

Dimitrios Matamis
Eleni Soilemezi
Matthew Tsagourias
Evangelia Akoumianaki
Saoussen Dimassi
Filippo Boroli
Jean-Christophe M. Richard
Laurent Brochard

Sonographic evaluation of the diaphragm in critically ill patients. Technique and clinical applications

Received: 20 September 2012
Accepted: 18 December 2012
Published online: 24 January 2013
© Springer-Verlag Berlin Heidelberg and ESICM 2013

Electronic supplementary material

The online version of this article (doi:[10.1007/s00134-013-2823-1](https://doi.org/10.1007/s00134-013-2823-1)) contains supplementary material, which is available to authorized users.

D. Matamis (✉) · E. Soilemezi · M. Tsagourias
Intensive Care Unit, Papageorgiou General Hospital, Peripheral Ring Road, N. Efkarpia, Thessaloniki, Greece
e-mail: dmatamis@gmail.com
Tel.: +30-2313323361
Fax: +30-2313323367

E. Akoumianaki · S. Dimassi · F. Boroli · J.-C. M. Richard · L. Brochard
Intensive Care Unit, University Hospital of Geneva, Geneva, Switzerland
e-mail: akoumianakievangelia@gmail.com

J.-C. M. Richard · L. Brochard
School of Medicine, University of Geneva, Geneva, Switzerland

Abstract The use of ultrasonography has become increasingly popular in the everyday management of critically ill patients. It has been demonstrated to be a safe and handy bedside tool that allows rapid hemodynamic assessment and visualization of the thoracic, abdominal and major vessels structures. More recently, M-mode ultrasonography has been used in the assessment of diaphragm kinetics. Ultrasounds provide a simple, non-invasive method of quantifying diaphragmatic movement in a variety of normal and pathological conditions. Ultrasonography can assess the characteristics of diaphragmatic movement such as amplitude, force and velocity of contraction, special patterns of

motion and changes in diaphragmatic thickness during inspiration. These sonographic diaphragmatic parameters can provide valuable information in the assessment and follow up of patients with diaphragmatic weakness or paralysis, in terms of patient-ventilator interactions during controlled or assisted modalities of mechanical ventilation, and can potentially help to understand post-operative pulmonary dysfunction or weaning failure from mechanical ventilation. This article reviews the technique and the clinical applications of ultrasonography in the evaluation of diaphragmatic function in ICU patients.

Keywords Bedside ultrasonography · Critically ill · Diaphragmatic dysfunction

Introduction

Bedside ultrasonography has become a valuable tool in the management of intensive care unit patients [1, 2]. This is especially true in emergency situations where an adequate imaging technique is frequently limited by a variety of factors, including difficulty of patient transportation to the radiology department due to illness severity. Ultrasonography is a noninvasive technique, which has proved to be an accurate, safe, easy to use bedside modality, overcoming many of the standard limitations of imaging techniques.

The diaphragm is the principal respiratory muscle, and its dysfunction predisposes to respiratory complications and can prolong the duration of mechanical ventilation [3–5]. Sonographic evaluation of the diaphragm has recently started to gain popularity in the ICU as specific needs for assessing diaphragmatic function arise in many clinical situations. Abnormal diaphragmatic motion is observed in conditions such as phrenic nerve injury, neuromuscular diseases [6–11], after abdominal [12] or cardiac surgery [4, 13] and in critically ill patients under mechanical ventilation [14–17]. Since diaphragmatic motion plays a prominent role in spontaneous respiration,

observation of the diaphragm kinetics seems essential. The use of tools previously available for this purpose is limited due to the associated risks of ionizing radiation (fluoroscopy, computed tomography) or due to their complex and/or highly specialized nature, requiring a skilled operator (transdiaphragmatic pressure measurement, diaphragmatic electromyography, phrenic nerve stimulation, magnetic resonance imaging). Sonography receives increasing recognition as a fast, easy and accurate method of noninvasively evaluating diaphragmatic function at the bedside. In the ICU population, it can quantify normal and abnormal movements in a variety of clinical conditions. In this review, we will show that it can be used for diagnosing diaphragmatic paralysis and recovery [3, 18, 19], serve as a bedside screening test for investigating postoperative diaphragmatic dysfunction [4, 15, 20, 21] and detect synchronization of spontaneous breathing efforts with the ventilator, potentially allowing an optimized adjustment of the ventilator settings.

Sonographic technique of diaphragmatic evaluation

Diaphragmatic sonography is performed using a 3.5–5 MHz phased array probe. The probe is placed immediately below the right or left costal margin in the mid-clavicular line, or in the right or left anterior axillary line and is directed medially, cephalad and dorsally, so that the ultrasound beam reaches perpendicularly the posterior third of the corresponding hemi-diaphragm (Fig. 1a). The two-dimensional (2D) mode is initially used to obtain the best approach and select the exploration line; the M-mode is then used to display the motion of the anatomical structures along the selected line (Fig. 1b). This is illustrated in a video placed in the on-line supplement. Patients are scanned along the long axis of the intercostal spaces, with the liver serving as an acoustic window to the right, and the spleen to the left. Normal inspiratory diaphragmatic movement is caudal, since the diaphragm moves toward the probe; normal expiratory trace is cranial, as the diaphragm moves away from the probe (Fig. 1c). In the M mode, the diaphragmatic excursion (displacement, cm), the speed of diaphragmatic contraction (slope, cm/s), the inspiratory time (T_{insp}, s) and the duration of the cycle (T_{tot}, s) can be measured. In mechanically ventilated patients, evaluation of diaphragmatic motion sometimes necessitates to briefly disconnect the patient from the ventilator to better visualize spontaneous breathing efforts. Of note, many ICU patients may have pleural effusions, consolidation or atelectasis, which, in contrast to what one might expect, allow an easier identification of the hemidiaphragms. The values of diaphragmatic excursion in healthy individuals were reported to be 1.8 ± 0.3 , 7.0 ± 0.6 and 2.9 ± 0.6 cm for males, and 1.6 ± 0.3 , 5.7 ± 1.0 , and 2.6 ± 0.5 cm for females,

