CHEST Original Research
CRITICAL CARE MEDICINE

A-Lines and B-Lines

Lung Ultrasound as a Bedside Tool for Predicting Pulmonary Artery Occlusion Pressure in the Critically Ill

Daniel A. Lichtenstein, MD, FCCP; Gilbert A. Mezière, MD; Jean-François Lagoueyte, MD; Philippe Biderman, MD; Ivan Goldstein, MD; and Agnès Gepner, MD

Background: **The risk of pulmonary edema is the main limiting factor in fluid therapy in the critically ill. Interstitial edema is a subclinical step that precedes alveolar edema. This study assesses a bedside tool for detecting interstitial edema, lung ultrasound. The A-line is a horizontal artifact indicating a normal lung surface. The B-line is a kind of comet-tail artifact indicating subpleural interstitial edema. The relationship between anterior interstitial edema detected by lung ultrasound and the pulmonary artery occlusion pressure (PAOP) value was investigated.**

Method: **We performed a prospective study in medicosurgical ICUs of university-affiliated teaching hospitals. We enrolled 102 consecutive mechanically ventilated patients who all underwent pulmonary artery catheterization. We defined A-predominance as a majority of anterior A-lines and B-predominance as a majority of anterior B-lines. These patterns were correlated with PAOP.**

Results: **For diagnosing PAOP ≤ 13 mm Hg, A-predominance had 90% specificity, 67% sensitivity, 91% positive predictive value, and 65% negative predictive value. For diagnosing PAOP ≤ 18 mm Hg, A-predominance had 93% specificity, 50% sensitivity, 97% positive predictive value, and 24% negative predictive value, respectively.**

Conclusions: **A-predominance indicates dry interlobular septa. Specific to predicting a low PAOP value, A-predominance suggests that fluid may be given without initial concern for the development of hydrostatic pulmonary edema. B-predominance indicates interstitial syndrome, which is usually related to interstitial edema. B-predominance is observed in a wide range of PAOP values, precluding conclusions about the need for fluid therapy. This bedside potential will be appreciated by those intensivists who envision fluid therapy based on low PAOP values and who consider that using the concept of a safety factor provided by lung ultrasound is logical.** ***(CHEST 2009; 136:1014–1020)***

Abbreviations: FN = false-negative; FP = false-positive; NPV = negative predictive value; PAC = pulmonary artery catheter; PAOP = pulmonary artery occlusion pressure; PEEP = positive end-expiratory pressure; PPV = positive predictive value; TN = true-negative; TP = true-positive

Early massive fluid therapy has been proven to benefit patients in septic shock.[1] How to assess the end point where the patient has received optimal fluid therapy, while the signs of circulatory failure persist, has not been satisfactorily ascertained. This question has long been approached by using the pulmonary artery occlusion pressure (PAOP), a value obtained using the pulmonary artery catheter (PAC). The PAOP provides information on left ventricular end-diastolic pressure,[2,3] which classically guides fluid therapy,[4] and defines the risk for hydrostatic pulmonary edema.

Lung ultrasound is increasingly becoming a diagnostic tool in the critical care setting, providing standardized data.[5] The B-line is an artifact that correlates with interstitial edema.[6,7] This study considers the interest of the A-line and B-line in predicting the PAOP value.

Materials and Methods

Methods

A prospective 5-year study evaluated 103 critically ill patients receiving a PAC in medicosurgical ICUs. These patients required hemodynamic measurements at the discretion of the managing team faced with instability or complex hemodynamic situations (Table 1). Patients were consecutive in the context of the part-time presence of the ultrasound operators (DL and GM), who were blinded to the hemodynamic measurements made by other members of the managing team. Hindrances to an examination (extensive dressings or pneumothorax) were a criterion for exclusion. The study was discontinued because PAC use was progressively discontinued.

The ultrasound examination was performed 1 to 10 min before hemodynamic measurements were made and lasted < 1 min. Through percutaneous introduction into the jugular, femoral, or subclavian veins, 7.5F right heart balloon flotation catheters (Swan-Ganz catheter; Baxter, Edwards; Irvine, CA) were inserted into the pulmonary artery. All patients were mechanically ventilated and adequately sedated. The following usual precautions were taken: pressure head (*ie*, the piece between the catheter [patient] and the screen, where hydrostatic pressure is converted into digital data displayed on a screen) at the fourth intercostal space at the middle axillary line; the catheter line was flushed; the zero level was checked before measurement; correct catheter placement was checked by using radiography; and appropriate pressure traces were determined surrounding balloon inflation. Only the PAOP curve displaying left auricle pressure curves (*ie*, characteristic a and v waves) with a value below the average pulmonary artery pressure was considered. Respiratory variations of PAOP remained under respiratory variations of pulmonary artery diastolic pressure. The values recorded for PAOP measurements were obtained from graphic recordings on end-expiration (determined by using pressure curves), without withdrawing patients from mechanical ventilation, with a null or moderate positive end-expiratory pressure (PEEP) level unchanged.

