Mechanical Ventilation Strategies in the Operating Theatre

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Summary

Mechanical ventilation is indispensable for the maintenance of pulmonary gas exchange during general anaesthesia but may in and of itself contribute to injury of the lungs in the context of ventilator-induced lung injury (VILI). With regard to perioperative medicine, the consequences of VILI can manifest themselves in postoperative pulmonary complications which can lead to a relevant increase in morbidity and mortality following surgical procedures. The following review describes the impact of postoperative pulmonary complications on clinical outcome and depicts important mechanisms involved in VILI. Current clinical evidence for key ventilation parameters and interventions such as tidal volume, end-expiratory pressure (PEEP) and recruitment manoeuvre, and more recently introduced personalised ventilation strategies based on dynamic ventilation parameters, compliance and driving pressure (ΔP) are analysed with respect to their potential to reduce postoperative pulmonary complications. In addition, mechanical ventilation strategies for special situations, namely obesity and pneumoperitoneum, are discussed.

Introduction

Mechanical ventilation is a prerequisite for sufficient pulmonary gas exchange and adequate tissue oxygenation during general anaesthesia. Having said that, however, it has been shown that mechanical ventilation can in and of itself induce or aggravate lung injury [3] and as such promote development of postoperative pulmonary complications which in turn can negatively impact the patient's prognosis [4]. These negative effects can be minimised using lung protective ventilation [5].

Subsequently,

- the significance of postoperative pulmonary complications and
- the most important mechanisms involved in **ventilator induced lung injury (VILI)** leading to those complications will be portrayed,
- current evidence for lung protective ventilation strategies presented and
- recommendations made for the implementation of such strategies.

In addition, strategies for mechanical ventilation in special situations (obesity, pneumoperitoneum) are discussed. Due to the complexity of the pathophysiological mechanisms and resulting ventilation strategies involved in one lung ventilation, this review will not cover that special situation, and would instead refer the reader to appropriate review articles [1,2].

Significance of postoperative pulmonary complications

Postoperative pulmonary complications constitute an important clinical problem which contributes to a relevant extent to an increase in morbidity and mortality

Conflict of interest

The authors have no financial or other competing interest to disclose.

Keywords

Intraoperative mechanical ventilation – Ventilator induced lung injury – Postoperative pulmonary complications

following surgery. Approximately 8% of patients undergoing surgery develop at least one postoperative pulmonary complication [4]. Current scoring systems comprising of both patient and procedure related factors show that the rate of diagnosed postoperative pulmonary complications rises from 3.4% for low risk, to 13% for moderate risk, and to 38% for high risk scores [4,6]. The fact that 28% of those undergoing general anaesthesia can be assigned to the latter two groups [7] taken together with the fact that 230 million surgical procedures are performed worldwide annually illustrates the magnitude of the problem [8]. The number of postoperative pulmonary complications correlates with in-hospital mortality, rising from 1.4% for one to 23.5% for four or more postoperative pulmonary complications [4,6]. In total, 19% of perioperative all-cause mortality can be attributed to postoperative pulmonary complications [9]. In addition, these complications significantly contribute to rising hospital costs [10].

Postoperative pulmonary complications constitute a significant clinical challenge. Rigorous implementation of lung protective ventilation strategies and elimination of other risk factors takes on fundamental importance in the perioperative phase.

Mechanisms involved in ventilator induced lung injury (VILI)

High tidal volumes (volutrauma) and **high driving pressure** (barotrauma) are key pathophysiologic mechanisms causing VILI by creating **stress** (transpulmonary pressure) and **strain** (distension of the lungs over and above their resting position volume) leading to damage to the lung parenchyma. Cyclic collapse and reopening (recruitment) of atelectatic areas of the lungs (atelectrauma) is also a contributor [11]. Strain consists of a **static component** (distention of the lungs over and above their functional residual capacity in their normal state through application of positive end-expiratory pressure [PEEP]) and a **dynamic component** (dynamic distension of the lungs over and above their current end-expiratory lung volume (EELV) through cyclic application of the tidal volume [V_T]) [12]. Until recently, the dynamic component of strain was assumed to be the more important with regard to the pathophysiology of VILI. As such, lung protective strategies aimed to increase EELV through use of recruitment manoeuvres and high levels of PEEP, thereby reducing the dynamic component of strain for a constant V_T [13]. In contrast, however, data based on current research shows an increase in ventilator induced lung injury with an increased static component of strain as a result of high PEEP [14].

