

The Use of Intraoperative Positive End Expiratory Pressure

Ahmed Zaky* and John D. Lang

Department of Anesthesiology and Pain Medicine, Puget Sound Health Care System, The University of Washington, Seattle, WA, USA

Abstract

General anesthesia is associated with impaired gas exchange mainly because of increased shunt due to atelectasis in the dependent regions of the lung. Postoperative atelectasis is associated with adverse clinical outcomes in terms of hypoxic respiratory failure requiring endotracheal intubation and pneumonia secondary to impairment of ciliary and lymphatic functions. Prevention of atelectasis and/or airway closure could be a mechanism by which positive end expiratory pressure (PEEP) improves oxygenation. Positive end expiratory pressure has been used intraoperatively as a part of open lung and protective lung ventilation strategies. However, it is unclear at the present time whether the intraoperative use of PEEP is associated with a decrease in mortality or in the incidence of other important clinical surrogates of outcome such as postoperative respiratory failure. The aim of this review is to review the physiologic effects and history of PEEP, to present some of the current uses in specific surgical populations and comment on potential benefits on postoperative mortality and pulmonary complications that may be ascribed to intraoperative PEEP use.

Keywords: Positive end expiratory pressure, Lung recruitment, General anesthesia, Atelectasis

Abbreviations: ARDS: Adult respiratory Distress Syndrome; CABG: Coronary Artery Bypass Grafting; CPAP: Continuous Positive Airway Pressure; EELV: End Expiratory Lung Volume; FiO₂: Fraction of Inspired Oxygen; FRC: Functional Residual Capacity; OLC: Open Lung Ventilation; PEEP: Positive End Expiratory Pressure; PLV: Protective Lung Ventilation; RM: Recruitment Maneuver; TLC: Total Lung Capacity; VT: Tidal Volume; PBW; Predicted Body Weight; PV: Pressure Volume

Background

Positive end expiratory pressure (PEEP) is a mechanical ventilatory maneuver of exerting a supra-atmospheric pressure in the lungs at end exhalation. It is important to recognize that PEEP is not a ventilator mode by itself; rather it is an adjunctive treatment that can be applied to all forms of mechanical ventilation; controlled, assisted or spontaneous [1].

Creation of a positive pressure at end exhalation increases the functional residual capacity (FRC) of the lungs by decreasing the collapse of the small airways thus, reducing atelectasis [2]. Furthermore, PEEP shifts the tidal volume to a more compliant portion of the pressure volume curve [3], prevents the intermittent loss of compliance during mechanical ventilation [4] and reduces the work of breathing [5]. By virtue of these effects, PEEP is capable of increasing arterial oxygenation [6]. On the other hand, if inappropriately applied, PEEP may have detrimental effects including impaired gas exchange [7], decreased cardiac output [8], splanchnic [9] and renal blood flows [10].

General anesthesia causes a reduction in the FRC as a result of increased intra-abdominal pressure, loss of inspiratory muscle tone, and a change in thoracic blood volume [11]. As a result, general anesthesia is associated with postoperative atelectasis [12]. Post-operative atelectasis has been implicated as a main cause of postoperative hypoxemia [13]. In addition, postoperative atelectasis impairs clearance of secretions [14] and lymphatic flow [15]; both which are predisposing factors to pneumonia. Taken together, there would seem to be an association between general anesthesia-induced atelectasis and postoperative adverse outcomes such as respiratory failure, pneumonia and mortality. Thus, PEEP with its potential to reduce postoperative atelectasis may serve as a protective maneuver to prevent the occurrence of these adverse outcomes.

Historical Perspectives of PEEP

The application of PEEP in conjunction with mechanical ventilation was initially introduced experimentally by Cournand et al. [16]. However, the researchers found that compared with mechanical ventilation with ambient end expiratory pressure, mechanical ventilation with PEEP was associated with decreased cardiac output as a result of a decrease in venous blood return. As a result, the interest in the application of PEEP to mechanical ventilation was tempered by its proposed hemodynamic consequences. The incorporation of PEEP in a mechanical ventilator was first introduced in the Engstrom mechanical ventilator in Sweden in the 1950s. The first clinical indication that PEEP increased the FRC in correlation with an increase in arterial oxygenation measured by oxygen saturation was accomplished by Frumin and colleagues in 1959 [17]. The researchers hypothesized that the decline in the alveolar-arterial oxygen gradient by increasing levels of PEEP is attributed to a reduction in the 'shunt-in-time' that takes place as a result of intermittent alveolar collapse at end exhalation. In their landmark study, Ashbaugh and colleagues [18] demonstrated that the addition of PEEP was capable of relieving life-threatening hypoxemia in patients with hypoxemic respiratory failure in a clinical scenario which they termed Adult Respiratory Distress Syndrome (ARDS). The publication of this study fueled further research investigating optimal ventilatory techniques for ARDS. The quest for optimal PEEP as an adjunctive ventilator maneuver for ARDS showed that dynamic lung compliance increases only with lower levels of PEEP [19]. The 1970's were notorious for the implementation of an assist ventilator mode of ventilation consisting of low tidal volumes, high PEEP and high respiratory frequency

***Corresponding author:** Ahmed Zaky, Department of Anesthesiology and Pain Medicine, Puget Sound Health Care System, The University of Washington, 1660 S Columbian Way, Seattle, WA 98108, S-112-ANES, USA, Tel: 206.277.6723; Fax: 206.764.2914; E-mail: ahmed.zaky@va.gov

Received September 01, 2011; **Accepted** October 11, 2011; **Published** October 15, 2011

Citation: Zaky A, Lang JD (2011) The Use of Intraoperative Positive End Expiratory Pressure. J Anesth Clin Res 4:308. doi:10.4172/2155-6148.1000308

Copyright: © 2011 Zaky A, et al.. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

'pressure panting technique' that further elucidated some of the advantages of PEEP in reversing hypoxemia and improving lung compliance [20]. The introduction of pulmonary computed tomography in the 1980, together with pioneering reports from Gattinoni et al. on the 'baby lung' concept gave more rationale about the use of PEEP in ARDS [21].

