AUCTORES
Globalize your Research

Journal of Thoracic Disease and Cardiothoracic Surgery
Gallardo Adrián *

Open Access

Review Article

Harms and Benefits of the Diaphragm's Eccentric Contraction

Alcaráz Melina [1], Silvero Aldana [1], Díaz-Cabrera Armando [2,3,4], Gallardo Adrián [1,4]*

[1] servicio de Kinesiología, Sanatorio Clínica Modelo de Morón, Morón, Buenos Aires, Argentina.

[2] unidad de paciente crítico, Hospital San Juan de Dios, Santiago, Chile.

[3] Exercise and Rehabilitation Sciences Institute, School of Physical Therapy, Faculty of Rehabilitation Sciences, Universidad Andres Bello, Santiago, 7591538, Chile.

[4] MoVICU Group, Mobility and Ventilation in Intensive Care Unit, La Serena, Chile.

***Corresponding Author:** Gallardo Adrián, Servicio de Kinesiología, Sanatorio Clínica Modelo de Morón, Morón, Buenos Aires, Argentina.

Received date: September 01, 2024; Accepted date: September 15, 2024; Published date: September 30, 2024

Citation: Alcaráz Melina, Silvero Aldana, Díaz-Cabrera Armando, Gallardo Adrián, (2024), Harms and Benefits of the Diaphragm's Eccentric Contraction, *J Thoracic Disease and Cardiothoracic Surgery*, 5(5); **DOI:10.31579/2693-2156/103**

Abstract

The diaphragm, the principal muscle of the inspiratory pump, is part of a group of respiratory muscles responsible for ensuring alveolar ventilation to meet metabolic demands. In patients requiring mechanical ventilatory assistance, multiple factors contribute to its alteration and condition its performance during different stages. Some of the situations that determine its alteration are related to excessive unloading or insufficient assistance, secondary to the programming of ventilatory parameters; in both cases resulting in harmful changes to its structure and function

Keywords: metabolic demands; respiratory; myotrauma

Introduction

The diaphragm, the principal muscle of the inspiratory pump, is part of a group of respiratory muscles responsible for ensuring alveolar ventilation to meet metabolic demands. In patients requiring mechanical ventilatory assistance, multiple factors contribute to its alteration and condition its performance during different stages. Some of the situations that determine its alteration are related to excessive unloading or insufficient assistance, secondary to the programming of ventilatory parameters; in both cases resulting in harmful changes to its structure and function [1].

While mechanical ventilation is used to offload the respiratory muscles, the main mechanisms that induce myotrauma are directly related to inappropriate mechanical ventilator settings. These include the application of high positive end-expiratory pressure (PEEP), excessive or insufficient assist pressure, and the presence of asynchronies that lead to eccentric diaphragmatic contractions. These factors cause muscle fiber injury, alterations in contractile function, weakness or atrophy, and are associated with longer stays in the intensive care unit (ICU), higher risk of complications, and increased mortality [2-4]. Clinical studies show that after 24 hours of mechanical ventilation, 64% of patients exhibit diaphragmatic weakness, and at the time of weaning, this weakness is observed in up to 80% of patients during their stay in the ICU [1,2,5,6]. This situation is of significant relevance for achieving successful weaning, as diaphragmatic dysfunction predisposes patients to sustained respiratory insufficiency, greatly prolonging the time required to disconnect them from mechanical ventilation [1,7].

These considerations highlight the importance of using ventilatory strategies aimed at protecting not only the lungs but also the diaphragm. Therefore, it becomes necessary to understand the mechanisms that cause injury.

Mechanisms of diaphragmatic injury

Myotrauma from over-assistance is caused by the application of excessive levels of ventilatory support, which reduces the patient's respiratory drive, resulting in a deterioration of muscle contraction. [1,2,8]. Reduced muscle activity leads to the loss of contractile proteins, resulting in disuse muscle atrophy [3,4]. In this situation, the diaphragm would have fewer contractile units to perform the same mechanical task, which predisposes to lower performance and the early onset of ventilatory failure.