Both static and dynamic distension of the lungs are important mechanisms in VILI.

In the presence of preexisting damage to the lungs in particular, barotrauma, volutrauma and atelectrauma show heterogenic distribution along the dorsoventral axis of the lungs. Atony of the dorsal aspects of the diaphragm associated with general anaesthesia and neuromuscular block leads to a cranial shift of intraabdominal organs, causing increased end-expiratory collapse with consecutive reopening. In addition, the weight of the heart and the lungs themselves contributes to the development of atelectrauma. In contrast, the well-ventilated ventral areas of the lungs are prone to both barotrauma and volutrauma [15].

At a cellular level, the mechanical stimuli are converted to biochemical signals. This activation of pro-inflammatory and anti-inflammatory mediators is known as **mechanotransduction** and triggers proapoptotic and profibrotic mechanisms whilst also activating the cellular immune response [16]. The endothelial, epithelial and extracellular components of the alveolocapillary unit can all be involved in this process. Damage to the endothelium increases its permeability and leads to development of interstitial or alveolar oedema. Damage to the epithelium decreases its ability to reduce alveolar oedema and synthesise surfactant [17]. The inflammatory reaction in the lungs can be initiated or reinforced by destruction of the extracellular matrix [18]. Where the inflammatory response to mechanical ventilation is not limited to the lung parenchyma, multiorgan failure can ensue as a result of a systemic inflammatory reaction [19].

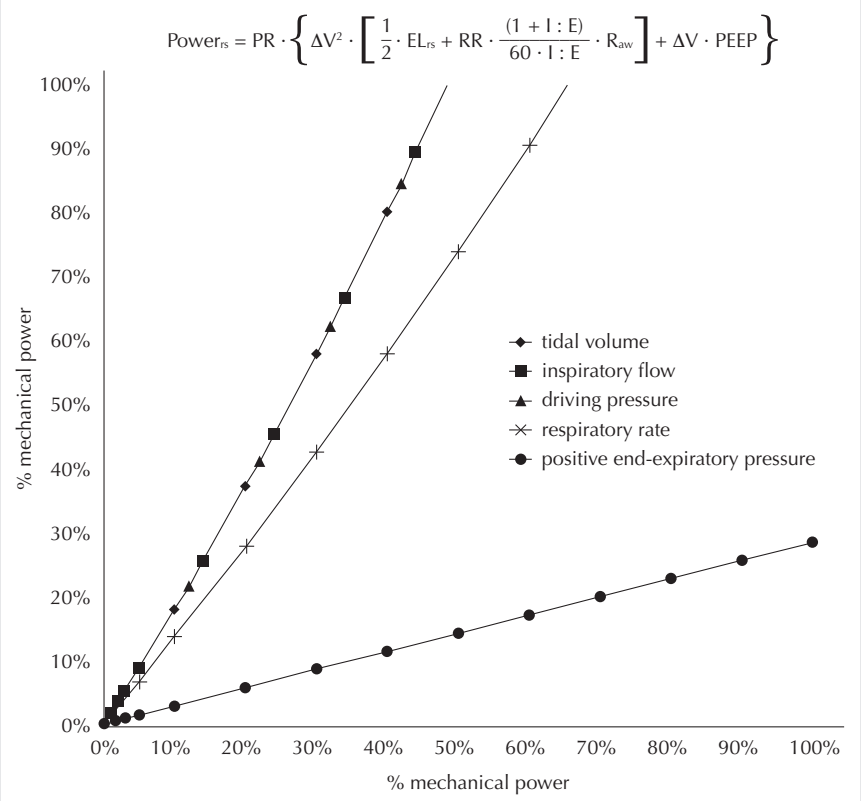
Rather than concentrating on the impact of individual variables, a more recent concept describes the **mechanical power** transferred from the ventilator to the respiratory system as a whole as the measure of and risk for potential damage. Hence the tidal volume and inspiratory flow, the **driving pressure** and respiratory rate, and PEEP contribute to varying degrees to overcoming resistive and elastic forces in the lungs and chest (Fig. 1) [20]. Because the mechanical power can be quantified based on ventilation parameters, ideal tuning of those parameters could be used to apply the lowest possible individual power providing optimum gas exchange.

