

Lung recruitment in trauma patients

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Abstract

The ventilatory management of trauma patients can present significant challenges. Its main aims focus on the reduction of the work of breathing, facilitation of an improvement in gaseous exchange, and on the avoidance of ventilation-induced lung injury (VILI). VILI is a pathophysiologic process consisting of several changes seen in ventilated lungs that have been attributed to the ventilation strategy employed subsequent to the traumatic lung injury. In order to avoid the major components of VILI, both maximal alveolar aeration and recruitment are needed to minimize the shear stresses in the injured lung tissue. Alveolar recruitment maneuvers have become the major goals of mechanical ventilatory support for patients with severe trauma over the last few decades. The importance of early lung recruitment with an 'open lung' approach has been well documented in several studies and its application has been shown to preserve lung mechanics, attenuate lung mechanotrauma, and thereby reduce mortality during ventilation. Controversies nonetheless exist regarding the most appropriate form of their use in patients with pulmonary injuries as recruitment maneuvers may have several adverse effects. The main concerns center on exploiting high airway pressures during the recruitment process and its potential harmful consequences, such as barotrauma and hemodynamic compromise. The purpose of this article is to review the major concepts in the mechanical ventilation literature that outline the principles for the use of the open lung management strategy in patients that have been subjected to significant trauma and to delineate both the major techniques of lung recruitment as well as their potential complications.

Introduction

Trauma is a major source of morbidity and mortality in the United States [1]. In patients with multiple systemic injuries, chest trauma is the cause of death in twenty to forty percent of cases [1] and may result in several pulmonary complications including tension pneumothorax and flail chest with pulmonary contusion [2]. A different approach and ventilatory strategy is required for each of these conditions.

Mechanical ventilation has become a critical therapy for such patients with impaired pulmonary function [3]. Certain modes of mechanical ventilation may however in themselves be associated with multiple potential adverse effects, such as the development of atelectasis, pneumonia, pulmonary fibrosis and pulmonary edema in addition to decreases in lung compliance and gaseous exchange [2,3]. Many of these pathophysiologic changes seen in trauma victims are attributed to the ventilation strategies used and are therefore called ventilator-induced lung injury (VILI) [4]. The main objective in the ventilatory management of patients with severe trauma should therefore focus not only on the reduction of work of breathing and improvement of gaseous exchange but also on the minimization of VILI.

This article reviews the pathophysiology and the principles governing the use of mechanical ventilation with alveolar recruitment in the management of trauma victims. The first part of this paper describes the role of VILI on the lung mechanics, the second part describes the recruitment principles of opening the lung, and the last part briefly outlines the complications of this strategy on different organ systems.

The role of ventilator-induced lung injury on lung mechanics

Pulmonary injury may result from underlying traumatic lung process or by way of the injurious application of mechanical ventilation. Evidence suggests that the pathophysiology of VILI is multifactorial (Table 1) and results from the combined effects of barotrauma, volutrauma, biotrauma and atelectrauma. These mechanisms can initiate a cascade of events that result in an increase in systemic inflammation. Stress failure, which is the mechanical disruption of the alveolar-capillary barrier, can subsequently cause the release of local inflammatory mediators that can spill into the systemic circulation [5]. Such inflammatory mediators that have been detected in the systemic circulation of lung injury patients, include interleukin-1, interleukin-6, interleukin-8, interleukin-10 and tumor necrosis factor- α (TNF- α) [6].

The predominant factors that contribute to VILI are a combination of overdistension of aerated lung units and high distending transalveolar pressures. This is related to high volumes and pressures that occur at the end of inspiration due to excessive stress at the margins between the

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Table 1. Pathophysiological Components of Ventilator-Induced Lung Injury [4].