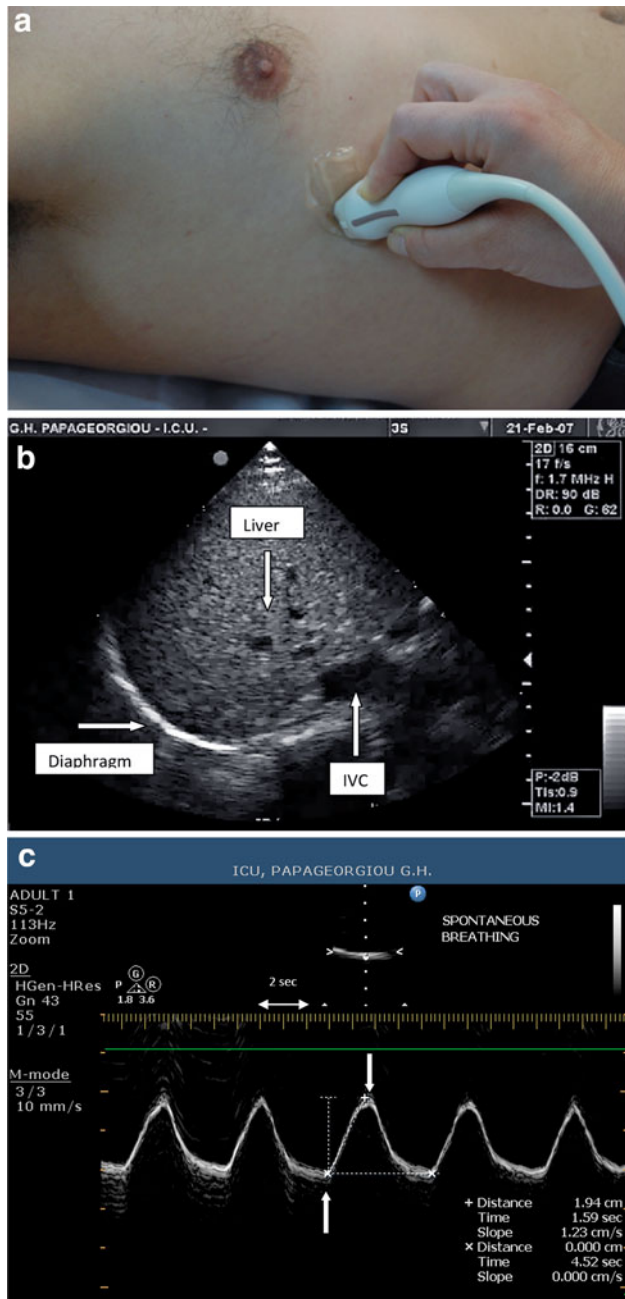


Fig. 1 a Probe position for B and M mode diaphragmatic excursion measurements with 3.5–5 MHz probe. b B-mode diaphragm sonography. The *bright line* reflects the diaphragm. c M-mode diaphragm sonography. *Arrows* indicate the beginning and the end of the diaphragmatic contraction. The distance between the *arrows*, indicate an excursion (displacement) of 1.9 cm. The inspiratory time (T_{insp}) is measured at 1.6 s, the cycle duration (T_{tot}) is 4.5 s, and the speed of diaphragmatic contraction (slope), calculated as the diaphragmatic excursion divided by the T_{insp}, is 1.2 cm/s

during quiet, deep breathing and voluntary sniffing, respectively [22]. Interestingly, the same diaphragmatic excursion values (1.8 cm) were found in ventilated patients who had succeeded in a weaning trial [16], with

no difference between the right and the left hemidiaphragm in both studies. In addition to the measurements of diaphragmatic excursion, the velocity of diaphragmatic contraction (slope, cm/s, Fig. 1c) can also be measured, like during the assessment of a maximal sniff. The latter is defined as a short, sharp inspiratory effort through the nose and it is thought to be a reproducible and quantitative assessment of diaphragmatic strength [23] although its role in the respiratory assessment of ICU patients remains to be determined. The slope (speed) of diaphragmatic contraction, during quiet breathing, has been measured at 1.3 ± 0.4 cm/s in forty healthy individuals without any significant differences between males and females [24].

Ultrasound has also been used to evaluate diaphragmatic thickness (tdi, mm) in the zone of apposition of the diaphragm to the rib cage. The zone of apposition is the area of the chest wall where the abdominal contents reach the lower rib cage (Fig. 2a). In this area, the diaphragm is observed as a structure made of three distinct layers (Fig. 2b): a non-echogenic central layer bordered by two echogenic layers, the peritoneum and the diaphragmatic pleurae [25]. To obtain adequate images of diaphragmatic thickness in M mode and 2D mode, a linear high-frequency probe (≥ 10 MHz) is necessary. The diaphragmatic thickness can be measured during quiet spontaneous breathing (Fig. 3) and during a maximal inspiratory and expiratory effort. An index of diaphragmatic thickening, the thickening fraction (TF) can be calculated using the M mode (TF = thickness at end-inspiration – thickness at end-expiration / thickness at end-expiration). Diaphragmatic thickening fraction can be used as an index of diaphragmatic efficiency as a pressure generator [26].

Normal values of diaphragmatic thickening: In normal individuals, there is a wide range of tdi at functional residual capacity (FRC), ranging between 1.8 to 3 mm. As lung volume increases from the residual volume (RV) to total lung capacity (TLC) there is a mean tdi increase of 54 % (range 42–78 %). Furthermore, the diaphragm also thickens during a maximal inspiratory pressure (P_{imax}) maneuver at FRC. A thickening ratio of 2.6 can be measured, dividing the diaphragmatic thickness during P_{imax} at FRC by the diaphragmatic thickness while relaxing at FRC [27, 28].

Accuracy and reproducibility

Several studies have addressed the subject of accuracy and reproducibility of ultrasounds to measure the diaphragmatic displacement and thickness in healthy volunteers and in ICU patients.

