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UNDERSTANDING THE DISEASE

Mechanical power: meaning, uses and limitations

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Ventilator-induced lung injury (VILI) carries significant attributable mortality in acute respiratory distress syndrome (ARDS). Even though all the ventilatory variables contribute to VILI, current guidelines focus almost exclusively on tidal volume (VT) and plateau pressure/driving pressure (ΔP).

In 2016, we formalised the concept of mechanical power (MP) with the aim to: (1) quantify the contribution of respiratory rate (RR) and the positive end-expiratory pressure (PEEP) to the total power delivered by the ventilator; (2) aggregate these variables into a single physical measure, whose value might relate to the risk of VILI [1].

In this manuscript, we aim to review the conceptual and analytical derivation of the MP, discuss the role of each component in the generation of VILI and the association between MP and outcome.

Derivation of mechanical power

To develop the MP formula, we started from the classical equation of motion and multiplied each pressure component (elastic, resistive and static) by the VT (i.e., expressing the work, or energy to the system), and then by the RR to obtain power in Joules/minute [1].

This original computation was subsequently adapted for different flow delivery (e.g., volume or pressure-controlled ventilation), and simplified for easier calculation and applicability to conditions different from passive ventilation [2, 3] Fig. 1.

Components of mechanical power and risk of VILI

The role of each MP component in the generation of VILI is still debated. The main controversy relates to whether PEEP should be excluded from MP computation given that PEEP is a static pressure (i.e., not associated with dynamic volume change) [4]. While this objection may seem plausible, it must be considered that PEEP generates a volume when first applied to the respiratory system, and the energy required to move the lung from its new energy position is equal to the sum of the internal energy of the system (i.e., VT × PEEP) and the energy needed to reach the desired inspiratory volume (i.e., VT × ΔP). In other words, it is the absolute pressure, not just the change in pressure that determines VILI [5]. This has been demonstrated in models where increasing PEEP while keeping the other components of the MP (e.g., VT, RR) constant [6], caused a proportional increase in VILI. Despite these debates, PEEP is included in all the equations proposed for the MP computation [2, 3].

Association between MP and outcome

Several studies including ARDS [7] and non-ARDS patients [8] have now demonstrated the association between MP and mortality. Its prognostic value has been compared with single variables (e.g., ΔP), and more recently with a combination of driving pressure and frequency: $4 \times \Delta P + RR$ [9] which reflects the relative effect on the odds of death. All these proposed variables have, however, predictive values comparable with MP, which is unsurprising given that they all share the same component.

Important questions are about the definition of a safe MP is, and the appropriate upper and lower safety thresholds. A "normal" MP provides acceptable PO_2 and PCO_2 and is included within safe boundaries. Experimentally we found in 30 kg pigs, a safe threshold was between an upper threshold of 12 J/min and a lower threshold of 4–7 J/min [10]. However, these are just average values, and in theory the distribution of MP within the respiratory cycle may play an equally important role. Indeed, MP is concentrated at the beginning of inspiration during pressure support

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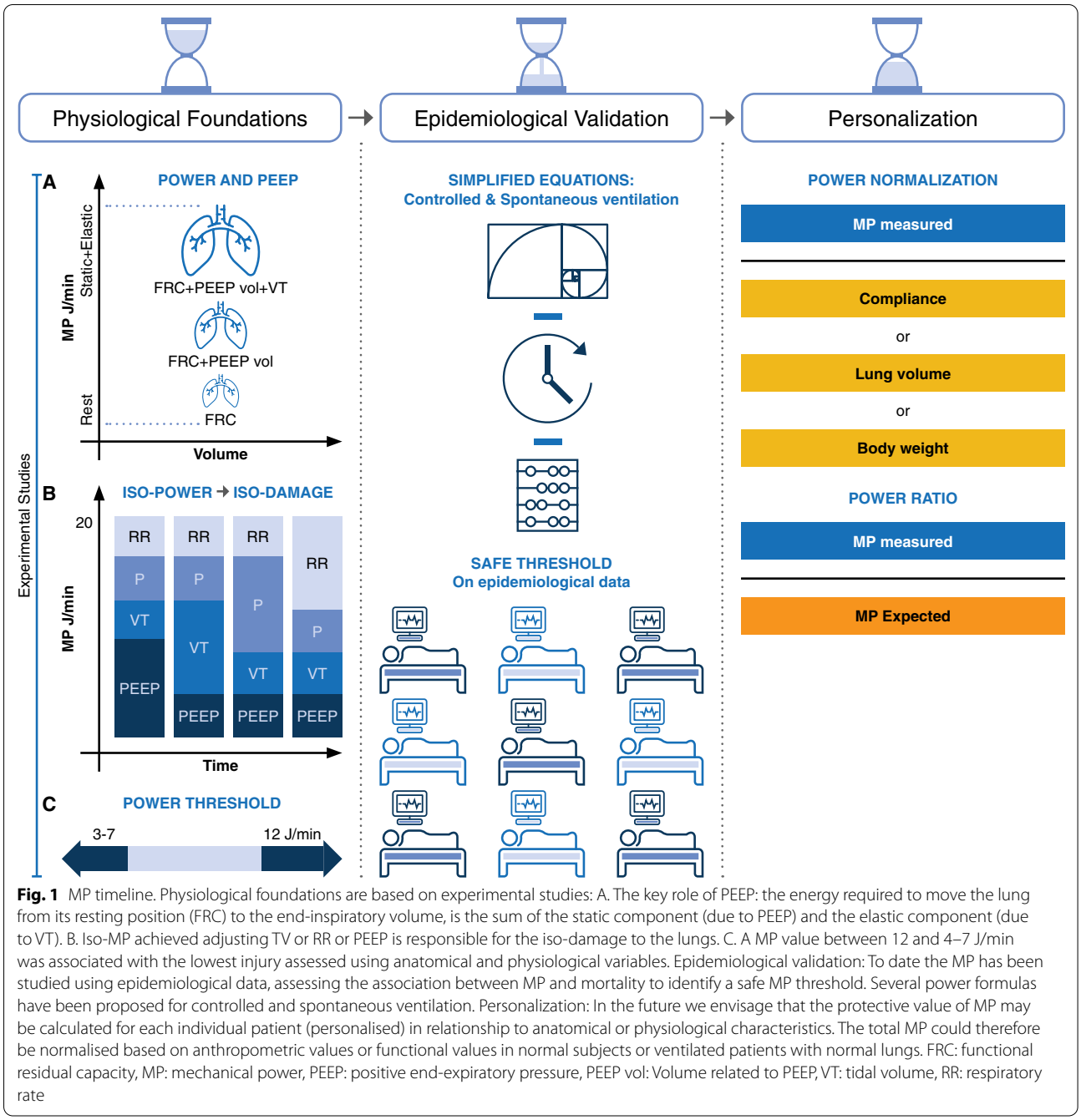

