FOCUS ON: MECHANICAL VENTILATION

Treatment of anesthesia-induced lung collapse with lung recruitment maneuvers

Gerardo Tusman a,*, Javier F. Belda b

a Department of Anesthesiology, Hospital Privado de Comunidad, 7600 Mar del Plata, Argentina
b Department of Anesthesiology, Hospital Universitario, Valencia, Spain

SUMMARY

General anesthesia causes atelectasis and airway closure in dependent areas of the lung. Both kinds of collapse induce a deterioration of gas exchange characterized by a decrease in arterial oxygenation and an increase in dead space. The severity of this lung dysfunction is proportional to the amount of collapsed tissue that depends on anesthesia, surgical and patient’s factors.

Lung collapse can be partially prevented by decreasing FiO2 and/or by applying CPAP during the induction of anesthesia. However, only lung recruitment maneuvers can resolve atelectasis completely. These recruitment maneuvers are ventilatory strategies aimed to restore the normal aeration of the lungs. The maneuvers consist in a brief and controlled increase in airway pressure to open up those pulmonary areas with collapse. Afterward, the lungs are ventilated with a protective strategy setting keeping the lungs open in time with enough positive-end expiratory pressure and low driving pressure.

This article describes the physiological and clinical background of lung recruitment maneuvers applied during the intra-operative period.

© 2010 Elsevier Ltd. All rights reserved.

1. Introduction

Anesthesia-induced lung collapse is a well known entity observed in approximately 90% of patients undergoing general anesthesia. This collapse begins at the induction of anesthesia and persists several hours after the end of the surgery. Such lung collapse is not related to age, gender, anesthetic agents or muscle relaxant drugs.1-3 Lung collapse can affect the whole acini (atelectasis), the respiratory bronchioli (airway closure) and/or the pulmonary capillaries.1-3 The functional lose of lung units makes ventilation and perfusion more heterogeneously distributed within the pulmonary parenchyma.4 The obvious consequence is an impairment in gas exchange due to local dysfunction of the alveolar-capillary membrane. The ventilation/perfusion (V/Q) mismatch observed is due to a mix of shunt and increasing numbers of low V/Q units in dependent zones of the lungs, and dead space and an increasing number of high V/Q units in the more ventral zones.

The mechanism of lung collapse can be multi-factorial. It can be related to the loss of respiratory muscle tone induced by a central effect of anesthetic drugs, to a displacement of blood outside the thorax, to surfactant inactivation by anesthetics or to a change in the shape of the thorax among many other factors.5-7 There is a strong evidence that the key factor involved in origin of the lung collapse is the diaphragmatic dysfunction observed during anesthesia.5 It has been postulated that abdominal pressure is transmitted into the thoracic cage through this dysfunctional diaphragm and compresses the pulmonary parenchyma in the more dependent areas. These are the lung zones with the lowest transpulmonary pressure (Ptp) and thus, are predisposed to collapse at the end of expiration. This kind of lung collapse is called compressive atelectasis.1-3

Other important factors related to lung collapse are the high FiO2 used during the anesthesia induction.8 High FiO2 induces atelectasis by reabsorption of O2 in pulmonary areas with low V/Q;9 because the rate of O2 diffusion into capillary blood is greater than the amount of ventilation of those poorly ventilated units. The consequence is a progressive loss of volume within these pulmonary units until a complete collapse takes place. Reabsorption atelectasis mainly develops in patients with a certain amount of low V/Q areas like smokers or the elderly.9 However, as anesthesia reduces functional residual capacity (FRC) and impairs ventilation in some dependent zones of the lung in almost all patients, reabsortion atelectasis can be observed even in young patients with healthy lungs.10

Lung collapse is associated with negative clinical consequences and complications that influence patient’s outcome. It is not easy to make a correlation between lung collapse and later respiratory complications because the diagnosis of atelectasis at the bedside...
is not simple. The gold-standard CT scan shows that approximately 90% of patients develop atelectasis during anesthesia, which is invisible to simple chest X-ray. Therefore, due to the complexity in diagnosis of lung collapse at the bedside with standard technologies, the link between atelectasis, respiratory complications and patient's outcome is impossible to determine with accuracy.