In a large study measuring diaphragmatic excursion in healthy volunteers, Boussuges [22] reported that the intraobserver reproducibility was 96 and 94 %, and the

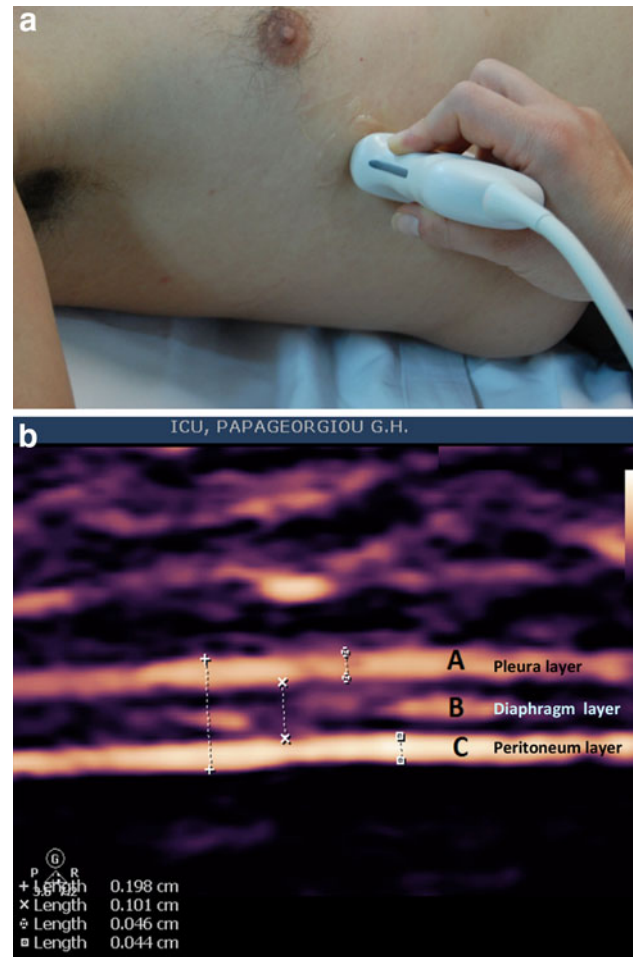


Fig. 2 a Probe position for B and M mode diaphragmatic thickness measurements in the zone of apposition with 10–12 MHz probe. b B-mode sonography of the diaphragm in the zone of apposition. A Echogenic diaphragmatic pleura, B non-echogenic central layer, C echogenic peritoneal layer. Notice the thickness measurement of each layer

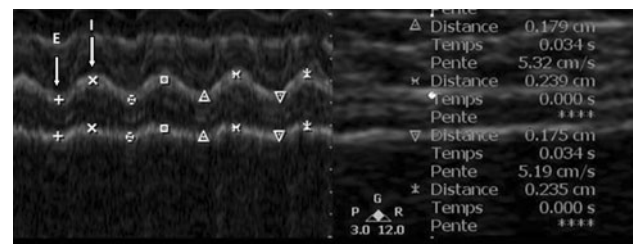


Fig. 3 Sonography of the diaphragm in the zone of apposition, in B-mode (right) and M-mode (left) during quiet breathing. E and I arrows indicates expiration and inspiration, respectively. Notice the diaphragmatic thickening during inspiration and the reproducibility of the thickness measurements during expiration (0.179 and 0.175 cm) and inspiration (0.239 and 0.235 cm)

interobserver reproducibility 95 and 91 %, during quiet breathing for the right and left diaphragm, respectively. The intraobserver and interobserver reproducibility (intra-

class correlation coefficients) of the diaphragmatic excursion measurements reported in ICU patients were found in the same range, between 88 and 99 % [4, 16].

Concerning the reproducibility of diaphragmatic thickness measurements during the same session, Vivier et al. [26] assessed analyser reproducibility, *intra-analyser* reproducibility (same settings analyzed repeatedly) and *inter-analyser* reproducibility (same recordings obtained separately by two different ultrasonographers). The values reported for repeatability (intra-class correlation coefficients) were all above 0.97. Coefficients of repeatability ranged around 7–8 % for *intra- or inter-analyser* repeatability and around 15–18 % for *intra- or inter-observer* repeatability.

To enhance reproducibility, there are some technical tips for the diaphragmatic echographer. First, one must know that there is little difference in the diaphragmatic excursion between the middle and the posterior part of the diaphragm [20]. Therefore, there is little reason to bother about the exact location, and the best diaphragmatic delineation in B-mode must be chosen before applying the M-mode. Second, the cursor for diaphragmatic excursion measurements in M-mode should always be as strictly perpendicular as possible with regards to the middle or posterior part of the diaphragm. This can be obtained by rotating the probe or by correcting the M-mode angle with a specific knob on the echo machine. Finally, for diaphragmatic thickness, use of the higher resolution linear probe (≥ 10 MHz) is necessary.

Limitations of the technique

There are limitations to diaphragmatic sonography, as well as some rules to be respected in order to avoid mistakes and errors in data collection and interpretation. One obvious limitation of diaphragmatic sonography, especially in ICU patients, is a poor acoustic window (poor quality images), and this has been reported to occur between 2 and 10 % [15, 16, 26].

When measuring diaphragmatic excursion, the sonographer should be as perpendicular as possible to the diaphragmatic excursion line, otherwise the accuracy and the repeatability of the diaphragmatic excursion measurements can be seriously affected. If the end point is the diaphragmatic excursion measurement, and the patient is under assisted modes of mechanical ventilation, the measured excursion (cm) will represent the sum of two forces working in the same direction; first, the force of the diaphragmatic contraction by itself, and second, the passive displacement of the diaphragm by the pressure applied by the ventilator. In this case, there is no means to distinguish which part of diaphragmatic displacement is passive, due to the external applied force, or active by the diaphragmatic contraction acting as a negative pressure generator. If the goal is to evaluate diaphragmatic excursion

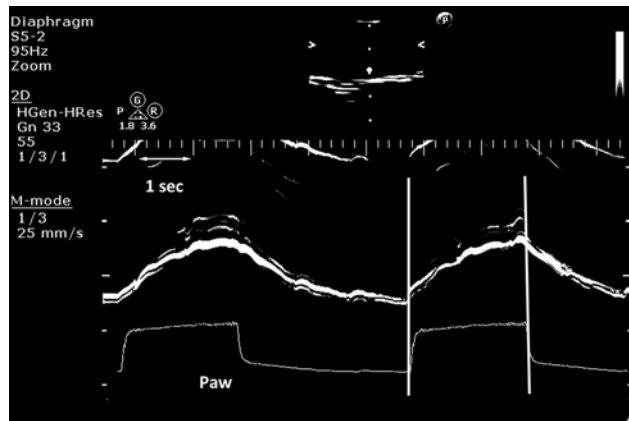


Fig. 4 Simultaneous recordings of diaphragmatic contraction in M-mode sonography and airway pressure waveform (Paw), in a patient under pressure support ventilation. Patient–ventilator synchrony is confirmed by the perfect synchronization of the beginning (first vertical line) and the end of the diaphragmatic contraction (second vertical line) and the triggering and the cycling off of the ventilator

as a force generator without the ventilator assistance, a brief recording (5–10 min) during spontaneous breathing is necessary. On the contrary, if one wants to detect diaphragmatic contractions and better understand patient–ventilator interactions (Figs. 4, 5) including the triggering delay of the ventilator (Fig. 6), the above-mentioned technical precautions are not mandatory.

