

We know that the distribution of ventilation and perfusion within the lung is not homogeneous, and related to gravitational and anatomical changes. This heterogeneity increases during mechanical ventilation; even if the lung is “healthy”. In “sick” lungs, it is even more exaggerated.

The approach of “optimal PEEP” is based of determination of the pressure value where the alveoli remain open; this approach (albeit with different definitions) is used and recommended during mechanical ventilation.

HOWEVER: It is very probably, that different regions of the lungs have different “optimal” PEEP’s, as been shown in Hedenstierna-school studies during lateral decubitus position. This MAY mean that the “average” (or “global”) optimal PEEP is not optimal on the whole lung: In some areas, it may be insufficient to keep the alveoli open; and (maybe more important) in some areas it may lead to overdistension.

Can it be argued that the “optimal PEEP” approach is actually a compromise between the areas with higher and lower levels?

Should “optimal PEEP” be reviewed in this sense; especially in “healthy” lungs?

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General Anesthesia Closes the Lungs: Keep Them Resting

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In patients undergoing high risk surgery, postoperative pulmonary complications (PPCs) are frequent and associated with increased morbidity and mortality (1). Different factors may promote PPCs, including clinical characteristics of the patients, type and duration of surgery, the amount and type of fluids administered, as well as pain control (2). The lung structure during surgery is altered due to a first hit induced by release of inflammatory mediators and bacterial translocation. Additionally, anesthesia and neuromuscular paralysis induce relaxation of respiratory muscles with a cranial shift of the diaphragm promoting a reduction in lung volume and formation of atelectasis in the most dependent lung regions. Atelectasis and reduction in lung volume are associated with impaired intraoperative oxygenation, increased dead space as well as reduced lung compliance. An inspiratory oxygen fraction higher than 80% has been also reported to facilitate atelectasis formation. It has been hypothesized that intraoperative atelectasis and development of ventilator induced lung injury (VILI) may promote postoperative respiratory complications and worsen clinical outcome (3). The major determinants of VILI are: 1) increased inspiratory lung stress by high tidal volumes (V_T), and 2) repetitive collapse and re-expansion of dependent lung parts, inducing the release of pro-inflammatory mediators; thus, leading to lung and distal organ injury. By using computed tomography of the lung, several clinical studies showed that a level of at least 10 cmH₂O positive end-expiratory pressure (PEEP), with or without previous recruitment maneuver was required to keep the lung open during anesthesia and paralysis (4). Therefore, an intraoperative ventilation strategy with the use of low V_T (6-8 mL/kg Predicted Body Weight, PBW) combined with moderate to high PEEP and intermittent recruitment manuevres, so called protective mechanical ventilation, compared to high V_T (higher than 9 mL/kg PBW) and low PEEP (less than 5 cm H₂O) and no recruitment manuevres could prevent lung injury, attenuating pulmonary inflammation, reducing PPC and improving postoperative outcome (5). However, based on these findings, we are unable to determine if the beneficial effects of protective mechanical ventilation may be ascribed to reduced V_T , increased PEEP or both. Furthermore, other studies showed that the amount of atelectasis in the majority of patients undergoing anesthesia are minor and limited to basal regions (6). In patients undergoing open abdominal surgery, mechanical ventilation with low V_T , PEEP of 12 cm H₂O and recruitment manuevres, compared with the use of PEEP levels of 0 to 2 cmH₂O, was associated with a higher dynamic compliance of the respiratory system during surgery, suggesting augmented

alveolar recruitment. Nevertheless, the development of postoperative pulmonary complications was similar in both groups. Additionally, the need of fluids, clinically relevant episodes of intraoperative hypotension, as well as requirement for vasoactive drugs were higher in patients mechanically ventilated with higher compared to lower levels of PEEP (7). A recent metaanalysis, including the individual data from largest randomized controlled trials, showed that lower V_T but not PEEP was the main determinant of reduced PPCs and improved outcome (8). Interestingly, moderate levels of PEEP between 5 and 8 cm H_2O were associated with increased risk to develop PPC. Bringing all these results together, higher levels of PEEP may indeed be associated with less intratidal recruitment and derecruitment, but negatively affect hemodynamics and do not result in any beneficial effect on postoperative outcome. Interestingly, the optimal level of PEEP, as individually titrated based according to the best respiratory compliance or dead space, was equivalent to 10 cm H_2O (9). These findings do not apply to morbidly obese patients, laparoscopic surgery in Trendelenburg positioning and duration of anesthesia of more than 4 hours. In these specific cases, further large controlled randomized trials are required. Further, inspiratory stress and plateau pressure higher 16 cm H_2O and PEEP 5 cm H_2O have associated with increased risk for PPC (10) More recently, the concept of driving pressure (ΔP) has been proposed as the “polar star” to optimize mechanical ventilation and explain the beneficial effects of lower V_T and not of PEEP to prevent VILI. ΔP is defined as the difference between pressure within the respiratory system at the end of inspiration and at the end of expiration, at zero flow during an end-inspiratory and end-expiratory pause. The real determinant of respiratory system ΔP is the transpulmonary ΔP , particularly when there is a change in chest wall mechanics, such as in obesity, abdominal distension, or surgery. It has been shown that higher ΔP is the independent risk factor associated with PPCs, instead of V_T and PEEP (11). Moreover, PPC increased in patients in which PEEP was associated with a higher ΔP . The association between ΔP and PPC might be explained through the mechanical energy, or power, delivered to the lung structures by the mechanical ventilator. The energy, is determined by the ΔP changes with time multiplied by the V_T , per breath. Power is the energy delivered in a specific time, and therefore increases at higher respiratory rates. Intensity (i.e., power delivered per unit of ventilated area) is even more important in determining VILI. Thus ΔP , energy, and power are key drivers of VILI, promoting PPC. In short, we introduced the concept of “intraoperative permissive atelectasis”, gently ventilating the aerated lung regions, and keeping the atelectatic areas resting (12). During anesthesia, we suggest the following intraoperative ventilation settings: low V_T (6–8 mL/kg predicted body weight), low plateau pressure (<16 cm H_2O), low PEEP (5 cm H_2O or lower), low ΔP (<13 cm H_2O), low inspiratory oxygen fractions and respiratory rate to maintain adequate gas-exchange, while recruitment manoeuvres should not be used routinely. Phys-

iology is important to better understand the clinical mechanisms and suggest possible therapeutic managements. However, before translating physiological findings to clinical practice, large randomized controlled trials are warranted. During general anesthesia keep the atelectatic lungs resting. So far, there has been no physiological parameter to estimate atelectasis and change mechanical ventilation during anesthesia. Certainly, prevention is better than treatment!

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