Fig. 1 MP timeline. Physiological foundations are based on experimental studies: A. The key role of PEEP: the energy required to move the lung from its resting position (FRC) to the end-inspiratory volume, is the sum of the static component (due to PEEP) and the elastic component (due to VT). B. Iso-MP achieved adjusting TV or RR or PEEP is responsible for the iso-damage to the lungs. C. A MP value between 12 and 4–7 J/min was associated with the lowest injury assessed using anatomical and physiological variables. Epidemiological validation: To date the MP has been studied using epidemiological data, assessing the association between MP and mortality to identify a safe MP threshold. Several power formulas have been proposed for controlled and spontaneous ventilation. Personalization: In the future we envisage that the protective value of MP may be calculated for each individual patient (personalised) in relationship to anatomical or physiological characteristics. The total MP could therefore be normalised based on anthropometric values or functional values in normal subjects or ventilated patients with normal lungs. FRC: functional residual capacity, MP: mechanical power, PEEP: positive end-expiratory pressure, PEEP vol: Volume related to PEEP, VT: tidal volume, RR: respiratory rate

ventilation, while in volume-controlled ventilation the MP distribution is more equally distributed throughout the inspiratory time [11]. Moreover, the MP dissipation during expiration may be more uniform if the expiratory flow is constant [12, 13]. Also, not all the elements of MP have equal weight: doubling the VT leads to a fourfold increase in MP; doubling RR leads to a 1.4-fold increase, and doubling PEEP to a twofold increase in MP [1]. Experimentally, three iso-power packages of 20 J/min, achieved adjusting VT or RR or PEEP [14] resulted in similar lung weight and wet-to-dry ratio at 48-h suggesting that iso-power, however, achieved, produces the same injury. Of note, the lung damage obtained at iso-power with the increasing of VT resulted in greater impairment in lung mechanics, while the iso-power damage induced by increasing PEEP resulted in greater systemic complications such as hemodynamic impairment and water retention. The concept of iso-power seems a convenient way to guide titration of the ventilator settings if MP goes beyond the – yet to be defined – upper thresholds of safety.

Mechanical power normalisation

The additional essential step for the clinical use of MP is its "normalisation" to the size of the lung as to move the respiratory system of an elephant or a mouse will obviously require different energy, and this will affect the safety thresholds. Scaling, however, must be applied to humans of different ages, sex and lung dimensions. Currently, this problem remains unsolved although attempts have been made normalising MP for body weight, functional residual capacity, and compliance. We are currently investigating the possibility of MP normalisation through the use of an MP ratio–analogously to what was done to derive the ventilatory ratio –between the measured MP and the MP applied to normal lungs to eliminate a normal amount of VCO_2.

Importantly, using oesophageal pressure as an estimate of the changes in pleural pressure it is possible to quantify the proportion of the total MP which is delivered to the lungs – an essential information for critical patients with different chest wall elastance.

MP is a summary variable derived from solid physical and biological foundations, that can be included in any ventilator to monitor the safety of mechanical ventilation and guide lung protective strategies. In a sense, MP is to ventilation what SvO_2 is to the body's homeostasis a variable that reflects the combination of different elements (e.g. arterial oxygenation, oxygen consumption, cardiac output or haemoglobin) all potentially associated with mortality. In the same way MP indicates that one or more variables are excessive and expose patients to the risk of VILI. Protective ventilation may be achieved by the combination of variables that achieves the lowest MP compatible with adequate gas exchange and hemodynamics.

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