Thickening is only influenced by active contraction, but can be affected by several factors evaluated in normal

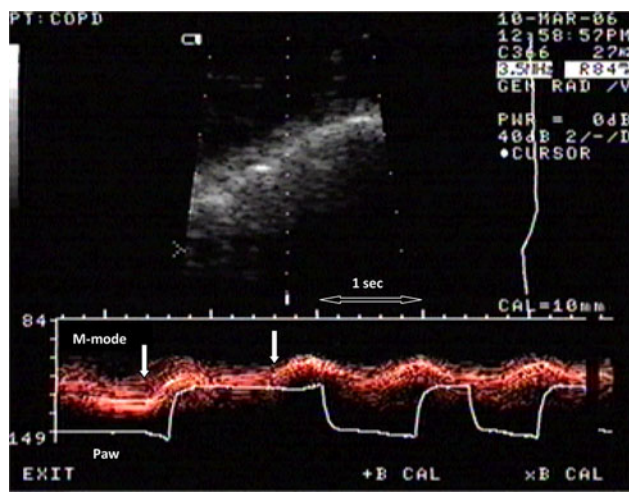


Fig. 5 Diaphragmatic contraction in M-mode sonography and Paw in a COPD patient under pressure support ventilation, indicating patient–ventilator asynchrony. In the first assisted breath, ventilator inspiratory time is much longer compared to the second breath. In the first assisted breath, we notice two diaphragmatic contractions (arrows); the second diaphragmatic contraction prolongs the inspiratory time of the assisted breath

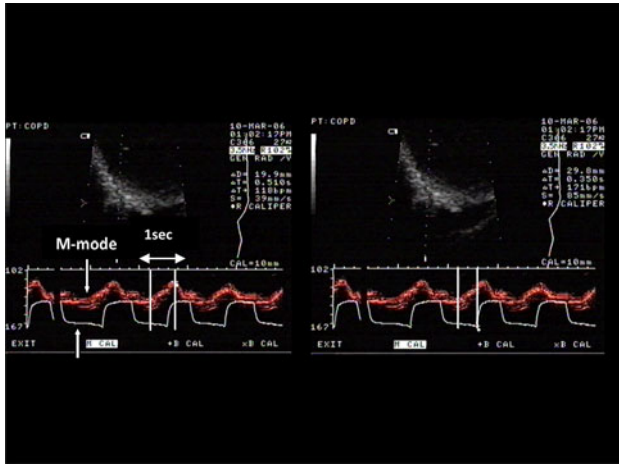


Fig. 6 Diaphragmatic contraction in M-mode sonography and airway pressure waveform in a COPD patient under pressure support ventilation illustrating triggering delay. The same picture is on the *right* and on the *left*. On the *left*, the duration of the diaphragmatic contraction is measured at 510 ms (*two vertical lines*). On the *right*, the time between the beginning of the diaphragmatic contraction and the triggering of the same assisted breath is measured at 350 ms (*two vertical lines*). This indicates that 350 out of 510 ms of the total diaphragmatic contraction has been wasted to overcome intrinsic PEEP before triggering the ventilator

or sick individuals; most of the studies have been performed in spontaneously breathing subjects [8, 9, 11, 19, 27, 28]. Thickness measurements during spontaneous breathing may be influenced by lung volume in a non-linear relationship [27, 28]. The diaphragmatic thickening is more pronounced above 50 % of the vital capacity [27], and there is a large increase in thickness between relaxation and 10 % of the inspiratory effort [28]. Furthermore, there are very few data on thickness in mechanically ventilated patients interacting [26], or not [14], with the ventilator.

Sonographic evaluation of diaphragmatic weakness and paralysis

Diaphragmatic paralysis

The diaphragm is the principal respiratory muscle during quiet breathing, and its dysfunction or paralysis can be observed in many clinical circumstances, such as major cardiac, thoracic or abdominal surgery, spinal injury, critical illness polyneuromyopathy, direct injury of the phrenic nerve or polyradiculoneuritis. Traditionally, methods to diagnose diaphragmatic weakness and paralysis examine the thoracic and abdominal pressures generated during spontaneous inspiration [13]. Measurement of transdiaphragmatic (Pdi) pressure remains the gold standard for diagnosing bilateral diaphragmatic

paralysis. However, Pdi is poorly sensitive and thus ineffective to diagnose unilateral diaphragmatic paralysis, since one efficient hemi-diaphragm is sufficient to generate adequate trans-diaphragmatic pressure during quiet breathing, and this is illustrated by the fact that 6 % of asymptomatic subjects have a dyskinetic diaphragm [29, 30]. Fluoroscopic examination of hemi-diaphragmatic motion during a sniff test may be useful in patients with unilateral diaphragmatic paralysis but false negative results can frequently occur [3, 18, 31]. Chest radiographs have a sensitivity of 90 % and a specificity of only 44 % in detecting unilateral diaphragmatic paralysis [32]. Pulmonary function tests used for the diagnosis of respiratory muscle weakness are highly dependent on lung volumes and patient effort [33, 34]. Magnetic resonance imaging (MRI) quantitative evaluation may allow the assessment of the excursion, synchronicity and velocity of diaphragmatic motion [35, 36]. However, all the above-mentioned techniques are not easily applicable in ICU patients, especially when they are intubated and mechanically ventilated.

Sonography is a simple, noninvasive alternative method of diaphragmatic imaging, ideal for repeated or prolonged examinations, such as those required for the diagnosis and follow up of uni- or bilateral diaphragmatic paralysis. Ultrasound has been used to assess the motion of the diaphragmatic dome [20, 25, 37–39]. Theoretically it should share some of the diagnostic limitations of fluoroscopy. Nonetheless, Houston et al. [40] diagnosed diaphragm motion abnormalities in 22 patients, of which only seven were also identified by fluoroscopy. This may be related to the fact that fluoroscopy images the highest portion of the diaphragm, which is the least moving part of the diaphragm [38, 41].