On the other hand, insufficient ventilatory assistance could trigger an extremely high respiratory drive and excessive activity of the respiratory muscles, causing sarcolemma rupture and diaphragmatic dysfunction [2,3]. Additionally, in the presence of lung injury, the negative pleural pressure generated by diaphragmatic contraction is not transmitted uniformly, potentially concentrating in dependent lung regions and

generating a pendelluft effect. This causes changes in the distribution of regional ventilation without changes in total ventilatory volume. Therefore, the presence of an intense inspiratory effort can lead to overdistension of dependent lung regions, which is detrimental to the lungs. [4,9,10]

Positive end-expiratory pressure (PEEP) is routinely applied in mechanically ventilated patients to improve gas exchange and respiratory mechanics by increasing end-expiratory lung volume (EELV). However, myotrauma from excessive PEEP occurs when the increase in EELV displaces the diaphragm caudally, causing flattening of the diaphragmatic dome. This shortens the zone of apposition and places the diaphragm in a mechanically disadvantageous position for generating force [2,6,11]. If this condition persists over time, it may promote longitudinal atrophy with the loss of sarcomeres in series, leading to changes in the diaphragm's length-tension relationship and impairing its contractile function and neuromechanical efficiency [11]. The acute withdrawal of PEEP (e.g., during the weaning from mechanical ventilation) substantially decreases EELV, which can cause the diaphragm fibers to overstretch in an attempt to adapt. This overstretching can have a detrimental effect on diaphragmatic strength, placing it at a mechanical disadvantage. [1,2,4,9,11].

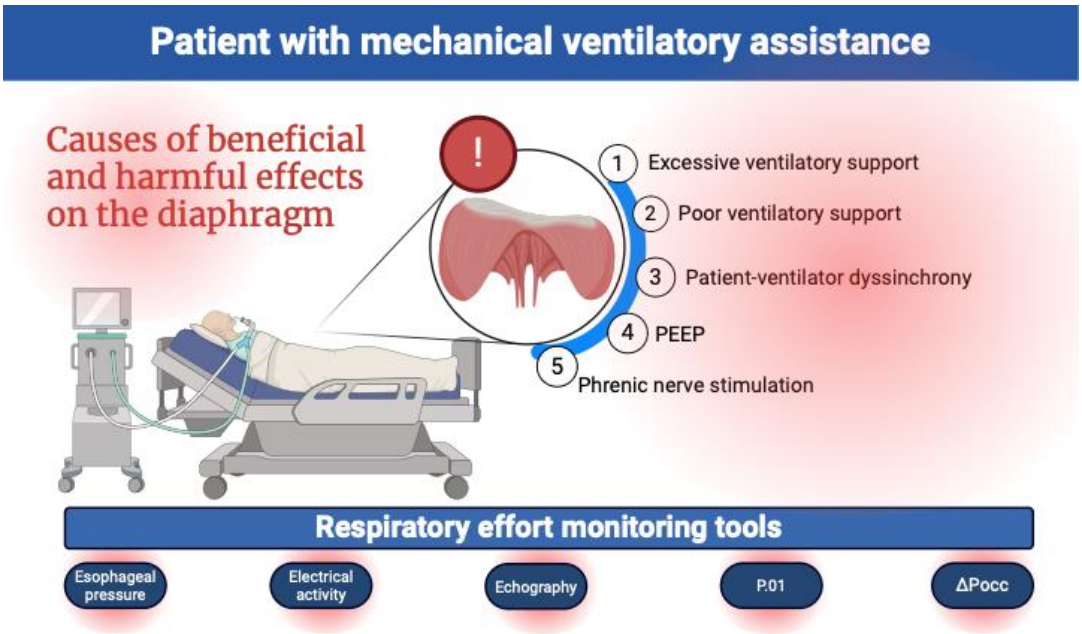
Another injury mechanism is eccentric contractions, which can occur when the ventilator cycles to initiate the expiratory phase (opening the expiratory valve) before the neural inspiratory phase is complete. This post-inspiratory load happens during various types of patient-ventilator asynchronies and under conditions of expiratory braking [12]. This expiratory braking effect, caused by the eccentric contraction of the diaphragm, occurs when muscle contraction continues after the cessation of mechanical inspiratory flow. In other words, there is active relaxation of the diaphragm during the expiratory phase. These prolonged contractions maintain EELV, prevent alveolar collapse, and reduce the formation of possible atelectasis. However, eccentric contraction may be influenced by PEEP levels: the higher the PEEP level, the lower the expiratory activity of the diaphragm, suppressing its braking effect. [3,4,12,13].

