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# Alveolar Recruitment Maneuvers for One-Lung Ventilation During Thoracic Anesthesia

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**Abstract** Especially during thoracic surgery, anesthesiologists have to cope with both the drastic alteration of normal pulmonary physiology and the mechanical stress on the lung tissue induced by ventilation. These problems result from the total collapse of the lung being operated on and the partial collapse of the ventilated other lung. In addition, technical factors inherent to thoracic surgery and the patient's condition potentiate such adverse effects. Traditional ventilatory settings described for thoracic surgery have not led to a decrease in the incidence of hypoxemia or perioperative atelectasis. However, lung recruitment maneuvers and ventilatory strategies are aimed at resolving lung collapse in mechanically ventilated patients, improving lung function, and decreasing the rate of post-operative pulmonary complications directly related to lung collapse. This article summarizes recent evidence for the role that lung recruitment maneuvers play during one-lung ventilation anesthesia.

**Keywords** Lung recruitment · PEEP · Atelectasis · Thoracic surgery · One-lung ventilation · Ventilator-induced lung injury (VILI) · Hypoxemia

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## Introduction

One-lung ventilation (OLV)—a technique that excludes the operated lung from ventilation—is one of the most challenging procedures for anesthesiologists. OLV must assure an optimal surgical field while at the same time maintaining an adequate gas exchange and minimizing the mechanical injury to the ventilated lung [1, 2]. Individualized positive-pressure mechanical ventilation is the key to accomplishing the above goals but very much depends on how well the operated non-ventilated lung is isolated from the other, ventilated lung.[1] However, mechanical ventilation under these circumstances is a two-edged sword: it is life-saving and injurious at the same time. To date, it is well known that ventilator-induced lung injury (VILI) has a negative impact on patient outcome [2–5, 6•, 7••].

Increasing evidence suggests that mechanical ventilation induces VILI even in patients with healthy lungs, such as during general anesthesia [5, 6•, 7••]. The origin of VILI resides in the high stress that mechanical breaths induce within collapsed lung zones and the high strain on normally ventilated areas which receive excessive ventilation [5, 8]. VILI is clearly associated with post-operative complications such as atelectasis, hypoxemia, pneumonia, or acute respiratory distress syndrome (ARDS) [4, 7••]. VILI has an even higher adverse impact on critically ill patients undergoing complex surgery like thoracic interventions performed under OLV [2, 4].

This new knowledge calls for the intra-operative use of protective ventilatory strategies based on low tidal volume

[1] Nowadays, many thoracic surgeries are performed with different body positioning besides the classical lateral decubitus. Therefore, in this paper, we write about *ventilated* or *non-ventilated* lungs instead *dependent* or *non-dependent* ones.

