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SPECIAL ISSUE INSIGHT

Lung and diaphragm protective ventilation guided by the esophageal pressure

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The undesired effects of mechanical ventilation on the lung [1] and the diaphragm [2] are increasingly appreciated.

Lung protective ventilatory strategies aim to limit tidal volume, driving pressure and plateau pressure to minimize lung stress and strain and consequently reduce the morbidity and mortality risks associated with mechanical ventilation [3]. This approach, however, often involves concomitant deep sedation and the administration of neuro-muscular blocking (NMBA) agents to control the potentially harmful patient-ventilator asynchrony and facilitate adherence to stringent ventilatory settings. However, excessive unloading of the diaphragm contributes to its weakness and atrophy which is associated with greater risk of weaning failure and worse outcomes [4]. A 'lung and diaphragm protective ventilation' (LDPV) strategy has been proposed as an approach aimed at addressing simultaneously the risk of ventilator-induced lung injury (VILI) and diaphragmatic dysfunction [4]. The aim of this strategy is to maintain or restore diaphragm activity during mechanical ventilation by titrating a respiratory drive to allow for sufficient diaphragm activity to avoid atrophy while preventing load-induced injury and hypertrophy, and in addition to also provide a safe lung-distending pressures minimizing patient-ventilator asynchrony. In accordance with the equation of motion, the pressure generated by the respiratory muscles—or by the mechanical ventilator—must overcome the elastic and resistive load on the respiratory system to move the lung and the chest wall, and generate a change in volume. During active breathing, this inspiratory pressure is mainly generated by the contraction of the diaphragm. In patients with acute respiratory failure, the load imposed on the diaphragm can be excessive [5] and unsustainable, thus a mechanical support is instituted to reduce the work of breathing and fatigue. Indirect evidence suggest that excessive diaphragm loading promotes diaphragm injury. First, a study suggested that an excessive acute inspiratory loading may induce significant sarcomere disruption of the diaphragm in patients with chronic obstructive pulmonary disease undergoing a respiratory muscles training program before elective surgery [6]. Second, an ultrasound study in invasively ventilated patients reported both a decrease in diaphragm thickness (atrophy)—from low inspiratory effort—and increase in diaphragmatic thickness from excessive effort in up to 25% of patients. Both abnormalities were associated with prolonged mechanical ventilation [7]. A persistently high inspiratory effort, if not recognized and corrected, can promote a diaphragm inflammation and injury. In addition, the increased diaphragmatic contraction can cause significant changes in pleural and transpulmonary pressure particularly of the dependent lung regions and an increase in trans-vascular pressure, all factors associated with the development of lung injury [8]. Conversely, a ventilatory over-assistance, sedation or NMBA can favor the generation of atelectasis and diaphragm atrophy.

Diaphragm dysfunction may be present in up of 64% of mechanically ventilated patients admitted to the intensive care and can be related to sepsis and is associated to severity of the disease [9].

Although no randomized controlled trials have so far evaluated a ventilatory strategy according to a higher or lower diaphragm activity, several experimental data indicate deleterious effects of an absent or excessive diaphragm respiratory effort [4]. Recently, a panel of experts

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suggested that the maintenance of a modest amount of respiratory effort—near a physiological normal activity—is desirable to prevent diaphragm atrophy or hypertrophy [10].

Thus, the first step towards a LDPV and allow personalization of sedation and ventilatory assistance is the assessment of the inspiratory effort and muscular pressure.

Traditionally, inspiratory effort and work of breathing are measured using esophageal manometry. Esophageal pressure swings, the pressure–time product, and transdiaphragmatic pressure (which requires the additional measurement of gastric pressure and computed as the difference between the changes in gastric and esophageal pressure during inflation) are the most accurate method of measuring inspiratory effort. A recent study showed the possibility in critically ill patients to set a lung–diaphragm protective ventilation according to a predefined algorithm based on the transpulmonary pressure [11]. The esophageal pressure, which requires only one catheter is more frequently used. However, the esophageal pressure being a surrogate of the pleural pressure assess not only the diaphragm but all the respiratory muscles activity [12]. According to the esophageal pressure swing, strictly upper and lower limits are still uncertain; however, it has been suggested to avoid an excessive or an insufficient effort an esophageal pressure above − 12/− 8 cmH_2O and below than − 3/− 2 cmH_2O [4].

Furthermore, the esophageal pressure during ventilation can also provide additional invaluable data such as the computation of the total lung distending pressure (i.e., lung stress), the assessment of the transmural vascular pressure and the presence of patient's ventilation asynchrony.

However, several clinical parameters can be implemented easily at the bedside without the need of the above monitoring tools. These parameters are derived from ventilator: brief expiratory occlusion maneuvers; or using the diaphragm ultrasound.

A simple and readily available parameter to estimate respiratory drive is the P0.1 or the inspiratory pressure measured at 0.1 s (P0.1) of inspiration during a brief airway occlusion derived from the ventilator. It has been suggested that P0.1 should be between 1.5 and 5 cmH_2O [4].