Barotrauma	Ventilation with excessive end-inspiratory or plateau pressures
Volutrauma	Ventilation with excessive tidal or end-expiratory volumes
Biotrauma	Local and systemic release of inflammatory mediators
Atelectrauma	Ventilation below the lower inflection point on pressure volume curve with cyclic opening and closing of alveoli

nonaerated and aerated lung units [7]. When the transalveolar pressure falls below the critical closing pressure of alveolar units at the end of expiration it induces the repeated opening and closing of small airways such as alveoli and bronchioles that are atelectatic at end-expiration. It is these processes that define atelectrauma and are associated with an increased activation of pulmonary inflammatory mediators, which are released in both the lung and systemic circulation causing histological changes that are indistinguishable from acute respiratory lung syndrome (ARDS) [6].

Haitsma *et al.* [8] demonstrated that there is loss of compartmentalization of cytokines in VILI, causing an imbalance in the inflammatory response. These high levels of cytokines (particularly TNF- α) can trigger severe organ dysfunction. According to this study, the application of 10 cmH₂O of positive end-expiratory pressure (PEEP) significantly diminished this loss of compartmentalization. Furthermore, both the absence of PEEP and high peak pressures have been shown to increase bacterial translocation from the lung into the systemic circulation in several further animal studies [9]. D'angelo *et al.* [10] illustrated that the application of prolonged low-volume ventilation with no PEEP even in normally functioning lungs causes injury to peripheral airways.

There have been numerous mechanisms proposed to explain the change in lung mechanics in the presence of injury. Gattinoni *et al.* [11,12] reinforced the idea that large portions of an injured lung are derecruited and not aerated during positive pressure ventilation. Additionally, they found that dependent portions of the injured lung are exposed to a compressive pressure and collapse after examining the effects of ventilator settings and posture. Their findings showed that dependent atelectasis was due to the increased weight of edematous lung and that subsequent lung injury was determined to be caused by large stresses in the parenchyma surrounding these atelectatic regions.

It has also been shown that a critical factor leading to VILI is inadequate lung recruitment. The reason for this is that the adaptive processes to lung injury can initiate vascular cell proliferation and collagen deposition [13]. The potential for such detrimental changes can be avoided by lung recruitment and prevention of derecruitment thus avoiding the repetitive shear stress associated with opening and closing of unstable lung units [14]. Maximal alveolar aeration and recruitment are needed to minimize shear stresses in the lung tissue during inspiration [14]. The application of alveolar recruitment maneuvers should therefore be the major goals of mechanical ventilatory support for patients with severe trauma.

Recruitment principles

The use of low tidal volume and limited airway pressure ventilation is the only ventilatory strategy that has shown a decrease in acute respiratory distress syndrome (ARDS) mortality in several randomized studies. Two major randomized controlled clinical trials demonstrated that this protective ventilation approach leads to a marked improvement in clinical outcome [15,16]. It is however known that low tidal volumes lead to progressive lung derecruitment that can actually be detrimental

in patients with significant pulmonary contusions. Richard *et al.* [17] illustrated that the reduction of tidal volumes from 10 to 6 mL/kg, while keeping the PEEP constant, was responsible for significant lung volume loss corresponding to alveolar derecruitment. Furthermore, Cereda *et al.* [18] demonstrated the principle of derecruitment in a study showing that low tidal volume ventilation could induce a progressive decrease in compliance which could be prevented by using a higher PEEP level. The traditional mechanical ventilation approach as seen in patients with pulmonary contusion uses low to moderate levels of PEEP to support oxygenation by preventing or reversing alveolar atelectasis or flooding.

Halter *et al.* [19] demonstrated that when alveoli are opened with a recruitment maneuver they can still collapse without the use of adequate PEEP. Concurrently, those recruited alveoli that do not collapse are unstable and vulnerable to shear stress-induced damage. Therefore, a recruitment maneuver used without adequate PEEP could potentially exacerbate VILI [19]. Ranieri *et al.* [20] showed that PEEP mainly caused hyperinflation of those alveoli previously recruited by high tidal volume ventilation. On the other hand, with the use of low tidal volumes, the effect of PEEP was one of alveolar recruitment counterbalanced by derecruitment [20].