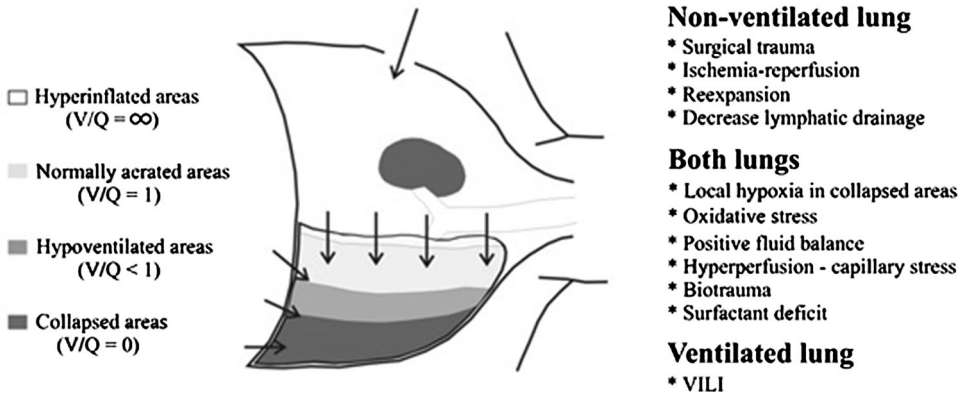


**Fig. 1** Schematic representation of the mechanisms behind deficiencies in exchange and lung injury in thoracic surgeries with one-lung ventilation. In thoracic surgery, the operated lung is totally collapsed and the ventilated lung becomes compressed by abdominal viscera through a dysfunctional diaphragm, by the mediastinal weight, and by the surgeon's activity (*arrows*). Causes of a deterioration of gas exchange are schematically depicted on the *left*. These range from pulmonary areas without ventilation (shunt = *V*/*Q* 0) to overinflated areas (dead space = *V*/*Q* ∞). Main potential causes of lung injury during thoracic surgeries are listed on the *right*

ventilation and limited airway pressures [5, 6•, 7••]. However, the main shortcoming of such protective concepts is that the root cause of lung injury—mechanical ventilation of a partially collapsed lung—although attenuated is not totally abolished. Therefore, an optional ventilator treatment necessarily consists of a deliberate combination of the above protective ventilation with a lung recruitment maneuver (RM) [9, 10••].

RMs are ventilatory strategies aimed at restoring a normal functional residual capacity even during adverse circumstances such as thoracic anesthesia [11–13]. RM re-aerate atelectasis and areas of airway closure thereby increasing the alveolar surface for gas exchange and decreasing the mechanical stresses which are the origin of VILI.

The objective of this paper is to analyze the impact that RMs have on gas exchange and lung injury during thoracic surgery with OLV.

## Gas Exchange During Thoracic Surgeries with One-Lung Ventilation

The mechanisms leading to an impaired gas exchange during OLV are depicted in Fig. 1. The unfavorable ratios of local ventilation to perfusion (*V*/*Q*) created by both the total collapse of the operated/non-ventilated lung and the different states of aeration and perfusion in the ventilated lung lead to inefficiencies of gas exchange, witnessed clinically as hypoxemia and hypercapnia of different degrees [14, 15].

The incidence of hypoxemia during OLV ranges from 1 to 24 % and depends, among other factors, on the correct placement of the double-lumen tube, the use of bronchoscopes, the patient's underlying disease, the body position during surgery, the kind of surgery performed, the ventilatory settings used, and anesthesiologist's experience in thoracic surgery [16, 17]. Originally, shunting through the non-ventilated lung was thought to be the main reason for hypoxemia [18], although a considerable part of the total shunting also results from the perfusion of atelectatic areas within the ventilated lung [14, 19]. Beyond pure atelectasis, areas with a low *V*/*Q* due to airway collapse also contribute significantly to the deficiencies in arterial oxygenation typically seen during OLV [20].

Hypercapnia is another common problem of gas exchange observed in 10–30 % of all thoracic surgeries [21]. Nowadays, the incidence of hypercapnia is increasing due to the use of low tidal volumes for reasons of lung protection. Hypercapnia depends on the same factors already described above for hypoxemia but with another confounding factor, the re-inhalation of $CO_2$ from the large instrumental dead space of typical double-lumen tubes. Although hypercapnia induces respiratory acidosis, a slight increase in $PaCO_2$ might even be beneficial for most patients, as it is known to augment the hypoxic pulmonary vasoconstriction reflex, to increase oxygen delivery to tissues, to protect the lungs from injury, and to decrease the hospital length of stay after major surgery [22].