Evidence for intraoperative protective ventilation

Limits of current clinical evidence

Because of their bearing on postoperative morbidity and mortality, reducing postoperative pulmonary complications is an important goal in perioperative care. Numerous clinical trials looking into ideal ventilation parameters have been performed, aiming to reduce the risk of postoperative pulmonary complications by decreasing VILI using lung protective ventilation. Many of these trials exhibit limitations, however, making interpretation of results and especially extrapolation of clear recommendations for intraoperative ventilation difficult. Care provided to the control group in some trials was, for example, not equivalent to the clinical standards of the day for intraoperative ventilation, making it difficult to gauge the actual

Figure 1



Concept of mechanical (ventilation) power. The figure shows the mechanical power transferred by the ventilator as a function of five individual factors. Whilst tidal volume, inspiratory flow and driving pressure increase the mechanical power in equal measure, PEEP and respiratory rate contribute a lesser increase. A 20% increase in volume, flow and driving pressure leads to a 37% increase in total power applied; a 20% increase in PEEP only leads to approx. 6% increase in power (modified from [20]).

benefit provided by the trial intervention as compared to standard care. In addition, a number of trials combined several respiratory interventions (e.g. reduction of VT combined with increased PEEP and use of recruitment manoeuvres), comparing these bundles with standard care. This approach makes it impossible to quantify the effect of each individual measure [21]. As such, Severgnini et al. [22] and Futier et al. [23] were able to demonstrate improved postoperative pulmonary function and clinical outcome with lung protective ventilation using reduced VT combined with increased PEEP and recruitment manoeuvres when compared with standard care. The trial design, however, made it impossible to show a causal relationship between any one intervention and the effect.

Significance of the tidal volume

Use of small tidal volumes of 6 ml/kg predicted body weight (PBW) represents the clinical standard in treatment of acute respiratory distress syndrome (ARDS); this is the only respiratory intervention proven to be associated with a reduction in mortality [24]. With increasing understanding of the role V_T plays in the pathophysiology of VILI in patients with ARDS, attention was directed towards those being ventilated but not suffering acute respiratory failure, particularly those patients receiving intraoperative mechanical ventilation. Although experimental research [25] and clinical trials [26] were able to show that high tidal volumes did not necessarily cause increased lung damage in those not originally suffering from lung disease, a trend has been seen in everyday clinical routine towards reduction of VT in intraoperative ventilation [27]. This development is founded in newer research which has shown that a reduced V_T leads to a decrease in pulmonary inflammation [28] and improves postoperative outcome. As such, Sundar et al. were able to reduce the rate of postoperative reintubation by ventilating using low V_T [29]. Although these results were not reproduced in every subsequent trial [30], meta-analyses show a clear positive effect of low V_T on clinical outcome in patients without ARDS. One meta-analysis of ventilation in such patients in intensive care and the operating theatre showed that use of low V_T reduced

- the incidence of lung injury,
- the rate of pulmonary infections and
- mortality when compared to ventilation using high V_T [31].

A further meta-analysis was able to show that use of $V_T \leq 7$ ml/kg as opposed to >10 ml/kg PBW was associated with a significantly decreased rate of postoperative pulmonary complications [32]. In a Cochrane Review updated in 2018, Joanne Guay et al. likewise came to the conclusion that use of low tidal volumes can positively impact the rate of postoperative pneumonia and the requirement for postoperative invasive or non-invasive ventilation. The overall mortality rate across the literature cited in the review was so low that an influence of low V_T on mortality could not be shown. A recent prospective observational study also confirmed the association between reduction of V_T for intraoperative ventilation and the rate of postoperative pulmonary complications [33].

Use of low tidal volumes can reduce postoperative pulmonary complications.

