

Protective Ventilation during Anesthesia

Is It Meaningful?

Göran Hedenstierna, M.D., Ph.D., Lennart Edmark, M.D., Ph.D.

SEVERAL clinical trials on protective ventilation during anesthesia and postoperative pulmonary complications have been performed during the past years and assumed to be suitable as guidelines for clinical treatment.¹⁻⁸ Comprehensive reviews and meta-analysis have also been performed.^{9,10} Why then do these studies differ in their recommendations? One reason may be that the three major tools that have been used for creating protective ventilation have been taken over from intensive care and thus from a different category of patients with severely sick lungs that may be more vulnerable to forces caused by mechanical ventilation. These tools are (1) a low tidal volume or low driving pressure, assumed to reduce stress and strain of the lung; (2) a recruitment maneuver, assumed to reopen any collapsed alveoli; and (3) a positive end-expiratory pressure (PEEP), assumed to keep the lung open during ongoing anesthesia and surgery. Protective ventilation covers a period from induction to emergence from anesthesia, and whether any positive effects remain in the postoperative period is unknown. The atelectasis that develops intraoperatively may last for some days after surgery¹¹ and may be a cause of postoperative pulmonary complications. Without knowing how successful the protective ventilation during anesthesia is in keeping the lung open without excessive strain, and if any positive effects remain in the postoperative period, reduction in postoperative pulmonary complications can hardly be attributed to protective ventilation. One may even ask if



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the concept protective ventilation can be replaced by nonharmful ventilation during anesthesia. It must also be realized that postoperative lung complications may be related to events other than intraoperative pulmonary dysfunction. That mentioned, let us return to the three tools that make up the framework of protective ventilation and discuss if the physiology makes sense.

First, a low tidal volume has been claimed to be the most important aspect of the protective ventilation concept. Tidal volume has been reduced (or recommended to be reduced) from 10 to 15 ml/kg ideal bodyweight (IBW), which was standard some 15 to 20 yr ago, to 6 ml/kg in the ventilation of patients with acute respiratory failure¹² and during anesthesia.⁹ This results in a substantial decrease in stress and strain. In table 1, it can be seen that in a patient with severe pulmonary disorders weighing 70 kg, being ventilated with 10 ml/kg IBW, and having a very low end-expiratory lung volume (EELV), in the worst-case half a liter (baby lung),¹³ tidal volume causes a 140% increase in strain. It is likely that such strain causes damage to the lung and possibly inflammation, so-called ventilator-induced lung injury. A decrease in tidal volume to 6 ml/kg IBW causes a smaller increase in strain (by 84%). This should be compared with the strain in an awake healthy subject breathing spontaneously, with a tidal volume of half a liter and an EELV (similar to functional residual capacity [FRC] when airway pressure is atmospheric) of 2.8 l. The increase in strain is 18%. With a tidal volume of 0.7 l, corresponding to 10 ml/kg IBW,

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Table 1. Relations between Tidal Volume and Lung Volumes in Awake and Anesthetized Man and in Severe Acute Lung Injury

FRC awake: 2,800 ml
VT 500 ml → 7 ml/kg or 18% of FRC
VT 700 ml → 10 ml/kg or 25% of FRC
FRC during anesthesia: 2,400 ml
VT 500 ml → 7 ml/kg or 21% of FRC
VT 700 ml → 10 ml/kg or 29% of FRC
EELV in ARDS: 500 ml
VT 700 ml → 10 ml/kg or 140% of EELV
VT 420 ml → 6 ml/kg or 84% of EELV

Tidal volume (VT) in an awake man and during anesthesia and intensive care. VT expressed as % of resting lung volume reflects lung strain and may be a better measure of mechanical load than VT in % of ideal body weight, here set at 70 kg. Note the low % values (strain) in an awake and anesthetized man compared to those in the ARDS patient.

ARDS = acute respiratory distress syndrome; EELV = end-expiratory lung volume at the applied positive end-expiratory pressure level; FRC = end-expiratory lung volume at an airway pressure of 0 cm H₂O (atmospheric pressure).

increase in strain is no larger than 25%. These are likely numbers in a spontaneously breathing subject, but it should be remembered that the frequency and size of tidal volume vary during breathing. During anesthesia, EELV is reduced by an average of 0.4 l,¹⁴ and with the same tidal volumes (0.5 to 0.7 L), the increase in strain is no higher than 21 to 29%. A tidal volume of 1 l still causes no larger increases in strain than 42%. Thus, applying a concept from intensive care to a healthy lung during anesthesia may not be of similar importance. Moreover, duration of mechanical ventilation differs.