Patient-ventilator asynchronies, resulting from a mismatch between the patient's respiratory drive and mechanical assistance, can also trigger vigorous diaphragmatic contractions during the expiratory phase [4], leading to lung injury [14,15]. Among the potential asynchronies that can generate harmful diaphragmatic contractions are reverse triggering, ineffective effort, and premature cycling. In the presence of reverse triggering, for instance, muscle contraction is triggered in response to passive mechanical insufflation, typically in sedated patients or those with high ventilatory support [15]. This means that muscle contraction occurs after a mandatory ventilator cycle, creating conditions of eccentric loading [6]. These phenomena are common in mechanical ventilation and have been associated with higher mortality rates. [2,12].

This type of asynchrony, however, could generate opposite effects on the diaphragm, either preventing or inducing myotrauma depending on the level of respiratory effort of the patient. Reverse triggering with low respiratory effort could have positive effects on diaphragm strength by preventing disuse and atrophy of its fibers. Conversely, when occurring in the context of high respiratory effort, it induces harmful eccentric contractions that could affect its function and lead to lung injury [4,16-18]. In an experimental study conducted in an animal model with acute respiratory distress syndrome, it was observed that the presence of reverse triggering affects diaphragm function differently depending on the level of respiratory effort: the combination of reverse triggering and high inspiratory effort was associated with a deterioration of diaphragm function and structure after 3 hours, thus leading to increased muscle injury. [18].

Controversies

There is controversy regarding the potential beneficial or harmful effects that eccentric contractions may generate. When respiratory effort is high, it can induce eccentric myotrauma, muscle fiber injury, and functional impairment. This situation is mediated by the presence of elevated respiratory drive, which can be detrimental to both the lung and the diaphragm [4,6].



On the other hand, low respiratory effort in the presence of eccentric contractions can preserve diaphragm function [1,6,16,18]. Even with low respiratory effort, a certain level of reverse triggering may be allowed, thus promoting an increase in muscle strength. It has been demonstrated that repetitive eccentric contractions in limb muscles have beneficial effects, but further research is needed to understand how this approach would affect the diaphragm [17]. At the same time, in patients in the early phase of severe ARDS, there is a discrepancy between lung-protective and diaphragmatic ventilation [2]. In this regard, it has been reported that

neuromuscular blockade in the first 48 hours can improve outcomes, as diaphragm inactivity could prevent ventilator-induced injury [3].

Therefore, it is important to maintain an optimal level of inspiratory effort in patients under mechanical ventilation, as diaphragmatic dysfunction has been associated with difficulty in weaning, prolonged mechanical ventilation, and increased mortality [3,4,15]. To achieve this goal, a reliable estimation of respiratory effort should be sought, and appropriate adjustments should be made in ventilator settings [15,19]. Tools such as esophageal pressure monitoring, diaphragmatic electrical activity measurement, ultrasound, and assessment of respiratory drive through P0.1 have shown to be useful in this regard [2-4].

Finally, a new approach based on phrenic nerve stimulation has been recently proposed to prevent muscle dysfunction due to disuse. However, further research on the outcomes of this training is still needed, but it could be considered for future approaches [20].

Conclusion

The diaphragm, as the main respiratory muscle, can go through different clinical stages that may lead to alterations in its function and, consequently, have deleterious effects on outcomes. However, there are also situations that promote its activation and subsequent performance with a direct impact on weaning times and ventilator-free days. Utilizing monitoring tools and making changes in ventilator programming are vital steps to reduce harmful structural changes and optimize outcomes.

