avoid the effects of positive end-expiratory pressure (PEEP) on expiratory flow during rib cage compression, PEEP was not applied. Tidal volume was set to achieve P_{aCO_2} of 40 ± 5 mm Hg and was maintained until 5 min after mucus infusion. Anesthesia and muscle paralysis were maintained by a continuous infusion of sodium pentobarbital (20 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. Expiratory tidal volume (measured with an NVM-1, Bear Medical Systems, Riverside, California), end-tidal carbon dioxide (measured with a Capnomac Ultima, Datex Instrumentarium, Helsinki, Finland), arterial blood pressure, and heart rate

were continuously measured. Rectal temperature was monitored, and an electric heat blanket was used to maintain rectal temperature at 38–39°C.

Model of Atelectasis

To create atelectasis, artificial mucus was infused through the catheter into the airways, as we previously described.³ The artificial mucus was made of 1.6% (weight/volume) polyethylene oxide powder (Polyox [average molecular weight 5,000,000 atomic mass units], 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.⁴ The loss modulus (G'') of the artificial mucus was 45.2 dyn/cm², and the storage modulus (G') was 28.8 dyn/cm². The mucus infusion was continued at a rate of 0.2 mL/min for 10 min. After a stabilization period of 5 min, arterial blood gases, expiratory tidal volume, and peak inspiratory pressure were recorded, and those values were defined as baseline. All animals were then switched to pressure-controlled ventilation using a pressure-relief valve, an F_{IO_2} of 1.0, peak inspiratory pressure of 18 cm H₂O, inspiratory flow of 15 L/min, and zero PEEP.

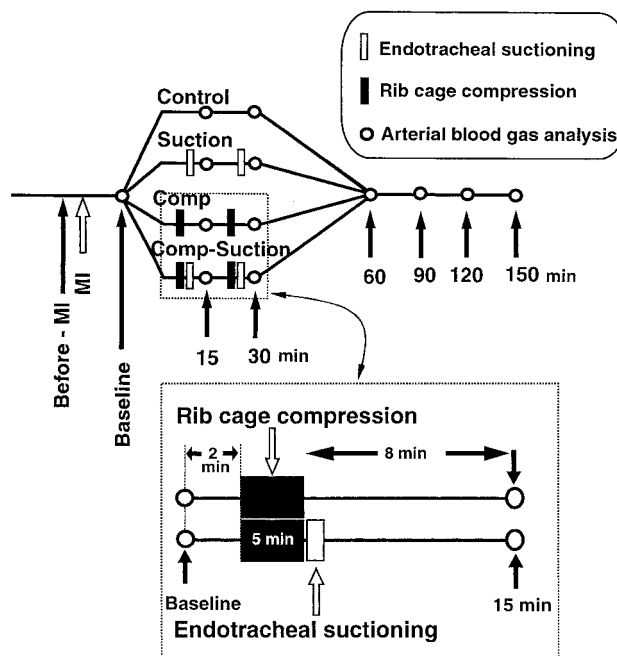


Fig. 1. Experimental protocol. Open circles represent the points at which arterial blood was sampled for gas analysis. Comp = compression. MI = mucus infusion.

Protocol

Figure 1 illustrates the experimental protocol. After baseline measurement all rabbits were randomly assigned to one of 4 groups:

1. The control group ($n = 7$) received no intervention after mucus infusion.
2. The suction group ($n = 7$) received endotracheal suctioning but no rib cage compression.
3. The compression group ($n = 7$) received rib cage compression but no endotracheal suctioning.

The compression-suction group ($n = 7$) received both rib cage compression and endotracheal suctioning.

Arterial blood gas measurements were performed 15, 30, 60, 90, 120, and 150 min after baseline. In the compression and compression-suction groups rib cage compression was applied to every breath for 5 min and was performed at 2 and 17 min after baseline. Each rabbit received 2 rib cage compression sessions. In the suction and compression-suction groups endotracheal suctioning was performed at 7 and 22 min after baseline (in the compression-suction groups, endotracheal suctioning was performed immediately after rib cage compression).

After that 30 min intervention period all the rabbits were observed for 120 min and then sacrificed with intravenous injection of 2 mL potassium chloride (2 mol/L) solution.

Expiratory Rib Cage Compression

Manual bilateral expiratory rib cage compression was performed by a single operator (author TU), who attempted to use consistent technique, applying the same force with each animal. The rib cage compression method was based on the standard technique for clinical use.¹ The operator gradually applies bilateral squeeze to the lower rib cage during the expiration. With both spontaneously breathing and mechanically ventilated subjects compression is stopped at the end of expiration to allow free inspiration. Special care was taken to ensure that compression was applied only during expiration.