Amato *et al.* [15] demonstrated significant improvement in pulmonary function and a higher rate of weaning from the ventilator when lung protective ventilation was used in patients with ARDS. Their ventilation strategy was based on maintaining low inspiratory driving pressure with low tidal volumes and the preferential use of limited airway pressure, simultaneously using a higher PEEP to keep the end-expiratory pressures above the lower inflection point of the static pressure volume curve of the respiratory system.

It has also been shown, that in patients with ARDS, the use of a higher PEEP combined with lower tidal volumes is associated with a lower concentration of inflammatory cytokines and mediators in bronchoalveolar lavage fluid and blood [6]. One can thus say that both the PEEP and the tidal volume are interactive variables the appropriate selection of which determines the extent of lung recruitment.

Optimal positive end-expiratory pressure selection

The application of PEEP plays a fundamental role in the lung protective strategy with the aim of minimizing lung collapse [17]. It has several beneficial effects on gaseous exchange, edema formation, release of inflammation mediators and lung mechanics [2,3,17]. The extent of end-expiratory collapse depends on two phenomena: the maximum volume pressure achieved during the previous inspiration and the gravitational forces, which are the superimposed pressures that compress the most dependent regions of the lung. Crotti *et al.* [21] have shown that there are unique opening pressures related to different lung regions. It is the lowest in the nondependent lung, intermediate in the mid-lung, and highest in the most dependent lung region. Recruitment of the most dependent lung region associated with atelectasis typically occurred at pressures as high as 45 cmH₂O. Derecruitment was shown to occur by decreasing the PEEP level from the total lung capacity thereby causing progressive collapse of the most dependent regions, which were formally subjected to the greatest superimposed pressure. Furthermore, alveolar collapse was shown to occur maximally between a PEEP of 0 and 15 cmH₂O in patients with acute lung injury (ALI) or ARDS. The recruited lung thus, tends to stay open at pressures lower than the initial pressure that had been used to open it.

Based on the assumption that a lower inflection point (LIP)

represents alveolar recruitment, it is the LIP on the total respiratory system pressure-volume curve that is used extensively to set the PEEP in patients with acute respiratory failure [22]. This means that the LIP on the pressure-volume curve reflects the average critical pressure needed to reopen those regions of the lung that close during expiration [22].

The relationship between the LIP and the amount of alveolar recruitment has only been shown in a few studies. Muscedere *et al.* [23] described saline-lavaged lungs that were ventilated with PEEP both above and below the LIP of their static pressure volume curves in a rat model. Their findings showed that lungs that were ventilated without PEEP had significant decreases in compliance and marked histological injury. This is likely due to the resulting shear stress in airway walls from the distal airway collapse and inflammatory mediator release. The development of VILI in patients with ARDS may be a consequence of the same mechanism.

The relationship between LIP and alveolar recruitment was further investigated by Mergoni *et al.* [24]. The authors showed that there is an insignificant relationship between the amount of the alveolar recruitment determined by application of PEEP and the level of the LIP suggesting that recruitment had also occurred when the level of PEEP was raised above the LIP. The recruitment obtained by increasing PEEP to 10 to 15 cmH₂O was only forty percent of the whole recruited volume, when the initial level of LIP was less than 10 cmH₂O (in all but four patients). This suggested that a large amount of lung tissue was still recruitable when patients were ventilated with a PEEP level marginally above the LIP.

Using a model of acute lung injury, Crotti *et al.* [21] studied five consecutive patients, who had ALI/ARDS according to the criteria suggested by the European American Consensus Conference on ARDS [25] performing thoracic CT scans at different PEEP levels and plateau pressures. They found that alveolar recruitment occurred along the entire pressure-volume curve, independent of the upper and lower inflection points and occurred progressively from nondependent to dependent lung regions.

