Bedside Ultrasound for Weaning from Mechanical Ventilation

The Diaphragm Is Not Enough!

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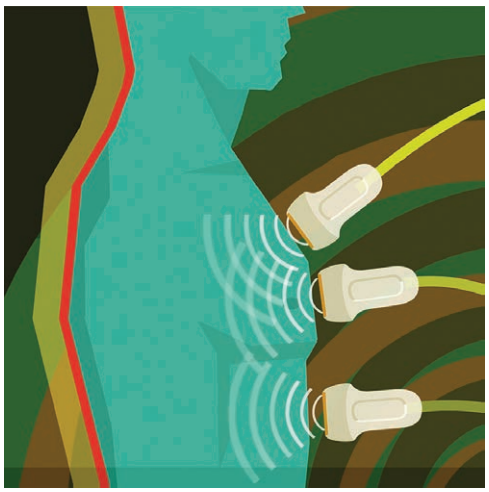
Despite many advances in mechanical ventilation over the past decades, patient–ventilator interaction remains difficult to assess in patients in the intensive care unit, especially during the weaning phase. Easy detection and measurement of breathing effort remains an important objective. Ultrasonography, a simple, widely available bedside technique, can provide valuable information in this context to complement clinical examination.[1] Ultrasonography has been used in patients in the intensive care unit for more than a decade to evaluate excursion or thickening of the diaphragm, the main respiratory muscle.[2] In particular, it can be used to detect anatomical atrophy or functional impairment related to mechanical ventilation or sepsis.[3,4] However, the diaphragm is not the only inspiratory muscle involved in ventilation, especially when it is damaged or weakened. During tidal ventilation, the diaphragm works in synergy with the scalene and external intercostal muscles to trigger inspiration, as well as with the dilator muscles of the upper airway. In cases of respiratory distress, the sternocleidomastoid muscles and the trapezius are also recruited.



"It may be possible with ultrasonography to assess the mechanics, thickness, and strength of all the respiratory muscles."

It may be possible with ultrasonography to assess the mechanics, thickness, and strength of all the respiratory muscles.[1] In this issue of Anesthesiology, Dres *et al.*[5] investigate whether intercostal muscle function can be observed and quantified by ultrasound imaging. The authors proceed in three stages: (1) they demonstrate in 23 healthy volunteers that ultrasound measurements of the thickness and thickening of the anterior intercostal muscles are feasible and reproducible; (2) in 16 ventilated patients, they show that there is a negative correlation between thickening of the intercostal muscles and the level of inspiratory support, suggesting that ultrasonography may help to detect mobilization of the intercostal respiratory muscles when ventilatory support is insufficient; and (3) in 54 patients being weaned from mechanical ventilation, they show that diaphragm dysfunction is associated with intercostal muscle recruitment. Patients with diaphragmatic dysfunction and high levels of intercostal muscle thickening had a higher failure rate in spontaneous breathing trials.

This preliminary study is important because it outlines a new and simple way to assess excessive respiratory effort at the bedside. During inspiration, the muscle fibers of the parasternal intercostal muscles contract, displacing the rib cage cranially and anteriorly. Because their mass remains constant, increases in thickness can be observed using ultrasound imaging. Recruitment of the intercostal muscles at the start of inspiration increases tidal volume and depends on the intensity of the respiratory drive. It has been shown that extubation outcomes cannot be predicted using isolated ultrasound observations of the diaphragm.[6] There are

Image: A. Johnson, Vivo Visuals.

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two possible explanations for reduced diaphragm thickening depending on the context: adaptive weak diaphragm contraction secondary to excessive ventilator support (over assistance) or intrinsic diaphragm dysfunction secondary to sepsis and disuse atrophy. Observations of intercostal muscle thickening could help clinicians distinguish the former (reduced intercostal thickening) from the latter (increased intercostal thickening).

One of the major limitations of muscle ultrasonography is its reproducibility. The small size of intercostal muscles (between 3 and 4 mm thick), probe orientation difficulties, muscle edema, and the pressure of the transducer and operator's hand on the wall can all affect measurements. It is also not possible with the proposed technique to analyze the internal and external intercostal muscles separately. Moreover, the technique may be of limited value in patients with permanent mobilization of the intercostal muscles (*e.g.*, chronic obstructive pulmonary disease). Finally, it is unclear whether ultrasound observations of intercostal muscle thickening are more sensitive than simple clinical observations of inspiratory patterns and whether this approach is accessible to nonexperts. Dres *et al*. have proposed a new and interesting technique. Their results need to be first confirmed in a larger number of patients. If confirmed, they pave the way for subsequent studies assessing bedside ultrasound of both diaphragm and parasternal intercostal muscles, to improve the standard clinical practice of ventilator weaning. In this quest, the diaphragm will no longer be observed alone.

Competing Interests

The authors are not supported by, nor maintain any financial interest in, any commercial activity that may be associated with the topic of this article.

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