Endotracheal Suctioning

An experienced intensive care nurse (author TU) performed endotracheal suctioning according to the guidelines of the American Association for Respiratory Care.⁵ Neither hyperventilation nor hyperinflation were performed, before or after endotracheal suctioning. A 6 French, 46-cm suction catheter was connected to an electric vacuum device (MMC-1500W, Sanko, Ohmiya, Japan) set at -255 cm H₂O. Before and after suctioning the catheter was weighed (electric balance ER182A, A&D Company, Tokyo, Japan), and the weight of the suctioned mucus was

calculated by subtracting the weight of the catheter before suctioning from that after suctioning.

Proportion of Atelectatic Lung

With 24 of the rabbits (6 from each group) we calculated what percent of the total lung surface was atelectatic.⁶ Immediately after the rabbit was sacrificed, the ETT was clamped at zero PEEP, and then the chest was opened. The total lung area and atelectatic area were traced on paper.

Statistical Analysis

All values are reported as mean \pm SD unless otherwise specified. Group differences and changes from baseline in physiologic variables and the proportion of atelectatic area were analyzed with repeated-measures 1-way analysis of variance. Statistically significant differences were followed up with post hoc analysis (Scheffé's multiple comparison test). Differences between the suction and compression-suction groups and between the 2 body positions with regard to the weight of aspirated mucus were compared with the unpaired t test. Differences were considered statistically significant when $p < 0.05$.

Results

Figure 2 shows the results for P_{aO_2} , P_{aCO_2} , and C_{RS} . There were no significant P_{aO_2} differences between the groups before mucus infusion or at baseline. After mucus infusion P_{aO_2} decreased in all rabbits, by a mean of 312.3 ± 101.9 mm Hg.

In the control and suction groups P_{aO_2} gradually increased after baseline, whereas P_{aO_2} markedly decreased in the compression and compression-suction groups. Throughout the experiment, there was no significant P_{aO_2} difference between the control and suction groups nor between the compression and compression-suction groups.

There were no significant P_{aCO_2} differences between the groups before mucus infusion nor at baseline. At baseline P_{aCO_2} had increased in all rabbits, by a mean of 7.8 ± 5.0 mm Hg. After the baseline period the control and suction groups' P_{aCO_2} did not change (range 60–80 mm Hg throughout the experiment [ie, post-baseline period]), whereas P_{aCO_2} gradually increased in the compression and compression-suction groups. There were no significant P_{aCO_2} differences between the control and suction groups, throughout the experiment. And there were no significant P_{aCO_2} differences between the compression and compression-suction groups.

There were no significant differences in C_{RS} between the groups before mucus infusion nor at baseline. At base-