The lower inflection point on the pressure-volume curve should therefore not be used as the method of choice to calculate the level of PEEP as recruitment occurs along the entire volume-pressure curve, independent of lower and upper inflection points [22]. It would therefore seem more useful to obtain a direct measurement of recruitment at the level when PEEP is applied when putting into practice a ventilatory strategy aimed at recruiting most of the collapsed lung units.

It should be noted however that whilst PEEP holds unstable alveoli open it does not actually open them [17]. Instead, it is the sustained high pressures that open the alveoli and reopen the closed airway. For that reason, the PEEP should be increased to some degree after the recruitment maneuver is complete to preserve the newly recruited units since the normal lung becomes maximally inflated at a transpulmonary pressure of 30 to 35 cmH₂O. Transpulmonary pressure can be defined as the alveolar pressure minus the pleural pressure (assumed to be close to 0 cmH₂O) and it is the most important determinant of alveolar distension as opposed to the plateau pressure or peak inspiratory pressure [26]. Acute respiratory distress syndrome in the lungs of trauma patients associated with either indirect lung injury or extrapulmonary causes, responds more favorably to ventilatory strategies which alter the transpulmonary pressure [27]. This is because the predominant finding seen in such patients is a reversible or compressive atelectasis

rather than a consolidated airspace. Thus it appears that recruitment is ultimately impacted by the pathophysiologic process producing a particular lung injury [28].

Recruitment techniques: Review of the literature

Open lung management (OLM) is a ventilation strategy aimed at preventing atelectasis and preserving surfactant function whilst facilitating optimal gaseous exchange [29]. The prevention of atelectasis by the application of OLM attenuates mechanotrauma, preserves lung mechanics, and reduces mortality during ventilation [30,31]. In a study by Schreiter *et al.* [32] in patients with severe chest trauma, OLM assessed by computed tomography examinations significantly reduced the amount of atelectasis [33]. Furthermore, OLM has been shown to reduce protein leakage into the alveolus, which inactivates the surfactant system [31]. A vicious cycle is subsequently triggered by an impaired surfactant system involving increased shear forces and mechanotrauma. As shown in cardiac surgical patients, VILI can also be reduced or may even be prevented, by ventilating with OLM and thus reducing atelectasis [34,35]. The early application of OLM was also shown to significantly increase functional residual capacity after extubation, as compared with conventional ventilation and this effect was maintained up to 5 days after extubation [35].

The primary objective of OLM has three important steps: finding the pressure that is needed to open the lung concurrently with the collapse pressure for the patient's lung; opening the lung; and keeping the lung open to minimize any cyclic alveolar opening-closing. The pressure needed to open the lung is reached with the peak inspiratory pressure, and the collapse pressure is determined by PEEP [29]. The use of recruitment maneuvers involves a sustained increase in airway pressure for thirty to ninety seconds or repeated periodic increases in inspiratory pressure over a short period of time [2,22,29]. This technique usually facilitates alveolar recruitment through the application of continuous positive airway pressure (CPAP) by setting the PEEP to the required pressure or via application of pressure-controlled ventilation (PCV) [2,22].

In a study by Pelosi *et al.* [36], ten patients with ARDS were ventilated with a lung protective strategy using low tidal volumes and plateau pressures less than 35 cmH₂O and subsequently recruited with the application of three consecutive sighs per minute for one hour at plateau pressures of 45 cmH₂O in a volume control mode with an average PEEP of 14 cmH₂O. The results showed that there was a marked decrease in intrapulmonary shunt as well as a significant increase in end-expiratory lung volume after 1 hour of sigh, which correlated with improvement in arterial oxygenation. In another study using a CPAP mode to apply sustained inflation pressure of 30 to 45 cmH₂O for 20 seconds in fourteen patients with hypoxic respiratory failure and bilateral pulmonary infiltrates who had been ventilated less than seventy-two hours, Lapinsky *et al.* [37] found a significant improvement in the level of oxygen saturation that was maintained in ten of the fourteen patients for at least a period of four hours. In the four patients without an initial improvement the use of repeat recruitment maneuvers and institution of a higher PEEP similarly improved oxygenation. Using a lung-protective ventilatory strategy, Grasso *et al.* [38] studied twenty-two patients with ARDS. The authors applied 40 cmH₂O of CPAP for forty seconds as a recruitment maneuver and defined patients as being responders if their arterial oxygenation increased by more than fifty percent from baseline. The outcome was a significant improvement in arterial oxygenation independent of the underlying cause of the ARDS. Finally, Schreiter *et*