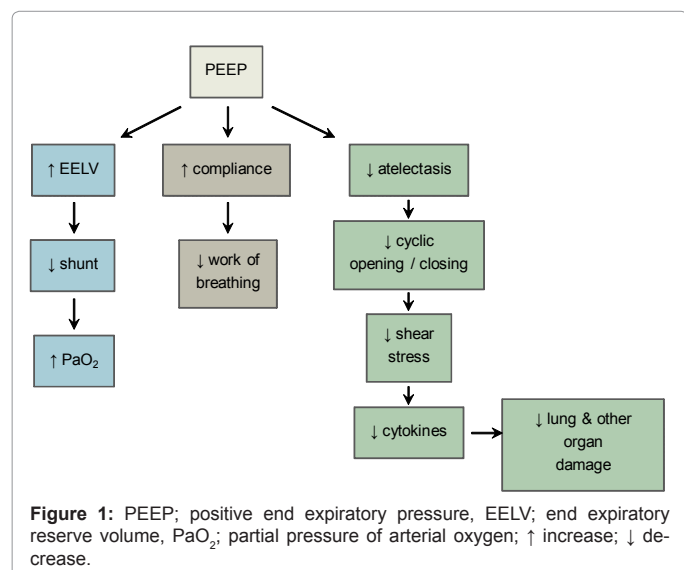
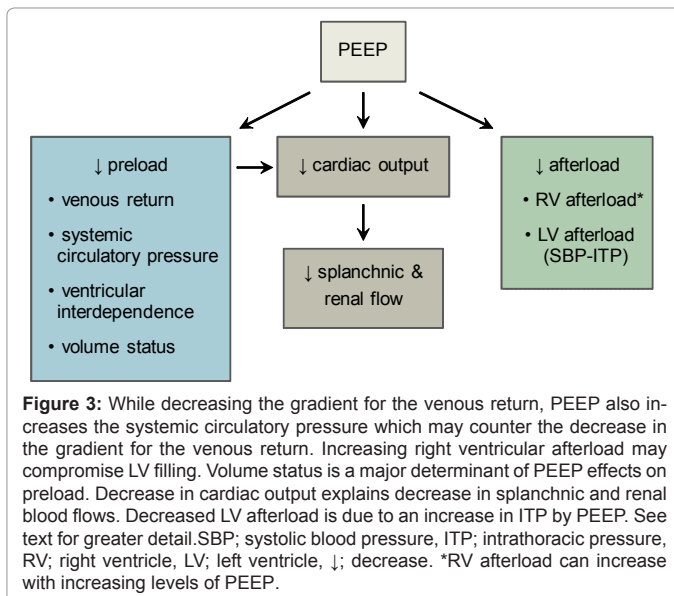
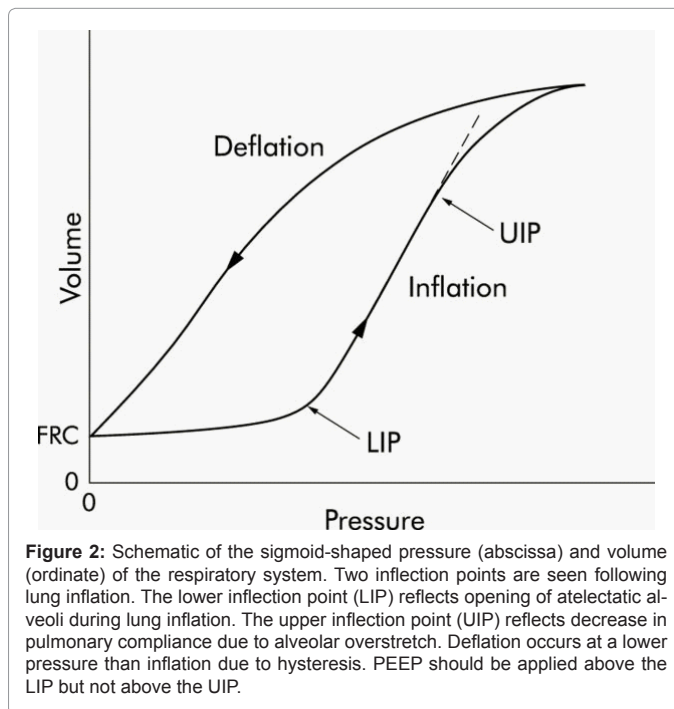
Physiologic Effects of PEEP

Effects of PEEP on pulmonary mechanics

When applied, PEEP influences both the elastic and resistive properties of the lungs (Figure 1). The elastic properties of the lungs can be appreciated from the pressure volume curve. The PV curve extending from the residual volume (RV) to the total lung capacity (TLC) has a sigmoidal shape [22] (Figure 2). However, the curve becomes linear at volumes above the FRC. The PV curve reveals 2 inflection points: an upper inflection point UIP at volumes about 75-80% of TLC when the lungs are overly stretched and less compliant. The lower inflection point (LIP) denotes the collapse of small airways at lung volumes below FRC under the surface tension of the alveolar fluid. Subsequent inflation requires a much higher pressure to open these collapsed alveoli [23]. From this point on, compliance increases as more alveoli are open. The difference between closing and opening pressures is created by hysteresis in surface tension which is greater during inflation than during deflation. Theoretically, application of PEEP above the LIP would decrease the pressure needed to open the alveoli, increase lung compliance and decrease the atelect-trauma that results from repetitive cyclic opening of collapsed alveoli. On the other hand, if PEEP exceeds the UIP, the alveoli will be over-stretched and decreasing lung compliance. As a result of a PEEP-induced increase in lung's end expiratory volume, there is a resultant decrease in airway resistance [24].

Effects of PEEP on cardiovascular functions

The effects of PEEP on cardiac preload, contractility and afterload are not fully- understood but do occur (Figure 3). Positive end expiratory pressure decreases cardiac output that was initially thought to be due to a decrease in the pressure gradient for venous return as result of elevated right atrial pressure [25]. However, the PEEP-mediated decrease in the gradient for venous return is less than expected because PEEP produces a concomitant increase in mean circulatory pressure which is the upstream



pressure of the venous return [26]. The rise in the mean filling pressure is thought to be due to reflex decrease in venous capacitance [27], and increased intra-abdominal pressure secondary to diaphragmatic descent [28]. One previous investigation demonstrated that increasing the airway pressure from 0-15 cmH₂O lead to a drop in the left ventricular stroke volume where both right atrial and mean systemic pressures increased equally. The PEEP-mediated decrease in cardiac output can be counteracted by adequate volume expansion [29]. Thus, PEEP leads to a decrease in cardiac output secondary to a decrease in venous return that is not fully explained by a decrease in its pressure gradient and that this decrease is responsive to volume expansion. Another mechanism by which PEEP leads to a decrease in the cardiac output is through a decrease in right ventricular afterload and leftward displacement of the interventricular septum thereby restricting left ventricular filling [8].

