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Potentially beneficial patient-ventilator dyssynchrony: is it possible?

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Abstract

Patient-ventilator dyssynchronies are entities commonly observed during mechanical ventilation. Their persistence can be injurious to lung tissue and the diaphragm. However, these entities have shown different clinical impacts, with reports of diaphragm function preservation in the presence of reverse trigger. Therefore, we hypothesize the existence of a potentially beneficial dyssynchrony.

Keywords: Dyssynchronies, Reverse trigger

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Patient-ventilator dyssynchronies are entities commonly observed during mechanical ventilation. Their persistence can be injurious to lung tissue and the diaphragm, and they are associated with longer mechanical ventilation time, longer ICU stays, and higher mortality. [1,2] However, these entities have shown different clinical impacts, with reports of diaphragm function preservation in the presence of reverse trigger. [3,4] Therefore, we hypothesize the existence of a potentially beneficial dyssynchrony. Is this clinically possible?

Diaphragm inactivity, along with mechanical ventilation, can induce muscle atrophy and adverse changes in respiratory drive. [5,6] Clinical studies show that after 24 hours of mechanical ventilation, 64% of patients exhibit diaphragm weakness, which can rise to 80% at the time of weaning. [5,7] The patient's clinical condition will determine the impact of diaphragm activity during ventilatory support: subjects without lung injury or systemic inflammatory processes could benefit from diaphragm activity triggered by reverse trigger (RT), for example, whereas patients with moderate to severe acute respiratory distress syndrome may not benefit from muscular activity.

To our knowledge, mechanical ventilation can impact in an uneven manner according to the programming of established ventilatory variables. [8] Insufficient contractions, such as elevated diaphragm contractions, have been associated with longer durations of mechanical ventilation, [9] while there is experimental evidence linking RT and the presence of eccentric contractions (lengthening of the muscle while contractile tension is exerted) with improved diaphragm trophism. [3]

Furthermore, different levels of PEEP may impact both the respiratory drive and diaphragm architecture, [10,11] conditioning muscular performance. Additionally, eccentric contraction resulting from dyssynchronies may be deleterious if sustained over time, [12,13] generating varying degrees of dysfunction.

On the other hand, studies have reported a reduction in diaphragm atrophy in the presence of RT with variable levels of respiratory effort and drag patterns, showing that high effort is associated with deterioration of diaphragm function, while effort within considered adequate limits (7-12 cmH_2O) is associated with preservation of strength. [3,4]

Moreover, the presence of clusters of dyssynchronies has also been associated with increased survival. [14] Additionally, it has been shown that increased peak expiratory flow could predispose to alveolar collapse, making eccentric diaphragm activity important to prevent the occurrence of atelectasis. [15] The above suggests that respiratory effort, in the form of eccentric contractions, could preserve diaphragm function. [3,7,12,16] Even with low respiratory effort, some level of RT could be permitted, thus favoring an increase in muscular strength. It has been demonstrated that repeated eccentric contractions in limb muscles generate beneficial effects, although it remains to be studied how this approach would affect the diaphragm. [17] The quantification of eccentric contraction needs to be clarified to determine whether this activity could be beneficial or deleterious.

Quantification of the magnitude of eccentric work

The level of effort during the presence of RT has shown to be highly variable, ranging from minimal activity to efforts close to 37 cmH_2O, and at the same time, it has been reported that breath stacking occurs in less than 25% of cycles with RT present. [18] Esophageal pressure, diaphragmatic electrical activity, and transdiaphragmatic pressure have been proposed as measurements of muscular effort, but these are not always readily available at the bedside and require dedicated equipment and skilled personnel for signal interpretation. Therefore, we propose the use of a manual inspiratory pause to quantify the maximum deflection of the pressure-time curve secondary to the presence of reverse trigger as a non-invasive method that is easy to apply and available on all ventilators. Pelosi and colleagues observed that esophageal pressure better reflects variations in pleural pressure than the pleural pressure value itself. [19] In this sense, employing this technique as a surrogate for changes in pleural pressure could yield valuable information.

The use of this technique allows, once the inspiratory tidal volume is completed, to quantify the pressure drop within the system (observable in the pressure-time curve) and obtain the value of the maximum deflection. The activity should occur within a maximum of 1.5 seconds after the start of a mandatory cycle, [18] having performed a prior expiratory pause of 4 seconds to rule out the presence of a spontaneous cycle. Once this value is obtained, it is subtracted from the maximum pressure point, yielding the magnitude of the force exerted by the diaphragm (Figure 1). Considering previous reports, [18] those values that are ≤ 8.7 cmH_2O could be considered as efforts that keep the diaphragm active and prevent atrophy due to disuse.