al. [32] found increased arterial oxygenation and increased total lung volume in patients with severe chest trauma after utilizing intrinsic PEEP by pressure-cycled, high-frequency, inverse-ratio ventilation. These studies suggest that the principles of recruitment remain the same and should fulfill the fundamental concept of their use regardless of the strategy that is employed [2,39].

The majority of trauma patients are managed with pressure controlled ventilation and since its initial use, this ventilation mode has been a mainstay in the treatment of severe pulmonary injury [2]. Amato *et al.* [15] were one of the first groups to use in mode in combination with alveolar recruitment higher and showed an improved weaning rate as well as 28-day. The use of PCV allows one to control the ventilatory pressure throughout the ventilator cycle in order to generate the appropriate pressure necessary to expand the collapsed alveoli during a recruitment maneuver [2,3,22,39]. This is especially important in patients with severe chest trauma in whom the pressure needed for alveolar recruitment may reach values as high as 60 to 70 cmH₂O after which the peak inspiratory pressure is adjusted to the lowest level necessary to maintain an open lung. This so-called ideal pressure is typically 15 to 30 cmH₂O lower than the obligatory recruitment pressure [2,3,29]. Such a pressure is achieved when arterial blood gases are found to be satisfactory in the context of stable tidal volumes. Concomitantly, the level of PEEP that is chosen, typically 10 to 20 cmH₂O, should preserve hemodynamics and demonstrate the most significant improvement in the PaO₂ [39]. It should be noted however that although PEEP holds unstable alveoli open, it does not open them. If sufficient PEEP is not applied after the recruitment maneuver is complete, the improvement in lung mechanics and oxygenation will not be sustained and alveoli will collapse within seconds [40]. Thus it is necessary to increase the PEEP to some degree after the recruitment maneuver is complete in order to maintain patency of the recruited units [41]. Maintaining the lung open after the recruitment maneuver is especially important for the success of alveolar recruitment and it can functionally be monitored by measuring the partial pressure of oxygen in arterial blood, which reliably correlates with the amount of lung parenchyma taking part in gaseous exchange [29].

The process of derecruitment upon the sudden withdrawal of PEEP is most evident in those patients that require sustained elevated levels of PEEP to attain optimal recruitment. Using an animal model, Suh *et al.* [42] and demonstrated that repeated derecruitment accentuated lung injury during mechanical ventilation and found significant pathologic changes at the bronchiolar level, with a profound inflammatory cell infiltration as well as desquamation and necrosis of epithelium. Similar changes were found in another study in the surfactant-deficient lung by Enhorning and Robertson in an animal model [43]. Sudden derecruitment may occur during patient transport, suctioning of the airways and during aerosol therapy. Added to which, the composition of inspired gas may play a role on recurrence of atelectasis and in maintaining the recruitment effect, as more rapid derecruitment occurs at a higher fraction of inspired oxygen [44].

To summarize, upon completion of a recruitment maneuver, the lung should be generally ventilated in a pressure controlled mode with sufficient PEEP to keep the lung open at a level just higher than closing pressure (the pressure at which the alveoli collapse). The ventilator is typically set to obtain the lowest possible airway pressure as well as the lowest possible tidal volume and driving pressure (defined as the peak inspiratory pressure minus total PEEP) [28]. Ideally, the tidal volume should be between 4 and 6 mL/kg and the driving pressure less than 15 cm H₂O [29]. This is to ensure proper elimination of carbon

dioxide elimination and to avoid possible shear forces to the lung [29].