The most important consequences of lung collapse are those associated with deterioration in gas exchange (hypoxemia and hypercapnia) \(^{(11)}\) and the detrimental effect to arterial O\(_2\) content and O\(_2\) delivery to body tissues. This is the reason why a FiO\(_2\) higher than 21% is needed during anesthesia and also in the immediate post-operative period. The higher the venous admixture induced by lung collapse, the higher the FiO\(_2\) should be in the peri-operative period to avoid hypoxemia. In specific groups of patients (morbidly obese or critically ill of any cause) and surgery (cardiac, thoracic or laparoscopy) hypoxemia can be observed even when using high FiO\(_2\). Increasing FiO\(_2\) is effective for treating the "symptom" but not the patho-physiological mechanisms behind hypoxemia and hypercapnia. Moreover, increasing FiO\(_2\) promotes reabsorption atelectasis and can impair the V/Q ratio after surgery. \(^{8,12}\)

Beyond the known deleterious and dangerous effect of acute hypoxemia and/or hypercapnia, the persistence of low arterial O\(_2\) content and delivery to the tissues is associated to organ failure caused by a local disequilibrium between O\(_2\) supplied and demand. Low urine output rate and a predisposition to bacterial translocation in the gut are both clinical examples that are said to be associated with inappropriate delivery of O\(_2\) to body tissues. In the post-operative period, a low delivery of O\(_2\) was associated to wound infection, nausea/vomiting and acute myocardial ischemia.

Another important complication of lung collapse is the lung injury induced by mechanical ventilation (VILI). \(^{(16)}\) There is increasing evidences showing that positive-pressure ventilation induces pulmonary injury when lungs are partially \(^{-20}\) collapsed, not only in sick but also in "healthy" lungs. Lung collapse induced lung injury can be due to two main mechanisms;

1) One of them is the strain and stress that normally aerated areas suffer when they receive an excess of volume and positive-pressure. As the collapsed areas cannot be totally recruited using a standard ventilatory setting, the inspiratory flow goes to normal ventilated areas. These are zones of the lungs placed more ventrally related to the gravitational plane.

2) The other mechanism is the tidal recruitment; i.e. a cycling inspiratory recruitment followed by an expiratory collapse of some lung units induced by a mechanical breath. This kind of mechanism takes place in those pulmonary zones where their opening (plateau pressure) and closing (PEEP) pressures fall within the tidal range. The involved parenchyma suffers a stress with shearing forces on bronchiolar/alveolar walls calculated as high as 100 cm H\(_2\)O. \(^{21}\)

Experimental and clinical studies revealed a progressive pulmonary immune dysfunction in healthy lungs during anesthesia and surgery. An injurious ventilatory pattern with high VT and low PEEP has been associated with increased cytokine production which was observed in BAL samples. This relation between the inflammatory response and the size of VT is related to mortality in acute lung injury; where a decrement in VT to 5 ml/kg is associated with an increase in survival rate. \(^{(23)}\) These consequences and complications of lung collapse in "healthy" lungs call for a solution beyond the cosmetic effect of increasing FiO\(_2\) in the peri-operative period.

2. Mechanism of lung collapse and recruitment

Lung collapse as well as lung recruitment are pressure-dependent phenomena because the main factor responsible for acini integrity during mechanical ventilation is trans-pulmonary pressure (Ptp) i.e. the pressure differences between airways and pleural space.

