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Ultrasound of the diaphragm—an essential tool for pulmonologists and intensivists

Luigi Vetrugno^{1,2}, Daniele Orso^{1,2}, Tiziana Bove^{1,2}

"It is always foolish to give advice: but giving good advice is fatal." Oscar Wilde

The diaphragm is the major inspiratory muscle, and its continual rise and fall can be likened to the monotonous up-and-down movement of an engine piston. The diaphragm never stops, contracting and relaxing throughout a lifetime, except, of course, during anesthesia or if it is blocked through the use of paralyzing agents in the ICU. The perpetual movement of the diaphragm generates the so-called transdiaphragmatic pressure, the value of which directly correlates with the strength required to achieve adequate ventilation. In the past, only specialized centers equipped for research purposes had the appropriate tools that were necessary for assessing the strength of this muscle, which could be evaluated in two ways. The first modality involves measuring the transdiaphragmatic pressure (in cmH_2O) and employs a double-balloon probe-one balloon is inserted into the esophagus and one is inserted into the stomach. In the second modality, diaphragmatic force is measured indirectly by means of the evaluation of the twitch pressure (in cmH₂O)—the pressure generated at the outside tip of the endotracheal tube. However, in both cases, in order to generate a maximal volitional inspiratory effort in uncooperative patients, cervical magnetic stimulation of the phrenic nerve is required. Needless to say, both techniques are highly invasive and accompanied by limitations. The most important limitation is that unilateral diaphragmatic paralysis can never be excluded, because one hemidiaphragm can compensate for the contralateral one.(1)

Bearing in mind the limitations of the abovementioned techniques, the contemporary use of ultrasound for the assessment of diaphragm function in clinical practice stands to offer some important advantages. Although the ultrasound technique for the evaluation of the diaphragm was originally published some 20 years ago by Wait et al.,⁽²⁾ Santana et al.,⁽³⁾ in the article published in the present issue of *Jornal Brasileiro de Pneumologia* (JBP), clearly and accurately described that it is only in the past 10 years that the importance of this approach has been explored in more detail, especially within the ICU.

More than 60% of the patients admitted to the ICU show some form of diaphragm dysfunction in terms of a decrease in unilateral or bilateral diaphragm activity (weakness), abolished function (paralysis), or paradoxical movement. In addition, 80% of the patients develop diaphragm dysfunction during mechanical ventilation (MV).⁽¹⁻⁴⁾ A recent study⁽⁵⁾ has shown that the most frequent type of shock in the ICU (septic shock) is associated with

preferential diaphragmatic atrophy. This preferential loss of diaphragm muscle volume, when compared with that of the psoas muscle, creates a condition called sepsisinduced diaphragmatic dysfunction. That study provides evidence supporting the notion that the loss of diaphragm muscle volume is associated with a loss of strength.⁽⁵⁾

MV is the most frequently used short-term life support technique worldwide. However, although MV provides an unarguably vital form of life support, saving patients from an underlying disease, relieving the work of the diaphragm can rapidly lead to diaphragmatic atrophy and strongly impact the clinical outcome of the patient.⁽⁶⁾ The first group to introduce the concept of ventilator-induced diaphragm dysfunction was Vassilakopoulos et al.,⁽⁷⁾ who postulated that the rapid disuse of diaphragm fibers during MV is the underlying cause of this condition. Five years later, Levine et al.⁽⁸⁾ provided crucial evidence, demonstrating that diaphragm inactivity during MV results in marked atrophy of human diaphragm myofibers. Grosu et al.⁽⁹⁾ substantiated that evidence, reporting a 6% reduction in diaphragm thickness within 48 h after initiation of MV in patients in the ICU. Therefore, sepsis and MV in the ICU are now recognized as the two key players that are responsible for diaphragm dysfunction in critically ill patients, and the term "critical illness-associated diaphragm weakness" has been coined in order to refer to all of these mechanisms.⁽¹⁾ However, many other disease processes can affect diaphragm function in terms of its contractile properties, innervation, or indeed both; for example, traumatic injuries, mass effect, inflammatory disease, neurological disease, regional anesthesia, and idiopathic conditions.

In the present issue of the JBP, Santana et al.⁽³⁾ have provided a detailed review of the literature regarding the technical aspects of how diaphragmatic ultrasound can be used in order to assess diaphragm function during normal breathing, deep breathing, and sniffing. Main findings and clinical applications in critically ill patients are clearly presented. The authors have also evaluated other conditions that can potentially induce diaphragm dysfunction, including asthma, cystic fibrosis, COPD, and neuromuscular disorders.⁽³⁾ With regards to COPD patients in the ER, diaphragmatic ultrasound has only recently been identified as a tool suited to monitoring patients with acute hypercapnic respiratory failure and severe dyspnea undergoing noninvasive ventilation.⁽¹⁰⁾

^{1.} Dipartimento di Medicina, Anestesia e Terapia Intensiva Clinica, Università degli Studi di Udine, Udine, Italia.

^{2.} Dipartimento di Anestesia e Terapia Intensiva, Azienda Sanitaria universitaria Friuli Centrale-ASUFC-Ospedale Santa Maria della Misericordia of Udine, Udine, Italia.





Figure 1. Looking at the diaphragm with different "eyes".

Santana et al.⁽³⁾ have also reported how to evaluate diaphragmatic excursion by using the subcostal view in B mode and transverse scanning. This is of particular interest because the longitudinal approach has been reported to be preferable.⁽¹¹⁾ Normal values of diaphragmatic excursion in healthy volunteers have previously been described.⁽¹¹⁾ However, the measurement of diaphragmatic excursion (displacement) can only be performed in patients with spontaneous breathing, such as those just admitted to the ICU or intubated patients undergoing a spontaneous breathing trial for weaning purposes. In contrast, ultrasound assessment of diaphragmatic excursion during MV provides erroneous results, because it measures not only the effort exerted by the patient but also the power of the ventilator. Santana et al.⁽³⁾ claim that the use of the thickening fraction (TF), calculated by means of the diaphragm thickness at end-inspiration (Tdi-insp) and end-expiration (Tdi-exp) at the zone of apposition-TF = [(Tdi-insp - Tdi-exp)/Tdi-exp] × 100 -could be a better indicator of diaphragm activity during MV. TF is an expression of muscular contraction and can therefore be used in order to measure diaphragm activity and assess whether MV support can be managed by the patient in question. Indeed, some patients on MV are exposed to overassistance (excessive unloading of the diaphragm by the ventilator reduces or abolishes inspiratory effort), or underassistance of MV (excessive loading of the diaphragm owing to insufficient ventilator assistance)-both of which causing diaphragm myotrauma. We also know that patient-ventilator dyssynchrony leads to diaphragmatic myotrauma due to eccentric muscle loading.⁽¹²⁾ The general consensus to date is that TF should be kept within the normal range, between 15% and 30%, such as in healthy subjects breathing at rest, which seems to be associated with a shorter duration of MV.⁽⁶⁾

At this point, the limitations of diaphragmatic ultrasound also need to be acknowledged. It might be impossible to evaluate a patient presenting with a poor acoustic window; the left hemidiaphragm is difficult to explore; and the level of experience of the operator in performing diaphragmatic ultrasound is important. That being said, with a adequate amount of training proficiency, diaphragmatic ultrasound is a technique that is easy to perform. In conclusion, the well-evidenced central message of the review by Santana et al.⁽³⁾ is clear: ultrasound is a highly appropriate technique for examining the diaphragm. Use it.

AUTHOR CONTRIBUTIONS

LV designed the editorial. All of the authors participated in drafting and revising the editorial. All authors have read and approved the final version of the manuscript.

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