## Lung Injury in Thoracic Surgeries with One-Lung Ventilation

A non-cardiogenic protein-rich inflammatory edema is characteristic for the injured alveolar-capillary membrane after thoracic surgery with OLV [23]. The mechanisms leading to this lung injury are complex and are present in both the ventilated and the non-ventilated lung (Fig. 1; Table 1). The synergistic negative effects of these

**Table 1** A rationale approach to decrease lung injury during thoracic surgeries

| Mechanisms of lung injury | Evidence | Author's suggestions |
|---|---|---|
| Surgical trauma | Lung injury to the operated lung is proportional to the surgical aggression | Consider minimally invasive and video-assisted surgery whenever possible |
| Ischemia–reperfusion (I–R)<br>Oxidative stress | Lung injury to the operated lung caused by I–R and oxidative stress are well-known problems during and after OLV | Decrease OLV time; use the lowest $FiO_2$ possible; re-expand the operated lung with a low $FiO_2$ |
| Fast lung re-expansion after OLV | Abrupt and fast re-expansion of the operated lung with high driving pressures and volumes increases the stress in lung tissue | Re-expand the operated lung with a ventilator instead of a bag using a controlled cyclic step-wise and slow recruitment maneuver similar to the alveolar recruitment strategy |
| Local hypoxia by atelectasis | A mild inflammatory response develops locally in collapsed areas of the lungs | Decrease OLV time<br>Treat lung collapse in the ventilated lung by a recruitment maneuver |
| Positive fluid balance | Excess of intravascular fluid is an independent risk factor for ARDS in patients undergoing thoracic surgeries | Apply goal-directed fluid therapy keeping normal cardiac output and oxygen delivered at the lowest amount of i.v fluids possible<br>Low doses of vasoactive drugs help maintain a neutral fluid balance in case of vasodilation<br>Consider inhaled B2 agents to decrease pulmonary edema<br>Consider hydrocortisone to preserve the endothelial glycocalix |
| Capillary stress failure | Hyperperfusion of lung tissue caused by OLV, vascular clamping and declamping, and excess of inotropic vasoactive drugs injures the alveolar-capillary membrane | Perform vascular clamping and declamping slowly and avoid high doses of inotropic and vasoactive drugs |
| VILI | Tidal recruitment and overdistension is the main causes of VILI | Apply a goal-directed ventilatory strategy that consists of an active recruitment maneuver and sufficiently high PEEP ($10 \pm 2$ cm $H_2O$) to keep the lungs open<br>Ventilate the open lung with a protective pattern at very low VTs (4 mL/kg) and low plateau pressures |

damaging mechanisms support the *multiple hit theory* which tries to link the local inflammation process within the lungs with the systemic inflammatory response [24]. Both these inflammations can potentiate each other and may even lead to an ARDS in 1–7 % of lung resections [2, 3].

These multiple insults affect the lungs in different ways and to different degrees. The operated lung has to cope with an injury that is related to the extent of the *surgical trauma* [25, 26]. In general, the degree of lung injury is proportional to the surgical aggression which has already been described for all kind of thoracic procedures including minimally invasive ones. Thus, Yim et al. [25] demonstrated that video-assisted surgery is related to lower plasmatic cytokine concentrations when compared to open surgeries. More invasive surgeries for cancer may directly affect the pulmonary *lymphatic drainage* which aggravates non-cardiogenic edema formation.

Another important mechanism of lung injury during OLV is the *ischemia* of the non-ventilated lung and its sequential *reperfusion* when ventilation is restored [27]. Hyperoxia in the ischemic operated lung at the onset of ventilation leads to an increase in reactive oxygen species, in cell damage, and in local leukocyte infiltration. The generation of free oxygen radicals is proportional to the duration of OLV [27–29].

The *re-expansion* of the operated lung at the end of surgery—a kind of lung recruitment "maneuver" commonly performed by manual bagging—could be an additional cause of lung injury beyond the one caused by ischemia reperfusion. There are a few reports showing that RMs enhance the expression of inflammatory mediators [30, 31, 32••, 33]. However, the potentially deleterious effect of RMs is related to the way they are performed and especially to their timing, the driving pressures, and volumes applied [9, 34••]. There is evidence that "fast" lung

recruitments induced local inflammation in an experimental model of ARDS. Recruitment maneuvers performed as sustained inflation (SI) typically increase airway pressures and lung volumes abruptly, and by their non-physiological nature create stress on the fragile lung tissue. Such stresses can be minimized by applying cyclic step-wise RMs [30, 31, 32••, 33, 34••].