References

1. Goligher EC, Jonkman AH, Dianti J, Vaporidi K, Beitler JR, Heunks L. et al. (2020). Clinical strategies for implementing lung and diaphragm-protective ventilation: avoiding insufficient and excessive effort. *Intensive Care Med*, 46(12):2314-2326.
2. Shi, Zhonghua & Zhou, Jian-Xin & Heunks, Leo. (2022). Diaphragm-Protective Mechanical Ventilation: Saving the Diaphragm from the Detrimental Effects of Mechanical Ventilation. *Journal of Translational Critical Care Medicine*. 4(5).
3. Itagaki T. (2022). Diaphragm-protective mechanical ventilation in acute respiratory failure. *J Med Invest*; 69(3.4):165-172.
4. Goligher EC, Brochard LJ, Reid WD, Fan E, Saarela O, Slutsky AS, Kavanagh BP, Rubenfeld GD, Ferguson ND. (2019). Diaphragmatic myotrauma: a mediator of prolonged ventilation and poor patient outcomes in acute respiratory failure. *Lancet Respir Med*;7(1):90-98.
5. Dres M, Goligher EC, Heunks LMA, Brochard LJ. (2017). Critical illness-associated diaphragm weakness. Intensive Care Med, 43(10):1441-1452.
6. García-Valdés P, Fernández T, Jalil Y, Peñailillo L, Damiani LF. (2023). Eccentric Contractions of the Diaphragm During Mechanical Ventilation. *Respir Care.* 25;68(12):1757-1762.
7. Jaber S, Petrof BJ, Jung B, Chanques G, Berthet JP, Rabuel C, Matecki S. et al. (2011). Rapidly progressive diaphragmatic weakness and injury during mechanical ventilation in humans. *Am J Respir Crit Care Med*, 1;183(3):364-371.
8. de Vries H, Jonkman A, Shi ZH, Spoelstra-de Man A, Heunks L. (2018). Assessing breathing effort in mechanical ventilation: physiology and clinical implications. *Ann Transl Med*, 6(19):387.
9. (2023).Toro Contreras, Celina Del. Mechanical ventilation for lung and diaphragm protection in intensive care. *Med. crít. (Col. Mex. Med. Crít.)* [online]. 37(2);122-133.
10. Yoshida T, Torsani V, Gomes S, De Santis RR, Beraldo MA, Costa EL, Tucci MR, Zin WA, Kavanagh BP, Amato MB. (2013). Spontaneous effort causes occult pendelluft during mechanical ventilation. *Am J Respir Crit Care Med.* 15;188(12):1420-1427.
11. Jansen D, Jonkman AH, Vries HJ, Wennen M, Elshof J, Hoofs MA, van den Berg Heunks L. et al. (2021). Positive end-expiratory pressure affects geometry and function of the human diaphragm. *J Appl Physiol* (1985). 1;131(4):1328-1339.
12. Coiffard B, Dianti J, Telias I, Brochard LJ, Slutsky AS, Beck J, Sinderby C, Ferguson ND, Goligher EC. (2024). Dyssynchronous diaphragm contractions impair diaphragm function in mechanically ventilated patients. *Crit Care.* 2;28(1):107.
13. Pellegrini M, Hedenstierna G, Roneus A, Segelsjö M, Larsson A, Perchiazzi G. (2017). The Diaphragm Acts as a Brake during Expiration to Prevent Lung Collapse. *Am J Respir Crit Care Med*, 15;195(12):1608-1616.
14. Esperanza JA, Sarlabous L, de Haro C, Magrans R, Lopez-Aguilar J, Blanch L. (2020). Monitoring Asynchrony During Invasive Mechanical Ventilation. *Respir Care*, 65(6):847-869.
15. Mauri T, Yoshida T, Bellani G, Goligher EC, Carteaux G, Rittayamai N, Mojoli F, Chiumello D, et al. (2016). PLeUral pressure working Group (PLUG—Acute Respiratory Failure section of the European Society of Intensive Care Medicine). Esophageal and transpulmonary pressure in the clinical setting: meaning, usefulness and perspectives. *Intensive Care Med*, 42(9):1360-1373.
16. Mellado Artigas R, Damiani LF, Piraino T, Pham T, Chen L, Rauseo M, Telias I, Brochard L. et al. (2021). Reverse Triggering Dyssynchrony 24 h after Initiation of Mechanical Ventilation. *Anesthesiology.* 1;134(5):760-769.
17. Gea J, Zhu E, Gáldiz JB, Comtois N, Salazkin I, Fiz JA, Grassino A. (2009). Consecuencias de las contracciones excéntricas del diafragma sobre su función [Functional consequences of eccentric contractions of the diaphragm]. *Arch Bronconeumol*, 45(2):68-74.
18. Damiani LF, Engelberts D, Bastia L, Osada K, Katira BH, Post M, Kavanagh BP, Brochard LJ. et al. (2022). Impact of Reverse Triggering Dyssynchrony during Lung-Protective Ventilation on Diaphragm Function: An Experimental Model. *Am J Respir Crit Care Med.* 15;205(6):663-673.
19. Roesthuis L, van den Berg M, van der Hoeven H. (2021). Non-invasive method to detect high respiratory effort and transpulmonary driving pressures in COVID-19 patients during mechanical ventilation. *Ann Intensive Care.* 8;11(1):26.
20. Panelli A, Verfuß MA, Dres M, Brochard L, Schaller SJ. (2023). Phrenic nerve stimulation to prevent diaphragmatic dysfunction and ventilator-induced lung injury. Intensive Care Med Exp. 18;11(1):94.

DOI:10.31579/2693-2156/103