MECHANICAL ventilation may cause harm to the lung, at least if the lung is already hurt as is the case in intensive care. Can it be harmful also when ventilating the "lung-healthy" patient during anesthesia? Probably not during anesthesia alone, as suggested by animal experiments on prolonged mechanical ventilation,1,2 but inflammatory events,3 such as those associated with surgery, may trigger adverse effects. The comprehensive review on intraoperative protective mechanical ventilation by Güldner et al.4 in the current issue of Anesthesiology begins with a summary of potential mechanisms of lung damage in the anesthetized, mechanically ventilated, surgical patient. The list is long. However, increased knowledge about how the lung is affected by mechanical ventilation may help us improve methods of ventilator support used during anesthesia and intensive care. The advance in knowledge has followed two main tracks: studies that identify mechanisms of impairment and large multicenter trials that test these new findings in the clinical environment. So, what have we learned from these studies? A consistent finding in general anesthesia is the loss of muscle tone with a subsequent decrease in resting lung volume, that is, functional residual capacity. This promotes airway closure. Gas trapped behind a continuously closed airway will be absorbed and eventually result in atelectasis.5 Atelectasis may promote bacterial translocation and trigger inflammation.6,7 Large tidal volume and high airway pressure increase lung stress and strain with a potential impact on inflammation, at least in the intensive care setting.8

The current review focuses on atelectasis and how to prevent it. Use of recruitment maneuvers to reopen collapsed lung and the application of positive end-expiratory pressure (PEEP) to prevent collapse have been analyzed by comparing different single and multicenter studies. In addition, small and large tidal volumes have been compared, and several studies have investigated all three interventions (recruitment, PEEP, and small tidal volume) to design a "protective ventilation" strategy. Findings have not been consistent. A study by Futier et al.9 used all three techniques for protective ventilation although the title of their article referred solely to small tidal volume. Goldenberg et al.10 made a comment on this limitation, and Futier et al. wrote in another article a year later that PEEP should be used from induction of anesthesia until emergence.11 A still larger study compared PEEP less than 2 cm H\textsubscript{2}O in one group with another group receiving PEEP of 12 cm H\textsubscript{2}O. Both groups were ventilated with the same tidal volume, 8 ml/kg body weight.12 No benefit was seen in the group receiving high PEEP and postoperative pulmonary complications were equally high (approximately 40%). High PEEP caused some circulatory impairment and required more fluid and vasoactive drugs than low PEEP. A recent meta-analysis examined 3,365 abdominal or thoracic surgery patients. Postoperative lung complications were fewer in those who had received a small tidal volume and a high PEEP.13 So, how can we reconcile these different findings? The authors of the current review conclude that a low tidal volume (6 to 8 ml/kg) no or low PEEP (≤2 cm H\textsubscript{2}O) and intermittent recruitment maneuvers should be the basic settings. This suggestion deserves not only attention but also a critical analysis.

**Image:** J. P. Rathmell.

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First, the focus of most of the larger studies has been to keep the lung open during anesthesia. A recruitment maneuver may be performed immediately before extubation of the trachea, but whether there is a benefit persisting into the postoperative period depends on the inspired oxygen concentration. One may ask how important it is to maintain an open lung during one or few hours of anesthesia if atelectasis appears in the postoperative period and lasts for several hours or even days.

Second, one PEEP may not fit all. PEEP that is too high may compromise circulation, whereas too little PEEP may not prevent collapse. A slender patient needs less PEEP, whereas an obese patient may need more to keep the lung open. An individualized PEEP, based, for example, on the body mass index, may be a better approach. Whether postoperative lung complications are decreased by an individualized PEEP remains to be shown.

Third, there is no discussion regarding the inspired oxygen concentration in any of the larger multicenter studies. An alveolus full of oxygen behind a closed airway, for example, after induction of anesthesia or after a recruitment maneuver, will not stay open for more than 4 to 5 min because of the rapid absorption of the oxygen into the capillary blood. If the same recruitment maneuver is performed with lower oxygen concentration (e.g., 40%) in nitrogen, the alveolus will stay open for half an hour or longer, nitrogen being slowly absorbed and acting as a scaffold. Increased inspired oxygen to treat hypoxemia during anesthesia, as suggested by Gündner et al., has immediate effect on arterial oxygenation but may promote atelectasis formation that eventually impairs oxygenation. Simultaneous use of PEEP should prevent lung collapse. If the patient is "postoxygenated," possibly together with airway suctioning minutes before extubation of the trachea, atelectasis is most likely produced. A patient with no atelectasis during anesthesia may then be delivered to the postoperative ward with collapsed lungs.

In summary, the review by Gündner et al. is a careful analysis of presently used strategies for keeping the lung open during anesthesia and whether low or high tidal volume is important. The authors conclude that a small tidal volume is of importance, whereas PEEP is not. Different opinions may persist, one reason being that focus has been on the lung during anesthesia, whereas emergence from anesthesia to wakefulness may be of even greater importance. The inspired oxygen concentration should also be considered. How much is needed for safety reasons and does a high oxygen concentration prevent wound infection? Should high oxygen be provided together with PEEP to stabilize the lung and prevent collapse? And, does lung collapse really matter if sufficient oxygenation is achieved? The review by Gündner et al. gives stimulus to further studies in this area.

Competing Interests
The author is not supported by, nor maintains any financial interest in, any commercial activity that may be associated with the topic of this article.

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References
EDITORIAL VIEWS


ANESTHESIOLOGY REFLECTIONS FROM THE WOOD LIBRARY-MUSEUM

Unlucky Year Thirteen for Needham’s Odd Fellows Building: Did Leach’s Ether Contribute to the Conflagration?

A signer of the Declaration of Independence who was elected the Governor of Massachusetts, Elbridge Gerry was dubiously memorialized by the coining of “gerrymandering,” a word combining his surname with the word “salamander,” for the amphibian-shaped redrawing of geographic boundaries for political advantage. Also named after Governor Gerry, dentist Elbridge Gerry Leach, D.D.S. (1814–1898), taught at Harvard Dental School (1868–1870) before establishing his weekend dental office in Needham, Massachusetts during non-summer months. Dr. Leach offered ether, dental extraction and a “new set” of teeth for $15 (about $360 in today’s U.S. dollars). The commercial shelter for Leach’s Needham office, the Odd Fellows Building was built in 1874 but burned to the ground in 1887. Consequently, Leach’s advertising card (above) could have been distributed during a 13-year window of time. Both flammable and explosive, the ether stored in Leach’s office could easily have caused or accelerated the total destruction of Needham’s Odd Fellows Building…. (Copyright © the American Society of Anesthesiologists, Inc.)

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