The *local hypoxia* observed in atelectatic areas also induces a mild lung injury with an inflammatory response mediated by macrophages [35]. Duggan et al. [36] described histological damage and increased lung permeability in atelectatic areas of rat lungs, which are attenuated by oxygen.

The chance of developing lung injury after major surgery increases with a *positive fluid balance* [37]. This is also true for thoracic surgery as described by Licker et al. [38], who found that a positive fluid balance was an independent risk factor for developing ARDS. A positive fluid balance augments edema formation in previously injured lungs due to both an increased hydrostatic capillary pressure and a decreased plasmatic oncotic pressure. In addition, the endothelial glycocalix—an internal protective layer that prevents plasmatic proteins and water from leaving the vascular system—can be damaged in thoracic surgery by fluid overload as well as by lung trauma, ischemia reperfusion, local hypoxia, and inflammatory response [39].

*Transfusion-related lung injury* is a known entity that can especially affect critically ill patients undergoing thoracic surgery. Even if euvolemia is maintained, blood products are known to cause neutrophil sequestration and activation within the lungs [40].

A sudden increase in pulmonary blood flow with hyperperfusion of the ventilated lung during OLV can damage the lungs by a *capillary stress failure* [41]. Temporary hyperperfusion is commonly observed in thoracic surgery [20], especially during the clamping and de-clamping of the pulmonary artery and with the abuse of inotropic and vasoactive drugs. López-Aguilar et al. [41] determined the role that capillary blood flow and pressure play in inducing lung damage. They found in an animal model that high pulmonary capillary blood flow and pressure increased the score of histological alveolar and endothelial injury when compared to low flow and pressure conditions.

The main mechanism of lung damage in the ventilated lung is the mechanical injury that the ventilator induces—in the true sense of the term *ventilator-induced lung injury*. However, the concept of VILI has changed over the last 30 years [9]. Initially, the main causes of VILI were thought to be *barotrauma* and *volutrauma*, and thus the damage caused by high airway pressures and VTs, respectively. Despite the fact that airway pressures and tidal volumes have progressively been reduced over past decades, to date, VILI remains an important issue. It is now considered to be mainly due to *tidal recruitment* and *tidal overdistension* [42–44]. Tidal recruitment refers to the cyclic opening and closing of unstable airways and alveoli during a breathing cycle. This repetitive opening and closing of lung units induces shear stress within the boundary between stable and unstable units. Such damage can thus occur whenever ventilated lungs are partially collapsed. Tidal overdistension, on the other hand, can be observed whenever excessive inspiratory flows are directed primarily towards normally ventilated areas. Such airflow strains these areas beyond their elastic limit.

## Ventilatory Management During OLV Anesthesia

### Protective Ventilation

Radical changes in the way patients with acute lung injury are ventilated in intensive care medicine have been observed during the last years. It is now an accepted fact that mortality and ventilator-free days in ARDS patients are lower if they are ventilated with 6 rather than 12 mL/kg predicted body weight (31 vs. 40 %, respectively; $p = 0.07$) [45]. In many institutions throughout the world, such *protective ventilation* with low VT and plateau pressures with an associated permissive hypercapnea and the use of PEEP are considered standard treatment for ARDS patients.

