

flation. Specific emphasis on allowing an adequate expiratory time, as dictated by the resistance and compliance of the lung, is critical to properly manage intrinsic positive end-expiratory pressure and dynamic hyperinflation.

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The authors reply:

We agree.

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Expiratory Rib-Cage Compression, Airway Suctioning, and Atelectasis

Unoki et al¹ recently reported, in *RESPIRATORY CARE*, their evaluation of the effects of rib-cage compression with and without airway suctioning on P_{aO_2} , P_{aCO_2} , respiratory-system compliance, and mucus clearance in 28 rabbits with atelectasis induced by tracheal infusion of artificial mucus. The

rabbits were intubated, paralyzed, mechanically ventilated, tracheostomized, and randomized to one of 4 groups: (1) control, (2) suctioning only, (3) rib-cage compression only, and (4) rib-cage compression plus suctioning.

In the group that received rib-cage compression, P_{aO_2} , P_{aCO_2} , and respiratory-system compliance were significantly worse than the groups that did not receive rib-cage compressions. Endotracheal suctioning with and without rib-cage compression did not improve P_{aO_2} , P_{aCO_2} , respiratory-system compliance, or mucus clearance. There were no significant differences in the weight of aspirated mucus between the groups.

Unoki et al are to be commended for evaluating the effects of a commonly used chest physical therapy procedure with an animal model. However, their conclusions are seriously flawed, because of problems with the study design and inappropriate interpretation of the study results.

All the animals were anesthetized and paralyzed during the study, and were mechanically ventilated initially in a volume-controlled mode. Following the induced atelectasis, the animals were then switched over to a pressure-controlled mode, for reasons unexplained.

First, the pharmacologic paralysis would have ablated the ability to stimulate a cough reflex during endotracheal airway suctioning, which obviously would have reduced the effectiveness of airway suctioning to generate expiratory flow and assist mucus clearance. The use of chest wall compressions to improve expiratory flow and move the airway secretions from the peripheral airways to the more central airways is evidenced by the significant reductions in dynamic lung/thorax compliance in the groups that received compressions. As the rabbits were ventilated in a pressure-controlled mode, the significant reductions in dynamic lung/thorax compliance obviously explains the resultant deterioration in oxygenation and hypercarbia. With the rabbits paralyzed, the central airway secretions could not be cleared, because of the absence of cough reflex, which explains the deterioration in ventilation variables. This must be further explored and underlies their seeming lack of understanding of the effects of these chest physical therapy procedures on these outcome measures.

Recent work by Main et al² with pediatric ventilated patients supports this reason-

ing. Main et al demonstrated that these chest physical therapy procedures can improve airways resistance, which was not measured by Unoki et al. As Unoki et al did not monitor expiratory flow rate or airway resistance,¹ we are unsure as to the effectiveness of the interventions in their animal model.

In summary then, Unoki et al used a pressure-cycled mode, so the significant reductions in dynamic airway compliance support the deduction that the chest wall compressions in fact enhanced the movement of airway secretions to more central airways, resulting in the deterioration in ventilation and oxygenation.

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The authors reply:

We thank Dr Ntoumenopoulos for his interest in our article¹ and stimulating comments. We would like to address the issues raised by his insights. First, we emphasize that our study was carried out under experimental conditions, using an induced-atelectasis model, so we should be cautious about extrapolating the results to humans, as we mentioned in our article. Second, rib-cage compression has been advocated as an effective technique to treat atelectasis in mechanically ventilated patients, despite little clinical or experimental evidence. Therefore, to elucidate the effects of rib-cage compression under well-controlled and consistent circumstances, we chose an animal study.

Dr Ntoumenopoulos wondered why we chose pressure-controlled ventilation following the induced atelectasis. There are