In unilateral or bilateral diaphragmatic paralysis, the negative pressure generated by the other respiratory muscles during inspiration, causes the diaphragm to passively move cranially instead of its normal caudal movement. The M-mode trace of the paralyzed side shows the absence of active or a paradoxical (i.e., cranial) movement (Fig. 2) particularly with the sniff test [18]. Moreover, the M-mode tracing of the diaphragmatic movement direction (cranial vs. caudal) allows distinguishing diaphragmatic weakness from paralysis. During inspiration, in patients with diaphragmatic weakness, one observes a reduced diaphragmatic caudal movement, and in patients with diaphragmatic paralysis, a paradoxical motion (Fig. 7) [22, 42]. Repeated documentation of diaphragmatic excursion for the same individual in follow-up examinations can provide information regarding the evolution of the paralysis. All the above sonographic measurements are applicable to diaphragmatic paralysis of variable etiology, including brachial plexus neuritis, phrenic nerve injury following cardiac or other surgery [11, 16, 35], spinal cord injury [10] or idiopathic cases. The qualitative discrimination between reduced and

paradoxical inspiratory movement may be of critical importance, since the latter is associated with delayed recovery of the phrenic nerve in cardiothoracic surgery patients, and is responsible for prolonged ventilatory support and hospital stay [21] probably due to more severe nerve injury.

Diaphragmatic weakness

In neuromuscular diseases, sonography can be used to evaluate the motion of the diaphragmatic dome (diaphragmatic displacement and speed of contraction) and the diaphragmatic thickening in the zone of apposition. In an ultrasonographic study of three patients with amyotrophic lateral sclerosis, Yoshioka et al. [11] described no change in diaphragmatic excursion and thickness between quiet breathing and maximal inspiratory effort, suggesting a severe impairment of the contractile function of the diaphragm. DeBruin et al. [8] studied the diaphragmatic thickness in children with Duchenne muscular dystrophy and found that, despite a greater diaphragmatic thickness at FRC, the thickening fraction was less than that of controls during maximum inspiratory effort (1.6 vs. 2.3). Such studies underline the usefulness of diaphragmatic sonography as a noninvasive and handy tool for the diagnosis of diaphragmatic dysfunction in patients with neuromuscular diseases and thus potentially enable the early discrimination of a subpopulation who may eventually need mechanical ventilator support [4, 43].

The evaluation of the diaphragm thickness (tdi) in the zone of apposition of the diaphragm to the rib cage can

also be an informative approach for weakness or paralysis. Gottesman and McCool [3] found that tdi of the paralyzed diaphragms was less than 2.0 mm, significantly thinner than that of the normally functioning diaphragms, a finding consistent with their hypothesis that chronic diaphragm paralysis results in atrophy. Measurements of tdi alone, however, may lead to false negative results in the settings of acute paralysis where atrophy has not yet occurred, or to false positive results in small individuals, since tdi varies with weight and height [44]. Due to the above limitations, to safely diagnose diaphragmatic paralysis, diaphragmatic thickening should be calculated during inspiration according to the formula $\Delta tdi = (tdi_{TLC} - tdi_{FRC})/tdi_{FRC}$ (where Δtdi is the change in diaphragm thickness, tdi_{TLC} is diaphragm thickness at TLC and tdi_{FRC} is diaphragm thickness at FRC) [3, 19]; all patients with a paralyzed diaphragm exhibited less than 20 % thickening of the diaphragm during inspiration to TLC [3]. The criterion of Δtdi is useful not only for the initial diagnosis of diaphragmatic weakness or paralysis, but also for monitoring subsequent recovery. The change in Δtdi strongly correlated with changes in vital capacity and the maximal inspiratory pressure reflecting inspiratory muscle strength [19]. Recently, in ICU patients under mechanical ventilation, it has been shown that diaphragmatic thickening fraction decreased in parallel with the diaphragmatic pressure time product, as soon as the work of breathing was alleviated by incremental levels of pressure support [26].

Diaphragmatic sonography in ICU patients during partial ventilatory support

A balloon-tipped catheter is traditionally used to measure esophageal and gastric pressure, and to evaluate inspiratory effort. Studies in healthy volunteers and also in patients under assisted modes of ventilation confirm that diaphragmatic M-mode sonography provides a mirror image of the changes in esophageal pressure (Fig. 8). Indeed, during inspiration, as the diaphragm contracts and the dome descends, the progressively decreasing esophageal pressure coincides with positive traces on M-mode diaphragmatic sonography; during expiration, esophageal pressure increases while the sonographic trace descends. Therefore, ultrasound can provide a modality that allows demonstration of the patient's initiation and completion of inspiratory effort in real time, obliterating the need for invasively inserting esophageal balloon catheters for that purpose.

Diaphragmatic M-mode sonography can provide valuable information in the evaluation of patients during partial ventilatory support. Simultaneous recordings of M-mode diaphragmatic sonography and airway pressures waveforms can allow visualizing that each patient's

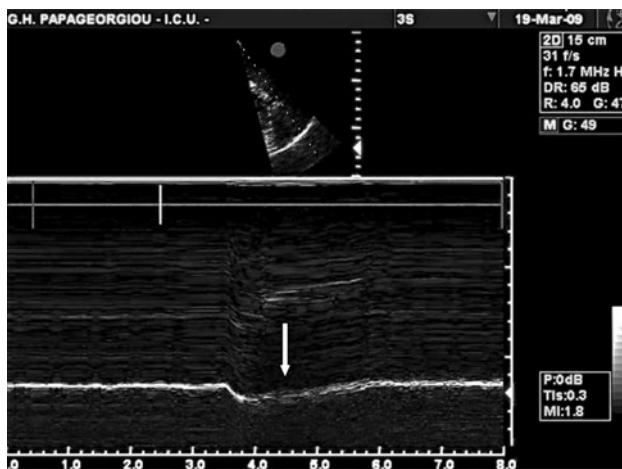


Fig. 7 Paradoxical diaphragmatic motion on M-mode sonography in a patient with Guillain-Barré syndrome (the scale at the bottom represents time in seconds). There is a cranial diaphragmatic movement (away from the probe) during spontaneous breathing due to diaphragmatic paralysis. The intercostal muscles recover earlier than the diaphragm and create a negative intrathoracic pressure which displaces the paralytic diaphragm inwards, into the thorax and away from the probe

inspiratory effort triggers the ventilator appropriately (Fig. 4). Therefore, real-time hemi-diaphragmatic sonography could be used in the evaluation of patient–ventilator interactions in clinical practice, in order to detect cases of patient–ventilator asynchrony (Figs. 5, 6). In these cases, diaphragmatic sonography could even allow a proper adjustment of the ventilator settings in order to optimize synchronization of the patient’s inspiratory effort with the assisted mechanical breath. This hypothesis, however, needs to be prospectively tested. Mechanical ventilation in controlled mode and possibly with high levels of partial ventilatory assist, can also result in ventilator-induced diaphragm dysfunction [17, 45]. Recent preliminary data suggest that sonographic assessment of the diaphragm can provide a noninvasive measurement of diaphragmatic thickness and allows to observe a progressive diaphragm thinning, as shown in seven patients receiving MV [14].