Significance of PEEP

90% of patients undergoing general anaesthesia develop atelectasis, which can be especially readily detected in the intraoperative phase, but which can

also persist postoperatively [34]. Up to 4% of lung volume comprising of 16–20% of lung tissue can be affected by this phenomenon [35]. A whole host of mechanisms, including

- compression of pulmonary structures caused by a cranial shift of the diaphragm and intraabdominal organs,
- surgical manipulation,
- small airway collapse,
- absorption of intra-alveolar gas associated with high inspiratory fractions of oxygen, and
- limited production of pulmonary surfactant

are responsible for the development of atelectasis [21]. Atelectasis can increase pulmonary inflammation when atelectrauma occurs as shear forces arise at the interface between ventilated and non-ventilated areas of the lungs [36].

Although PEEP can eliminate atelectasis [37] and retrospective observational studies have shown that PEEP ≥5 cm H2O is associated with a reduction in postoperative pulmonary complications [38], prospective randomised trials have failed to show a positive effect of PEEP on postoperative outcome. PEEP of 12 cm H_2O together with recruitment manoeuvres was compared to PEEP of 2 cm H_2O in the PROVHILO trial, using tidal volumes of 7 ml/kg PBW in both cases. Whilst intraoperative pulmonary function improved with the intervention, the requirement for catecholamines was increased and neither the postoperative outcome nor postoperative pulmonary function were improved [39,40].

These results led to the concept of **permissive** atelectasis – already established as the **lung rest strategy** in the treatment of ARDS [41,42] – being applied to intraoperative ventilation. Minimal PEEP is used, and the development of atelectasis tolerated under the assumption that only a small area of lung will be affected, and that lung tissue within that area is protected from ventilation induced forces. In return, the negative haemodynamic consequences of high PEEP are avoided and global static stress on the lungs is reduced [21]. It is important to mention, however, that the concept of permissive atelectasis is based on the PROVHILO trial, which examined patients of normal weight without severe pulmonary disease undergoing open abdominal surgery, the results of which may therefore not apply to other clinical situations. As such, in a prospective randomised trial on hypoxic patients following cardiac surgery, Costa Leme et al. were able to show a decrease in postoperative pulmonary complications and reduced intensive care and hospital stay when an intervention consisting of high PEEP and aggressive recruitment was implemented [43].

There is currently no evidence suggesting that pulmonary complications in patients undergoing non-cardiac surgery can be avoided by using high PEEP. Other than when required to avoid intraoperative hypoxia, routine use of PEEP >10 cm H_2O cannot be recommended.

Significance of recruitment manoeuvres

PEEP together with recruitment manoeuvres, which involve applying airway pressure above the opening pressure of atelectatic areas, form the so-called **open lung strategy**, the aim of which is to recruit atelectatic areas of lung and stabilise those areas in an open condition. Recruitment manoeuvres without subsequent application of PEEP lead to reopening of atelectatic areas of lung, but renewed collapse occurs, increasing atelectrauma. On the other hand, applying PEEP without prior recruitment increases the global static pressure on the lungs, increasing the risk of barotrauma and volutrauma. Clinical trials have shown opening pressures of up to 40 cm H_2O for patients of normal weight, increasing to up to 50 cm H_2O in the overweight [44,45]. Various recruitment manoeuvres have become established in clinical practice. For volume-controlled ventilation, the required opening pressure can be achieved by a stepwise increase in tidal volume whilst ventilating at a low respiratory rate. For pressure-controlled ventilation, the effect can be achieved ventilating at a low respiratory rate and increasing PEEP step by step whilst maintaining a fixed driving pressure. When compared with the classic **bag squeeze**, this approach provides more haemodynamic stability and better control over inspiratory pressure [46].

The majority of currently available clinical trials compared ventilation strategies comprising of PEEP plus recruitment manoeuvres with ZEEP (**zero PEEP**) or minimal PEEP without recruitment manoeuvres as the clinical standard, and the intervention was typically associated with improved intraoperative pulmonary function. Individual trials have also shown that recruitment manoeuvres alone improved gas exchange during intraoperative lung protective ventilation [47]. With the exception of Costa Leme et al., whose trial included a highly select patient cohort [43], no other trials have shown an effect of PEEP plus recruitment manoeuvres or recruitment manoeuvres alone on clinical outcome. A meta-analysis confirms this [48]. Based on atelectasis detected on computed tomography, a relatively small trial involving non-abdominal surgical patients recently confirmed that routine lung recruitment is unnecessary for a good many surgical interventions, so long as moderate PEEP (7–9 cm H_2O) is applied [49].

