Pre-emergence Oxygenation and Postoperative Atelectasis

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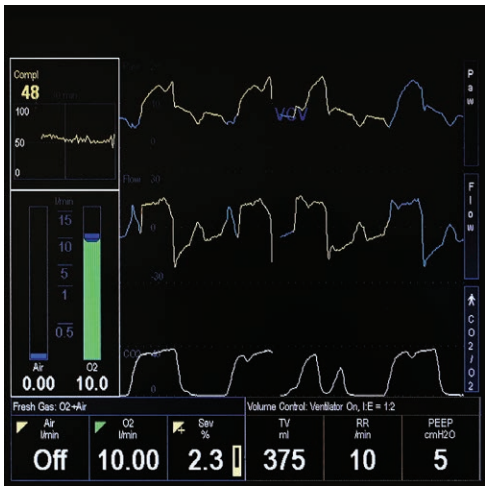
General anesthesia profoundly affects pulmonary function with a decrease in resting lung volume due to loss of muscle tone.[1] Functional residual capacity decreases irrespective of whether ventilation is controlled or spontaneous, whether anesthesia is inhaled or intravenous (with the exception of ketamine), or whether muscle relaxants are administered.[1] As functional residual capacity falls below closing capacity, airway closure and atelectasis occurs, especially in dependent lung regions. More than 20 yr ago, computed tomography scanning in humans demonstrated atelectasis formation with induction of general anesthesia.[2] In this issue of Anesthesiology, Östberg *et al.*[3] utilized computed tomography scanning to investigate whether removal of positive end-expiratory pressure (PEEP) before pre-emergence oxygenation would reduce postoperative atelectasis in humans. The study by Östberg *et al.* [3] is important because it investigates the effects of anesthesia, PEEP, and pre-emergence oxygenation on normal human respiratory physiology, without the influence of patient disease.



"[A]dministration of 100% oxygen to confer patient safety during emergence is unlikely to increase atelectasis if intraoperative PEEP is used."

Most previous research has focused upon changes in pulmonary function and development of atelectasis with induction of general anesthesia. The atelectasis occurs in dependent lung areas near the diaphragm in 90% of patients,[4] generally affecting 3 to 4% of total lung area, but as high as 15 to 30% in some patients.[1] As the amount of lung that is collapsed is greater than the amount of atelectasis visualized on computed tomography scanning, 15 to 20% of the lung is collapsed during general anesthesia.[1] Atelectasis after thoracic surgery and cardiopulmonary bypass is larger.[5] Atelectasis may cause hypoxemia due to ventilation–perfusion mismatch and increased pulmonary shunting.[2] More concerning, atelectasis persists in the postoperative period, especially after major surgery, and may contribute to postoperative pulmonary complications.[1,6] Reduction of atelectasis decreased bacterial growth and translocation in experimental pneumonia, suggesting a role of atelectasis in postoperative pneumonia.[7]

The mechanisms behind general anesthesia-induced atelectasis include compression of lung tissue from changes in the shape of the diaphragm and chest wall with increases in intraabdominal pressure; airway closure due to a decrease in functional residual capacity below closing capacity; and rapid absorption of oxygen from alveoli in areas of airway closure.[6,8] Absorption of alveolar gas accelerates alveolar collapse. Nitrogen is poorly soluble in plasma and is slowly absorbed in areas of closed airways. In contrast, oxygen is readily absorbed from alveoli in these regions. High levels of inspired oxygen used in preoxygenation prior to induction of anesthesia are instrumental in the development of absorption atelectasis. Atelectasis occurred in all patients preoxygenated with 100% oxygen and was nearly absent in patients preoxygenated with 60% oxygen.[9,10] Use of intraoperative recruitment maneuvers[11] and PEEP[12] reduce atelectasis. PEEP increases end-expiratory volume and reduces airway closure.

Although most of past research has focused on the use of high concentrations of oxygen during induction and maintenance of anesthesia, anesthesiologists also routinely administer high concentrations of oxygen during emergence. Pre-emergence oxygenation is used to increase patient safety in the advent of postemergence airway obstruction, laryngospasm, or inadequate respiratory effort. However, absorption atelectasis returns rapidly when high concentrations of oxygen are resumed.[1]

Östberg *et al.*[3] investigated whether removal of PEEP before pre-emergence oxygenation would reduce postoperative atelectasis formation in a physiologic study of thirty

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consenting healthy patients undergoing nonabdominal ambulatory surgery under general anesthesia.[3] Continuation of PEEP during pre-emergence oxygenation would be expected to increase F_{IO_2} in dependent alveoli kept open by PEEP, creating more regions for postoperative absorption atelectasis. In contrast, withdrawing PEEP because of pre-emergence oxygenation would theoretically prevent 100% oxygen from getting to areas of closed airways, thereby reducing postoperative absorption atelectasis. Patients were anesthetized with propofol, remifentanil, and rocuronium to facilitate endotracheal intubation. Mechanical ventilation was maintained with F_{IO_2} 0.35, tidal volume of 7 ml/kg ideal body weight, and PEEP 7 or 9 cm H_2O (the higher PEEP level was used if the subject's body mass index was above or equal to 25). A computed tomography scan was performed at the end of surgery while the subject was still anesthetized. Subjects were randomly allocated to either maintained PEEP or zero PEEP during pre-emergence oxygenation and the computed tomography scan repeated 30 min after awakening and tracheal extubation. The primary outcome for the study was a change in the amount of atelectasis. Oxygenation was also measured.

