

Open is Better Than Closed

Göran Hedenstierna

Hedenstierna Laboratory, Department of Medical Sciences, Clinical Physiology, Uppsala University Hospital, Sweden

Dr. Pelosi has focused completely on the lung during anesthesia and I will therefore limit my comments to this situation. However, I would like to emphasize that the efforts to create a more even expansion of the lung, to enable recruitment without concomitant over-expansion of other lung regions requires certain invasiveness that may be reasonable in the intensive care patient but not during routine anesthesia.

I believe that much of the “protective ventilation” concept with low tidal volume, repeated recruitment maneuvers and PEEP (with varying recommendations on the magnitude of PEEP) is much ado about nothing. The three corner stones in this concept have been taken from intensive care for patients with severely sick lungs who are ventilated for days, not a few hours as during anesthesia. A large tidal volume will cause less damage in a healthy lung with a much larger resting lung volume than the intensive care patient. Take as an example an ARDS patient with a “baby lung” where lung volume may be as low as 0.5 L. A tidal volume of 1 L, corresponding to approximately 15 mL/kg ideal body weight, causes an increase in strain (Tidal volume / FRC) (here FRC is approximated to equal resting lung volume) of 200%. Even a decrease in tidal volume to 0.42 L, corresponding to a recommended 6 mL/kg, causes considerable increase in strain, by 84%. Compare that with an anesthetized patient with a resting lung volume of 2.4 L (1) - even a tidal volume of 1 L causes no more increase in strain than 42%, and a tidal volume of 0.7 L increases strain by 29%. This almost similar to waking, spontaneously breathing healthy man.

A recruitment maneuver can open up collapsed lung regions but an airway and alveolar pressure of 40 cm H₂O or more is needed (2, 3), perhaps not without any risk. The recruitment may not last if the ventilation is provided with high oxygen concentration. Thus, ventilation with 100% O₂ after a recruitment maneuver causes recurrence of atelectasis within 5 minutes (4).

PEEP prevents recurrence of atelectasis by keeping airways open. Atelectasis is caused by closure of distal airways that promotes absorption atelectasis by continuous uptake of oxygen from the alveoli. In a study on closing pressure, i.e. the airway pressure at which airways begin to close, was increased during anesthesia and as a rule of thumb it will be around 7 cmH₂O in a normal weight subject (5). Lower PEEP will not be enough to keep the airways open and may therefore not be expected to keep all lung open. A higher PEEP may compromise hemodynamics and in one study a PEEP of 12 cmH₂O was used in mostly normal weight subjects and that PEEP level required increased fluid administration and use of vasoactive drugs (6). Another aspect is that PEEP forces blood flow downwards in the lung, towards more dependent regions with possible persisting atelectasis. The effect of PEEP may be improved oxygenation but also a worsening! This was noticed already 40 years ago (7).

With all these possible consequences (or absence of effects), the value of “protective ventilation” can be discussed. I therefore agree with Pelosi that “general

Address for Correspondence:

Dr. Göran Hedenstierna

E-mail: goran.hedenstierna@medsci.uu.se

Turk J Anaesthesiol Reanim 2016; 44: 167-8

DOI: 10.5152/TJAR.2016.004

©Copyright 2016 by Turkish Anaesthesiology and Intensive Care Society
Available online at www.jtaics.org

anesthesia closes the lungs” and, perhaps, that one can let them rest. But I do not see any value in itself to allow the lung to collapse. Oxygenation will be impeded – but you may say that it can be corrected by increasing the inspired oxygen concentration. However, it increases the atelectasis formation and increasing inspired oxygen further causes a vicious circle with more atelectasis and more need of oxygen. Moreover, atelectasis is a locus of inflammation (8). Thus, an open lung is in my opinion a better lung than a closed lung.

The emergence from anesthesia may be the most important part of the peri-operative period and focus should be switched from the anesthesia per se to the emergence. Post-oxygenation is common and may be combined with airway suctioning. This combination might be the ultimate way of producing atelectasis (9). Thus, airway suctioning should be done with caution, proposedly by clear indications, and ventilation with pure oxygen should be discontinued and switched to lower oxygen concentration while the lung is still kept open. By using continuous positive airway pressure (CPAP) and PEEP from induction to, and including, emergence the fall in FRC during anesthesia may be prevented or attenuated. This will reduce atelectasis formation in the immediate postoperative period (10).

To conclude, the fall in FRC during anesthesia promotes airway closure that causes atelectasis, and rapidly so if the lungs are filled with oxygen, e.g. by a routine pre-oxygenation procedure. To allow this to happen, i.e. to let the lung to close and rest, is not to optimize anesthesia. I would favor an open lung with a suitable PEEP, not too much and not too little, and to make efforts to deliver an open lung to the post-operative ward.

References

1. Wahba RW. Perioperative functional residual capacity. *Can J Anaesth* 1991; 38: 384-400. [\[CrossRef\]](#)
2. Hartland BL, Newell TJ, Damico N. Alveolar recruitment maneuvers under general anesthesia: a systematic review of the literature. *Respir Care* 2015; 60: 609-20. [\[CrossRef\]](#)
3. 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. [\[CrossRef\]](#)
4. Rothen HU, Sporre B, Engberg G, Wegenius G, Hogman M, Hedenstierna G. Influence of gas composition on recurrence of atelectasis after a reexpansion maneuver during general anesthesia. *Anesthesiology* 1995; 82: 832-42. [\[CrossRef\]](#)
5. Hedenstierna G, McCarthy GS. Airway closure and closing pressure during mechanical ventilation. *Acta Anaesthesiol Scand* 1980; 24: 299-304. [\[CrossRef\]](#)
6. Hemmes SN, Gama de Abreu M, Pelosi P, Schultz MJ. High versus low positive end-expiratory pressure during general anaesthesia for open abdominal surgery (PROVHILO trial): a multicentre randomised controlled trial. *Lancet* 2014; 384: 495-503. [\[CrossRef\]](#)
7. Hewlett AM, Hulands GH, Nunn JF, Milledge JS. Functional residual capacity during anaesthesia III: Artificial ventilation. *Br J Anaesth* 1974; 46: 495-503. [\[CrossRef\]](#)
8. van Kaam AH, Lachmann RA, Herting E, De Jaegere A, van Iwaarden F, Noorduyt LA, et al. Reducing atelectasis attenuates bacterial growth and translocation in experimental pneumonia. *Am J Respir Crit Care Med* 2004; 169: 1046-53. [\[CrossRef\]](#)
9. Benoit Z, Wicky S, Fischer JF, Frascarolo P, Chapuis C, Spahn DR, et al. The effect of increased FIO₂ before tracheal extubation on postoperative atelectasis. *Anesth Analg* 2002; 95: 1777-81. [\[CrossRef\]](#)
10. Edmark L, Auner U, Hallen J, Lassinantti-Olowsson L, Hedenstierna G, Enlund M. A ventilation strategy during general anaesthesia to reduce postoperative atelectasis. *Ups J Med Sci* 2014; 119: 242-50. [\[CrossRef\]](#)