Recruitment manoeuvres can improve intraoperative pulmonary function, but not clinical outcome. When impaired intraoperative pulmonary function makes a recruitment manoeuvre necessary, PEEP should subsequently be increased.

Significance of individualised ventilation strategies

Despite discussions of the large randomised trials calling for individualised ventilation strategies, there is still no evidence that this approach can avoid postoperative pulmonary complications.

It would appear intuitive that ventilation parameters should be adjusted according to individual patient characteristics such as body height, approximating lung size. One simple way to accommodate that wish is to use predicted body weight, which can be determined either using a simplified calculation (body height [cm] - 100) or appropriate formulae (males: 50 + 0.91 x (body height [cm] - 152.4); females: 45.5 + 0.91 x (body height [cm] - 152.4)). The tidal volume should then be adjusted in accordance with the predicted body weight (e.g. 6–8 ml/kg). To judge inspiratory distension of the lungs (the relationship between V_T and lung capacity) and as such strain as a potential indicator of risk of ventilator induced lung injury would require intraoperative determination of end-expiratory lung volume, something which is currently only possible using elaborate technical means (computed tomography, nitrogen washout). In contrast, the **driving pressure** (defined as Pplat – PEEP [cm H_2O]) or **dynamic compliance of the respiratory system** are readily available parameters displayed by the ventilator which – taken together with V_T – represent the power transferred by the ventilator to the respiratory system. A meta-analysis based on individual patient data showed that higher intraoperative driving pressure was associated with an increased rate of pulmonary complications [50]. However, to date it remains unclear whether individual measures aimed at decreasing the driving pressure – such as recruitment manoeuvres or adjusting PEEP – can directly avoid complications.

A Spanish multicentre trial involving patients undergoing abdominal surgery (IPROVE) was not able to avoid postoperative pulmonary complications despite using individualised PEEP settings and recruitment manoeuvres [51]. The average titrated individualised PEEP based on the maximum compliance of the respiratory system was 8 cm H_2O, so that patients in the study groups were ventilated with an average PEEP of 10 cm H_2O, whilst PEEP of 5 cm H_2O was used for the control group. Although the intraoperative driving pressure was 2–3 cm H_2O lower during ventilation with higher static pressures when compared with the control group, and intraoperative rescue manoeuvres and hypoxia were avoided completely, no significant effect on morbidity was shown despite additional individualised postoperative measures such as non-invasive CPAP ventilation.

The question upon which criteria individualised PEEP should be based is also intriguing. In addition to the compliance of the respiratory system as used in the IPROVE trial, select centres and clinical trials have been using bedside **electrical impedance tomography** (EIT) with the aid of which intra-tidal overdistension and end-expiratory collapse of lung units can be visualised. An individually optimised PEEP could aim to minimise both these key mechanisms of lung injury. For patients with a median BMI of approx. 30 kg/m² PEEP titrated by this means lies between 6 and 16 cm H_2O and so shows significant heterogenicity (Fig. 2) [52]. In view of the questionable impact on clinical outcome, these methods are still too complex for everyday clinical use; instead, relatively simple monitoring of ventilation pressure curves with regard to tidal overdistension or recruitment (so-called **stress index under volume-controlled ventilation**) may be a serious alternative [53].

Individualised ventilation strategies can improve intraoperative pulmonary function, but do not seem to exert a positive influence on clinical outcome.

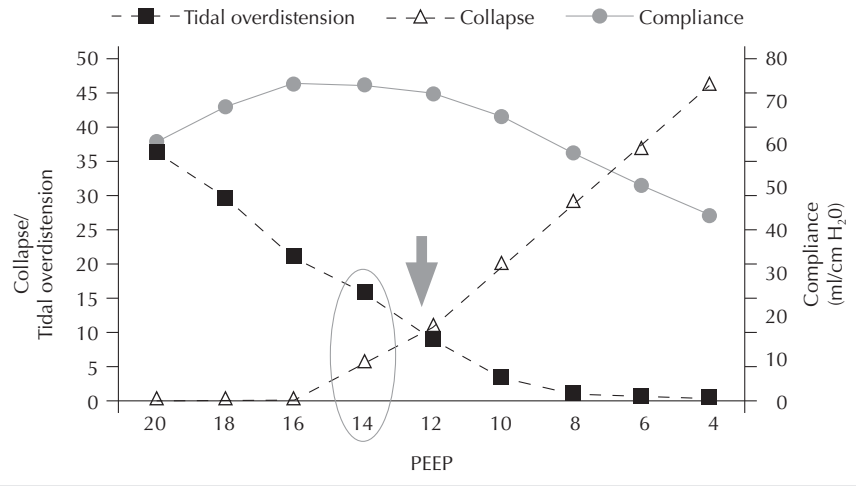
Intraoperative protective ventilation for special situations

