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FOCUS ON: MECHANICAL VENTILATION IN THE OR

Mechanical ventilation in cardiac surgery

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SUMMARY

Postoperative pulmonary dysfunction (PPD) is a frequent complication after cardiac surgery. Its pathogenesis is related to pulmonary inflammation, but this appears to be secondary to multiple etiological factors, including the surgical procedure itself, extra corporeal circulation (ECC), ischemia-reperfusion injury, and mechanical ventilation (MV). On the other hand, the presence of atelectasis remains one of the principal causes of PPD. The open lung approach (OLA) is a protective ventilation strategy, typically initiated after orotracheal intubation and maintained until extubation of the patient. Compared to a conventional ventilation strategy, OLA improves gas exchange parameters, induces a minor elevation of inflammatory mediators, and retains more residual functional capacity. Finally, recent studies have shown that the addition of low frequency ventilation during ECC can decrease the incidence of PPD after cardiac surgery.

1. Introduction

Postoperative pulmonary dysfunction (PPD) is a common complication after cardiac surgery,[1] but it is not clear what mechanisms are involved in its development. Therefore, it is unknown which therapeutic maneuvers might reduce the incidence of PPD. The manifestations of PPD include a frequent presence of pleural effusion (27–95%)[2] and atelectasis (16.6–88%),[3] detectable postoperative hypoxemia without clinical symptoms (3–10%),[4] and the development of acute respiratory distress syndrome (ARDS). The incidence of ARDS is low (0.5–1.7%),[5] but it is associated with high mortality (50–90%).[6] 40% of patients readmitted into intensive care units (ICU) present with respiratory failure.[7] Chung et al[8] found that, after cardiac surgery, a high inspiratory oxygen requirement during the ICU stay was related to increasing risk of readmission (odds ratio 1.09, $p < 0.05$). In addition, after cardiac surgery, the systemic inflammatory response was typically associated with a moderate pulmonary component, characterized by reduced pulmonary compliance, pulmonary edema, an increased intrapulmonary shunt fraction, and reduced functional residual capacity (FRC).[9]

1.1. Mechanisms involved in the development of PPD after cardiac surgery

1.1.1. Mechanical ventilation (MV) with general anesthesia

After cardiac surgery, the FRC typically diminishes by 40–50% during the first hours after extubation[10]; in contrast to other types of surgery where FRC is reduced by about 20% after extubation.[11] The exaggerated effect observed after cardiac surgery remains unexplained, but seems to be related to pulmonary inflammation. Many etiological factors play an important role in this effect, including extra corporeal circulation (ECC), injuries due to ischemia-reperfusion, the surgical intervention, and MV. The presence of atelectasis is one of the principal causes of PPD,[12] and there is a correlation between the amount of atelectasis and the intrapulmonary shunt.[13]

Pulmonary inflammation induced by MV is the result of combined mechanical and biological trauma. Mechanical trauma refers to the stress related to alveolar overdistension due to large tidal volumes (volutrauma) or pressures (barotrauma). This stress causes epithelial injury, loss of epithelial integrity, and edema. In particular, stress is produced in the presence of large areas of atelectasis, when tidal volume inflates fewer areas of alveoli. Biological trauma occurs upon ventilating normal lungs with high inspiratory volumes, which triggers local and systemic inflammatory responses. Consequently, the release of cytokines from a variety of lung cells alters cellular pathways that are important for normal function of tissues and organs; this effect has been called biotrauma.

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The development of atelectasis is closely related to the loss of surfactant and to repetitive closing and re-opening of the alveoli. Atelectasis is a primary factor in the development of pulmonary inflammation.[14] When dependent parts of the lung are atelectatic when the lung is exposed to high ventilating pressures, the traction forces applied to junctional tissues can be very high; for example, the traction force can approximate 140 cm H_2O with an alveolar pressure of 30 cm H_2O.[15] This "stress induced failure" of the alveolar capillary membrane is responsible for increased microvascular permeability, edema, and an influx of plasmatic proteins, which[16,17] inactivate the surfactant. Dreyfuss et al. demonstrated that the determining factor for pulmonary injury and inflammation was ventilation with high tidal volumes (producing high transpulmonary pressure), but not ventilation with high pressures (producing low transpulmonary pressure).

Biotrauma results from the mechanical, or "shear" forces between the open and closed alveoli and overdistension of alveoli, which provoke inflammatory responses at the pulmonary level. However, it is not clear how mechanical forces are translated into the biochemical signals that produce biotrauma. Theories proposed have implied mechanoreceptors, stretch-sensitive channels, activation of the inflammatory cascade,[14] and activation of the transcription of the nuclear factor kappa.[18] Many experimental studies have studied the relationship of mechanical distension of the alveolocapillary membrane and the production of mediators. [19–25]

During MV, pulmonary endothelial cells are exposed to stress, particularly during repetitive opening and closing of alveoli in atelectatic regions, but also during changes in transluminal pressure with alveolar inflation. Mechanical stress in the alveolocapillary membrane can affect the structural proteins of the membrane, the ion channels, and the cellular cytoskeleton; subsequent changes in cellular signaling cascades can produce diverse effects, including liberation of cytokines and other mediators, activation of transcription factors, altered expression of genes and proteins, cellular division, or even cellular death.[26] The exaggerated inflammatory response (production of interleukins, inflammatory mediators, etc) is not limited to the pulmonary tissues, but may cross into the systemic circulation[27] and cause a systemic inflammatory response.

