ICU
MANAGEMENT & PRACTICE

Ventilator-Induced Lung Injury and Mechanical Power

Mechanical ventilation is essential for managing acute respiratory failure but can cause ventilator-induced lung injury (VILI). VILI arises from four main mechanisms: alveolar over-distention (volutrauma), over-pressurisation (barotrauma), cyclic opening and collapse of alveoli (atelectotrauma), and inflammation (biotrauma), leading to alveolar damage and impaired gas exchange. Despite lung-protective strategies like limiting tidal volume and inspiratory pressures, mortality in ARDS patients remains high.

Mechanical power (MP), a measure of the energy transferred by the ventilator to the lungs, has been proposed as a predictor of VILI. High MP is linked to worse outcomes, making it a target for lung-protective strategies. However, practical challenges in calculating MP and a lack of RCT evidence limit its use in clinical settings. This review synthesises current research on MP, its role in VILI, and challenges in implementation.

Lung-protective ventilation strategies aim to reduce mechanical stress and strain on the lungs to prevent VILI. A key study by the ARDS Network demonstrated that limiting tidal volume (VT) and plateau pressure (Pplat) reduced mortality and increased ventilator-free days in patients. These findings were supported by meta-analyses and benefited patients with and without ARDS, establishing low VT and Pplat as core components of lung-protective ventilation.

In 2016, MP emerged as a novel strategy to reduce VILI by applying thermodynamic principles to quantify the energy transferred between the ventilator and the lungs during each breath. MP consists of three components: elastic static component - the energy delivered once when positive end expiratory pressure (PEEP) is applied, which stretches the lung from its resting state. This is not included in MP calculations; elastic dynamic component - the energy required to counteract the lung's recoil during each breath, including the recoil from PEEP and the volume variation (VT) stretch and resistive component - the work needed to overcome airway resistance and the ventilator tubing during airflow ($\dot{V}$).

The role of PEEP in MP and its relationship to VILI is still debated. Two key issues are: (1) PEEP is static and does not cause dynamic volume changes, and (2) current models assume a simple linear relationship between PEEP and MP, overlooking a U-shaped link between PEEP and VILI. PEEP helps prevent alveolar collapse (atelectrauma) and improves lung compliance (CRS) by increasing functional residual capacity (FRC). However, changes in CRS affect MP values, making PEEP's impact on MP complex in both volume-controlled and pressure-controlled ventilation.

Other factors like peak pressure (Ppeak) and respiratory rate (RR), traditionally not considered in lung-protective strategies, are included in MP and may also influence VILI. RR is also significant, as higher rates increase the risk of VILI. Experimental studies suggest that reducing RR can improve lung outcomes by minimising oedema and haemorrhage. Recommendations suggest maintaining RR ≤ 35 breaths per minute to reduce VILI and mortality.

The original power equation for calculating MP was validated in volume-controlled ventilation (VCV) using the geometric method in patients with both healthy lungs and ARDS. However, its complexity and need for inspiratory and expiratory holds to calculate airway pressure components, as well as CRS and resistance of the RRS, limit its real-time clinical application.

To improve usability, two simplified equations were developed for MP calculation in VCV with constant inspiratory flow. The first can estimate MP based on bedside lung characteristics but still requires flow holds and assumes constant CRS and RRS. The second uses flow measurements instead of plateau pressure (Pplat) for breath-by-breath MP calculation, assuming a constant RRS value of 10 $cmH_2O \times sec/L$.

To address the limitations of static respiratory parameters, dynamic MP has been proposed, using the difference between peak pressure (Ppeak) and PEEP (ΔPdyn). Dynamic MP offers the advantage of real-time, continuous monitoring by ventilators, providing insights into physiological changes. Studies show a significant association between increases in dynamic MP and higher 30-day mortality and postoperative complications. This suggests that early monitoring and limiting exposure to high dynamic MP may reduce mortality and VILI risk in mechanically ventilated patients.

Although higher MP is linked to increased ICU mortality, there are no RCTs proving the efficacy of a ventilatory strategy focused on limiting MP. The lack of a clear threshold for preventing VILI, along with variations in how MP is calculated, complicates the design of such a strategy for RCTs. As a result, routine clinical use of MP is not yet recommended.

Future research should further investigate the relationship between MP and patient outcomes, especially dynamic MP. Additionally, MP should be studied in broader populations, such as those with acute hypoxaemic respiratory failure, to simplify its use and extend its benefits to more critically ill patients.

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