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FOCUS ON: MECHANICAL VENTILATION IN THE OR

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

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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 FiO_2 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.

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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 FiO_2 used during the anesthesia induction.[8] High FiO_2 induces atelectasis by reabsorption of O_2 in pulmonary areas with low V/Q; because the rate of O_2 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, reabsorption 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

* Corresponding author. Tel.: +54 223 4990074; fax: +54 223 4990099.
E-mail address: gtusman@hotmail.com (G. Tusman).

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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.[1] 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[8,9,12] impair the V/Q ratio after surgery.

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 dysequilibrium 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[13–15] 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[17–20]collapsed, not only in sick but also in "healthy" lungs. Lung collapse induced lung[16,20]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_2O.[21]

Experimental and clinical studies revealed a progressive pulmonary immune[18–20]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[19,22] 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 6 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.[1,5]

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.

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 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.[8,25,26] 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

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