The legacy of such novel insights was delayed in the anesthesia world since the traditional opinion was that "healthy" lungs do not need any special attention. However, new evidence unequivocally shows that even healthy lungs can be damaged by inadequate mechanical ventilation. Thus, Gagic et al. reported that 24 % of 332 patients without ARDS who received mechanical ventilation in the ICU developed acute lung injury after a few days. The main risk factor associated with such lung injury was the size of the VT applied (odds ratio 1.3 for each mL above 6 mL/kg of predicted body weight; $p < 0.001$) [5]. Similarly, Serpa Neto et al. [6•] in a meta-analysis of 2.822 ICU patients without ARDS showed a decrease in lung injury, pulmonary infection, hospital length of stay, and mortality with low VT ventilation. Comparable findings were observed in the operating theatre in patients with healthy lungs. In a recent meta-analysis including 1.669 anesthetized patients, Hemmes et al. [7••] showed that low VT at high PEEP ventilation (with and without RM) decreased the risk of lung injury, atelectasis, and pulmonary infection compared with high VT ventilation at low PEEP.

Of all ventilated lungs, those treated with OLV during thoracic surgeries are the ones most prone to VILI, mainly because the traditional ventilatory strategies still

commonly used in clinical practice are not only "non-protective" but must be considered potentially injurious, as they employ relatively high VTs for just one single lung together with high $FiO_2$ and low or no PEEP at all. Thus, studies could show a pulmonary production of pro-inflammatory cytokines during OLV [46, 47], which was more pronounced in the ventilated than in the operated lung [48••, 49]. This local inflammation in thoracic surgery was associated with a high incidence of postoperative pulmonary complications [50].

The latest evidence suggests that protective ventilation decreases VILI during OLV. Schilling et al. [51] demonstrated that ventilation during OLV with low VT (5 mL/kg) compared to high VT (10 mL/kg) partially decreases the pulmonary expression of pro-inflammatory cytokines and neutrophil infiltration. Similarly, Michelet et al. [52] found that patients undergoing esophagectomies with OLV who were ventilated with VT 5 mL/kg at 5 cm $H_2O$ of PEEP showed lower cytokine levels than those ventilated with a VT of 9 mL/kg without PEEP.

The attenuation of the pulmonary inflammatory response by using protective ventilation during OLV has important positive clinical repercussions. Protective ventilation during OLV compared with high VT and ZEEP showed fewer postoperative pulmonary dysfunctions and a more satisfactory gas exchange [53••]. In a retrospective analysis, Licker et al. [2] showed that ventilation with VT of 5 but not 7 mL/kg during OLV was associated with a lower incidence of atelectasis and acute lung injury, fewer admissions to the ICU, and a shorter hospital stay.

Although ventilation with low VT, plateau pressures, and PEEP decreases the pulmonary inflammatory response, such a ventilatory strategy cannot totally abolish VILI during OLV [48••, 49]. This is because protective ventilation inherently promotes lung collapse due to the use of low VT and high $FiO_2$ [54], and thus the damaging effects of tidal recruitment can still act in the partially collapsed ventilated lung.

## Recruitment Maneuvers and the Open Lung Concept

Lachmann proposed another ventilatory strategy for ARDS patients, which consists of two main components: (1) an active opening up of collapsed lung units by a RM, and (2) the use of sufficiently high levels of PEEP to keep lung units open [55]. The rationale of any RM is to restore the FRC in order to normalize lung function and to avoid the mechanisms of VILI. Amato et al. [56] described an improved outcome of patients with ARDS when protective ventilation was combined with a RM.

The adoption of such a kind of protective ventilation in the field of anesthesia was delayed compared to intensive care medicine. Already in 1993, Hedenstierna's group in Sweden had described the patho-physiology around lung collapse and the physiological basis of sustained inflations—then called vital capacity maneuvers [11]. Shortly thereafter, our group demonstrated the beneficial clinical effect of a cycling RM on anesthesia-induced atelectasis [12, 13]. RM has been demonstrated to be safe in a wide variety of species, patients, and surgical procedures, including thoracic ones performed under OLV [13, 57••].

