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FROM THE INSIDE

Is mechanical ventilation a cure for ARDS?

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Introduction

In general, medical treatment intending to cure a syndrome like the acute respiratory distress syndrome (ARDS) should treat the etiology, the pathophysiology, and the symptoms. The cure of the etiology is specifically directed against the disease leading to ARDS. The cure of the pathophysiology is the control of the process by which the causative agent triggers the damage. The cure of the symptoms consists in the treatment of the actual manifestation of the disease. A variety of etiological agents can induce widespread inflammatory processes in the lungs and cause edema ("heavy" lung) either via biological pathways, e.g., bacteria, or by physical means, e.g., acid or smoke. All pathways lead to the symptoms by which we define ARDS. The weight of the lungs, increased by the edema, induces a collapse of the lowermost lung regions. This is responsible for (a) the refractory hypoxemia that results from blood perfusing collapsed or consolidated lung regions and (b) the reduced lung compliance due to the decreased lung volume that can still be ventilated (the baby lung).

Mechanical ventilation clearly cannot remedy the etiology no matter how it is applied. However, it can "cure" the impaired gas exchange, one of the main symptoms, and, depending on how it is performed, it may act on the pathophysiology of edema formation and clearance. To fully understand how mechanical ventilation acts, it is useful to consider its components separately. These are dynamic, i.e., the driving airway pressure and its associated tidal volume, and static, i.e., positive end-expiratory pressure and the associated end-expiratory volume.

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Mechanical ventilation and gas exchange

Positive end-expiratory pressure (PEEP)

Depending on its level, PEEP completely or partially prevents end-expiratory lung collapse by counteracting the compressive forces of the increased weight of the lungs. If the previously collapsed regions had been perfused this effect would reduce intrapulmonary shunt. PEEP is definitely one of the major contributors to the symptomatic "cure" of impaired oxygenation in ARDS. In addition, PEEP may interact with pulmonary edema in several ways. It reduces its formation by lowering cardiac output and increasing interstitial pressure, and it diminishes its resolution by interfering with the factors involved in edema clearance, i.e., lymphatic circulation, pleural pressure, and venous capillary pressure. Therefore, while it is disputable whether PEEP is a cure of ARDS pathophysiology, it can without doubt symptomatically "cure" hypoxemia by preventing collapse of the recruitable lung regions, and by reducing right-to-left shunt by lowering cardiac output.

Tidal volume

Mechanical ventilation refers to a process by which gas is moved in and out of the lungs by the forces provided by the ventilator. The ventilator thus takes over the function of the respiratory muscles. In ARDS the respiratory muscles are exerted to their limit by an excessive respiratory drive and by the effort of moving heavy lungs. Under these conditions the respiratory work required to maintain sufficient ventilation cannot be sustained indefinitely. Muscle fatigue ensues, leading to the need for mechanical support.

Mechanical ventilation improves oxygenation by allowing precise control of the inspiratory oxygen concentration and by opening collapsed lung regions during inflation. This accounts for the reduction of shunt-related hypoxemia during the inspiratory phase. More importantly, mechanical ventilation is essential for CO_2

removal. By relieving the respiratory muscles, mechanical ventilation can correct or prevent hypercapnia.

Mechanical ventilation and related risks

Like all effective therapies, mechanical ventilation has risks and benefits. Minimizing the risks requires defining the acceptable minimum beneficial effects, e.g., on PaO_2 and $PaCO_2$. Since the risks involved in obtaining normal values of PO_2 and PCO_2 are too high, especially in most severe ARDS patients one should/could accept values of approximately 60 mmHg for both variables.

PEEP

Every increase of PEEP, even if associated with a recruitment-related increase in oxygenation, causes unnecessary distension of already open pulmonary units and lowers cardiac output. This is the reason for achieving the desired benefits with the lowest possible PEEP. It has been shown that setting PEEP according to the FIO_2-PEEP table from the higher PEEP arm of the recent PEEP trial allows one to choose a PEEP proportional to the recruitability (lower PEEP in patients with a lower recruitable lung volume and higher PEEP in patients with greater recruitable lung volume). In contrast other approaches to PEEP adjustment based on lung mechanics do not offer this advantage.

Tidal volume

The use of large tidal volumes and high driving pressures in the small ARDS lungs damages the lung parenchyma available for ventilation and is considered unacceptable practice today. The need to reduce the tidal volume means accepting some degree of hypercapnia. Increased PCO_2 levels are usually well tolerated but the risks of hypoventilation should not be underestimated. These are not associated with hypercapnia per se but with the unavoidable development of reabsorption atelectasis and worsening of the shunt-related hypoxemia, which increase the risk of right heart failure. Therefore, both high and extremely low tidal volume ventilation (without adapting PEEP) should be discouraged.

PEEP and tidal volume interaction

Tidal volumes and PEEP are usually linked when ventilating ARDS patients. When PEEP is increased to improve oxygenation or to prevent intratidal opening and closing of lung units, tidal volume should be decreased if the plateau pressure increases to above a level associated with a greater risk, i.e., above 30 cmH_2O. In our opinion, the "protective" effects of PEEP seen in animal studies are primarily due to the reduction of tidal volumes and are not a direct effect of PEEP. One should note that the so-called protective lung strategy (including PEEP) actually protects against the deleterious effects of mechanical ventilation, and not against the development of ARDS. Conversely, when tidal volumes are reduced to protect the lungs against the effects of mechanical ventilation, PEEP should be increased, at least initially, to maintain the mean airway pressure and prevent further alveolar collapse. This may be particularly relevant if extracorporeal CO_2 elimination is used to maintain normocapnia. Under these conditions a large reduction in tidal volume without a concomitant increase in PEEP may lead to severe hypoxemia and right heart failure.

In our opinion, mechanical ventilation is a symptomatic measure to ameliorate the deterioration in the gas exchange. It is not a causal cure for ARDS since it acts neither on its etiology nor on its pathophysiology. It is a procedure that buys time for causal treatment to take effect and for natural healing processes to take place. Great efforts must be taken to prevent the risks associated with mechanical ventilation from outweighing its benefits.

Compliance with ethical standards

Conflicts of interest

The authors declare no conflict of interest.

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