The PAOP was considered a marker of lung filtration pressure rather than end-diastolic left ventricle pressure. If there was mitral regurgitation, the PAOP measurement considered the entire curve (not the nadir value).

The data from this observational study were collected during routine ultrasound examinations, which were not invasive and did not involve therapeutic changes or randomization. Our Ethics Committee stated that approval was not required and waived the requirement for informed consent.

Ultrasound Technique

Ultrasonography is routine in our ICU.[8] Its use has been extended from the heart to the whole body since 1989.[9] An ultrasound device (ADR-4000; ATL; Tempe, AZ) and a 29-cm-wide ultrasound device (Hitachi-405; Hitachi; Tokyo, Japan) with a 5-MHz microconvex probe without Doppler were used. The following briefly reviews the ultrasound technique used. We defined the stages of investigation by combining a zone and the patient's position. Stage 1 considers the anterior wall in supine patients. The probe is longitudinally applied perpendicular to the wall. For acute respiratory failure assessment, we used one upper point and one lower point per lung, requiring < 30 s.[10] For hemodynamic assessment, we divide the anterior surface into four areas, inserting the probe at the center of each, making four points of investigation per lung with dichotomous answer, requiring < 1 min. The features of the pleural line are illustrated in Figure 1.

The air/fluid ratio, gradually increasing from pleural effusion (pure fluid) to alveolar consolidation, interstitial syndrome, normal lung, and pneumothorax (pure air), generates characteristic patterns.[10] The nomenclature for describing air artifacts has been published.[11]

Normal lung tissue yields lung sliding and horizontal repetition artifacts arising from the pleural line, which have been termed *A-lines* (Fig 1). Normal interlobular septa are not detected. Lung sliding is a to-and-fro dynamic of the pleural line, indicating movement of the visceral pleura past the parietal pleura from the respiratory craniocaudal excursion of the lung.

The following seven types of B-lines have long been described by their characteristic features[12]: comet-tail artifact (roughly vertical); artifact arising from the pleural line; hyperechoic artifact (isoechoic to the pleural line); well-defined artifact (laser-beam-like); an artifact spreading up to the edge of the screen without fading; an artifact erasing the physiologic A-lines; and an artifact moving with lung sliding (Fig 2). The B-line is generated by an air-fluid mixture, which occurs when subpleural interlobular septa surrounded by subpleural air-filled alveoli become edematous. Three or more B-lines visible between two ribs define B+ lines, or *lung rockets*. With a CT scan as a reference, lung rockets appear fully sensitive and specific for demonstrating the subpleural thickened interlobular septa and/or

Table 1—*Patient Characteristics*

Characteristics	Values
Patients, No.	102
Sex	
Male	62
Female	40
Age, yr	
Mean (SD)	57 (19)
Range	20–88
PEEP, cm H_2O	0–7
Mean (± SD) tidal volume, mL/kg	7 ± 1
Plateau pressure, cm H_2O	< 32
Patients sedated on mechanical ventilation	All
Indications for pulmonary artery catheterization, No.	
Septic shock	24
ARDS	28
Acute hemodynamic pulmonary edema	13
Severe trauma	9
Complications following various surgeries	8
Hypovolemic or anaphyllactic shock	6
Severe pulmonary disorders	5
Severe abdominal disorders	4
Severe cardiac disorders	3
Various	2
Patients with pericardial tamponade	0
Patients that had mitral regurgitation with V wave	5

Manuscript received January 1, 2009; revision accepted May 17, 2009.
Affiliations: From the Medical ICU (Drs. Lichtenstein and Biderman), Hôpital Ambroise-Paré, Boulogne, France; ICU (Drs. Mezière and Gepner), Centre Hospitalier, Saint-Cloud, France; Hôpital Bichat (Dr. Lagoueyte), Paris, France; and Surgical ICU (Dr. Goldstein), Hôpital Pitié, Paris, France.
Correspondence to: Daniel A. Lichtenstein, MD, FCCP, Service de Réanimation Médicale, Hôpital Ambroise-Paré, F-92100 Boulogne (Paris-Ouest), France; e-mail: dlicht@free.fr