Post-operative diaphragmatic dysfunction

Diaphragmatic dysfunction contributes to the etiology of postoperative pulmonary complications after thoracic and abdominal surgery, leading to delayed weaning and prolonged stay in ICU. Kim et al. [12] demonstrated that the diaphragmatic inspiratory amplitude during deep breathing predicted changes in vital capacity throughout seven postoperative days in patients undergoing liver lobectomy. The best cutoff values of diaphragmatic inspiratory amplitude for detecting 30 and 50 % decreases of vital capacity from preoperative values, as calculated by receiver operating characteristic analysis, were 36 and

24 mm, with sensitivity of 94 and 81 % and specificity of 76 and 91 %, respectively ($p < 0.001$). In another study in cardiothoracic surgery patients by Lerolle et al. [4], a maximal positive diaphragmatic excursion of less than 25 mm was associated with severe diaphragmatic dysfunction as defined by a negative value of the Gilbert index [46]. The latter is an index which evaluates the diaphragm contribution to respiratory pressure swings during quiet ventilation and is calculated as the ratio of gastric pressure swing to transdiaphragmatic pressure swing; a negative value indicates a paradoxical motion of the diaphragm. This ultrasonographic threshold had an excellent negative likelihood ratio, which was confirmed by assessing patients with uncomplicated postoperative course, none of them having their maximal diaphragmatic excursion <25 mm [4]. Such studies highlight the advantages of a fully noninvasive technique, which is now increasingly available in the ICU, to focus on patients at high risk for postoperative respiratory complications.

Weaning from mechanical ventilation

Sonography may also be of help during weaning from mechanical ventilation. Jiang et al. [15] performed a B-mode ultrasonographic evaluation of the diaphragmatic movements by measuring the liver/spleen displacement during spontaneous breathing trials. This examination proved to be a good predictor for extubation outcome. Using a mean cutoff value of 1.1 cm of liver and spleen displacement, the sensitivity and specificity to predict successful extubation was 84.4 and 82.6 % respectively, better than traditional weaning parameters used in the trial, such as rapid shallow breathing index and Pi max [15]. Patients with adequate spontaneous tidal volume but poor diaphragmatic excursion were more likely to fail a breathing trial compared to patients with adequate spontaneous tidal volume and good diaphragmatic movement; this can be explained by the fact that spontaneous tidal volume represents the result of the combined activation of all respiratory muscles used without specifically measuring the contribution of the diaphragm, whereas diaphragmatic excursion represents the final result of combined diaphragmatic strength, intrathoracic and intra-abdominal pressures [15]. The authors suggested that diaphragmatic movement was a more sensitive and specific parameter than volume-associated weaning parameters in predicting extubation outcome. Patients who recruit accessory respiratory muscles to maintain adequate tidal volumes may therefore experience more difficulties to sustain spontaneous breathing and fail extubation more often [15]. Kim et al. [16] investigated diaphragmatic dysfunction diagnosed by M-mode ultrasonography (vertical excursion <10 mm or paradoxical

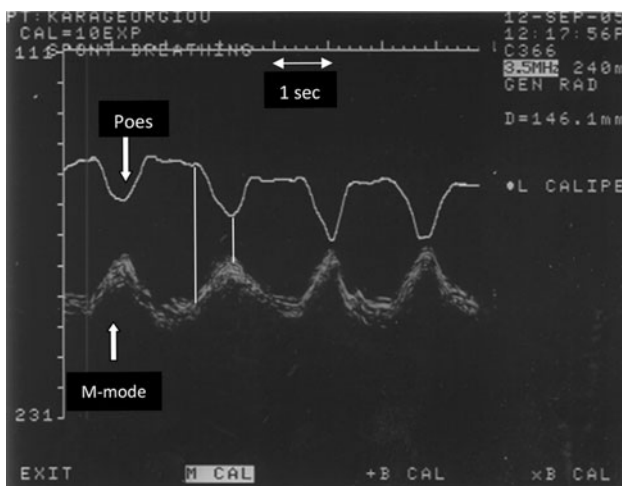


Fig. 8 Simultaneous recording of the esophageal pressure and M-mode diaphragmatic sonography. Notice the perfect synchronization of the beginning of diaphragmatic contraction and the drop of the esophageal pressure (first vertical line). The second vertical line indicates the end of inspiration and the maximal pressure drop in the esophageal pressure

movements) in 88 medical intensive care unit patients, and they found a prevalence of ultrasonographic diaphragmatic dysfunction of 29 %. Patients with diaphragmatic dysfunction had longer weaning times and total ventilation times than patients without diaphragmatic dysfunction. Their results also suggest that ultrasonography of the diaphragm may be useful in identifying patients at high risk of difficult weaning. However, the role of diaphragmatic excursion as a predictor of extubation outcome in the weaning process remains to be further evaluated.

Future applications and conclusions

Data on diaphragmatic sonography are still scarce compared to cardiac or lung ultrasound applications in ICU patients. For the ICU physician, already familiar with the ECHO machine for cardiac or lung applications, the learning curve of the diaphragmatic sonography is very short. Future research on diaphragmatic sonography in the ICU environment might evaluate the relationship between diaphragmatic thickness and displacement, assess patient-ventilator asynchrony, compare thickness and diaphragmatic displacement with more invasive parameters evaluating the diaphragmatic strength (transdiaphragmatic pressure at rest and during maximal efforts), titrate external PEEP to overcome auto-PEEP in order to improve ventilator trigger delay and synchrony, follow diaphragmatic atrophy or recovery from atrophy in patients suffering from critical illness polyneuromyopathy or assess diaphragmatic function in patients during prolonged or difficult weaning from mechanical ventilation. One additional interesting point could be the assessment of diaphragmatic relaxation. Abnormalities of diaphragmatic relaxation have been reported as a marker of impaired contractile performance [47, 48]. Diaphragmatic relaxation rate is so far measured using the trans-diaphragmatic pressure. As illustrated by Figs. 1c, 4 and 8, diaphragmatic relaxation could be evaluated noninvasively by diaphragmatic sonography.