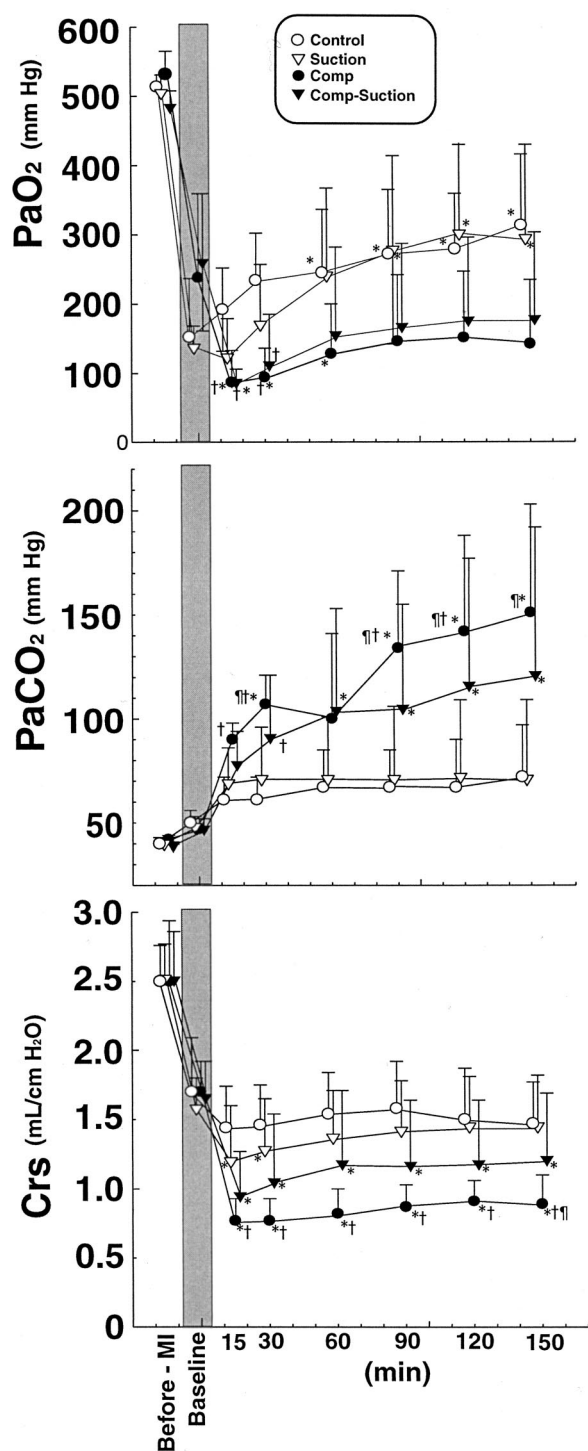


Fig. 2. Mean \pm SD P_{aO_2} , P_{aCO_2} , and dynamic compliance of the respiratory system (C_{RS}) among 4 study groups: control, suctioning, compression (comp), and compression plus suctioning (comp-suction). The shaded area represents the baseline period (immediately following infusion of artificial mucus [MI]). * = $p < 0.05$ versus baseline. † = $p < 0.05$ versus control. ‡ = $p < 0.05$ versus suction.

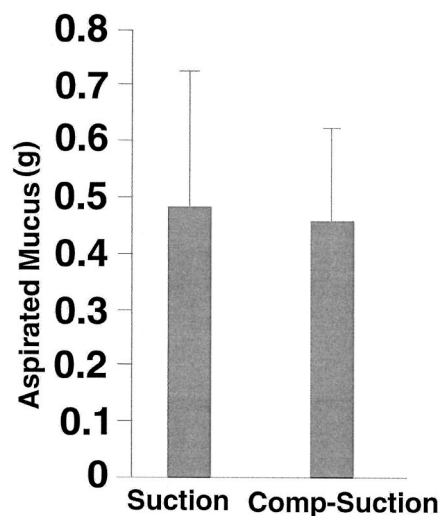


Fig. 3. Mean \pm SD weight of aspirated mucus in the suction group (suctioning only) and the compression-suction group (suctioning plus rib cage compression).

line C_{RS} had decreased in all the rabbits, by a mean of 0.85 ± 0.32 mL/cm H_2O .

In the compression and compression-suction groups C_{RS} significantly decreased after baseline. In the suction group C_{RS} was significantly lower than baseline at 15 and 30 min. However, the C_{RS} of the compression-suction group was significantly lower than the C_{RS} of the control group after baseline. Throughout the experiment there were no significant C_{RS} differences between the control and the suction groups, nor between the compression group and the compression-suction group.

Figure 3 shows the aspirated mucus measurements. There were no significant differences between the suction and compression-suction groups.

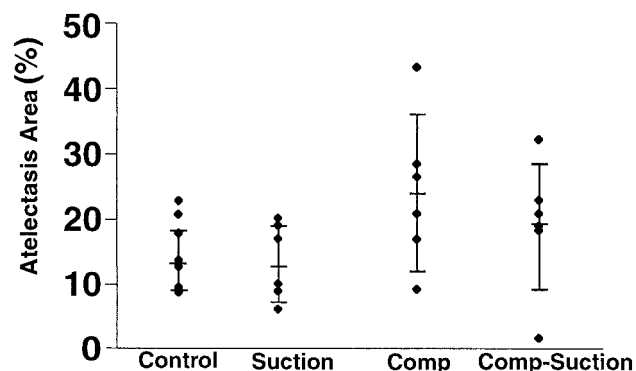


Fig. 4. Percent of total lung surface that was atelectatic ($n = 6$ in each study group). On the vertical bars the middle tick marks represent the means and the high and low tick marks indicate the ranges of the standard deviations.

Fig. 4 shows the values for percent of atelectatic area. There were no significant differences between the 4 groups ($p = 0.12$).

Discussion

The present study demonstrates that rib cage compression of mechanically ventilated rabbits with induced atelectasis had deleterious effects on gas exchange and lung mechanics and did not benefit mucus clearance. The results are contrary to our hypothesis.

Effects of Rib Cage Compression on Oxygenation and Ventilation

P_{aO_2} had decreased at 15 min in the compression and compression-suction groups, but not in the animals that did not receive rib cage compression; this indicates that rib cage compression was detrimental to oxygenation. The rabbits underwent rib cage compression had greater (though not significantly greater) atelectatic area, so we believe that rib cage compression probably exacerbated lung collapse. The finding that post-baseline C_{RS} decreased in the compression groups accords with our hypothesis. A possible cause of the greater atelectatic area with rib cage compression is a decrease in end-expiratory lung volume. Opie and Spalding⁷ showed that rib cage compression increased esophageal pressure during expiration. That increase in pleural pressure may decrease transpulmonary pressure, thereby exacerbating alveolar collapse. Also it is possible that the artificial mucus we used made airway closure more likely than does real mucus, but we assumed that the artificial mucus was similar to real mucus.³ When an airway closes, high airway pressure is needed to reopen it because of the adhesive force of the airway-lining fluid.⁸