Second, a recruitment maneuver can be performed in different ways, all being based on the increase in airway pressure to around 40 and even up to 50 cm H₂O.^{15,16} That they have been successful in the different protective ventilation studies might be assumed although not confirmed. The recruitment may not last if the ventilation is provided with high oxygen concentration. Thus, ventilation with 100% oxygen after a recruitment maneuver causes recurrence of atelectasis within 5 min.¹⁷ Application of a PEEP may prevent this return of atelectasis as will be discussed next.¹⁸

Third, an adequate level of PEEP prevents recurrence of atelectasis. Ventilation with PEEP may even recruit a normal lung in a subject with a normal body mass index (BMI).¹⁹ PEEP may be easier to use and safer to open the lung and keep it open than intermittent recruitment maneuvers. Moreover, if the lung after recruitment is ventilated with 100% oxygen, appropriate PEEP prevents the return of atelectasis.¹⁸ The question is then how much PEEP is required? It certainly depends on the BMI of the patient. In a subject with normal BMI, a PEEP of 6 to 8 cm H₂O might suffice, whereas in an obese subject, higher PEEP most likely is required (10 cm H₂O or more). The atelectasis is caused by closure of distal airways that promotes absorption atelectasis by continuous uptake of oxygen from the alveoli. Closing

pressure is increased during anesthesia, *i.e.*, the airway pressure at which airways begin to close during expiration, and as a rule of thumb, it is around 7 cm H₂O in a lung-healthy adult with a normal BMI.²⁰ There is certainly a variation around this number. This suggests that to keep airways open, a PEEP of 7 cm H₂O is appropriate during anesthesia. Lower PEEP will not be enough to keep the airways open and may therefore not be expected to recruit or even keep all lung open. A higher PEEP may compromise hemodynamics, and in one study, a PEEP of 12 cm H₂O was used in mostly normal-weight subjects and that PEEP level required increased fluid administration and use of vasoactive drugs.³ This should, however, not result in a conclusion that PEEP is of no use, only that unnecessarily high PEEP may be.

Finally, efforts to keep the lung open should also focus on the emergence from anesthesia. Using 100% oxygen during emergence and extubation is common and often combined with airway suctioning. However, this combination might be the ultimate way of producing atelectasis.^{21–23} Thus, performing airway suctioning should have clear indications. Vigorous coughing on the endotracheal tube, perhaps provoked by suctioning the airway, can also result in significant alveolar collapse.^{23,24} Even if both suctioning and coughing can be avoided before extubation, the transition from an assumed open lung that has been enriched with a high oxygen content to an extubated lung without any support for maintaining EELV is another scenario for rapid formation of atelectasis. The use of continuous positive airway pressure or PEEP from induction to, and including, emergence may reduce atelectasis formation in the immediate postoperative period, if combined with a low inspired oxygen concentration and continuous positive airway pressure introduced after

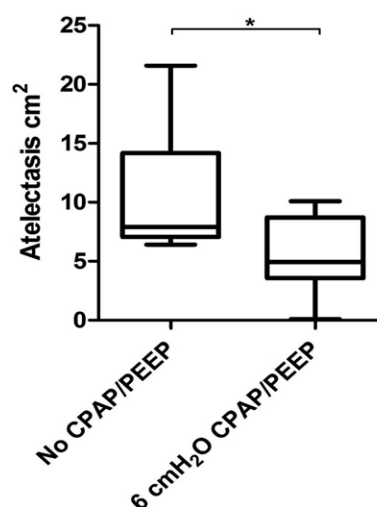


Fig. 1. Atelectasis 20 min after anesthesia and surgery during spontaneous breathing in patients with no positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) during emergence and patients who had this support. Note the almost 50% smaller atelectasis when end-expiratory lung volume has been kept elevated by PEEP/CPAP. Reprinted with permission from Edmark *et al.*²⁵

successful extubation (fig. 1).²⁵ It is also obvious that residual effects of drugs used during anesthesia, as well as opioids used postoperatively on the pulmonary function can be significant.²⁶ Thus, we believe that the lung during emergence and in the postoperative period should be given more attention than seems to be the case today. This does not preclude efforts to create non-harmful ventilation during the anesthesia. From a physiologic²⁷ and microbiologic²⁸ point of view, this suggests that the lung be kept open with as little strain as possible. A combination of a low driving pressure to ensure reasonable tidal volume¹⁰ and a PEEP just high enough to keep airways open may be a good mix. Just a low tidal volume, or driving pressure, without PEEP seems to us to increase the risk of atelectasis formation and was considered risky in 1963 in the classic article by Bendixen *et al.*²⁹

Taken together, it currently appears that during anesthesia of an adult patient with healthy lungs and a normal BMI, (1) tidal volume seems not to be a big issue; (2) recruitment maneuvers may not be necessary provided that a graded PEEP is applied that keeps airways open; (3) the patient should be delivered to the postoperative area with an open lung, and there the lung should be kept open.

The concept of protective ventilation of a sick lung in the intensive care unit is of utmost importance but may rather be considered nonharmful ventilation during anesthesia in an otherwise healthy lung in the operating room. Nonharmful ventilation and postoperative open lung may be important concepts in reducing pulmonary complications, and their design should thus be an interesting field for further research.

Competing Interests

The authors are not supported by, nor maintain any financial interest in, any commercial activity that may be associated with the topic of this article.

Correspondence

Address correspondence to Dr. Hedenstierna: goran.hedenstierna@medsci.uu.se

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