Left ventricular afterload is the force opposing contraction. It is determined by the systemic arterial resistance and the transmural pressure exerted on the left ventricle. The latter is determined by the difference between the systolic pressure and the intrathoracic pressure. An increase in the intrathoracic pressure by PEEP will lead to a decrease in the left ventricular transmural pressure and hence afterload, an effect that is more pronounced in patients with lower ejection fraction [30]. Positive end expiratory pressure has minimal effects on cardiac contractility [31]. Positive end expiratory pressure-induced decrease in renal and splanchnic circulations are related to its effects on cardiac output and the level of the PEEP applied as well as the volume status of the individual.

Alveolar Collapse during Anesthesia

Atelectasis occurs in 90% of patients undergoing general anesthesia. It is seen both with spontaneous breathing, after muscle paralysis, and under inhalational and intravenous anesthetics. Ketamine is probably the only intravenous anesthetic not associated with atelectasis [32].

Atelectasis occurs mainly near the diaphragm in the supine patient and can be severe enough to comprise 15 -20% of the lung tissue. Cardiopulmonary bypass is the surgical procedure that is mostly associated with atelectasis. Up to 50% of the lung can undergo atelectasis after cardiopulmonary bypass and this can occur within a very short time interval [33]. There is a linear correlation between atelectasis and impairment of gas exchange which can certainly lead to postoperative hypoxemia. In extreme cases not only is there alveolar collapse, but also collapse at the level of bronchiole and capillaries [34].

Three basic mechanisms explain alveolar collapse during general anesthesia:

Absorption atelectasis

This occurs as a result of a fall in the FRC from shifting from the erect to a supine position after induction of general anesthesia. The decrease in FRC and the loss of muscle tone by paralysis lead to cranial displacement of the diaphragm. Reduction in FRC leads to airway closure either during exhalation or if severe enough, throughout the respiratory cycle leading to air trapping. Continued uptake of the trapped air within the closed alveoli will, by mixing of venous and pulmonary arterial blood, lead to further atelectasis and shunting. The higher the concentration of inspired oxygen, the faster the occurrence of atelectasis [35]. Alveolar collapse is exaggerated by age when the closing volume exceeds the expiratory reserve volume in the supine position at age of 50 [36].

Compression atelectasis

This is the main mechanism by which atelectasis occurs in patients with ARDS. The weight of the edema fluid in the lungs extrudes air leading to alveolar collapse. Whether this is the mechanism in healthy lungs, has yet to be determined. Some reports speculate that compression atelectasis takes place as a result of the cephalad displacement of the diaphragm as a result of loss of abdominal muscle paralysis leading to compression of the dependent regions of the lung [37].

Surfactant deficiency

Surfactant is a lipoprotein secreted by type II alveolar cells to reduce the surface tension thus preventing collapse of small alveoli. Lack of intermittent deep breaths during general anesthesia affects surfactant function leading to alveolar collapse [38]. This collapse is also manifested

as soon as a PEEP of 10 cmH₂O was discontinued. A recruitment maneuver in the form of a forced vital capacity or a peak inspiratory pressure of 40 cmH₂O prevented alveolar re-collapse after PEEP, suggesting that a forceful inflation of the lung might redistribute surfactant and hence maintain alveolar stability [39].

Factors Affecting Alveolar Collapse during General Anesthesia

Fraction of inspired oxygen

There is increasing evidence that a FiO₂ of 1.0 is associated with faster atelectasis compared to lower FiO₂. An interesting study has shown that when a FiO₂ of 1.0 was used after a recruitment maneuver, alveolar collapse recurred after 5 minutes. When a FiO₂ of 0.4 was used, alveolar collapse occurred 40 minutes after the change [40]. In another study, induction of anesthesia with FiO₂ of 1 was associated with a shunt fraction increase from 0.3% to 6.5% and an area of atelectasis measuring 8.0 cm² on computed tomography (CT). In contrast, when using an FiO₂ of 0.3, the shunt fraction only increased to 2%, with an area of atelectasis of only 0.2cm² [41]. Similarly, increasing the FiO₂ to 1.0 before extubation was associated with atelectasis that persisted into the postoperative period [42]. Despite the established association with atelectasis, a FiO₂ of 1.0 is still used for preoxygenation to increase the oxygen reservoir in the lungs during prolonged intubation. Recently, the use of lower FiO₂s has been studied during preoxygenation for general anesthesia. Edmark and colleagues [43] compared different FiO₂s for preoxygenation during the conduct of a general anesthesia. The authors observed significantly smaller areas of atelectasis as measured by computed tomography with a FiO₂ of 0.8 compared with a FiO₂ of 1.0 (9.8 cm² vs. 1.2 cm²). The time to desaturation to 90% was significantly shorter (411 vs. 303 sec). Compared with a FiO₂ of 0.3, preoxygenation with a FiO₂ of 0.8 did not affect the incidence or severity of atelectasis in patients undergoing colon resection [44].

Do All Patients Need PEEP Applied Intra-operatively?

Cardiopulmonary bypass

The use of PEEP has been part of the mechanical ventilation strategies of patients undergoing cardiopulmonary bypass (CPB). Open lung ventilation (OLV) (which entails sustained high inflation pressures, with low tidal volumes, and high PEEP), protective lung ventilation (PLV) (ventilation with low tidal volume targeting a plateau pressure <30 cmH₂O), recruitment maneuvers, and the use of continuous positive airway pressure, are examples of mechanical ventilator strategies that incorporated PEEP in these patients. Outcomes assessed included oxygenation (with various indices), changes in concentrations of inflammatory mediators, hemodynamic parameters as well as pulmonary mechanical changes (Table 1).

In a prospective randomized controlled trial, Miranda and colleagues [45] randomized 62 patients undergoing Coronary Artery Bypass Grafting (CABG) and /or valve surgery into 3 groups with different ventilation strategies; the first group was ventilated according to an open lung ventilation (OLV) immediately after endotracheal intubation (early OLV), the second group was ventilated with the same strategy 30 minutes after arrival to ICU (late OLV), and the third received conventional lung ventilation from the beginning of the procedure. The outcome measured was serum levels of Interleukin (IL) 6, 8, and -10. Patients in first 2 groups showed a significant early decline in serum IL-8 compared with the conventional lung strategy group (CLV). Serum IL-10 decreased more rapidly in the early OLV group

Author	Design	Patients	Mechanical Ventilation	Procedure	Oxygenation Parameter	Outcome
Reis (59)	RCT	N = 69	1. Conventional 2. Early OLV 3. Late OLV	CABG/valve	SpO ₂	-CLV: 37% hypoxic on 3rd day after extubation. -OLV: none
Celebi (47)	RCT	N = 60	1. CPAP of 40 2. PEEP of 20 3. PEEP of 5	CPB	PaO ₂ /FiO ₂	PaO ₂ /FiO ₂ higher in CPAP 40, and PEEP 20 cm H ₂ O groups compared with PEEP of 5 group (30)
Reis (45)	RCT	N = 62	1. Conventional 2. Early OLV 3. Late OLV	CABG/valve	NA	-JIL-8 more rapidly in OLV groups -JIL-10 more rapidly only in early OLV
Dyhr (46)	RCT	N = 16	1. RM→PEEP 1 cm H ₂ O >LIP 2. RM only	CABG	PaO ₂	-PaO ₂ 16 +/- 16 KPa -No change from baseline

CLV; conventional lung ventilation, OLV; open lung ventilation, 1,2,3; refer to group numbers, LIP; lower inflection point, PEEP; positive end expiratory pressure, RM; recruitment maneuver, PaO₂; partial pressure of oxygen, RCT; randomized controlled trial.