The obese patient

Obesity seems to be a risk factor for use of higher intraoperative tidal volumes [54,55], although there is actually no reason to **routinely** ventilate obese patients with a higher tidal volume. The patients themselves are more likely to compensate their increased breathing effort with a higher respiratory rate.

The only study which primarily examined the effects of various tidal volumes in obese patients showed an improvement in oxygenation when higher tidal

Figure 2



Individually optimised PEEP based on electrical impedance tomography (EIT). Extent of tidal overdistension (squares) and alveolar collapse (triangles) determined using EIT at various levels of positive end-expiratory pressure (PEEP). An optimum individualised PEEP would appear to be present at a level of 12 to 14 cm H_2O, at which collapse and overdistension are equally minimised and ventilation undergoes best possible homogenous distribution. Compliance was simultaneously recorded and shows a similar optimum PEEP (modified from [52]).

volumes were used; alternatively, the same effect was also achieved using higher PEEP [56]. The results, however, were published in 1978, and as such at a point in time when the negative implications of extensive tidal volumes on lung structure were unknown or neglected, and their use represented the clinical standard. Today, ventilation with a tidal volume based on predicted body weight should be the standard for lung protective ventilation even in obese patients.

In obese patients, the loss of end-expiratory lung volume with induction of general anaesthesia is relatively greater than in patients of normal weight. That loss can be compensated in obese patients particularly by recruitment manoeuvres with subsequent sufficient PEEP, thereby reducing driving pressure and improving oxygenation [57]. To achieve best possible homogenous distribution of intraoperative ventilation, individual PEEP of up to 26 cm H_2O is required for morbidly obese patients [57]. Eichler et al. confirmed these results in patients with a median BMI of 50 kg/m^2 in whom end-expiratory transpulmonary pressure measured via oesophageal catheter (cf. section 'pneumoperitoneum') was adjusted to a positive value using suitable PEEP [59]. This concept assumes that alveoli are open under positive transpulmonary pressure but collapse under negative pressure. With respect to those criteria, this trial showed suitable PEEP to be approx. 17 cm H_2O and 24 cm H_2O for those undergoing pneumoperitoneum.

However, the positive intraoperative effects of high PEEP are temporary; when postoperative CPAP is forgone, the effects are no longer demonstrable briefly after weaning and extubation [57,58]. This observation is commensurate with the results of the large, multicentre, randomised PROBESE trial involving more than 2000 obese patients who were randomised to relatively low PEEP of 4 cm H_2O without planned recruitment manoeuvres or to PEEP of 12 cm H_2O with hourly recruitment [60]. The trial included predominantly abdominal surgical interventions, and of those most were laparoscopic. Although intraoperative driving pressure was reduced by more than 5 cm H_2O in those with higher PEEP, the authors did not find any difference in the occurrence of postoperative pulmonary complications. In contrast, higher PEEP protected from intraoperative hypoxia but was also associated with higher vasopressor requirements a well as a greater rate of intraoperative hypotension and bradycardia.

So, although measures taken to improve lung ventilation (**open lung strategy**) can have significant positive effects on intraoperative pulmonary function, to date evidence is lacking to show that such measures are associated with better postoperative outcome. As such, widespread use of higher levels of PEEP cannot be recommended even for obese patients.

Obese patients suffer a higher rate of perioperative pulmonary complications when compared with patients of normal weight. Tidal volumes should be strictly governed by predicted body weight – a fact currently not adequately recognised in everyday clinical routine. Use of higher levels of PEEP to improve postoperative outcome is not recommended.