Gravity transforms pleural pressure from negative to positive in dependent zones of the lungs due to the weight of pulmonary tissue within the thorax. Assuming that the pressure in the airways is homogeneously distributed within lungs, the vertical pressure gradient into the pleural space makes Ptp lower in dependent compared to the non-dependent lung areas. During anesthesia this vertical gradient in pleural pressure is exaggerated because the diaphragm becomes dysfunctional and it is displaced upward by the abdominal pressure, compressing the lungs in the lowermost zones. \(^{(11)}\)

Each lung unit has a closing pressure or Ptp threshold when this unit begins to collapse and an opening pressure or Ptp threshold when the collapsed unit becomes aerated again. The closing pressure is reached at the end of expiration because the Ptp is the lowest airway pressure possible during this part of the mechanical respiratory cycle. Contrarily, the opening pressure is reached at the end of inspiration because this pressure is the highest one. \(^{(20)}\)

Young–Laplace equation explains why a semi-spherical shape like alveolus needs high pressure to open up but lower pressure to keep it open, according to the expression:

\[
P = 2T/r
\]

where \(P\) is the airway pressure, \(T\) is the surface tension and \(r\) is the radius of the lung unit. The P necessary to open up and to keep a lung unit in the "open" state is inversely proportional to its radius. This means that the radius of a collapsed unit is "short" and needs high pressure during inspiration to open up. In an opposite fashion, an open unit with a "large" radius (at normal FRC) needs a lower airway pressure at the end of expiration to avoid its collapse.

In summary, we can say that lung recruitment is an inspiratory process that is performed by the plateau pressure while lung collapse is an expiratory one, which can be avoided by increasing the end-expiratory pressure beyond the closing pressure of dependent zones of the lungs.

3. Treatment of lung collapse

Lung collapse can be partially prevented by two main strategies: one of them is to use continuous positive airway pressure (CPAP) during the anesthesia induction. \(^{(24)}\) The main mechanism of CPAP is to avoid the fall in FRC by keeping airway pressure higher than the lung's closing pressure. It has been demonstrated in patients that the amount of atelectasis decreases when CPAP is applied during induction of anesthesia. The problem is that CPAP is useless in apneic patients when they fall sleep and that some atelectasis can appear quickly when CPAP is discontinued during the laryngoscopy.

The second strategy to prevent atelectasis is to reduce FiO\(_2\) during induction. \(^{(8,22)}\) It is well known that low FiO\(_2\) decreases reabsorption atelectasis in lungs with reduced FRC, but at the cost of reducing the available safe time of apnea during laryngoscopy. This is for surely a dangerous technique because difficult ventilation and/or intubation cannot be predicted with accuracy in all patients. Both of these "preventive" strategies could have a synergistic, although partial effect on the genesis of lung collapse.

Lung recruitment maneuvers are ventilatory strategies in which the main goal is to recover collapsed areas of the lungs. These maneuvers are based in the premise described by Lachmann \(^{(27)}\) a few decades ago, taking into account the Young–Laplace
The alveolar recruitment strategy (ARS) is a kind of cycling maneuver that was originally conceived to solve lung collapse in the intra-operative period. The ARS consists of a controlled and step-wise increment and decrement of airway pressures using a fixed setting in pressure control ventilation mode (driving pressure of 15 cm H₂O, a respiratory rate between 10 and 15 bpm, I:E 1:1 and FiO₂ of 1). The ARS is constituted by three well defined phases (Fig. 1) that can be conducted by the algorithm described in Fig. 2.

The rationale of each ARS phase is described in detail as follow.

3.1. Hemodynamic pre-conditioning phase

One main problem of positive-pressure ventilation is the potential of hemodynamic instability in patients who are hypovolemic. High airway pressure decreases right and left ventricle preload in hypovolemic patients without a direct effect on heart contractility. The consequence is a decrease in cardiac output and in systemic arterial pressure. These hemodynamic events are treated by adequate preload replacement with i.v. fluids.