Table 1: Studies of PEEP in Patients Undergoing Cardiopulmonary Bypass.

compared with the other 2 groups. There was a non- statistically significant trend towards better oxygenation (assessed by PaO₂/FiO₂) in the OLV groups compared with the CLV group. The authors concluded that OLV was associated with an attenuated inflammatory response compared with CLV. In another randomized controlled trial, Dyhr et al. [46], randomized 16 patients into 2 groups, a group receiving a recruitment maneuver (RM) with PEEP and the other receiving only RM (RM/zero PEEP, or ZEEP). It was seen that, compared with baseline, EELV and PaO₂ increased in the RM/PEEP while they remained unchanged in the RM/ZEEP group. After removal of PEEP, EELV volume decreased, while oxygenation remained unchanged. In an attempt to test the effects of adding PEEP to RM versus either alone, Celebi et al. [47] randomized 60 patients undergoing coronary artery revascularization into 3 groups, the first group received a RM and a decremented PEEP (CPAP of 40 cmH₂O/PEEP of 20 cmH₂O), the second group received only decremented PEEP of 20 cmH₂O, and the third received a PEEP of 5 cmH₂O. All maneuvers were employed in the immediate postoperative period. The most striking finding was that oxygenation, defined by the ratio of partial pressure of oxygen to its fraction of inspired concentration (PaO₂/FiO₂) was significantly higher in the first 2 groups compared with the third, and that effect persisted only for 4 hours after the RM. Besides, atelectasis score was significantly lower in the first 2 groups compared with the third. Interestingly, a protective lung ventilation strategy, a strategy known to decrease lung inflammation and increase survival in ARDS, was not superior to conventional lung ventilation in terms of levels of serum markers of inflammatory, pulmonary function or length of stay postoperatively [48].

The above-mentioned evidence reflects the inconsistencies in the patient population undergoing cardiopulmonary bypass, the ventilatory strategies of which PEEP was part of, the time of initiation of these ventilatory strategies, and the outcomes sought. More studies are needed to resolve these inconsistencies.

Thoracic surgery

Approximately, 9-27% of patients undergoing thoracic surgery with one lung ventilation develop arterial hypoxemia due to shunting of blood to the collapsed non-dependent lung [49]. Ventilating these patients with FiO₂ of 1.0 has been traditionally advocated to treat hypoxemia and maximize blood flow to the dependent lung. However such a high percentage of inspired oxygen may lead to the formation of reactive oxygen species (ROS) inducing oxidative lung injury, especially in diseased lungs [50]. In addition, the known risk of absorption atelectasis is also in play. Therefore, the use of PEEP and CPAP has been advocated to counteract

atelectasis induced with one-lung ventilation while preventing hyperoxic lung injury and absorption atelectasis. The use of PEEP in the setting of one-lung ventilation has yielded mixed results. Recently, Yang and colleagues randomized 100 patients undergoing elective lobectomy into either protective one lung ventilation (Vt 6 ml/kg, FiO₂ of 0.5, PEEP of 5 cmH₂O) or conventional one-lung ventilation (Vt 10 ml/kg, FiO₂ - 1.0, and ZEEP). The incidence of the primary end points of pulmonary dysfunction (PaO₂/FiO₂ <300, and/or newly developed lung infiltrate or atelectasis in the first 72 hours after the procedure) were significantly lower in the protective one-lung ventilation group compared with the conventional group [51]. In contrast, in a study performed by Hoftman and colleagues, a PEEP of 5 cm of H₂O and 10 cm of H₂O applied sequentially to 41 patients undergoing thoracic procedures under OLV failed to improve arterial oxygenation compared to pre-PEEP levels [52]. A small sample size was certainly a limitation of the study.

Due to the known mechanical perturbations PEEP has been used as an adjunct to RM and protective lung ventilation in thoracic procedures. In a prospective randomized trial of 42 patients undergoing thoracic surgery, Park et al. [53] randomized patients to receive either a tidal volume of 10 ml/kg or a RM of 10 manual breath of Inspired pressure of 40 cmH₂O followed by PEEP of 15 cmH₂O, before the commencement of one lung ventilation. The dependent lung was ventilated with tidal volume of 6 ml/kg and PEEP of 5 cmH₂O in both groups. Patients who had a RM prior to the commencement of one lung ventilation demonstrated significantly higher PaO₂'s (38.9+/-15 versus 28.8 +/- 14.4) and a lower alveolar arterial gradient at 15 minutes, and at the end of one lung ventilation (46.1+/- 14.4 vs. 55.9 +/-14.7, and 39.7+/-12.4 vs. 50.7+/-12.3 KPa, respectively).

An important clinical concept to consider is the interaction between the applied PEEP (extrinsic) and the existence of intrinsic or "auto PEEP" that is characteristically present in patients with Chronic Obstructive Pulmonary Disease (COPD) presenting for lung resection. Extrinsic PEEP should not exceed the intrinsic PEEP in these patients, otherwise it may exaggerate alveolar distension and further contribute to hypotension [54].

The "best PEEP" selected during thoracic procedures has been sought by many investigators. Slinger et al. [55] explored the relationship between extrinsic PEEP and lower inflection point and its effect on oxygenation. It appears that maximum oxygenation can be achieved as the PEEP moves towards the lower inflection point, and that oxygenation progressively declined as PEEP moved past that point. Maisch et al. [6] proposed that the 'best PEEP' should only be applied after a RM as evidenced by an increased compliance with the lowest dead space fraction. Additionally, Lachmann

et al. [56] demonstrated that excessive PEEP may lead to bacterial translocation in an experimental model of pneumonia.