Pneumoperitoneum

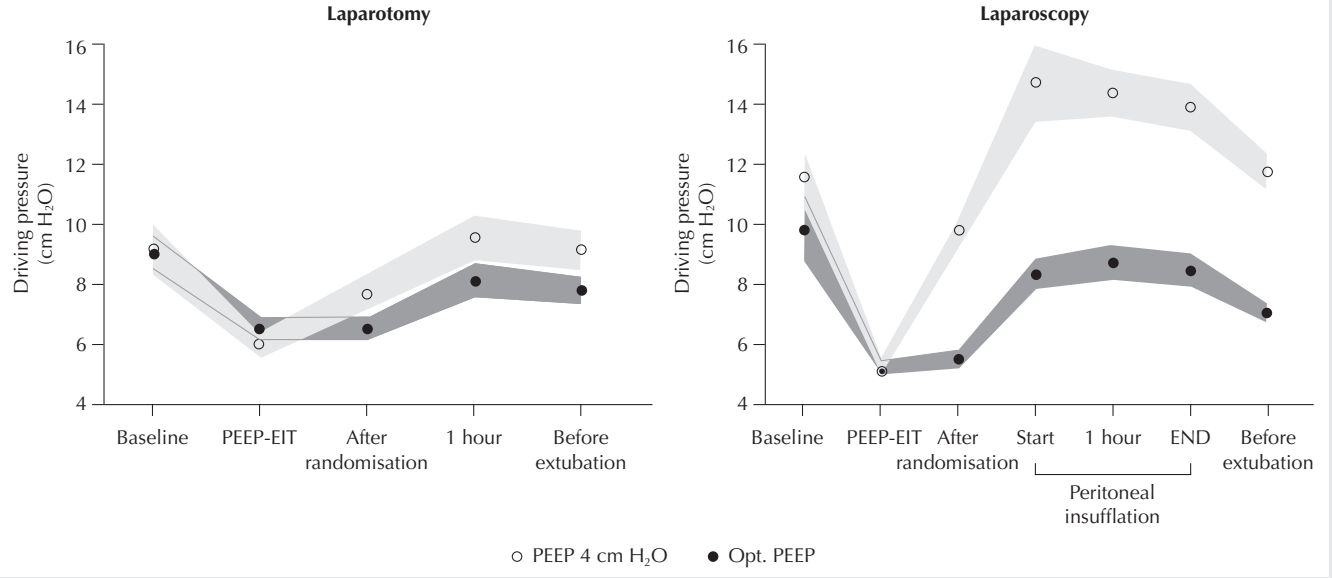
Insufflation of carbon dioxide (CO_2) into the abdomen in the context of minimally invasive surgery leads to significant changes to the mechanical characteristics of the respiratory system. On the one hand, thoracic compliance changes with cranial shift of the diaphragm; on the other hand, compressive atelectasis leads to a reduction in end-expiratory lung volume. Conventional monitoring, e.g. of parameters relating to respiratory mechanics as provided by the mechanical ventilator (plateau and peak pressures, compliance), is not suitable for quantifying these two (lung/thorax) mechanical components. As such, faced with an increase in plateau pressure during otherwise unchanged volume-controlled ventilation, the anaesthetist will be unable to discern whether this is solely as a result of CO_2 insufflation into the abdomen with a subsequent change in thoracic compliance or whether it instead represents an actual mismatch between tidal volume and end-expiratory lung volume causing increased strain and potentially lung damage.

Airway pressures resulting from insufflation and positioning of the patient mean that strict limitation of those pressures as is clinical standard in ventilation of patients with ARDS cannot as a concept simply be applied to the setting of minimally invasive surgery. One option to estimate pressure acting directly on the lungs is to measure transpulmonary pressure as the difference between airway and pleural pressure. Pleural pressure can be approximated by determining oesophageal pressure which, however, requires insertion of a special oesophageal catheter. To date, however, there is no evidence that adjusting ventilation parameters, and especially PEEP, in accordance with transpulmonary pressure measurements can avoid postoperative pulmonary complications. Patients undergoing prolonged surgical procedures or extreme positioning, such as required for robot-assisted surgery for example, might profit from such respiratory interventions.