In our previous study³ rib cage compression did not significantly change oxygenation in rabbits with induced atelectasis. It is likely that end-expiratory lung volume during rib cage compression was inversely correlated with collapse of airway and alveoli. In the present study PEEP was not applied, whereas 5 cm H_2O of PEEP was applied in our previous study.³ Panitch et al⁹ reported that continuous positive airway pressure prevented airway collapse during rapid thoracic compression techniques, which is one of the pulmonary function tests used with infants suffering acquired tracheobronchomalacia. Similarly, in our previous study it appeared that PEEP prevented airway and alveolar collapse.³ Furthermore, it is also possible that with zero PEEP rib cage compression caused more homogeneous and/or distal distribution of the artificial mucus, thereby causing greater atelectasis.

Effects of Endotracheal Suctioning on Oxygenation and Ventilation

Endotracheal suctioning is a routine procedure with patients who are intubated to maintain unobstructed airway. However, studies suggest that endotracheal suctioning can cause complications such as hypoxemia,¹⁰ cardiac arrhythmia,¹¹ intracranial pressure elevation,¹² and others.¹³ In the present study there were post-baseline C_{RS} decreases not only in the rabbits that received rib cage compression but also in those that received endotracheal suctioning without rib cage compression, although its duration was shorter. That finding indicates that endotracheal suctioning alone can decrease C_{RS} .

In the present study, changes in airway resistance were not evaluated, for methodological reasons. Guglielminotti et al reported that endotracheal suctioning evoked a transient bronchoconstriction response but thereafter did not reduce respiratory resistances below pre-suctioning values in mechanically ventilated patients.¹⁴ Another study showed that endotracheal suctioning could induce bronchoconstriction and atelectasis in ventilated sheep.¹⁵ Therefore in the present study suctioning might have transiently increased airway resistance and decreased lung compliance. Moreover, the failure to improve mucus clearance by endotracheal suctioning might aggravate pulmonary mechanics. In the present study we did not apply hyperinflation after endotracheal suctioning. Lu et al¹⁵ reported that, in mechanically ventilated sheep, suctioning-induced atelectasis and bronchoconstriction could be lessened by conducting a hyperoxygenation maneuver before and a recruitment maneuver after suctioning. A post-suctioning recruitment maneuver might recruit alveoli that are collapsed by expiratory rib cage compression.

Mucus Output

In the present study rib cage compression did not improve mucus clearance. Forced expiration, which may increase the expiratory flow rate, is likely to propel airway secretions. It has been reported that rib cage compression increases peak expiratory flow in intubated patients.^{16,17} In the present study, however, there was no difference in mucus collected with and without rib cage compression. In the present study it appears that rib cage compression did not increase expiratory flow rate, probably because of alveolar collapse caused by rib cage compression. Considering the changes in physiologic variables, further lung collapse ensued immediately after the application of rib cage compression. It is unlikely that rib cage compression could increase expiratory flow enough to remove airway mucus, because the distal alveoli are collapsed by the compression maneuver. Another possible explanation is that rib cage compression causes airway closure (in addition to

alveolar collapse).¹⁸ We think that airway closure is likely in the absence of applied PEEP.

Limitations

The present study suffered several limitations. First, there are anatomic and physiologic differences between rabbits and humans; so we must be cautious in attempting to extrapolate our findings to humans. The effects of rib cage compression on oxygenation and respiratory mechanics probably depend on chest wall mechanics. However, alveolar collapse by rib cage compression may occur in human infants, because the ratio of chest wall to lung compliance is similar in the human infant and the rabbit.¹⁹ Furthermore, because they have highly unstable chests, premature newborns and infants cannot distend their lung parenchyma as much as older humans can,¹⁸ so maintaining lung volume is more difficult.

Second, among animal species there seem to be considerable differences in morphology and function of collateral ventilation,²⁰ and it not yet known how those differences affect oxygenation and/or ventilation when there is substantial airway obstruction.

Third, the PEEP level may influence the effect of rib cage compression. We assumed that using no PEEP would minimize expiratory resistance and maximize expiratory flow and thus enhance mucus clearance, but that assumption might be wrong. Hence, the interaction between PEEP level and rib cage compression combined with endotracheal suctioning needs further research.

Conclusions

In intubated, atelectatic rabbits, rib cage compression without PEEP, even when combined with endotracheal suctioning, impairs oxygenation and ventilation and does not improve mucus clearance.

ACKNOWLEDGMENTS

We thank Ms Yumi Isaka for expert technical assistance.

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