Collectively taken, PEEP is an important maneuver in improving lung oxygenation during one lung ventilation especially if applied after a recruitment maneuver and in the setting of protective lung ventilation strategy. However, it should be applied cautiously to in order to prevent untoward effects such as alveolar overdistension.

Bariatric Surgery

Positive end expiratory pressure has been used both in laparoscopic and open bariatric surgeries. The application of PEEP varied, however, in timing, magnitude and whether it was used in conjunction with RM. Adding PEEP of 8 cmH₂O to a RM of 40 cmH₂O following abdominal incision was associated with a more significant improvement of oxygenation compared with a PEEP of 8 cmH₂O in a cohort of patients undergoing open bariatric procedure [57]. Interestingly, application of a PEEP of 10 cmH₂O was equivalent to a reverse Trendelenburg position in terms of improvement of arterial oxygenation (measured by the difference in the alveolar arterial pressure of oxygen A-a gradient), increase in total lung compliance and decrease in blood pressure in a cohort of 20 morbidly obese patients undergoing laparoscopic bariatric surgery [58]. In another cohort of 10 patients undergoing laparoscopic bariatric procedures, repetitive RMs of 50 cmH₂O followed by a PEEP of 12 cmH₂O were associated with a significantly improved oxygenation compared with a ventilator strategy using a PEEP of 4 cmH₂O. Interestingly, oxygenation returned to pre-recruitment levels 30 minutes after extubation [59]. Similarly, a vital capacity RM followed by a PEEP of 10 cmH₂O was associated with superior oxygenation and lower atelectasis scores compared with RM alone and with RM and a PEEP of 5 cmH₂O in obese patients undergoing laparoscopic bariatric surgery [60]. The combined effects of RMs and PEEP as compared to either strategy alone were elegantly demonstrated by CT scan in a recently published study [61]. In short, the combination of a RM + PEEP was superior to either a RM or PEEP alone in reducing atelectasis (Figure 4). In summary, small sample sizes and lack of a standard PEEP approach make it difficult to draw a firm conclusion. However, the application of PEEP appears to have a “positive” effect, but how beneficial is deserving of further more vigorous investigation as surgical procedures for this particular patient population continues to grow.

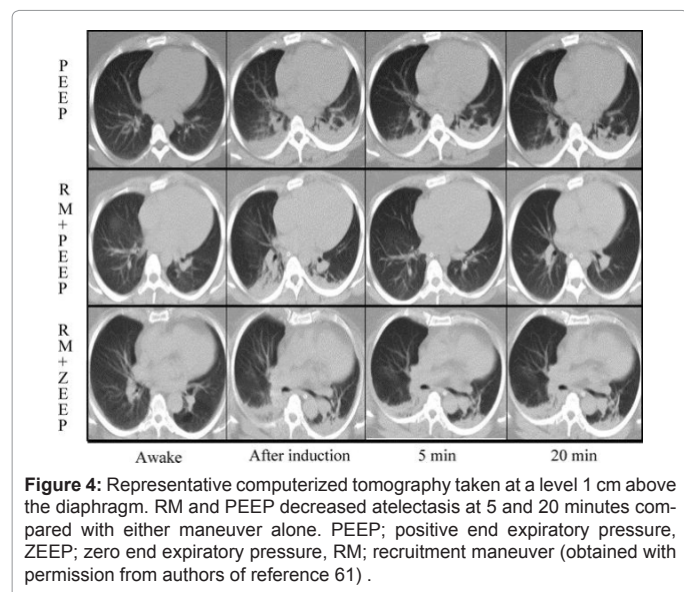


Figure 4: Representative computerized tomography taken at a level 1 cm above the diaphragm. RM and PEEP decreased atelectasis at 5 and 20 minutes compared with either maneuver alone. PEEP; positive end expiratory pressure, RM; recruitment maneuver, PLV; protective lung ventilation, FIO₂; fraction of inspired oxygen, V_T; tidal volume, RF; respiratory failure, ↓; decrease, ↑; increase, ? unknown.

Pneumoperitoneum

Pneumoperitoneum created by various laparoscopic surgical techniques has been demonstrated to impair pulmonary mechanics and gas exchange. These effects have been observed both in obese and non-obese patients. Compared with non-obese patients, obese patients manifest worsening of oxygenation under general anesthesia associated with Trendelenburg position and peritoneal insufflation [62]. The application of PEEP has been used to counter the upward shift of the diaphragm caused by the pneumoperitoneum and to improve pulmonary mechanics [63]. Positive end expiratory pressure has also been used in conjunction with RMs to maintain the increase in oxygenation achieved by the latter. Futier et al. [64] observed an increase in the EELV both in obese and non-obese patient who received 10 cmH₂O of PEEP. Interestingly, no increase in oxygenation was noticed. When a RM of 40 cmH₂O for 40 seconds was applied, PaO₂ significantly increased and was maintained for 30 minutes. In another study conducted exclusively in obese patients, repetitive recruitment maneuvers (every 10 minutes) produced the maximum improvement in lung compliance and PaO₂ [65]. In aggregate, the current evidence reveals that the creation of pneumoperitoneum can induce deleterious effects on lung mechanics in both obese and non-obese patients. Recruitment maneuvers coupled with PEEP appear to reverse these changes and improve gas exchange. In obese patients this effect is more pronounced if the RMs are repeated.

Patients with Pre-existing Acute Lung Injury (ALI) and Adult Respiratory Distress Syndrome (ARDS)

Mechanical ventilation of patients with ARDS using lower tidal volumes (6 ml/Predicted Body Weight (PBW)) with a goal of maintaining a plateau pressure of <30 cmH₂O has been shown to improve survival [66]. Considerable debate exists, however, regarding a survival benefit of high PEEP (11-15 cmH₂O) [67,68]. A recent meta-analysis has shown that, compared to a lower PEEP, a high PEEP was associated with a statistically significant decrease in hospital mortality in patients with ARDS. High PEEP was associated with a less benefit and a potential harm in patients with acute lung injury who did not have ARDS. In this meta analysis, higher PEEP was associated with a small risk of non-fatal pneumothorax in patients with ARDS [69].