The study found that the amount of atelectasis in these healthy patients under general anesthesia was small and increased slightly in both groups after emergence and extubation.[3] Postoperative atelectasis for all subjects was a median of 5.2 cm^2 (95% CI 4.3 to 5.7 cm^2) representing 2.5% of total lung area (95% CI, 2.0 to 3.0%). Postoperative oxygenation was unchanged compared to the awake state.[3] Thus, use of pre-emergence oxygenation did not substantially increase postoperative atelectasis formation in healthy patients undergoing ambulatory nonabdominal surgery, regardless of whether PEEP was continued or discontinued. An earlier study in healthy anesthetized patients without use of intraoperative PEEP found larger areas of postoperative atelectasis (mean 8.3%) after pre-emergence oxygenation.[13] It is likely that use of intraoperative PEEP maintained adequate airway opening to avoid airway closure during emergence.

Östberg *et al.*'s[3] findings reassure anesthesiologists caring for the many healthy, nonobese patients undergoing nonabdominal ambulatory surgery. In this healthy patient group, administration of 100% oxygen to confer patient safety during emergence is unlikely to increase atelectasis if intraoperative PEEP is used. Individual optimization of PEEP during general anesthesia for abdominal surgery may better reduce postoperative atelectasis than an arbitrary level of PEEP.[14] The logical next step is for the investigators to perform a similar study in patients at high-risk for postoperative respiratory complications. These results will inform optimal anesthesia care for complex high-risk patients.

As the authors acknowledge, the study results do not translate to patients undergoing intraabdominal surgery or patients who have preexisting pulmonary disease, smoking histories, advanced age, or morbid obesity. This population of patients is at much higher risk of postoperative pulmonary complications.[6] Administration of 100% oxygen with PEEP before emergence and tracheal extubation in the high group may be associated with more marked postoperative atelectasis, than if PEEP had first been discontinued. Additionally, some subjects in the study by Östberg *et al.*[3] had larger areas of atelectasis under anesthesia and after emergence in the absence of obvious differences in demographic, or surgical factors. Seven out of 30 patients across both groups were "outliers" with larger areas of atelectasis without obvious associated factors.[3] This finding emphasizes that understanding of why some patients are more likely to develop atelectasis during and after general anesthesia is incomplete and why human physiologic studies like the one by Östberg *et al.*[3] are so very important.

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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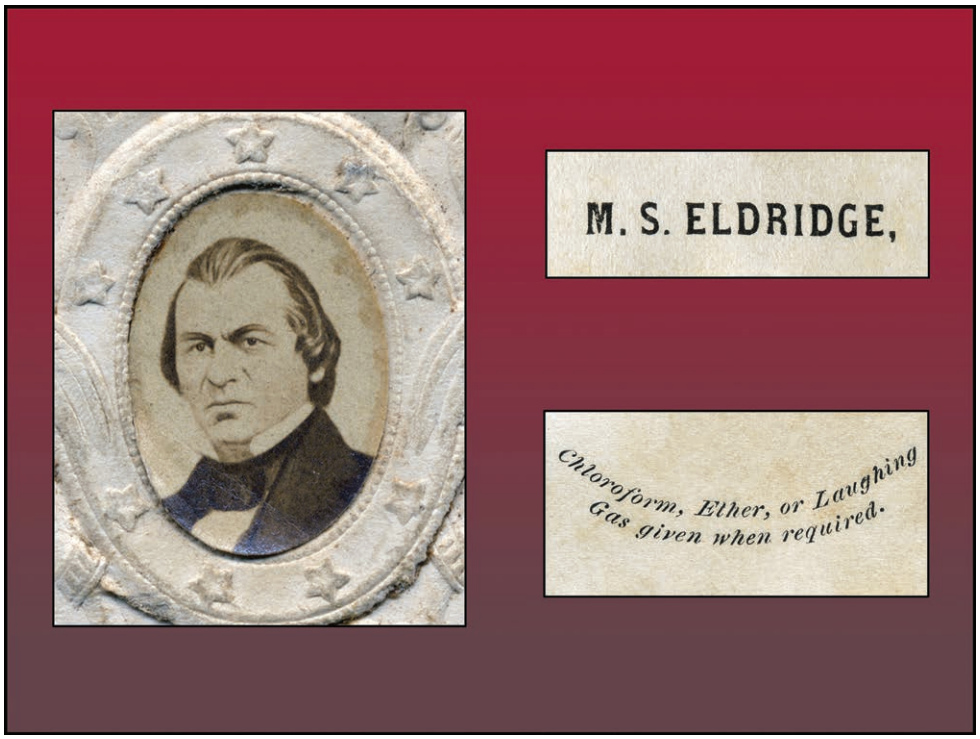
References