Information about RMs as potential triggers of the local inflammatory response or VILI is scarce. Only a few experimental and clinical studies provide first hints. On the one hand, experimental reports showed an increased expression of inflammatory mediators [30, 31, 32••, 33] with the SI-type RM but not with the cyclic type of RM (see below). On the other hand, additional experimental and clinical data showed that RM did not affect or even decreased the inflammatory response. Schilling et al. [57••] recently demonstrated that repetitive RMs did not induce any significant pro-inflammatory responses in healthy pigs undergoing OLV anesthesia. These data fit with clinical studies which showed that RM had no effect on the level of systemic inflammatory cytokines in critically ill ventilated patients with and without ARDS [58, 59]. Other experimental studies even showed a decreased inflammatory response when using RMs [60–62]. The cytokine attenuation during a RM could be due to the abolition of VILI as demonstrated by Kozian et al. [63••]. The authors showed that a RM combined with protective ventilation decreased tidal recruitment on CT images when compared to high VT ventilation in pigs undergoing OLV. Similar findings were obtained in cardiac surgery patients. Reis Miranda et al. [64] demonstrated that a RM associated with low VT and high PEEP decreased cytokines release as compared to a ventilation at VT 6–8 mL/kg and 5 cm $H_2O$ of PEEP.

These positive effects of RM on VILI could be responsible for the decreased rate of post-operative complications in anesthetized patients. A recent meta-analysis showed that the risk of lung injury, atelectasis, and pulmonary infection decreased with protective ventilation during anesthesia [7••]. However, this analysis included studies with and without RM and, therefore, a final conclusion in favor of RM cannot be drawn. Recently, Futier et al. [10••] described that protective ventilation combined with RM improved clinical outcomes and reduced health care utilization in patients undergoing major abdominal surgery. Taking all this encouraging evidence together, it seem that cycling of RMs have a complementary protective effect on the lung tissue not only in thoracic surgery with OLV but in all mechanically ventilated patients.

The management of OLV has changed over the last decades. In the early days of thoracic anesthesia, awareness and knowledge about VILI were rare and, thus, ventilation with high VT and $FiO_2$ without PEEP was promoted [18].

**Table 2** Clinical studies related to the use of RM during OLV

| Author, year | Kind of study (number of patients) | Kind of surgery | Kind of RM | Main effects |
|---|---|---|---|---|
| Tusman et al. 2002 [19] | Prospective, case-series ($n = 10$) | Open lobectomies | Cycling RM in the dependent lung: 40/20 cm $H_2O$ of Ppl/PEEP for 10 breaths | RM increased $PaO_2$ and lowered airway pressure during OLV |
| Tusman et al. 2004 [66] | Prospective, case-series ($n = 12$) | Open lobectomies, thoracoscopies, minimal-invasive CABG | Cycling RM in the dependent lung: 40/20 cm $H_2O$ of Ppl/PEEP for 10 breaths | RM increased $PaO_2$ and decreased dead space variables during OLV |
| Cinella et al. 2008 [67] | Prospective, case-series ($n = 13$) | Open lobectomies, lung resections | Cycling RM in the dependent lung: 40/20 cm $H_2O$ of Ppl/PEEP for 6 breaths | RM increased $PaO_2$ and decreased respiratory elastance during OLV Transient decrease in cardiac output during RM |
| Garutti et al. 2009 [68] | Prospective, case-series ($n = 40$) | Open thoracotomy | Cycling RM in the dependent lung: 40/20 cm $H_2O$ of Ppl/PEEP for 5 breaths | RM improved arterial and venous oxygenation. Slight and transient effects on hemodynamics during RM |
| Park et al. 2011 [69] | Prospective, randomized, controlled study ($n = 40$) | Open lobectomies, pneumonectomies, wedge resections | Cycling RM in the dependent lung of treated patients: 40/20 cm $H_2O$ of Ppl/PEEP for 12 min | RM increased $PaO_2$ and compliance during OLV in the treated group compared to control group |
| Unzueta et al. 2011 [70••] | Prospective, randomized, controlled study ($n = 40$) | Open lobectomies | Cycling RM during two lung ventilation in treated patients: 40/20 cm $H_2O$ of Ppl/PEEP for 10 breaths | RM increased $PaO_2$ and compliance and decreased dead space during OLV in the treated group compared to control group |

Later on, reduced tidal volumes and PEEP of no more than 5 cm $H_2O$ became popular, while RM was still considered a rescue intervention in case of severe hypoxemia [65].