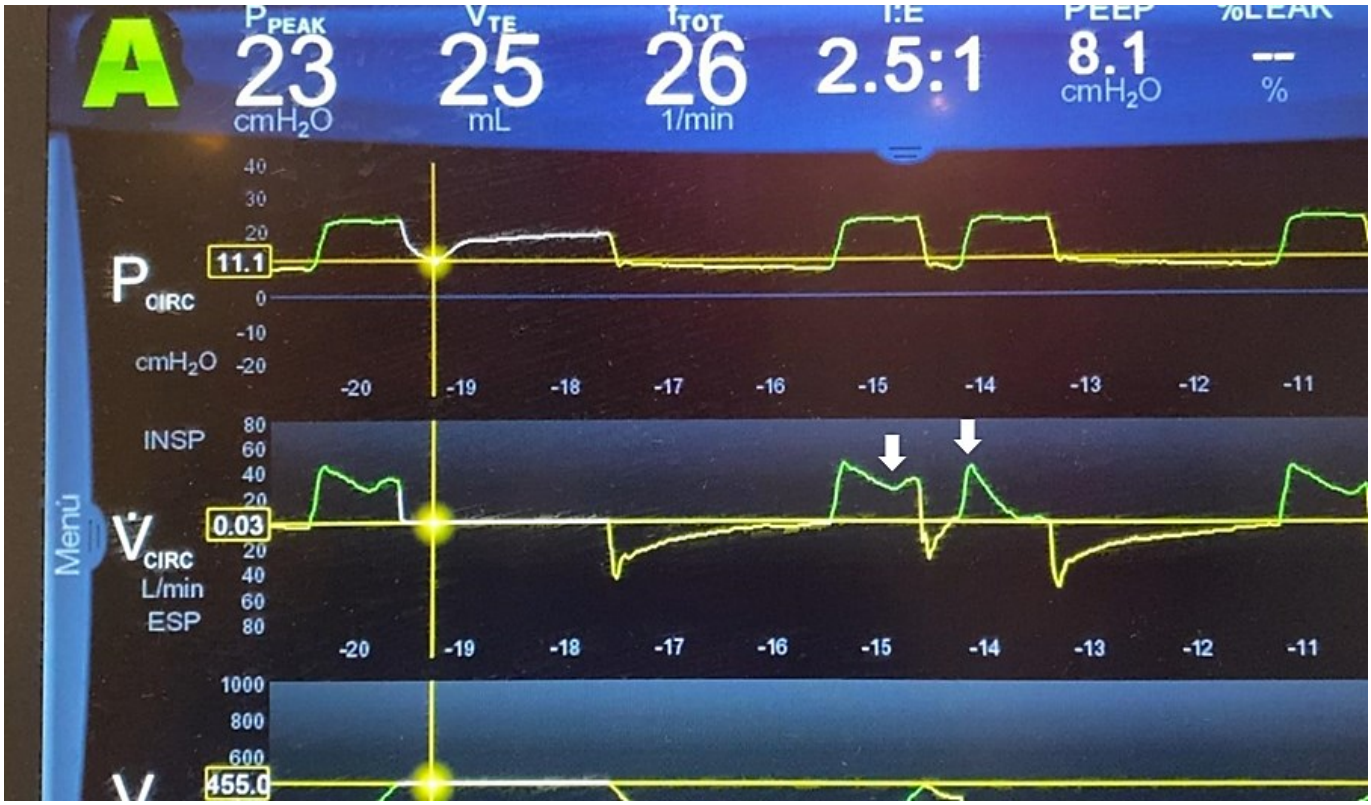

Figure 1. Maneuver of inspiratory pause and quantification of the maximum deflection in the pressure curve secondary to the presence of reverse trigger. The pressure drops to 11.1 cmH_2O, from a maximum pressure of 25 cmH_2O, representing a difference of 13.9 cmH_2O corresponding to diaphragmatic contraction. According to the values reported in previous studies, [18] this value exceeds the median muscular pressure found in reverse trigger, which could trigger stacking of breaths (this situation is visible in the second cycle, white arrows).

Limitations of quantification with manual inspiratory pause

The maneuver must be performed under controlled pressure ventilation, to reach and maintain a fixed pressure; this will be taken as the starting point to calculate the magnitude of the deflection secondary to diaphragmatic contraction. The possibility of performing an inspiratory pause and the subsequent pressure drop offers the chance to objectively estimate the amount of force exerted by the diaphragm, although this measurement is not as precise as esophageal pressure or transdiaphragmatic pressure. [20]

Another limitation could be considered when comparing this technique with the electrical activity of the diaphragm. Furthermore, the proposed technique assumes exerting force against a “fixed resistance” whereas in a real situation, the diaphragm will exert an eccentric contraction when air exits the system (because of the opening of the expiratory valve), which we could define as a dynamic situation. Thus, depending on the moment in the respiratory cycle when the eccentric contraction occurs, different pressure values could be observed, meaning that different RT phenotypes might yield different values; for instance, a phenotype of early activation and relaxation might not be properly quantified, as the maximum contraction of the diaphragm could occur during the pressurization phase and not be captured in a later period. [21]

It is worth mentioning, however, that the maneuver is non-invasive, easy to perform, does not require specialized equipment, and is very easy to interpret. In this regard, we believe it can be integrated into clinical practice without difficulties.

Conclusion

The above suggests that approximately 75% of dyssynchronies due to RT present eccentric activity of the diaphragm without breath stacking, and that this activity could favor muscular action, preventing deterioration due to disuse. Knowing the amount of force exerted by this muscle is of vital importance to optimize our patients' outcomes. Therefore, to complement existing classifications, we propose a

quantification methodology and the inclusion of potentially beneficial dyssynchrony as a clinical entity.

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