DOI: 10.1378/chest.09-0001

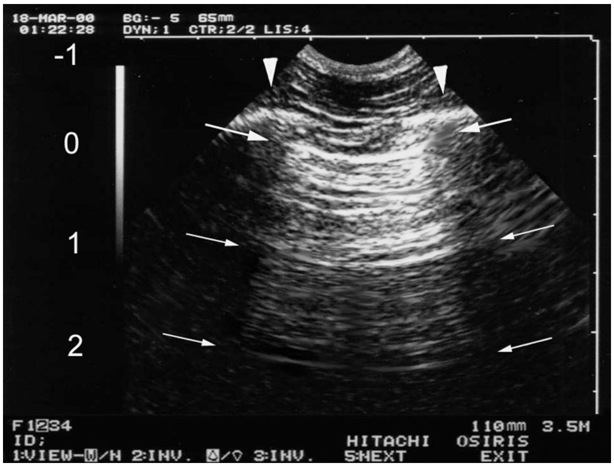

FIGURE 1. Pleural line and A-lines. Intercostal space, longitudinal view: the ribs (arrowheads) yield hyperechoic curves generating frank acoustic shadows. The pleural line is a roughly horizontal, hyperechoic line (large arrows), which is located 0.5 cm below the rib line in the adult. Its visible length between two ribs is 2.5 cm. The upper rib (pleural line)-lower rib pattern outlines the bat sign, a basic landmark. The A-lines (thin arrows) are those repetitive horizontal echoic lines that arise from the pleural line at regular intervals (skin-pleural line distance). They indicate subpleural air, which completely reflects the ultrasound beam. The length of an A-line can be roughly the same as the pleural line, but it can be shorter, and even not visible (see Fig 4). On the left margin: "-1" indicates the skin location, "0" the pleural line, "1" the first A-line, "2" a second A-line (lines A1, A2. . .). Note the equidistance between all these structures.

ground-glass areas (*ie*, interstitial edema) [Fig 3].[6] B-lines are observed at the lateral bases in 27% of healthy subjects.[6] Other comet-tail artifacts exist, none having B-line characteristics. Given the clinical importance of the description of the A-lines and B-lines, we provide here figures of some of them (Fig 4). Pneumothorax was excluded by observing lung sliding or its equivalents.[11]

Study Design

We used a previously described nomenclature[10] for stage 1 artifacts that was designed for assessing acute respiratory failure. The A-profile designates anterior-predominant bilateral A-lines associated with lung sliding. The A′-profile is an A-profile with abolished lung sliding. The B-profile designates anterior-predominant bilateral lung rockets associated with lung sliding. The B′-profile is a B-profile with abolished lung sliding. The A/B-profile designates anterior-predominant A-lines on one side and predominant lung rockets on the other.

We defined A-predominance as any profile without bilateral lung rockets, and the B-predominance as bilateral lung rockets. This concept focused on interstitial patterns and allowed a dichotomous approach to the lung.

The dependent conditions were PAOP ≤ 13 mm Hg and PAOP ≤ 18 mm Hg, the two usual cutoff points. The outcome variable was A-predominance or B-predominance.

Statistical Tests

The condition was low PAOP, and the sign was A-predominance, defining true-positive (TP), true-negative (TN), false-positive (FP), and false-negative (FN) results. The specificity of the tests was defined as TN/TN + FP, the sensitivity as TP/TP + FN, the positive predictive value (PPV) as TP/TP + FP, and the negative predictive value (NPV) as TN/TN + FN.

RESULTS

Of 103 patients, 1 patient experienced a pneumothorax and was excluded. The study enrolled 102 patients (Table 1), resulting in 102 comparisons of PAOP and lung ultrasound.

Distribution of the Respective Values

Figure 5 indicates the A-predominance to the left columns (with three subtypes), and B-predominance to the right (with two subtypes). A-predominance was observed in 45 cases, and B-predominance in 57 cases.

Results for Cutoff at 13 mm Hg

Sixty-one patients had PAOP ≤ 13 mm Hg. The A-profile was found in 29 cases, the A′-profile in 7