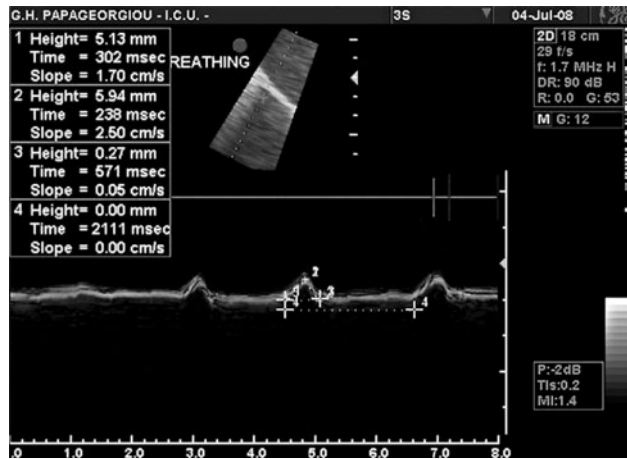


Fig. 9 Diaphragmatic contraction in M-mode sonography during a spontaneous breathing trial in a patient suffering from critical illness neuromyopathy (the *scale at the bottom* represent time in seconds). Diaphragmatic weakness is evidenced by the very small diaphragmatic displacement (0.5 cm)

Ultrasonography appears to be a promising tool in the evaluation of diaphragmatic function in ICU patients [49]. It has the advantage of being fully noninvasive and is becoming widely available in an increasing number of ICUs, bypassing limitations of previously used methods for this purpose. Diaphragmatic ultrasonography provides qualitative and quantitative information regarding diaphragmatic function, as part of an overall respiratory assessment in ICU patients. Apart from clear findings, such as during diaphragmatic paralysis, ultrasonographic evaluation of diaphragmatic function may become helpful in identifying a subpopulation of ICU patients at high risk of further respiratory complications. Further research regarding ultrasonographic diaphragmatic evaluation in pathologies such as sepsis, ventilator-induced diaphragmatic dysfunction and ICU neuromyopathy are anticipated with great interest.

Acknowledgments The authors are grateful to Aissam Lyazidi for technical assistance in preparation of the manuscript.

References

1. Beaulieu Y, Marik PE (2005) Bedside ultrasonography in the ICU: part 1. *Chest* 128:881–895
2. Beaulieu Y, Marik PE (2005) Bedside ultrasonography in the ICU: part 2. *Chest* 128:1766–1781
3. Gottesman E, McCool FD (1997) Ultrasound evaluation of the paralyzed diaphragm. *Am J Respir Crit Care Med* 155:1570–1574
4. Lerolle N, Guerot E, Dimassi S, Zegdi R, Faisy C, Fagon JY, Diehl JL (2009) Ultrasonographic diagnostic criterion for severe diaphragmatic dysfunction after cardiac surgery. *Chest* 135:401–407
5. Tobin MJ, Laghi F, Brochard L (2009) Role of the respiratory muscles in acute respiratory failure of COPD: lessons from weaning failure. *J Appl Physiol* 107:962–970
6. Ayoub J, Milane J, Targhetta R, Prioux J, Chamari K, Arbeille P, Jonquet O, Bourgeois JM, Prefaut C (2002) Diaphragm kinetics during pneumatic belt respiratory assistance: a sonographic study in Duchenne muscular dystrophy. *Neuromuscul Disord* 12:569–575

