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LETTER TO THE EDITOR

The customization of mechanical ventilation: Yes, but... Keep calm

La personalización de la ventilación mecánica: sí, pero... mantengamos la calma

Dear Editor,

Individualizing mechanical ventilation (MV) based on physiology and lung morphology, etiology, pulmonary images, and phenotypes should be understood as the titration of ventilation parameters based on close monitoring of specific physiological variables and individual objectives.[1]

However, individualizing mechanical ventilation parameters, and the speed at which ventilator variables are changed can have important biological effects. This is due to the viscoelastic nature of lung tissue where extracellular matrix components like epithelial/endothelial cells require adaptation time («stress relaxation») to alleviate the tension (stress).[2]

Under controlled mechanical ventilation (CMV), the ventilator needs to exceed elastic and resistive forces to trigger respiratory movement. Whichever the model used to explain lung deformation phenomena (strain), time must be taken into consideration in such a way that it should explain why alveoli don't close immediately or instantaneously fill up with air.[1,3]

In this sense, strain rate is referred to as the speed at which a material expands or contracts. Strain rate is inversely associated with time: with the same tension, the longest times (slow changes to the ventilator variables) are associated with lower strain rates. The shortest times, however, (quick changes to the ventilator variables) are associated with higher strain rates. Therefore, quick changes will end up fracturing interstitial alveolar structures following rapid displacement with poor accommodation to static or dynamic efforts. If these changes occur over time, more fractures of alveolar structures will happen as well. However, slow changes preserve interstitial alveolar structure thanks to a greater accommodation to deformation (strain). In time, and although alveoli may be heterogeneous, fractures of alveolar structures will be less likely.[1,2]

Therefore, sudden changes to any ventilation parameters whether static or dynamic (changes to tidal volume, positive end-expiratory pressure [PEEP], and inspiratory and expiratory flows) capable of changing deformation (strain) over time, can make ventilation-induced damage worse. This premise turns mechanical power into a particularly appealing variable for the management of ventilation parameters since with the basic equation of movement it includes all potential causes of ventilator-induced lung lesion: tidal volume, conduction pressure, flow, respiratory rate, and PEEP.[4] As a matter of fact, mechanical power keeps a significant correlation with mortality despite adapting to conduction pressure.[5] This proves that mechanical power provides an additional risk estimate beyond conduction pressure *per se* because it captures the energy applied in a different way compared to conduction pressure.

However, despite the repercussions this can have in the routine clinical practice[6,7] maybe little attention is being paid to expiratory phase during CMV. The active control of expiratory phase (in modes of flow-controlled ventilation)—providing constant and active gas flow during the entire respiratory cycle whether in inspiration or expiration—minimizes dissipated energy to the lowest possible level, improves lung ventilation in dependent lung regions and, consequently, gas exchange.[8,9]

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Conflicts of interest

None reported.

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