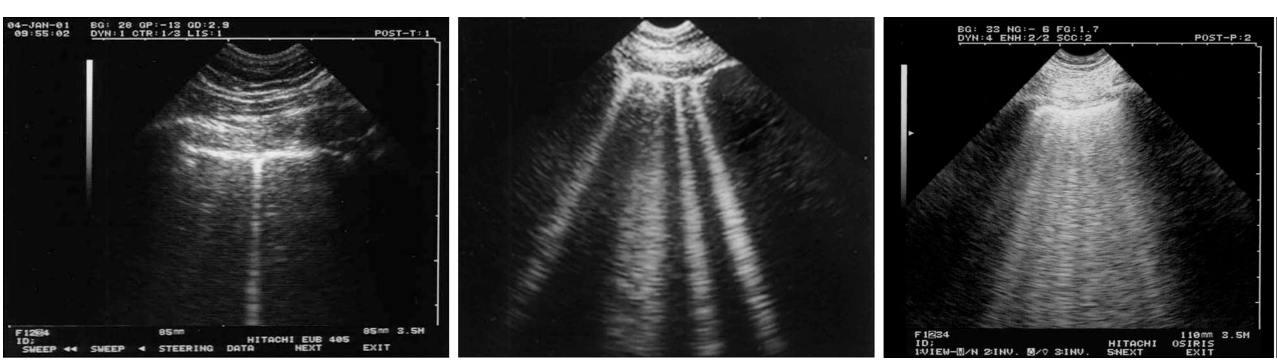

FIGURE 2. B-lines. *Left*: healthy subject. One isolated B-line, called "b-line," without pathologic meaning (possibly minor fissura). *Middle and right*: pulmonary edema. Several (three or more) B-lines are visible between two ribs, and define interstitial syndrome. This pattern was labeled lung rockets (or B+ lines). *Middle*: four or five B-lines are visible. The distance between two B-lines (at the pleural line) is roughly 7 mm in the adult, hence the name "B7-lines." B7-lines correlate with thickened subpleural interlobular septa. *Right*: seven or eight B-lines are visible, called *B-3 lines* (the distance between two B-lines at the pleural line is roughly 3 mm). B3-lines correlate with subpleural ground-glass lesions.

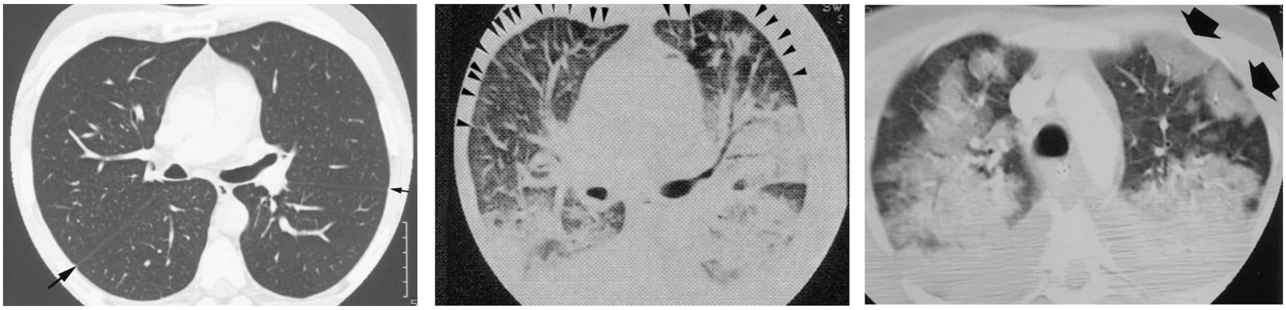

FIGURE 3. CT scan correlations. These three CT scan images correspond to the images in Figure 2. *Left*: normal CT scan. No element is visible at the anterior chest wall (apart from isolated fissures [arrows]). *Middle*: acute alveolar and interstitial syndrome. The subpleural interlobular septa are thickened and therefore visible on the CT scan (arrows). *Right*: acute alveolar and interstitial syndrome. Ground-glass areas abut the anterior left lung (arrows).

cases, the A/B-profile in 5 cases, the B′-profile in 7 cases, and the B-profile in 13 cases.

Forty-one patients had PAOP > 13 mm Hg. The A-profile was found in 3 cases, the A′-profile in none, the A/B-profile in 1 case, the B′-profile in 15 cases, and the B-profile in 22 cases.

Results for Cutoff at 18 mm Hg

Eighty-seven patients had PAOP ≤ 18 mm Hg. The A-profile was found in 31 cases, the A′-profile in 7 cases, the A/B-profile in 6 cases, the B′-profile in 21 cases, and the B-profile in 22 cases.

Fifteen patients had PAOP > 18 mm Hg. The A-profile was found in one case, the A′-profile in none, the A/B-profile in none, the B′-profile in 1 case, and the B-profile in 13 cases.

For diagnosing PAOP ≤ 13 mm Hg, A-predominance showed 90% specificity, 67% sensitivity, 91% PPV, and 65% NPV. For diagnosing PAOP ≤ 18 mm Hg, A-predominance showed 93% specificity, 50% sensitivity, 97% PPV, and 24% NPV.