7. Cohen E, Mier A, Heywood P, Murphy K, Boulton J, Guz A (1994) Diaphragmatic movement in hemiplegic patients measured by ultrasonography. *Thorax* 49:890–895
8. De Bruin PF, Ueki J, Bush A, Khan Y, Watson A, Pride NB (1997) Diaphragm thickness and inspiratory strength in patients with Duchenne muscular dystrophy. *Thorax* 52:472–475
9. DePalo VA, McCool FD (2002) Respiratory muscle evaluation of the patient with neuromuscular disease. *Semin Respir Crit Care Med* 23:201–209
10. Hardy F, Walker J, Sawyer T (2009) Sonographic measurement of diaphragm movement in patients with tetraplegia. *Spinal Cord* 47:832–834
11. Yoshioka Y, Ohwada A, Sekiya M, Takahashi F, Ueki J, Fukuchi Y (2007) Ultrasonographic evaluation of the diaphragm in patients with amyotrophic lateral sclerosis. *Respirology* 12:304–307
12. Kim SH, Na S, Choi JS, Na SH, Shin S, Koh SO (2010) An evaluation of diaphragmatic movement by M-mode sonography as a predictor of pulmonary dysfunction after upper abdominal surgery. *Anesth Analg* 110:1349–1354
13. Diehl JL, Lofaso F, Deleuze P, Similowski T, Lemaire F, Brochard L (1994) Clinically relevant diaphragmatic dysfunction after cardiac operations. *J Thorac Cardiovasc Surg* 107:487–498
14. Grosu HB, Lee YI, Lee J, Eden E, Eikermann M, Rose K (2012) Diaphragm muscle thinning in mechanically ventilated patients. *Chest*: PMID: 22722229
15. Jiang JR, Tsai TH, Jerng JS, Yu CJ, Wu HD, Yang PC (2004) Ultrasonographic evaluation of liver/spleen movements and extubation outcome. *Chest* 126:179–185
16. Kim WY, Suh HJ, Hong SB, Koh Y, Lim CM (2011) Diaphragm dysfunction assessed by ultrasonography: influence on weaning from mechanical ventilation. *Crit Care Med* 39:2627–2630
17. Petrof BJ, Jaber S, Matecki S (2010) Ventilator-induced diaphragmatic dysfunction. *Curr Opin Crit Care* 16:19–25
18. Lloyd T, Tang YM, Benson MD, King S (2006) Diaphragmatic paralysis: the use of M mode ultrasound for diagnosis in adults. *Spinal Cord* 44:505–508
19. Summerhill EM, El-Sameed YA, Glidden TJ, McCool FD (2008) Monitoring recovery from diaphragm paralysis with ultrasound. *Chest* 133:737–743
20. Harris RS, Giovannetti M, Kim BK (1983) Normal ventilatory movement of the right hemidiaphragm studied by ultrasonography and pneumotachography. *Radiology* 146:141–144
21. Kunovsky P, Gibson GA, Pollock JC, Stejskal L, Houston A, Jamieson MP (1993) Management of postoperative paralysis of diaphragm in infants and children. *Eur J Cardiothorac Surg* 7:342–346
22. Boussuges A, Gole Y, Blanc P (2009) Diaphragmatic motion studied by m-mode ultrasonography: methods, reproducibility, and normal values. *Chest* 135:391–400
23. Steier J, Kaul S, Seymour J, Jolley C, Rafferty G, Man W, Luo YM, Roughton M, Polkey MI, Moxham J (2007) The value of multiple tests of respiratory muscle strength. *Thorax* 62:975–980
24. Soilemezi E, Tsagourias M, Talias MA, Soteriades ES, Makrakis V, Zakynthinos E, Matamis D (2012) Sonographic assessment of changes in diaphragmatic kinetics induced by inspiratory resistive loading. *Respirology*. doi:10.1111/resp12011
25. Ayoub J, Cohendy R, Dauzat M, Targhetta R, De la Coussaye JE, Bourgeois JM, Ramonaxo M, Prefaut C, Pourcelot L (1997) Non-invasive quantification of diaphragm kinetics using m-mode sonography. *Can J Anaesth* 44:739–744
26. Vivier E, Mekontso Dessap A, Dimassi S, Vargas F, Lyazidi A, Thille AW, Brochard L (2012) Diaphragm ultrasonography to estimate the work of breathing during non-invasive ventilation. *Intensive Care Med* 38:796–803
27. Cohn D, Benditt JO, Eveloff S, McCool FD (1997) Diaphragm thickening during inspiration. *J Appl Physiol* 83:291–296
28. Ueki J, De Bruin PF, Pride NB (1995) In vivo assessment of diaphragm contraction by ultrasound in normal subjects. *Thorax* 50:1157–1161
29. Scillia P, Cappello M, De Troyer A (2004) Determinants of diaphragm motion in unilateral diaphragmatic paralysis. *J Appl Physiol* 96:96–100
30. Tobin M, Laghi F (1998) Monitoring of respiratory muscle function. In: Tobin M (ed) *Principle and practice of respiratory care monitoring*. McGraw-Hill, New York, pp 497–544
31. Alexander C (1966) Diaphragm movements and the diagnosis of diaphragmatic paralysis. *Clin Radiol* 17:79–83
32. Chetta A, Rehman AK, Moxham J, Carr DH, Polkey MI (2005) Chest radiography cannot predict diaphragm function. *Respir Med* 99:39–44
33. Fiz JA, Montserrat JM, Picado C, Plaza V, Agusti-Vidal A (1989) How many manoeuvres should be done to measure maximal inspiratory mouth pressure in patients with chronic airflow obstruction? *Thorax* 44:419–421
34. Wilcox PG, Pardy RL (1989) Diaphragmatic weakness and paralysis. *Lung* 167:323–341
35. Kiryu S, Loring SH, Mori Y, Rofsky NM, Hatabu H, Takahashi M (2006) Quantitative analysis of the velocity and synchronicity of diaphragmatic motion: dynamic MRI in different postures. *Magn Reson Imaging* 24:1325–1332
36. Kolar P, Sulc J, Kyncl M, Sanda J, Neuwirth J, Bokarius AV, Kriz J, Kobesova A (2010) Stabilizing function of the diaphragm: dynamic MRI and synchronized spirometric assessment. *J Appl Physiol* 109:1064–1071
37. Epelman M, Navarro OM, Daneman A, Miller SF (2005) M-mode sonography of diaphragmatic motion: description of technique and experience in 278 pediatric patients. *Pediatr Radiol* 35:661–667
38. Houston JG, Morris AD, Howie CA, Reid JL, McMillan N (1992) Technical report: quantitative assessment of diaphragmatic movement—a reproducible method using ultrasound. *Clin Radiol* 46:405–407
39. Riccabona M, Sorantin E, Ring E (1998) Application of M-mode sonography to functional evaluation in pediatric patients. *Eur Radiol* 8:1457–1461
40. Houston JG, Fleet M, Cowan MD, McMillan NC (1995) Comparison of ultrasound with fluoroscopy in the assessment of suspected hemidiaphragmatic movement abnormality. *Clin Radiol* 50:95–98
41. Young DA, Simon G (1972) Certain movements measured on inspiration-expiration chest radiographs correlated with pulmonary function studies. *Clin Radiol* 23:37–41
42. Gerscovich EO, Cronan M, McGahan JP, Jain K, Jones CD, McDonald C (2001) Ultrasonographic evaluation of diaphragmatic motion. *J Ultrasound Med* 20:597–604
43. Remerand F, Dellamonica J, Mao Z, Ferrari F, Bouhemad B, Jianxin Y, Arbelot C, Lu Q, Ichai C, Rouby JJ (2010) Multiplane ultrasound approach to quantify pleural effusion at the bedside. *Intensive Care Med* 36:656–664

-
44. McCool FD, Benditt JO, Conomos P, Anderson L, Sherman CB, Hoppin FG Jr (1997) Variability of diaphragm structure among healthy individuals. *Am J Respir Crit Care Med* 155:1323–1328
45. Hudson MB, Smuder AJ, Nelson WB, Bruells CS, Levine S, Powers SK (2012) Both high level pressure support ventilation and controlled mechanical ventilation induce diaphragm dysfunction and atrophy. *Crit Care Med* 40:1254–1260
46. Gilbert R, Auchincloss JH Jr, Peppi D (1981) Relationship of rib cage and abdomen motion to diaphragm function during quiet breathing. *Chest* 80:607–612
47. Coirault CCD, Lecarpentier Y (1999) Relaxation of diaphragm muscle. *J Appl Physiol* 87:1243–1252
48. Esau SABF, Grassino A, Permutt S, Roussos C, Prady RL (1983) Changes in relaxation rate with diaphragmatic fatigue in humans. *J Appl Physiol* 54:1353–1360
49. Lerolle N, Diehl JL (2011) Ultrasonographic evaluation of diaphragmatic function. *Crit Care Med* 39:2760–2761