Ranieri et al.[28] confirmed previous experimental findings in patients with ARDS. The levels of tumor necrosis factor-alpha (TNF-alpha), interleukin-6 (IL-6), and IL-8 in bronchoalveolar lavage (BAL) were lower with a ventilatory strategy titrated for optimal positive end-expiratory pressure (PEEP) and low tidal volumes than with a strategy that used high tidal volumes. In a multicentre study with 861 patients, ventilation with low tidal volumes (6 ml/kg) diminished plasma concentrations of IL-6 and significantly reduced the 28-day mortality of patients with ARDS. This suggested that the application of suitable ventilatory strategies clearly affected the development of an inflammatory response after cardiac surgery. Miranda et al.[29] found that an open lung strategy (tidal volume, 6 ml/kg; PEEP, 14 cm H_2O) applied immediately after intubation in cardiac surgery reduced the plasma levels of IL-6, IL-8, IL-10, TNF-alpha, and interferon-gamma.

Thus, the exaggerated pulmonary dysfunction after cardiac surgery was apparently due to two different types of stress. One was induced by MV (mechanical stress and biotrauma) and the second was the inflammatory response to the cardiac surgery. Other factors that contribute to the development of PPD after cardiac surgery include a ventilatory strategy that permits atelectasis and the setting of high volumes and low PEEP levels.[30]

1.1.2. Cardiovascular effects of MV

It is well known that MV can have potentially adverse cardiovascular effects, depending on the ventilatory strategy. Patients with cardiovascular pathology exhibit a hemodynamic response to MV that depends on numerous factors, including myocardial function, the state of intravascular volume, intrathoracic pressure, pulmonary distention, intrinsic autonomic tone, etc. Moreover, many patients present with pump failure and/or valvular pathology (fundamental indications for the cardiac surgery).

The MV per se, may produce changes in the right ventricle (RV) preload and afterload. Due to an increase in intrathoracic pressure, the gradient between the right atrium and the venous system may be reduced, diminishing the preload, and thus, the cardiac output of the RV. A reduction in lung volume at the end of expiration and alveolar collapse stimulate pulmonary hypoxic vasoconstriction (PHV), which diverts blood flow to areas that are better aerated.[31] When alveolar collapse is prevented with PEEP, recruiting maneuvers and a protective ventilatory strategy can reduce the vascular pulmonary resistance and prevent PHV.[32,33]

Miranda et al. studied the effects of an open lung strategy on the RV afterload after cardiac surgery. The patients were randomized into two groups, one with the open lung strategy (high levels of PEEP, tidal volumes of 4–6 ml/kg, and frequent recruitment maneuvers) and the other with conventional MV (PEEP 5 cm H_2O and tidal volumes of 6–8 ml/kg) after the surgery. No increases were observed in the RV afterload. In addition, the protective strategy group showed an improvement in the ratio of partial pressure of arterial oxygen to the fraction of inspired oxygen (PaO_2/FiO_2).

Left ventricle (LV) afterload is defined as the end-systolic volume or as the maximum tension in the wall of the left ventricle; in simpler terms, it is the pressure against the ejection of the LV. In patients with normal LV function, the increased intrathoracic pressure during MV reduces venous return and preload, which reduces cardiac output. Alternatively, cardiac output can increase due to a concomitant reduction in the afterload.[34] The influence of PEEP on the LV is complex. The PEEP improves arterial oxygenation and diminishes the intrapulmonary shunt, but PEEP also increases intrathoracic pressure. Therefore, the pressure difference between the LV and the systemic circulation is increased. Thus, with a constant blood pressure, less force is necessary for blood ejection from the LV.[35] Interestingly, this effect is observed more often in patients sensitive to afterload changes than in patients with congestive heart failure.

Conversely, during weaning from MV, when the patient starts breathing spontaneously, the negative intrathoracic pressure produces an increase in the LV afterload, and the previous increase in the LV ejection volume is lost. Normally, cardiac output increases in response to an increase in metabolic demand. Without this response, an increase in metabolic oxygen consumption can cause a reduction in the mixed venous oxygen saturation.[36] This, in some patients, can provoke myocardial ischemia and cause weaning failure.

1.2. Extracorporeal circulation (ECC)

The vascular contribution to the lungs depends almost exclusively on the pulmonary arteries. The principal function of the bronchial circulation is to feed the pulmonary structures; thus, it is responsible for approximately 1% of the pulmonary circulation. However, when the arterial circulation is chronically compromised, the bronchial circulation takes on a leading role. It was previously thought that when patients were submitted to ECC (on-pump surgery) without pulmonary perfusion, a "perfect" model of pulmonary ischemia would be produced. Indeed, compared to on-pump, off-pump surgery was associated with a reduced inflammatory response (cytokines) and lower levels of circulating neutrophils and monocytes.[37] Other studies in procedures without ECC have found lower pulmonary complication rates, earlier extubations, shorter MV durations, and a lower incidence of pneumonia compared to those with ECC.[38]