Discussion

Until now, lung ultrasound has been used for diagnosing the lung disorders: pneumothorax, pneumonia, COPD, asthma, and pulmonary embolism,[7,9–12] for which it showed high accuracy, as has been confirmed by other studies.[13–19] In particular, ultrasound proved to be an accurate test for diagnosing interstitial syndrome.[6] Pulmonary edema combines respiratory and hemodynamic phenomena. The present study extends

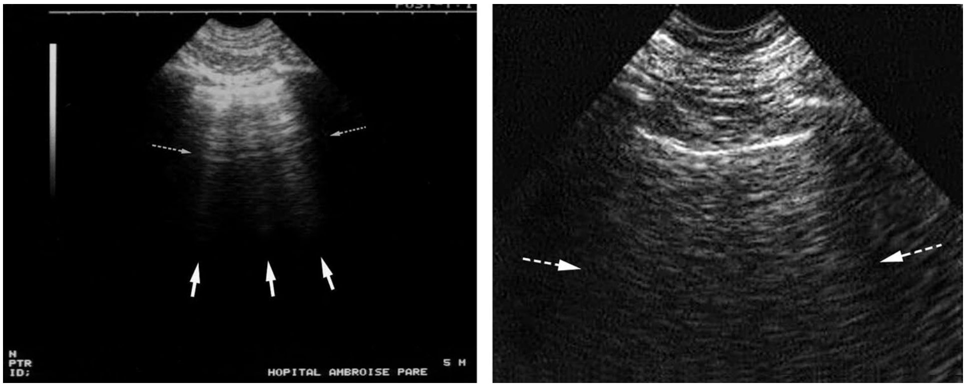

FIGURE 4. Z-lines and O-lines. *Left:* the Z-lines (vertical arrows) are comet-tail artifacts arising from the pleural line, with the following five features allowing distinction with B-lines: (1) less echoic than the pleural line; (2) ill-defined; (3) short, vanishing after 2 to 4 cm; (4) not erasing the A-lines (horizontal arrows); and (5) not moving with lung sliding. Three Z-lines are visible here. They are usually seen in healthy subjects and (as here) in those with pneumothorax. The Z-line seems, in our experience, devoid of meaning. *Right:* the O-line, one possible pattern: absence of any horizontal or vertical artifact. The expected location of an A-line would be at the exact level of the arrows. A slight movement of the probe often brings out A-lines. Example of an O-line (for non-A-line, non-B-line). This pattern should be considered as having the same meaning as an A-line.

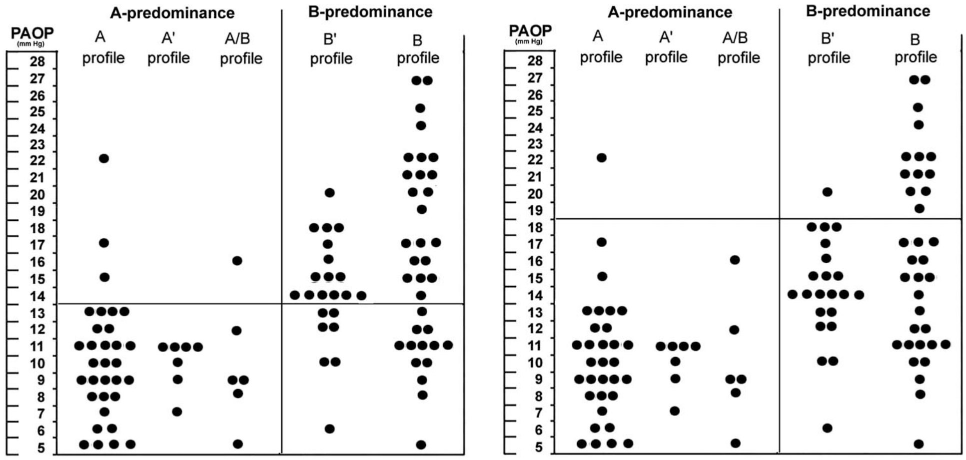

FIGURE 5. Overall results. *Left*: results with a cutoff point at 13 mm Hg. *Right*: results with a cutoff point at 18 mm Hg.

the potential of lung ultrasound to the hemodynamic management of critically ill patients. Our results indicate a high specificity of A-predominance for diagnosing low PAOP. The A-predominance is the normal pattern, indicating a dry lung surface. Wet lungs, which are seen in patients with hemodynamic pulmonary edema, yield B-predominance.[6,7,10,17,19] Acute hemodynamic pulmonary edema yields increased PAOP[2] with interlobular septal edema. Permeability-induced edema yields low PAOP,[20] usually with interlobular septal edema. The present study further validates the concept that B-lines derive from excess fluid accumulation along the interlobular septa caused by the elevation of PAOP with resultant hydrostatic pulmonary edema.