Despite the proven benefit of low tidal volume ventilation for patients with ARDS, its intraoperative application remains unclear. In a recent retrospective analysis of 249 patients who underwent surgery between 24 hours and 14 days after ALI/ARDS diagnosis, only 54 patients (53%)

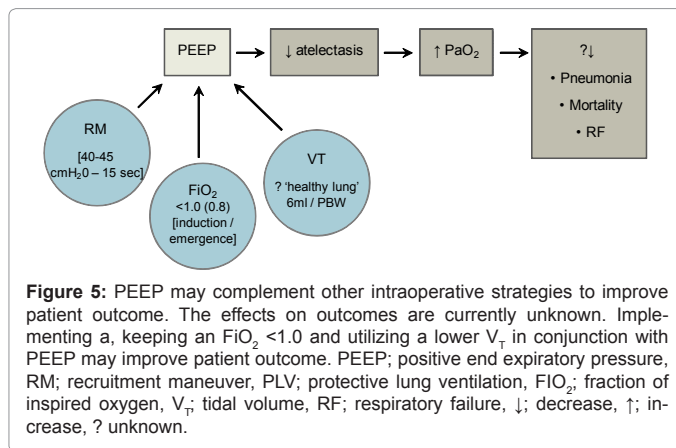


Figure 5: PEEP may complement other intraoperative strategies to improve patient outcome. The effects on outcomes are currently unknown. Implementing a, keeping an FIO₂ <1.0 and utilizing a lower V_T in conjunction with PEEP may improve patient outcome. PEEP; positive end expiratory pressure, RM; recruitment maneuver, PLV; protective lung ventilation, FIO₂; fraction of inspired oxygen, V_T; tidal volume, RF; respiratory failure, ↓; decrease, ↑; increase, ? unknown.

received low tidal volume ventilation. Intraoperative adherence to low tidal volume ventilation was not associated with a decrease in hospital mortality, in-hospital length of stay or improved oxygenation. Notably, there was no report of PEEP or of plateau pressures in this study [70].

In summary, in patients with ARDS/ALI, low tidal volume mechanical ventilation to a goal plateau pressure of <30 cm of H₂O is associated with decreased mortality. Higher PEEP may be beneficial in patients with ARDS but not ALI. The implications of intraoperative adherence to this strategy remain to be determined.

Does PEEP Affect Outcomes?

Despite of its beneficial effects on oxygenation and lung mechanics, the implementation of PEEP has not been shown to definitively affect patient outcomes. A recent meta-analysis [71] of 8 randomized clinical trials with a total of 388 patients demonstrated only 2 statistically significant effects of PEEP users compared with non-users; higher PaO₂/FiO₂ ratio on postoperative day 1 and reduced atelectasis (assessed by CT scan). There were no significant differences in mortality, incidence of barotrauma or adverse cardiac events between the 2 groups. Whether increased oxygenation or decreased atelectasis translates to fewer incidences of postoperative respiratory failure and pneumonia cannot be determined at the present time. A total of 21,200 further patients would need to be studied in order to have a statistically significant relationship between PEEP and mortality.

Conclusion

Positive end expiratory pressure is an adjunctive ventilatory modality. Its use in the operating room is associated with improved pulmonary mechanics and oxygenation, especially in surgical procedures associated with higher incidence of atelectasis. However, it is not clear whether these effects are associated with better patient outcomes. The use of PEEP is particularly indicated immediately after preoxygenation and immediately prior to extubation. The effects of PEEP are augmented and maintained by the use of RMs. A FiO₂ <1.0 followed by repetitive RMs and PEEP seem to be associated with the least incidence of atelectasis. Since there is no current consensus about the 'best PEEP', clinicians are encouraged to prescribe it on individual basis based on patient response (Figure 5).

References

1. Appendini L, Patessio A, Zanaboni S, Carone M, Gukov B, et al., (1994) Physiologic effects of positive end-expiratory pressure and mask pressure support during exacerbations of chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 149: 1069-1076.
2. East TD, in't Veen JC, Pace NL, McJames S (1988) Functional residual capacity as a noninvasive indicator of optimal positive end-expiratory pressure. *J Clin Monit* 4: 91-98.
3. Jonson B, Richard JC, Straus C, Mancebo J, Lemaire F, et al. (1999) Pressure-volume curves and compliance in acute lung injury: evidence of recruitment above the lower inflection point. *Am J Respir Crit Care Med* 159: 1172-1178.
4. Cereda M, Foti G, Musch G, Sparacino ME, Pesenti A (1996) Positive end-expiratory pressure prevents the loss of respiratory compliance during low tidal volume ventilation in acute lung injury patients. *Chest* 109: 480-485.
5. Katz JA, Marks JD (1985) Marks, Inspiratory work with and without continuous positive airway pressure in patients with acute respiratory failure. *Anesthesiology* 63: 598-607.
6. Maisch S, Reissmann H, Fuellekrug B, Weismann D, Rutkowski T, et al. (2008) Compliance and dead space fraction indicate an optimal level of positive end-

expiratory pressure after recruitment in anesthetized patients. *Anesth Analg* 106: 175-181.