It has been shown that recruitment manoeuvres, with increased PEEP where applicable, reduce atelectasis and improve intraoperative pulmonary function – to a fundamental extent in some cases (Fig. 3) [52,61,62].

The increase in arterial CO_2 caused by transperitoneal resorption of insufflated gas can require a significantly increased respiratory minute volume. In the context of reduced lung volumes and increased cyclic distension, increasing alveolar ventilation primarily by increasing tidal volumes may cause further VILI. Increasing the respiratory rate is in and of itself a useful measure but may, in the context of increased airway resistance in the setting of pneumoperitoneum, limit expiratory flow. It is for this reason that monitoring of intrinsic PEEP and/or

Figure 3



Intraoperative driving pressure during laparotomy compared with laparoscopy. The patient cohort ventilated with an optimised, higher PEEP (black dots; the dark shadow represents the 25th–75th percentile) shows a relatively small decrease in driving pressure during open surgery (left), but a significant reduction of approx. 6 cm H_2O during laparoscopic surgery when compared with ventilation with PEEP 4 cm H_2O. It is currently unknown whether this type of apparently lung protective ventilation results in a decrease in postoperative pulmonary complications (modified from [52]).

expiratory flow curves on the ventilator is paramount. Limitation of expiratory flow can be obviated by increasing expiratory time, which in turn is associated with higher peak airway pressure due to decreased inspiratory time. That higher peak airway pressure does not necessarily reflect the pressure at the level of the alveoli and may instead be primarily associated with higher airway resistance in the face of increased flow.

The aggressivity with which these respiratory measures are pursued can be tempered, however, if moderate hypercapnia based on pH is tolerated. Contraindications for this approach include any intracranial pathologies which would be associated with rising intracranial pressure in the face of increased p_aCO_2. Hypercapnia should also only be established with caution in those patients showing cardiocirculatory instability. Although positioning the patient can in theory significantly influence respiratory mechanics, positive use in the context of intraoperative respiratory difficulties is limited by surgical requirements.

Obesity and pneumoperitoneum are special challenges in intraoperative ventilation. In both cases higher PEEP and recruitment manoeuvres can improve intraoperative pulmonary function. However, it remains unclear whether clinical outcome can be influenced to the better.

Recommendations for intraoperative protective ventilation

Despite the fact that trials of intraoperative ventilation strategies have in some cases shown divergent results, it is possible to derive recommendations for lung protective ventilation, which could in turn contribute to a reduction in postoperative pulmonary complications. These recommendations can only apply to patients not suffering serious preexisting lung disease as those with such disease were not represented in relevant trials. Tidal volumes reduced to 6–8 ml/kg PBW should be the standard for intraoperative ventilation. Meta-analyses have demonstrated the potential for this measure to decrease postoperative pulmonary complications [31]. For patients of normal body weight, low levels of PEEP (such as 2 cm H_2O) can be used. Whilst higher levels of PEEP can improve intraoperative pulmonary function, they do not contribute to a decrease in postoperative pulmonary complications but do have the potential to impair haemodynamic stability. Even at low levels of PEEP sufficient oxygenation can be achieved in the majority of non-obese patients [39].

Hypoxic patients can profit from increased PEEP and recruitment manoeuvres [43]. Driving pressure can be used to find an adequate PEEP. If increasing of PEEP leads to an increase also of driving pressure, reduced compliance and with that increased stress on the lungs may be assumed. Conversely, if the driving pressure decreases, increased compliance and with that successful recruitment may be assumed [50].

Obese patients and those undergoing laparoscopy can profit from improved

pulmonary function when higher levels of PEEP and, when necessary, recruitment manoeuvres are used [57,61]. In obese patients, however, such measures have not been shown to have any further positive effect e.g. on postoperative complications, whilst instead intraoperative complications such as hypotension and bradycardia are more likely to occur.

New theoretical concepts which take the power transferred from the ventilator to the patient – rather than the two classical parameters tidal volume and airway pressure – to be the key aspect in the pathophysiology of ventilator induced lung injury, and which take all the relevant parameters – so tidal volume, airway pressure, flow, respiratory rate and I:E ratio – contributing to that power into account may, in future, help improve the lung protective aspect of mechanical ventilation [63].

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