Fluid Administration Limited by Lung Sonography

In patients with pulmonary edema, interstitial edema is a silent step that precedes alveolar edema.[21,22] The excess fluid first accumulates along the interlobular septa, which are not involved in gas exchanges. The fluid under pressure reaches the subpleural interlobular septa (accessible by using ultrasound analysis). When lymphatic resorption capacity is exceeded (*ie*, when the interstitial fluid increases > 50%), fluid begins to pour into the alveoli.[23] This step initiates alveolar edema, with gas exchange impairment and clinical signs.

Pulmonary edema is the main concern during fluid therapy.[2] The PAOP has long been used to evaluate this risk. The value of the PAC is currently being debated,[24–28] and its use is decreasing while Doppler echocardiography is being advocated.[8,29–34] The following alternatives are currently being used: central venous pressure; arterial systolic or pulse pressure variation; pulse contour analysis; continuous cardiac output devices; oxygen transport assessment; and microcirculation assessment.[35–40] This number of techniques possibly reflects the absence of an incontestable "gold standard." All these techniques have drawbacks (*eg*, invasiveness, delay to implementation, and difficulty in monitoring). None of them assesses interstitial edema directly, as does lung ultrasound. The concept of fluid responsiveness based on the variations in cardiac output is interesting and is being used increasingly. It could be coupled with lung ultrasound, which takes into account the tolerance of the lung to fluid therapy. The relevance of the PAOP can be debated for evaluating left ventricle pressures or fluid responsiveness,[4,28] but this parameter, linked with lung filtration pressure, still indicates the risk of pulmonary edema.

It should be understood that A-predominance does not define a need for fluid therapy, but rather is a criterion for lung tolerance to fluid therapy. If a B-predominance replaces an A-predominance following fluid therapy, this indicates recent interstitial syndrome (*ie*, the likelihood of acute pulmonary edema), likely from a hydrostatic mechanism. This suggests that the end point has been reached. The ideal aim is to correct the clinical signs of shock while remaining in an A-predominance. This setting does not consider cardiogenic shock, in which the B-profile is usual on hospital admission.[10]

The potential of ultrasound to detect interstitial edema (*ie*, to benefit from a precursory step) provides a safety margin, enhancing the concept of Guyton and Hall[23] of a safety factor but using modern tools.

Additional Points

Acute disorders abut the pleural line, creating an acoustic window, allowing their distinction.[10] CT scan analysis has shown that subpleural interstitial edema (accessible to ultrasound) is constantly associated with deeper interstitial syndrome[6] (Fig 3). Lung ultrasound can distinguish anterior interstitial from posterior alveolar patterns (a challenge for anteroposterior radiography).

Lung ultrasound is easily standardized, yielding increasing use.[13–19] The discernment of A-lines and B-lines shows high interobserver agreement.

Using lung ultrasound, the intensivist will find the advantages of a simple noninvasive, reproducible (allowing monitoring), low-cost bedside tool. Rapidity is a major advantage. In these unstable patients in whom saving time is vital, our machine switches on in 7 s, and the detection of anterior A-lines in one lung classifies the patient as being in A-predominance (even with contralateral B-lines) in < 30 additional seconds.

The ideal type of machine and probe for critical ultrasound has been discussed.[10] The dimensions and range of our microconvex probe allow optimal use for critical lung (and whole-body) ultrasound.[41]

The authors used a 1982 technology for describing A-lines and B-lines, and published the relation between lung ultrasound and PAOP in 2002.[41] Their lung rockets were subsequently used in cardiology.[17] The present report considers critically ill patients and proposes a simple, dichotomous (not continuous) approach to examining a limited area of the chest wall, considering a simple parameter for PAOP estimation. There is no intermediate pattern between A-lines and B-lines. A-lines are dichotomous to B-lines in a given area. The concept of A-predominance or B-predominance allows dichotomous analysis in a given patient. Observation shows that the transformation from dry subpleural interlobular septa to wet subpleural interlobular septa secondary to hemodynamic redistribution affects large territories simultaneously, with little place for intermediate situations, at least in an ICU population. The hydrostatic hyperpressure invades all septa of a given territory (lateral, anterior) simultaneously. Consequently, lung ultrasound allows a simple, qualitative approach. A comprehensive counting of B-lines is a fastidious task that can be interesting for subtle measurements in patients who are not critically ill.

Limitations

The present approach is qualitative. Documenting, for example, lateral walls, cardiac function, and vein calipers provides additional information, to the detriment of simplicity. In this preliminary approach, the authors did not focus on posterior changes since posterior B-lines may indicate gravitational changes.[42] They did not use sophisticated approaches considering the types of ventilation, PEEP variations, different points in the same patients, other imaging or hemodynamic modalities, and lung injury scores.