Patients with occult hypovolemia (i.e. an unsuspected hypovolemic condition) are a clinical challenge for anesthesiologists because the application of PEEP can induce episodes of hemodynamic instability in patients. For this reason, we include a simple test at the beginning of the ARS called hemodynamic "pre-conditioning" phase. This test is part of the ARS and checks the hemodynamic response in presence of 10 and/or 15 cm H₂O of PEEP before the highest airway pressures are reached during the recruitment phase. In our experience, these are adequate levels of PEEP at which an occult hypovolemia starts to be clinically manifest. The maneuver is interrupted if mean arterial pressure, heart rate and cardiac output (if available) change more than 15–20% from baseline or if mean arterial pressure fall below 55 mm Hg. If these cut-off values are observed, PEEP is reduced to a known safer level. Such occult hypovolemia is treated by i.v. infusion of crystalloids/colloids before the maneuver is reestablished. The amount of volume expansion will depend on the degree of hypovolemia but 3–5 mL/kg of crystalloids/colloids should be enough in most cases.

The pre-conditioning phase is part of the 5 cm H₂O step-wise increment in PEEP, from 0 to 20 cm H₂O (Fig. 1). Each PEEP step is maintained for at least 5 breaths. Clinicians should spend a few minutes at a PEEP of 10 and/or 15 cm H₂O to test the hemodynamic response described above. Then the maneuver can continue. The rationale of this step-wise increment in airway pressure is to achieve progressive recruitment with the plateau pressure keeping these new recruited areas open with PEEP. This strategy decreases the strain and stress on the lung parenchyma during the maneuver because the increased pressure and volume are distributed over a progressively larger tissue surface. In our experience this step-wise increment in PEEP also gives time for hemodynamic adaptation. The scenario is different for CPAP recruitment maneuvers, which produce a large and abrupt airway pressure gradient during the maneuver and also a high rate of hemodynamic instability.

3.2. Recruitment phase

It is important to stand out that the recruitment effect is observed along the ascending limb of the P–V curve of the lungs. This is why a progressive (although incomplete) recruitment effect is accomplished any time plateau pressure is increased by each PEEP step during the hemodynamic pre-conditioning phase. However, the opening pressure of the entire lungs is around 40 cm H₂O of alveolar pressure according to the findings of Rothen et al. Such alveolar pressure is the one that will open the "last" collapsed areas placed in the very dependent lung. This is the reason why we called this part of the ARS "recruitment phase" despite a considerable amount of pulmonary tissue become normally aerated during the previous phase. Once patient’s hemodynamics have been tested and stabilized, the recruitment phase consists in an increment in both, driving pressure to 20 cm H₂O and PEEP to 20 cm H₂O to reach the opening pressure at 40 cm H₂O of plateau pressure (Fig. 1). A driving pressure of 20 cm H₂O is commonly related to a safe tidal volume (VT) ≤8 mL/kg. In those patients with high respiratory compliance (Crts), driving pressure should keep at 15 cm H₂O to avoid larger VT. In this case, PEEP should be increased to 25 cm H₂O with the intention to reach the same target opening pressure while keeping VT within a normal range. Ten breaths maintaining this setting are enough to open up the whole lung in healthy patients.

3.3. Decremental PEEP titration phase

From an academic point of view, this phase would identify the lung’s closing pressure or the PEEP value at which lungs start to collapse again in dependent zones. Thus, the PEEP titration phase...
consists in a progressive decrease of PEEP in steps of 2 cm H₂O every few minutes. This closing pressure can be determined by observing atelectasis in lung images (CT scan, MRI, electrical impedance tomography), by a sudden fall in PaO₂, by a sudden increase in dead space or by obtaining non-invasive information from Crs or expired CO₂ kinetics (see below). Once this pressure has been determined, another recruitment phase is applied to recover any lung tissue that might have collapsed during the PEEP titration process. Baseline ventilation is then reassumed with a protective strategy (low VT) but setting a PEEP value a few cm H₂O above the closing pressure. This level of PEEP that keeps the lung open in time after the recruitment phase is called the “open-lung” PEEP (OL-PEEP). The setting of OL-PEEP is a hot topic in mechanical ventilation because the methods used to detect such level of PEEP are invasive or technically impossible for use at the bedside. In the operating room we commonly apply pre-defined values not only for closing but also for opening pressures. These target values were derived from physiologic studies performed in anesthetized patients. The opening pressure of normal lungs was assumed to be around 40 cm H₂O while the closing pressure was expected to be somewhere between 5 and 15 cm H₂O depending on the clinical circumstances.

