Driving pressure and intraoperative protective ventilation

Since its invention as a supportive therapy, mechanical ventilation has been associated with detrimental effects on pulmonary function even in healthy lungs. During the past 40 years, these findings have led to novel pathophysiological concepts (eg, ventilator-induced lung injury, barotrauma, biotrauma, atelectrauma, and lung stress and strain) and ventilator strategies (eg, lung rest, open-lung approach, extra-corpooreal CO₂ removal), mainly used in the treatment of acute respiratory distress syndrome. The stigmatisation of the additive harmful effect of mechanical ventilation reached its peak with findings of a reduced mortality associated with low tidal volume ventilation compared with conventional high tidal volume ventilation. Subsequently, driving pressure, which is the difference between plateau airway and positive end-expiratory pressure, seems to be the best ventilator parameter with which to predict an increased risk of death associated with mechanical ventilation.
In this issue, Ary S Neto and colleagues report an individual patient data meta-analysis further investigating the risk of mechanical ventilation in healthy individuals during general anaesthesia. The current study included 17 randomised controlled trials of protective ventilation; the primary outcome was incidence of postoperative pulmonary complications. After both a multivariate and mediation analysis, the driving pressure, but not the tidal volume or the positive end-expiratory pressure applied, seemed to be the only parameter that was associated with the development of postoperative pulmonary complications.

Two crucial aspects of doing an accurate meta-analysis are study selection and data retrieval. Therefore, all the inferences obtained thereafter in these types of study are based upon the pool of data collected. From this perspective, the study by Neto and colleagues has some limitations. As an example, in some of the trials included, the related sample size reported is larger than the original cohorts enrolled, the authors likely retrospectively obtained data from additional patients undergoing the study treatment. Furthermore, some studies did not report any parameter composing the primary endpoint of the meta-analysis. It is very likely that the primary endpoint has been retrieved retrospectively for those studies. Meta-analyses are a widespread and generally accepted method by which we obtain further evidence to help guide practice, but we should be aware of their possible limitations.

The strength of Neto and colleagues’ study is that it analysed all randomised controlled trials that compared different intraoperative ventilatory strategies independent of the type of surgery. Moreover, the investigators applied, for the first time in this population, a mediation analysis. This analysis allowed the investigators to work out the potential mediator determining the primary endpoint, thereby overcoming the inconclusive associational nature of a retrospective analysis.

Furthermore, the findings might be useful in focusing attention on the three adequately powered randomised controlled trials. Overall, the two studies combining different amounts of positive end-expiratory pressure and tidal volume (low positive end-expiratory pressure and high tidal volume vs high positive end-expiratory pressure and low tidal volume) have reported a reduced incidence of postoperative pulmonary complications as associated with protective ventilation. Of note, both trials included 0 cmH₂O positive end-expiratory pressure as a low positive end-expiratory pressure level in the conventional group. According to comparative physiology and experimental data on healthy animals, an excessive end-inspiratory alveolar deformation is unlikely to be the pathogenic determinant of ventilator-associated lung injury in human beings with healthy lungs. The only large randomised controlled trial comparing two levels of positive end-expiratory pressure during intraoperative ventilation, showing no difference between the treatments, applied 2 cmH₂O positive end-expiratory pressure, in the conventional group, in at least 50% of patients, which might be sufficient (although sub-optimum) in healthy lungs to reduce end-expiratory lung collapse. A simple interpretation of the beneficial effects of a protective ventilation might rely on the reduction of intra-tidal alveolar opening and closing by a level of positive end-expiratory pressure sufficient to prevent the collapse in a lung parenchyma characterised by relatively low opening pressures.

Additionally, because postoperative pulmonary complications are the result mainly of post-operative hypoxaemia, a positive end-expiratory pressure level (as compared with 0 cmH₂O positive end-expiratory pressure) might be sufficient to reduce the formation of lung atelectasis (and hypoxaemia) during sedation.
and paralysis, a disorder that is quite common without the application of recruitment manoeuvres (sequential hyperinflation of the lung). Of note, Futier and colleagues\(^1\) reported that patients ventilated with 0 cmH\(_2\)O positive end-expiratory pressure presented immediately after anaesthesia induction with a reduced respiratory system compliance compared with patients ventilated with 6–8 cmH\(_2\)O positive end-expiratory pressure.

Why is driving pressure during intraoperative ventilation important? Being equal to the tidal volume applied as a ratio of the respiratory system compliance, which is more tightly associated with lung resting volume than the association between lung resting volume and ideal body weight, the driving pressure might theoretically reflect the direct interaction between the tidal volume applied and the open lung receiving it. Nonetheless, as a ventilator parameter, the driving pressure also has some limitations, such as its uncertain safety in patients with abnormally elevated respiratory system compliance (obstructive lung disease or emphysema), in whom an extensive cycling alveolar deformation might not be associated with increased driving pressure. Moreover, other factors are crucial in the interaction of mechanical ventilation with lung parenchyma (either injured or uninjured), such as the resulting mean airway pressure applied, peak inspiratory flow, lung recruitability, and haemodynamics.

Further studies, particularly prospective ones, are necessary to highlight the actual role of driving pressure as a determinant of the risk associated with intraoperative mechanical ventilation.

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**Growth impairment in children with pulmonary hypertension**

Assessment of somatic growth is an essential component of paediatric health surveillance and serves as a universal marker of a child’s health status. Almost any problem within the social, interpersonal, and physiological domains can adversely affect growth. The most powerful tool in growth assessment is the growth chart. Growth is a process rather than a static quality, which is why repeated growth measurements are so important. Frequent causes of growth impairment are malnutrition, hormonal dysfunction or imbalance, genetic disorders, psychosocial factors, and chronic diseases—eg, chronic renal failure, lung disease, or chronic heart failure. Studies have emphasised poor growth as a cardinal feature in congenital heart