B-predominance detects interstitial edema, arising from fluid overload as well as permeability-induced pulmonary edema. B-predominance can indicate chronic interstitial syndrome (which would, therefore, present at hospital admission). The detection of B-predominance on the initial examination cannot provide information on the PAOP value. Particular patients who are in hypovolemic shock displaying B-lines or certain cases with massive pulmonary edema can benefit from fluid therapy. The number of cases in this study with B-predominance is explained by the high frequency of parenchymal disorders (hemodynamic or permeability-induced edema) in patients who have been referred for emergency PAC. These disorders are less frequent on hospital admission, which increases the value of our approach. Since patients were consecutive, those with ARDS were included, which may have created a bias (decreasing sensitivity), yet the B-predominance is not constant in ARDS patients.[10]

The learning curve for lung ultrasound is not null. It can, however, be favorably compared with other tools in use today.

Conclusions

Lung ultrasound provides a new approach for interstitial edema detection. A-predominance indicates dry anterior interlobular septa. Specific to predicting a low PAOP value, A-predominance suggests that fluid may be given without initial concern for the development of hydrostatic pulmonary edema. B-predominance indicates interstitial syndrome, possibly related to interstitial edema. B-predominance is observed in a wide range of PAOP values, precluding firm conclusions for the need for fluid therapy. It can be detected on initial management or may appear during fluid therapy. This bedside potential will be appreciated by those intensivists who envision fluid therapy based on low PAOP values and who consider that the concept of a safety factor provided by lung ultrasound is logical.

Acknowledgments

Author contributions: Dr. Lichtenstein collected the data and wrote the article. Dr. Mezière collected the data and cowrote the article. Dr. Lagoueyte reviewed the article. Dr. Biderman was the advisor. Dr. Goldstein collected data. Dr. Gepner was the advisor and collected data.

Financial/nonfinancial disclosures: The authors have reported to the ACCP that no significant conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

Other contributions: We are grateful to Larry Melniker who took the time to read and improve this article, to Marek Nalos who provided needed advice, and to François Jardin who made this work possible by opening his logistics to us.