In theory, the closing pressure (PEEP) must be determined in each patient at the bedside in order to personalize the recruitment maneuver; avoiding excessive or deficient airway pressure. To solve the problem, our group has described a non-invasive method for monitoring the OL-PEEP that can be easily applied at the bedside. This kind of monitoring has been validated in an experimental model of acute lung injury and works very well in anesthetized patients (personal unpublished data). The method consists in the analysis of the behavior of dynamic compliance Crs and airway resistance (Raw) and CO₂ elimination per breath (VTCO₂br) during the PEEP titration phase (Fig. 3). The “open-lung” condition is found immediately before the highest Crs was reached in an animal model of ALI. In our experience in the operating room, the OL-PEEP coincides with the highest Crs value and the lowest Raw during the PEEP titration trial. These findings make sense from the physiological point of view taking into account the relationship of Crs and Raw to lung volume: Crs is highest and Raw is lowest at normal FRC and the opposite is found at total lung capacity and at volumes close to the residual volume (Fig. 3).

Fig. 2. Algorithm for the alveolar recruitment strategy.
different types of surgery. The main clinical response to recruitment maneuvers is related to the improvement in lung function as observed through better gas exchange and ventilatory efficacy. These results are the consequence of the treatment of low ventilated zones (airway closure) and shunt areas (atelectasis) by applying the maneuver. These clinical studies, and also experimental data, have demonstrated that recruitment maneuvers are safe for patients.43

As the respiratory compliance and airway resistance are improved after recruitment, the likelihood of overdistending normal lung areas is lowered because ventilation is distributed more homogeneously within the pulmonary parenchyma. This effect is supported by the decrease in dead space observed after recruitment maneuvers, which is telling us that pulmonary capillaries are not compressed by alveolar pressure.38,39

As 90% of patients undergoing general anesthesia develop lung collapse,1 lung recruitment can be applied in almost all anesthetized patient. The remaining 10% are day-case and/or minor surgery in young, healthy and slim patients in which there is a small amount (if any) of lung collapse. In these few patients, recruitment maneuvers would not be indicated. In contrast, moderate or major surgery in patients ASA ≥ 2 will improve their clinical condition with lung recruitment maneuvers.

The ARS should be performed once after the induction of the anesthesia to get the clinical benefit during the entire period of the surgery. Whenever the airway is opened to the atmosphere (i.e. disconnection of the endotracheal tube from the anesthetic circuit) a new recruitment maneuver should be performed because lung collapse takes place in seconds. Thus, it is imperative to avoid circuit disconnection during surgery until patients are extubated at the end of the anesthesia.

Recruitment maneuvers are contraindicated in those patients in which airway are not sealed by a cufffed endotracheal tube (laryngeal or facial mask). In hemodynamically unstable patients of any origin, especially those who are hypovolemic, lung recruitment maneuvers are contraindicated until a stable state is reached following adequate treatment. Patients with bronchospasm, bronchial fistula, pneumothorax or high intracranial pressure are also unsuitable.

5. Conclusions

Lung collapse during general anesthesia is a well described entity that explains the impairment in lung function and some of the respiratory complications observed in the peri-operative period. Such collapse can be easily resolved by a lung recruitment maneuvers. These maneuvers normalize lung function which is proven by showing an increment in arterial oxygenation and respiratory compliance and by a decrease in dead space. Ventilator induced lung injury is theoretically minimized or avoided by lung recruitment maneuvers because a protective ventilatory setting can be applied in a normal FRC lungs, far away from the stress and strain that mechanical ventilation causes in collapsed lungs.

Conflict of interest statement

None.

References