RM can eliminate approx. 1/3 of the total venous admixture that comes from the ventilated lungs [19]. Restoring the FRC of the ventilated lung by actively recruiting it improves gas exchange, although it will never be entirely normalized because the second lung is totally collapsed and not ventilated at all. However, in nearly all cases, maintaining the ventilated lung in a perfectly open condition suffices to prevent hypoxemia and to avoid complementary CPAP in the operated lung. According to our group's experience, RM in conjunction with the use of 4 mL/kg VT provides an optimal arterial oxygenation at a slight hypercapnea (personal unpublished data). RM with sufficient PEEP should avoid tidal recruitment because the ventilated lung is no longer collapsed. At the same time, they minimize tidal overdistension because the inspiratory flow is more homogeneously distributed within the more compliant lung.

A summary of the main published clinical studies on the use of RM in thoracic surgeries is given in Table 2. The first clinical report of a lung recruitment procedure in thoracic surgery was in 2002 [19]. A cycling RM was successfully applied and the clinical results during OLV were similar to the one already described in other kinds of surgery: improved arterial oxygenation and respiratory system compliance at decreased dead spaces [19, 66]. Subsequent studies confirmed these results. Cinella et al. [67] performed a RM in the ventilated lung and showed a higher $PaO_2$ and a reduced respiratory elastance. Garutti et al. [68] showed similar effects on arterial oxygenation after RM in pulmonary resections with OLV lasting longer than 1 h. Park et al. [69] also confirmed the benefits of a RM performed before OLV on $PaO_2$ and lung mechanics. Unzueta et al. described a reduction in alveolar dead space and a better $PaO_2$ during OLV after RM [70••].

Some questions remain regarding the way RM should be performed. One of the key questions is related to the level of recruitment pressures recommended. Details of published RMs before or during OLV are summarized in Table 2. Since most of the lungs during OLV may be considered "healthy" with respect to their recruitability, they usually require no more than 40 cm $H_2O$ to be fully expanded. In order to limit the shear stresses between open and closed lung units during the cycling recruitment, a PEEP of 20 cm $H_2O$ appears appropriate.

Another question is related to the *way* such pressures are best delivered to the lungs. Thus, inappropriate *timing* is an important factor associated with negative RM effects.

Alveolar recruitment per se is a rapid phenomenon which happens quickly during the intervention. However, shear stresses within the fragile lung tissue and hemodynamic side effects are mainly associated with "fast" RM and not with "slow" cycling RM [33]. This way, one-step SI maneuvers are more harmful than *cycling* step-wise RMs because SIs abruptly change pressures and volumes within lungs [30, 31, 32••]. Riva et al. [30] showed in an experimental model of ARDS that a slow RM caused less stress for the lung tissue than a fast RM. In another experimental study, Silva et al. [32••] found that SI showed more hyperinflation and activation of pro-inflammatory and profibrogenic mediators than cycling RMs. Based on these data, Marini recently suggested that RMs of the SI-type in ARDS patients must be eradicated from clinical practice and replaced by slow, step-wise RMs [34••]. It is only logical that such advice from the ICU world should also be followed in thoracic anesthesia because the risk for VILI and non-cardiogenic inflammatory edema—although of different magnitude and clinical repercussions—is present in all mechanically ventilated patients irrespective of the reason for such ventilator treatment.

An important issue is the *duration* of the application of high airway pressures needed to reach an optimal therapeutic effect. The time required is proportional to the lung condition: it is as short as a few seconds in healthy lungs and increases up to 2–5 min in ARDS patients [9]. Therefore, 10 breaths at highest recruitment pressure should be enough to open up the lungs during OLV anesthesia (Table 2).