7. Hasan FM, Beller TA, Sobonya RE, Heller N, Brown GW (1982) Effect of positive end-expiratory pressure and body position in unilateral lung injury. *J Appl Physiol* 52: 147-154.
8. Jardin F, Farcot JC, Boisante L, Curien N, Margairaz A, et al. (1981) Influence of positive end-expiratory pressure on left ventricular performance. *N Engl J Med* 304: 387-392.
9. Fujita Y (1993) Effects of PEEP on splanchnic hemodynamics and blood volume. *Acta Anaesthesiol Scand* 37: 427-431.
10. Priebe HJ, Heimann JC, Hedley-Whyte J (1981) Mechanisms of renal dysfunction during positive end-expiratory pressure ventilation. *J Appl Physiol* 50: 643-649.
11. Hedenstierna G, Strandberg A, Brismar B, Lundquist H, Svensson L, et al. (1985) Functional residual capacity, thoracoabdominal dimensions, and central blood volume during general anesthesia with muscle paralysis and mechanical ventilation. *Anesthesiology* 62: 247-254.
12. Eichenberger A, Proietti S, Wicky S, Frascarolo P, Suter M, Spahn DR, et al. (2002) Morbid obesity and postoperative pulmonary atelectasis: an underestimated problem. *Anesth Analg* 95: 1788-1792.
13. Hedenstierna G, Edmark L (2005) The effects of anesthesia and muscle paralysis on the respiratory system. *Intensive Care Med* 31: 1327-1335.
14. Hedenstierna G (2002) Airway closure, atelectasis and gas exchange during anaesthesia. *Minerva Anesthesiol* 68: 332-336.
15. Pearse DB, Searcy RM, Mitzner W, Permutt S, Sylvester JT (2005) Effects of tidal volume and respiratory frequency on lung lymph flow. *J Appl Physiol* 99: 556-563.
16. Courmand A, Motley HL (1948) Physiological studies of the effects of intermittent positive pressure breathing on cardiac output in man. *Am J Physiol* 152: 162-174.
17. Frumin MJ, Bergman Na, Holaday Da, Rackow H, Salanitro E (1959) Alveolar-arterial O₂ differences during artificial respiration in man. *J Appl Physiol* 14: 694-700.
18. Ashbaugh DG, Bigelow DB, Petty TL, Levine BE (1967) Acute respiratory distress in adults. *Lancet* 2: 319-323.
19. Falke KJ, Pontoppidan H, Kumar A, Leith DE, Geffin B, et al. (1972) Ventilation with end-expiratory pressure in acute lung disease. *J Clin Invest* 51: 2315-2323.
20. Ashbaugh DG, Petty TL, Bigelow DB, Harris TM (1969) Continuous positive-pressure breathing (CPPB) in adult respiratory distress syndrome. *J Thorac Cardiovasc Surg* 57: 31-41.
21. Gattinoni L, Presenti A, Torresin A, Baglioni S, Rivolta M, et al. (1986) Adult respiratory distress syndrome profiles by computed tomography. *J Thorac Imaging* 1: 25-30.
22. Rahn H, Otis AB (1946) The pressure-volume diagram of the thorax and lung. *Am J Physiol* 146: 161-178.
23. Anthonisen NR (1964) Changes in Shunt Flow, Compliance, and Volume of Lungs during Apneic Oxygenation. *Am J Physiol* 207: 235-238.
24. Pelosi P, Ravagnan I, Giurati G, Panigada M, Bottino N, et al. (1999) Positive end-expiratory pressure improves respiratory function in obese but not in normal subjects during anesthesia and paralysis. *Anesthesiology* 91: 1221-1231.
25. Marini JJ, Culver BH, Butler J (1981) Mechanical effect of lung distention with positive pressure on cardiac function. *Am Rev Respir Dis* 124: 382-386.
26. Scharf SM, Caldini P, Ingram RH Jr (1977) Cardiovascular effects of increasing airway pressure in the dog. *Am J Physiol* 232: H35-43.
27. Fessler HE, Brower RG, Wise RA, Permutt S (1991) Effects of positive end-expiratory pressure on the gradient for venous return. *Am Rev Respir Dis* 143: 19-24.
28. van den Berg PC, Jansen JR, Pinsky MR (2002) Effect of positive pressure on venous return in volume-loaded cardiac surgical patients. *J Appl Physiol* 92: 1223-1231.
29. Jellinek H, Krenn H, Oczeni W, Veit F, Schwarz S, et al. (2000) Influence of