A more prolonged end-expiratory occlusion can allow the estimation of a total deflection in airway pressure (ΔPocc) calculated as the maximal deflection in airway pressure from PEEP during an end expiratory occlusion. This ΔPocc seems to correlate with the total pressure generated by the inspiratory muscles. It was recently shown that it possible to estimate muscular pressure by multiplying ΔPocc by 0.75 [13] and the total distending pressure as the difference between the driving pressure and 2/3 of ΔPocc [13]. A ΔPocc lower than 16–17 cmH_2O is suggestive for an excessive respiratory effort [13, 14].

Similarly, the measurement of diaphragm thickness fraction, computed as the change in diaphragm thickness during inspiration using ultrasound has been proposed as an alternative non-invasive method. However, only few studies have shown an acceptable correlation between the changes in diaphragm thickness fraction and the esophageal pressure or pressure–time product [14]. The possible explanations for these contradictory results are (1) the activation of intercostal and neck muscle during inspiration which can significantly affect the esophageal pressure without any change in thickness fraction and (2) the variability of the ratio between the chest and lung elastance which can differently affect the esophageal pressure changes for a similar diaphragmatic effort. Finally, the measurements of the diaphragm thickness has been reported to have only a moderate repeatability [14].

In conclusion, despite the availability of less invasive methods to estimate inspiratory effort, each suffers from limitations and thus, at present the esophageal pressure measurement remains an invaluable bedside tool to ensure consistent and reliable lung and diaphragm protective ventilation.

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Declarations

Conflicts of interest
All authors declare no conflict of interest in writing this paper.

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References

1. Slutsky AS, Ranieri VM (2013) Ventilator-induced lung injury. N Engl J Med 369:2126–2136. https://doi.org/10.1056/NEJMra1208707
2. Vassilakopoulos T, Petrof BJ (2017) A stimulating approach to ventilator-induced diaphragmatic dysfunction. Am J Respir Crit Care Med 195:281–282. https://doi.org/10.1164/rccm.201608-1619ED

3. Brower RG, Matthay MA, Morris A, Schoenfeld D, Thompson BT, Wheeler A (2000) Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. N Engl J Med 342:1301–1308. https://doi.org/10.1056/NEJM200005043421801
4. Goligher EC, Dres M, Patel BK, Sahetya SK, Beitler JR, Telias I et al (2020) Lung- and diaphragm-protective ventilation. Am J Respir Crit Care Med 202:950–961. https://doi.org/10.1164/rccm.202003-0655CP
5. Goligher EC, Jonkman AH, Dianti J, Vaporidi K, Beitler JR, Patel BK et al (2020) Clinical strategies for implementing lung and diaphragm-protective ventilation: avoiding insufficient and excessive effort. Intensive Care Med 46:2314–2326. https://doi.org/10.1007/s00134-020-06288-9
6. Orozco-Levi M, Lloreta J, Minguella J, Serrano S, Broquetas JM, Gea J (2001) Injury of the human diaphragm associated with exertion and chronic obstructive pulmonary disease. Am J Respir Crit Care Med 164:1734–1739. https://doi.org/10.1164/ajrccm.164.9.2011150
7. Goligher EC, Dres M, Fan E, Rubenfeld GD, Scales DC, Herridge MS et al (2018) Mechanical ventilation-induced diaphragm atrophy strongly impacts clinical outcomes. Am J Respir Crit Care Med 197:204–213. https://doi.org/10.1164/rccm.201703-0536OC
8. Brochard L, Slutsky A, Pesenti A (2017) Mechanical ventilation to minimize progression of lung injury in acute respiratory failure. Am J Respir Crit Care Med 195:438–442. https://doi.org/10.1164/rccm.201605-1081CP
9. Demoule A, Jung B, Prodanovic H, Molinari N, Chanques G, Coirault C et al (2013) Diaphragm dysfunction on admission to the intensive care unit: prevalence, risk factors, and prognostic impact—a prospective study. Am J Respir Crit Care Med 188:213–219. https://doi.org/10.1164/rccm.201209-1668OC
10. Dres M, Goligher EC, Heunks LMA, Brochard LJ (2017) Critical illness-associated diaphragm weakness. Intensive Care Med 43:1441–1452. https://doi.org/10.1007/s00134-017-4928-4
11. de Vries HJ, Jonkman AH, de Grooth HJ, Duitman JW, Girbes ARJ, Ottenheijm CAC et al (2022) Lung- and diaphragm-protective ventilation by titrating inspiratory support to diaphragm effort: a randomized clinical trial. Crit Care Med 50:192–203. https://doi.org/10.1097/CCM.0000000000005395
12. Mauri T, Yoshida T, Bellani G, Goligher EC, Carteaux G, Rittayamai N et al (2016) Esophageal and transpulmonary pressure in the clinical setting: meaning, usefulness and perspectives. Intensive Care Med 42:1360–1373. https://doi.org/10.1007/s00134-016-4400-x
13. Bertoni M, Telias I, Urner M, Long M, Del Sorbo L, Fan E et al (2019) A novel non-invasive method to detect excessively high respiratory effort and dynamic transpulmonary driving pressure during mechanical ventilation. Crit Care 23:1–10. https://doi.org/10.1186/s13054-019-2617-0
14. Yoshida T, Fujino Y (2021) Monitoring the patient for a safe assisted Ventilation. Curr Opin Crit Care 27:1–5. https://doi.org/10.1097/MCC.0000000000000788