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Perioperative Pulmonary Atelectasis: Comment

To the Editor:

We read the review articles by Zeng *et al.*¹ and Lagier *et al.*² with great interest, with their emphasis that atelectasis caused by peripheral airway closure is a common complication of mechanical positive pressure ventilation. This phenomenon was first detected during anesthesia by Hedenstierna *et al.*^{3,4} and was reviewed by Milic-Emili *et al.*⁵ It is well known that negative pleural pressure resolves peripheral airway closure and subsequent atelectasis. This can be achieved by synchronizing ventilation with the patient's efforts or by stimulating the phrenic nerve. However, a far simpler solution to avoid or treat atelectasis is to use negative pressure ventilation.

Before the polio pandemic in the 1950s, patients with atelectasis were treated with negative pressure ventilation in the iron lung. Its use was, however, abandoned for practical nursing reasons during and after the polio pandemic. After the introduction of positive pressure ventilation, the fight against ventilator-induced atelectasis started and is still going on.

A recent publication by Klassen *et al.*⁶ clearly shows the impact of peripheral airway closure in the context of positive and negative pressure ventilation. In an excised porcine lung, the driving pressure during positive pressure ventilation needed to be twice as large as during negative pressure ventilation to reach the same tidal volume. Moreover, the leakage from deliberate damage to the visceral pleura was five times larger during negative pressure ventilation. This demonstrates that positive pressure ventilation caused peripheral airway

closure that reduced ventilation of the peripheral parts of the lung, while negative pressure ventilation did not.

It has also been demonstrated in ventilated and perfused isolated rat⁷ and human⁸ lungs that negative pressure ventilation provides a superior method of preserving these organs for transplantation as compared to positive pressure ventilation.

Recent interest in negative pressure ventilation led to the Exovent⁹ project (www.Exovent.org; accessed January 29, 2022), in which a lightweight torso-only device was developed. This device can deliver continuous negative extrathoracic pressure to increase the functional residual capacity. It can also provide full negative pressure ventilation, with the addition of negative end-expiratory extrathoracic pressure, the analog of positive end-expiratory pressure in positive pressure ventilation. It is to be expected that later versions will be leaner than the first prototype described by Coulthard.⁹ It remains unknown whether using negative pressure ventilatory support to patients with diseased lungs (such as those seen with COVID-19 pneumonia) may influence lung damage from atelectasis, but studies on animals with surfactant-depleted lungs by Grasso *et al.*¹⁰ suggest this may be a possibility. Clinical trials to investigate whether this also applies to patients are needed.

Competing Interests

The authors declare no competing interests.

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Perioperative Pulmonary Atelectasis: Reply

In Reply:

We sincerely thank Jan van Egmond *et al.*¹ for their interest in our review articles^{2,3} and relevant comments. We entirely agree that atelectasis is a prominent clinical issue and that there is great need for intervention methods to mitigate it. We also concur that negative pressure ventilation can increase the end-expiratory lung volume as observed with delivery of continuous negative extrathoracic pressure in healthy adults⁴ and anesthetized, surfactant-depleted rabbits.⁵ Continuous negative abdominal pressure has also been reported to selectively recruit dorsal atelectatic lung and correspondingly increase the volume of ventilated lung in animal models under general anesthesia.^{6,7} Accordingly, negative pleural pressure could resolve peripheral airway closure and atelectasis *if* that closure is secondary to insufficient transpulmonary pressures.

Of note, our reviews are focused on perioperative pulmonary atelectasis. As detailed in the reviews, abdominal and thoracic surgery are particularly relevant in that context as they are related to mechanisms promoting atelectasis and are associated with significant incidence of atelectasis and postoperative pulmonary complications.^{2,3} Unfortunately, in such surgical conditions, the mentioned negative pressure ventilatory support system is not applicable intraoperatively and may be challenging postoperatively due to concerns about sterility and access to the surgical site and wound. Finally, it is unclear whether application of negative pressure ventilation during the postoperative period could interfere with wound healing. Future studies on interventions to mitigate perioperative atelectasis should consider the mechanisms producing the process and the clinical context for optimal design and implementation.

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Competing Interests

The authors declare no competing interests.

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