- positive airway pressure on the pressure gradient for venous return in humans. *J Appl Physiol* 88: 926-932.
30. Bernard GR, Pou NA, Coggeshall JW, Carroll FE, Snapper JR (1995) Comparison of the pulmonary dysfunction caused by cardiogenic and noncardiogenic pulmonary edema. *Chest* 108: 798-803.
31. Haynes JB, Carson SD, Whitney WP, Zerbe GO, Hyers TM, et al. (1980) Positive end-expiratory pressure shifts left ventricular diastolic pressure-area curves. *J Appl Physiol* 48: 670-676.
32. Tokics L, Hedenstierna G, Strandberg A, Brismar B, Lundquist H (1987) Lung collapse and gas exchange during general anesthesia: effects of spontaneous breathing, muscle paralysis, and positive end-expiratory pressure. *Anesthesiology* 66: 157-167.
33. Tenling A, Hachenberg T, Tydén H, Wegenius G, Hedenstierna G (1998) Atelectasis and gas exchange after cardiac surgery. *Anesthesiology* 89: 371-378.
34. Tusman G, Böhm SH (2010) Prevention and reversal of lung collapse during the intra-operative period. *Best Pract Res Clin Anaesthesiol* 24: 183-197.
35. Westbrook PR, Stubbs SE, Sessler AD, Rehder K, Hyatt RE (1973) Effects of anesthesia and muscle paralysis on respiratory mechanics in normal man. *J Appl Physiol* 34: 81-86.
36. Reber A, Nylund U, Hedenstierna G (1998) Position and shape of the diaphragm: implications for atelectasis formation. *Anaesthesia* 53: 1054-1061.
37. Brismar B, Hedenstierna G, Lundquist H, Strandberg A, Svensson L, et al. (1985) Pulmonary densities during anesthesia with muscular relaxation—a proposal of atelectasis. *Anesthesiology* 62: 422-428.
38. Wollmer P, Schairer W, Bos JA, Bakker W, Krenning EP, et al. (1990) Pulmonary clearance of ^{99m}Tc-DTPA during halothane anaesthesia. *Acta Anaesthesiol Scand* 34: 572-575.
39. Otis DR Jr, Johnson M, Pedley TJ, Kamm RD (1993) Role of pulmonary surfactant in airway closure: a computational study. *J Appl Physiol* 75: 1323-1333.
40. Rothen HU, Sporre B, Engberg G, Wegenius G, Högman M, et al. (1995) Influence of gas composition on recurrence of atelectasis after a reexpansion maneuver during general anesthesia. *Anesthesiology* 82: 832-842.
41. Rothen HU, Sporre B, Engberg G, Wegenius G, Reber A, et al. (1996) Atelectasis and pulmonary shunting during induction of general anaesthesia—can they be avoided? *Acta Anaesthesiol Scand* 40: 524-529.
42. Benoît Z, Wicky S, Fischer JF, Frascarolo P, Chapuis C, et al. (2002) The effect of increased FIO₂ before tracheal extubation on postoperative atelectasis. *Anesth Analg* 95: 1777-17781.
43. Edmark L, Kostova-Aherdan K, Enlund M, Hedenstierna G (2003) Optimal oxygen concentration during induction of general anesthesia. *Anesthesiology* 98: 28-33.
44. Akça O, Podolsky A, Eisenhuber E, Panzer O, Hetz H, et al. (1999) Comparable postoperative pulmonary atelectasis in patients given 30% or 80% oxygen during and 2 hours after colon resection. *Anesthesiology* 91: 991-998.
45. Reis Miranda D, Gommers D, Struijs A, Dekker R, Mekel J, et al., (2005) Ventilation according to the open lung concept attenuates pulmonary inflammatory response in cardiac surgery. *Eur J Cardiothorac Surg* 28: 889-895.
46. Dyhr T, Laursen N, Larsson A (2002) Effects of lung recruitment maneuver and positive end-expiratory pressure on lung volume, respiratory mechanics and alveolar gas mixing in patients ventilated after cardiac surgery. *Acta Anaesthesiol Scand* 46: 717-725.
47. Celebi S, Köner O, Menda F, Korkut K, Suzer K, et al. (2007) The pulmonary and hemodynamic effects of two different recruitment maneuvers after cardiac surgery. *Anesth Analg* 104: 384-390.
48. Koner O, Celebi S, Balci H, Cetin G, Karaoglu K, et al. (2004) Effects of protective and conventional mechanical ventilation on pulmonary function and systemic cytokine release after cardiopulmonary bypass. *Intensive Care Med* 30: 620-626.
49. Watanabe S, Noguchi E, Yamada S, Hamada N, Kano T (2000) Sequential changes of arterial oxygen tension in the supine position during one-lung ventilation. *Anesth Analg* 90: 28-34.
50. Witschi HR, Haschek WM, Klein-Szanto AJ, Hakkinen PJ (1981) Potentiation of diffuse lung damage by oxygen: determining variables. *Am Rev Respir Dis* 123: 98-103.
51. Yang M, Ahn HJ, Kim K, Kim JA, Yi CA, et al. (2011) Does a protective ventilation strategy reduce the risk of pulmonary complications after lung cancer surgery?: a randomized controlled trial. *Chest* 139: 530-537.
52. Hoftman N, Canales C, Leduc M, Mahajan A (2011) Positive end expiratory pressure during one-lung ventilation: Selecting ideal patients and ventilator settings with the aim of improving arterial oxygenation. *Ann Card Anaesth* 14: 183-187.
53. Park SH, Jeon YT, Hwang JW, Do SH, Kim JH, et al. (2011) A preemptive alveolar recruitment strategy before one-lung ventilation improves arterial oxygenation in patients undergoing thoracic surgery: a prospective randomised study. *Eur J Anaesthesiol* 28: 298-302.
54. Slinger PD, Hickey DR (1998) The interaction between applied PEEP and auto-PEEP during one-lung ventilation. *J Cardiothorac Vasc Anesth* 12: 133-136.
55. Slinger PD, Kruger M, McRae K, Winton T (2001) Relation of the static compliance curve and positive end-expiratory pressure to oxygenation during one-lung ventilation. *Anesthesiology* 95: 1096-1102.
56. Lachmann RA, van Kaam AH, Haitma JJ, Lachmann B (2007) High positive end-expiratory pressure levels promote bacterial translocation in experimental pneumonia. *Intensive Care Med* 33: 1800-1804.
57. Chalhoub V, Yazigi A, Sleilaty G, Haddad F, Noun R, et al. (2007) Effect of vital capacity manoeuvres on arterial oxygenation in morbidly obese patients undergoing open bariatric surgery. *Eur J Anaesthesiol* 24: 283-288.
58. Perilli V, Sollazzi L, Modesti C, Annetta MG, Sacco T, et al. (2003) Comparison of positive end-expiratory pressure with reverse Trendelenburg position in morbidly obese patients undergoing bariatric surgery: effects on hemodynamics and pulmonary gas exchange. *Obes Surg* 13: 605-609.
59. Whalen FX, Gajic O, Thompson GB, Kendrick ML, Que FL, et al. (2006) The effects of the alveolar recruitment maneuver and positive end-expiratory pressure on arterial oxygenation during laparoscopic bariatric surgery. *Anesth Analg* 102: 298-305.
60. Talab HF, Zabani IA, Abdelrahman HS, Bukhari WL, Mamoun I, et al. (2009) Intraoperative ventilatory strategies for prevention of pulmonary atelectasis in obese patients undergoing laparoscopic bariatric surgery. *Anesth Analg* 109: 1511-1516.
61. Reinius H, Jonsson L, Gustafsson S, Sundbom M, Duvernoy O, et al. (2009) Prevention of atelectasis in morbidly obese patients during general anesthesia and paralysis: a computerized tomography study. *Anesthesiology* 111: 979-987.
62. Meininger D, Zwissler B, Byhahn C, Probst M, Westphal K, et al. (2006) Impact of overweight and pneumoperitoneum on hemodynamics and oxygenation during prolonged laparoscopic surgery. *World J Surg* 30: 520-526.
63. Maracajá-Neto LF, Verçosa N, Roncally AC, Giannella A, Bozza FA, et al. (2009) Beneficial effects of high positive end-expiratory pressure in lung respiratory mechanics during laparoscopic surgery. *Acta Anaesthesiol Scand* 53: 210-217.
64. Futier E, Constantin JM, Pelosi P, Chanques G, Kwiatkowski F, et al. (2010) Intraoperative recruitment maneuver reverses detrimental pneumoperitoneum-induced respiratory effects in healthy weight and obese patients undergoing laparoscopy. *Anesthesiology* 113: 1310-1319.
65. Almarakbi WA, Fawzi HM, Alhashemi JA (2009) Effects of four intraoperative ventilatory strategies on respiratory compliance and gas exchange during laparoscopic gastric banding in obese patients. *Br J Anaesth* 102: 862-868.
66. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. The Acute Respiratory Distress Syndrome Network. *N Engl J Med* (2000) 342: 1301-1308.
67. Villar J, Kacmarek RM, Pérez-Méndez L, Aguirre-Jaime A (2006) A high positive end-expiratory pressure, low tidal volume ventilatory strategy improves outcome in persistent acute respiratory distress syndrome: a randomized, controlled trial. *Crit Care Med* 34: 1311-1318.
68. Amato MB, Barbas CS, Medeiros DM, Magaldi RB, Schettino GP, et al. (1998) Effect of a protective-ventilation strategy on mortality in the acute respiratory distress syndrome. *N Engl J Med* 338: 347-354.
69. Briel M, Meade M, Mercat A, Brower RG, Talmor D, et al. (2010) Higher vs lower positive end-expiratory pressure in patients with acute lung injury and acute respiratory distress syndrome: systematic review and meta-analysis. *JAMA* 303: 865-873.

70. Chaiwat O, Vavilala MS, Philip S, Malakouti A, Neff MJ, et al. (2011) Intraoperative adherence to a low tidal volume ventilation strategy in critically ill patients with preexisting acute lung injury. J Crit Care 26: 144-151.
71. Imberger G, McIlroy D, Pace NL, Wetterslev J, Brok J, et al. (2010) Positive end-expiratory pressure (PEEP) during anaesthesia for the prevention of mortality and postoperative pulmonary complications. Cochrane Database Syst Rev CD007922.

This article was originally published in a special issue, **Critical Care Anesthesia** handled by Editor(s). Dr. Porhomayon Jahan, State University of New York, USA; Dr. Siddappa R, Clarian North Medical Center, USA.