References

1 Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med 2001; 345:1368–1377
2 Braunwald E. Heart disease. Philadelphia, PA: WB Saunders Company, 1984; 173
3 Cholley BP, Payen D. Pulmonary artery catheters in high-risk surgical patients. N Engl J Med 2003; 348:2035–2037
4 Pinsky MR. Clinical significance of pulmonary artery occlusion pressure. Intensive Care Med 2003; 29:175–178
5 Mayo P, Beaulieu Y, Doelken P, et al. American College of Chest Physicians/La Société de Réanimation de Langue Française statement on competence in critical care ultrasonography. Chest 2009; 135:1050–1060
6 Lichtenstein D, Mezière G, Biderman P, et al. The comet-tail artifact: an ultrasound sign of alveolar-interstitial syndrome. Am J Respir Crit Care Med 1997; 156:1640–1646
7 Lichtenstein D, Mezière G. A lung ultrasound sign allowing bedside distinction between pulmonary edema and COPD: the comet-tail artifact. Intensive Care Med 1998; 24:1331–1334
8 Jardin F, Farcot JC, Boisante L, et al. Influence of positive end-expiratory pressure on left ventricle performance. N Engl J Med 1981; 304:387–392
9 Lichtenstein D, Axler O. Intensive use of general ultrasound in the intensive care unit, a prospective study of 150 consecutive patients. Intensive Care Med 1993; 19:353–355
10 Lichtenstein D, Mezière G. Relevance of lung ultrasound in the diagnosis of acute respiratory failure: the BLUE-protocol. Chest 2008; 134:117–125
11 Lichtenstein D, Mezière G, Lascols N, et al. Ultrasound diagnosis of occult pneumothorax. Crit Care Med 2005; 33:1231–1238
12 Lichtenstein D. Diagnostic échographique de l'œdème pulmonaire. Rev Im Med 1994; 6:561–562
13 Dulchavsky SA, Hamilton DR, Diebel LN, et al. Thoracic ultrasound diagnosis of pneumothorax. J Trauma 1999; 47: 970–971
14 Rowan KR, Kirkpatrick AW, Liu D, et al. Traumatic pneumothorax: detection with thoracic US; correlation with chest radiography and CT. Radiology 2002; 225:210–214
15 Mayo PH, Goltz HR, Tafreshi M, et al. Safety of ultrasound-guided thoracentesis in patients receiving mechanical ventilation. Chest 2004; 125:1059–1062
16 Blaivas M, Lyon M, Duggal S. A prospective comparison of supine chest radiography and bedside ultrasound for the diagnosis of traumatic pneumothorax. Acad Emerg Med 2005; 12:844–849
17 Agricola E, Bove T, Oppizzi M, et al. Ultrasound comet-tail images: a marker of pulmonary edema: a comparative study with wedge pressure and extravascular lung water. Chest 2005; 127:1690–1695
18 Soldati G, Testa A, Silva FR, et al. Chest ultrasonography in lung contusion. Chest 2006; 130:533–538
19 Volpicelli G, Mussa A, Garofalo G, et al. Bedside lung ultrasound in the assessment of alveolar-interstitial syndrome. Am J Emerg Med 2006; 24:689–696
20 Lemaire F, Brochard L. ARDS. In: Offenstadt G, ed. Réanimation médicale. Paris, France: Masson, 2001; 807–810
21 Staub NC. Pulmonary edema. Physiol Rev 1974; 54:678–811
22 Chait A, Cohen HE, Meltzer LE, et al. The bedside chest radiograph in the evaluation of incipient heart failure. Radiology 1972; 105:563–566
23 Guyton CA, Hall JE. Textbook of medical physiology. 9th ed. Philadelphia, PA: WB Saunders Company, 1996; 496–497
24 Packman RI, Rackow EC. Optimum left heart filling pressure during fluid resuscitation of patients with hypovolemic and septic shock. Crit Care Med 1983; 11:165–169
25 Wagner JG, Leatherman JW. Right ventricular end diastolic volume as a predictor of the hemodynamic response to a fluid challenge. Chest 1998; 113:1048–1054
26 Krausz MM, Perel A, Eimerl D, et al. Cardiopulmonary effects of volume loading in patients in septic shock. Ann Surg 1977; 185:429–434
27 Connors AF Jr, Speroff T, Dawson NV, et al. The effectiveness of right heart catheterization in the initial care of critically ill patients: SUPPORT Investigators. JAMA 1996; 276:889–897
28 Osman D, Ridel C, Rey P, et al. Cardiac filling pressures are not appropriate to predict hemodynamic response to volume challenge. Crit Care Med 2007; 35:64–68
29 Slama M, Masson H, Teboul JL, et al. Monitoring of respiratory variations of aortic blood flow velocity using esophageal Doppler. Intensive Care Med 2004; 30:1182–1187
30 Vieillard-Baron A, Chergui K, Rabiller A, et al. Superior vena caval collapsibility as a gauge of volume status in ventilated septic patients. Intensive Care Med 2004; 30:1734–1739
31 Lichtenstein D, Jardin F. Noninvasive assessment of CVP using inferior vena cava ultrasound measurement of the inferior vena cava in the critically ill. Réanimation Urgences 1994; 3:79–82
32 Boulain T, Achard JM, Teboul JL, et al. Changes in BP induced by passive leg raising predict response to fluid loading in critically ill patients. Chest 2002; 121:1245–1252
33 Via G, Braschi A. Echocardiographic assessment of cardiovascular failure. Minerva Anesthesiol 2006; 72:495–501
34 Poelaert JI, Schupfer G. Hemodynamic monitoring utilizing transesophageal echocardiography: the relationships among pressure, flow, and function. Chest 2005; 127:379–390
35 Pinsky MR. Using ventilation-induced aortic pressure and flow variation to diagnose preload responsiveness. Intensive Care Med 2004; 30:1008–1010
36 Tavernier B, Makhotine O, Lebuffe G, et al. Systolic pressure variation as a guide to fluid therapy in patients with sepsis-induced hypotension. Anesthesiology 1998; 89:1313–1321
37 Michard F, Boussat S, Chemla D, et al. Relation between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. Am J Respir Crit Care Med 2000; 162:134–138
38 Perel A, Minkovich L, Preisman S, et al. Assessing fluid-responsiveness by a standardized ventilatory maneuver: the respiratory systolic variation test. Anesth Analg 2005; 100: 942–945
39 Michard F, Teboul JL. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. Chest 2002; 121:2000–2008
40 Magder S. How to use central venous pressure measurements. Curr Opin Crit Care 2005; 11:264–270
41 Lichtenstein D, ed. Poumon. In: L'échographie générale en réanimation (General ultrasound in the critically ill). 2nd ed. New York, NY: Springer, 2002; 123–136
42 Rémy-Jardin M, Rémy J. Œdème interstitiel. In: Imagerie nouvelle de la pathologie thoracique quotidienne. Paris, France: Springer-Verlag, 1995; 137–143