1. Hedenstierna G, Rothen HU: Respiratory function during anesthesia: Effects on gas exchange. Compr Physiol 2012; 2:69–96
2. Tokics L, Hedenstierna G, Svensson L, Brismar B, Cederlund T, Lundquist H, Strandberg A: V/Q distribution and correlation to atelectasis in anesthetized paralyzed humans. J Appl Physiol (1985) 1996; 81:1822–33
3. Östberg E, Thorisson A, Enlund M, Zetterström H, Hedenstierna G, Edmark L: Positive end-expiratory pressure and postoperative atelectasis: A randomized controlled trial. Anesthesiology 2019; 131:809–17
4. Lundquist H, Hedenstierna G, Strandberg A, Tokics L, Brismar B: CT-assessment of dependent lung densities in man during general anaesthesia. Acta Radiol 1995; 36:626–32
5. Tenling A, Hachenberg T, Tydén H, Wegenius G, Hedenstierna G: Atelectasis and gas exchange after cardiac surgery. Anesthesiology 1998; 89:371–8
6. Miskovic A, Lumb AB: Postoperative pulmonary complications. Br J Anaesth 2017; 118:317–34
7. van Kaam AH, Lachmann RA, Herting E, De Jaegere A, van Iwaarden F, Noorduyn LA, Kok JH, Haitsma JJ, Lachmann B: Reducing atelectasis attenuates bacterial growth and translocation in experimental pneumonia. Am J Respir Crit Care Med 2004; 169:1046–53
8. Nimmagadda U, Salem MR, Crystal GJ: Preoxygenation: Physiologic Basis, Benefits, and Potential Risks. Anesth Analg 2017; 124:507–17
9. Edmark L, Kostova-Aherdan K, Enlund M, Hedenstierna G: Optimal oxygen concentration during induction of general anesthesia. Anesthesiology 2003; 98:28–33

10. Rothen HU, Sporre B, Engberg G, Wegenius G, Reber A, Hedenstierna G: Prevention of atelectasis during general anaesthesia. Lancet 1995; 345:1387–91
11. Rothen HU, Neumann P, Berglund JE, Valtysson J, Magnusson A, Hedenstierna G: Dynamics of re-expansion of atelectasis during general anaesthesia. Br J Anaesth 1999; 82:551–6
12. Östberg E, Thorisson A, Enlund M, Zetterström H, Hedenstierna G, Edmark L: Positive end-expiratory pressure alone minimizes atelectasis formation in non-abdominal surgery: A randomized controlled trial. Anesthesiology 2018; 128:1117–24
13. Benoît Z, Wicky S, Fischer JF, Frascarolo P, Chapuis C, Spahn DR, Magnusson L: The effect of increased FIO(2) before tracheal extubation on postoperative atelectasis. Anesth Analg 2002; 95:1777–81, table of contents
14. Pereira SM, Tucci MR, Morais CCA, Simões CM, Tonelotto BFF, Pompeo MS, Kay FU, Pelosi P, Vieira JE, Amato MBP: Individual positive end-expiratory pressure settings optimize intraoperative mechanical ventilation and reduce postoperative atelectasis. Anesthesiology 2018; 129:1070–81

ANESTHESIOLOGY REFLECTIONS FROM THE WOOD LIBRARY-MUSEUM

The Providential Discharge of Civil War Dentist-Anaesthetist Moses S. Eldridge



A diehard New Englander, Maine native Moses S. Eldridge (1833 to 1889) relocated south to New Hampshire and then Massachusetts before finally settling in Rhode Island. Initially offering dental patients in Providence chloroform or ether anesthetics, Eldridge left home to serve the Union forces during America's Civil War. The very month that Eldridge mustered out of the 11th Rhode Island Infantry, July of 1863, was the same month that Gardner Q. Colton revived dentists' use of nitrous-oxide by opening his Colton Dental Association in Manhattan. Under his albumen-print portrait (*left*) glued inside a nine-starred, oval-framed carte-de-visite, "M. S. Eldridge" (*upper right*) was inspired by Colton to advertise the former's willingness to administer "Chloroform, Ether or Laughing Gas when required" (*lower right*). Providence directories list Eldridge at this card's 37 Dorrance Street address from 1864 to 1866.

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