Complications of open lung management

In general, when OLM is used with caution, no complications are expected. However, OLM has the capability to affect several different organ systems. The main effect of ventilation with OLM on the circulatory system is a reduction in the preload [45]. More specifically, the effect on left ventricular contractility is directly related to the overall left ventricular function. Increasing the intrathoracic pressure will not affect left ventricular contractility if the left ventricular function is good [46]. However, an increase in intrathoracic pressure will lead to an augmentation of left ventricular contractility in the presence of left ventricular failure [46]. This is likely due to increased intrathoracic pressure causing a reduction in left ventricular afterload [46].

In addition, the elevation of intrathoracic pressure and its effect on the cardiac output and preload have also raised concerns regarding the use of recruitment maneuvers with high-PEEP levels [47]. The mechanism for this effect may involve a reduction in shunt through a preferential distribution of perfusion to functioning lung units secondary to a decrease in the cardiac output [47]. The reduction in cardiac preload induced by the elevation of intrathoracic pressure might however be counterbalanced by the systemic effects of hypercapnia [47]. This is because a moderate hypercapnia can cause an elevation of cardiac output and a reduction in systemic vascular resistance [47]. The two most frequently observed side effects of recruitment maneuvers are therefore transient hypotension and associated desaturation. In a study by Grasso *et al.* [38], no significant hemodynamic changes were detected in patients who responded to recruitment maneuvers. However the effect observed in patients who did not respond to recruitment maneuvers was of a twenty to thirty percent reduction in the mean arterial pressure and cardiac output.

With regards to patients with severe chest trauma, Scheiter *et al.* [32] did not observe an increased rate of pneumothorax while using ventilation with OLM granted that such recruitment maneuvers were performed with low driving pressures. However, the use of recruitment maneuver in unilateral lung injury should be avoided in general because of the potential to increase lung volume by overdistention of the more compliant aerated alveolar units that are already open [29,48]. Such overdistention causes increased intrapulmonary shunting by favoring capillary collapse in the more compliant parenchyma and diverting blood flow into the collapsed regions [29,48]. Furthermore, this overdistention may also lead to a combination of severe hypotension, bradycardia, decreased cardiac output and increased pulmonary arterial pressure [47].

In all probability, the use of high levels of PEEP during OLM, do not increase intracranial pressure (ICP) in either the normal or injured brain. Wolf *et al.* reported that ventilation with OLM using PEEP levels up to 15 cm H₂O did not increase ICP in severe brain injury [49]. However, there was an observation of a short and transient increase of ICP by the recruitment maneuver itself [50]. Furthermore, according to Huynh *et al.* [51], the ICP did not increase with PEEP levels up to 15 cm H₂O when high PEEP was used in the absence of recruitment maneuvers. However, the use of PEEP and recruitment in brain-injured multitrauma patients who develop neurologic pulmonary edema must be considered along with hemodynamics and oxygenation [52]. Patients who have sustained brain injury should be adequately fluid resuscitated to avoid the potential preload reduction (described above) that occurs during ventilation with OLM. This effect decreases

the cerebral perfusion pressure due to a reduction in the mean arterial pressure and cardiac output.

Conclusion

The primary objective of ventilation with OLM is to achieve an optimal level of gaseous exchange characterized by a maximal partial pressure of oxygen in arterial blood after performing a recruitment maneuver initiated by sufficient levels of positive end-expiratory pressure at the lowest possible pressure amplitude with a tidal volume less than 6 mL/kg. However, such a perfect scenario is not always achieved. In fact, the optimal method of performing the recruitment maneuver still remains elusive. Nonetheless, recruitment maneuvers appear to be warranted and may be life saving for trauma patients who require extremely high levels of positive end-expiratory pressure or the fraction of inspired oxygen to achieve adequate levels of oxygenation. Further studies are however still needed to evaluate the potential benefit of the recruitment maneuver in trauma patients.

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