Furthermore, the question of *how many* RM should be used during thoracic surgery needs to be answered. The answer depends on the details of the *surgery* and should be planned strategically. Some authors apply RMs during two lung ventilation and others only during OLV (Table 2). An initial RM must be performed immediately after anesthesia induction before OLV to make the patients benefit from the positive RM effects during the entire anesthesia time. In critically ill patients, in whom hemodynamic side effects of the RM are expected, such a maneuver can be applied during OLV, because one report demonstrated that a RM on the ventilated lung alone was hemodynamically well tolerated [68]. However, in general, the hemodynamic tolerance is related to a patient's preload status and is rarely a problem in normovolemic patients [9, 13, 71].

It must be emphasized that, in most cases, one single maneuver is enough to expand the lungs and to keep the lungs "open" during the entire anesthesia time provided sufficient PEEP is used [9, 13]. A second RM (or even more RMs) becomes necessary whenever the operated lung must be re-expanded, which is usually the case only once at the end of surgery. Following the reasoning described in this paper, a controlled and step-wise increase in lung volume and airway pressure performed using a ventilator should be safer than the traditional manual re-expansions with the anesthesia bag. A manual RM cannot control timing, volumes, or pressures and could thus be potentially harmful for the lungs [30, 31, 32••, 33, 34••].

The number of RMs needed to be performed in thoracic surgery also depends on how efficient such maneuvers are and on the level of PEEP chosen. The level of PEEP used after a RM must exceed the lung's closing pressure to keep the lungs "open" and free from collapse [9, 13]. Most studies conclude that RM effects are self-limited, and therefore RMs must be performed repetitively. However, according to Young–Laplace's law and our own experience, lung re-collapse after RM can only occur if the pressure within the lung is insufficient to keep them expanded. In other words, PEEP plays a central role in keeping the lungs expanded during the surgery. While sufficient PEEP is essential, it must be taken into account that too high levels of PEEP overdistend the ventilated lung and will increase its resistance to blood flow, which in turn shifts blood flow towards the shunting non-ventilated lung. As the lung's closing pressure varies among patients, it must be selected carefully to avoid de-recruitment. The optimum PEEP after RM is very difficult to determine at the bedside. We suggest using higher PEEP levels than the ones typically recommended for OLV (0–5 cm $H_2O$). In a recent study, Ferrando et al. [72] have determined in 15 patients that the optimum PEEP after a RM for OLV varies from 6 to 12 cm $H_2O$ (mean 10 $\pm$ 2 cm $H_2O$). These personalized levels of PEEP improved arterial oxygenation and decreased dead space compared to patients receiving a RM but only 5 cm $H_2O$ of PEEP.

## Conclusions

The paradigm of lung recruitment during anesthesia has shifted away from strategies primarily devised to improve gas exchange by re-aerating hypoventilated and collapsed areas towards ventilatory maneuvers that are aimed at protecting the lungs from VILI by reducing tidal recruitment and tidal overdistension. This is particularly important in thoracic surgery in which the risk for severe derangements of gas exchange and lung injury is particularly high. VILI is one of the causes of lung injury on which anesthesiologists can have a positive therapeutic impact. Beyond the clear benefits of alveolar recruitment maneuvers on VILI, its impact on patient outcome after thoracic surgery is as yet unknown and warrants further clinical studies.

### Compliance with Ethics Guidelines

**Conflict of Interest** Gerardo Tusman has received compensation from Maquet Critical Care for service as a consultant. Stephan H.

Bohm holds stock/stock options in Swisstom as part of salary. Fernando Suarez-Sipmann has received compensation from Maquet Critical Care for service as a consultant.

**Human and Animal Rights and Informed Consent** This article does not contain any studies with human or animal subjects performed by any of the authors.

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- • Of importance
- •• Of major importance


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