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Relevance of Lung Ultrasound in the Diagnosis of Acute Respiratory Failure*

The BLUE Protocol

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Background: **This study assesses the potential of lung ultrasonography to diagnose acute respiratory failure.**

Methods: **This observational study was conducted in university-affiliated teaching-hospital ICUs. We performed ultrasonography on consecutive patients admitted to the ICU with acute respiratory failure, comparing lung ultrasonography results on initial presentation with the final diagnosis by the ICU team. Uncertain diagnoses and rare causes (frequency < 2%) were excluded. We included 260 dyspneic patients with a definite diagnosis. Three items were assessed: artifacts (horizontal A lines or vertical B lines indicating interstitial syndrome), lung sliding, and alveolar consolidation and/or pleural effusion. Combined with venous analysis, these items were grouped to assess ultrasound profiles.**

Results: **Predominant A lines plus lung sliding indicated asthma (n = 34) or COPD (n = 49) with 89% sensitivity and 97% specificity. Multiple anterior diffuse B lines with lung sliding indicated pulmonary edema (n = 64) with 97% sensitivity and 95% specificity. A normal anterior profile plus deep venous thrombosis indicated pulmonary embolism (n = 21) with 81% sensitivity and 99% specificity. Anterior absent lung sliding plus A lines plus lung point indicated pneumothorax (n = 9) with 81% sensitivity and 100% specificity. Anterior alveolar consolidations, anterior diffuse B lines with abolished lung sliding, anterior asymmetric interstitial patterns, posterior consolidations or effusions without anterior diffuse B lines indicated pneumonia (n = 83) with 89% sensitivity and 94% specificity. The use of these profiles would have provided correct diagnoses in 90.5% of cases.**

Conclusions: **Lung ultrasound can help the clinician make a rapid diagnosis in patients with acute respiratory failure, thus meeting the priority objective of saving time.**

(CHEST 2008; 134:117–125)

Key words: chest ultrasonography; COPD; ICU; interstitial syndrome; lung, ultrasound diagnosis; pneumothorax; pulmonary edema; respiratory failure

Abbreviations: BLUE = Bedside Lung Ultrasound in Emergency; PLAPS = posterolateral alveolar and/or pleural syndrome

Acute respiratory failure is one of the most distressing situations for the patient. Emergency cases do not always present in conditions that are ideal for immediate diagnosis, which sometimes compromises outcome.[1–3] Physical examination and bedside radiography are imperfect,[4,5] resulting in a need for sophisticated test results that delay management.

Ultrasound has long shown its utility for plain organs.[6] Although the lung has traditionally been excluded from its repertoire,[7] studies have proven that this belief was unfounded.[8] Since 1989 in our ICU, using devoted logistics,[9] the concept of whole-body ultrasound was developed and extended to the lungs for managing critical situations.[10,11] Lung ultrasonography is becoming a standard tool in critical care. Accurate bedside detection of thoracic disorders should help diagnose acute respiratory failure.[12] This study examines this potential, as discussed previously.[13]

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Table 1—*Final Diagnoses and Methods of Diagnosis*

Diagnoses	Methods
For all patients	History, clinical examination, radiography read by radiologists, CT when available (n = 38), favorable clinical progression under treatment, and:
Cardiogenic pulmonary edema (referred to as *pulmonary edema*) [n = 64]	Evaluation of cardiac function using echocardiography, functional tests, and American Heart Association recommendations
Pneumonia (n = 83)	Infectious profile, radiologic asymmetry, microorganism isolated (blood, invasive tests), recovery with antibiotics. Included were infectious, aspiration, community, or hospital-acquired pneumonia. Pneumonia complicating chronic respiratory disease was classified as pneumonia. Beginning ARDS (n = 7) and massive atelectasis (n = 1) were included in this group
Decompensated chronic respiratory disease (referred to as *COPD*) [n = 49]	Condition defined as exacerbation of chronic respiratory disease without pneumonia, pneumothorax, pulmonary edema, pleurisy, or pulmonary embolism. COPD was confirmed by functional tests. Patients with simple bronchial superinfection were classified in this case. COPD patients with pneumonia, pneumothorax, etc, were first considered as pneumonia, pneumothorax, etc
Acute asthma (n = 34)	History, responds to bronchodilator treatment
Pulmonary embolism (n = 21)	Helical CT
Pneumothorax (n = 9)	Radiography (CT if necessary)
Excluded patients	
Rare (< 2%) causes (n = 9)	Chronic diffuse interstitial disease (n = 4), massive pleural effusion (n = 3), fat embolism (n = 1), tracheal stenosis (n = 1). Note: no dyspnea due to pericardial effusion in this consecutive series
No final diagnosis (n = 16)	Unknown diagnosis at the end of hospitalization, progression preventing conclusions
Several final diagnoses (n = 16)	Pulmonary edema plus pneumonia (n = 10), pulmonary edema plus COPD (n = 3), others (n = 3)

Materials and Methods

This was an observational study conducted in university-affiliated hospitals over 4 years investigating 301 consecutive adult patients with acute respiratory failure. The official diagnosis was established in the hospitalization report using standardized tests by the ICU staff and not including lung ultrasound data (Table 1). Sixteen patients never received a definite diagnosis, 16 patients had several official diagnoses, and 9 patients had rare (*ie*, frequency < 2%) diagnoses. To simplify this study, these patients were subsequently excluded (Table 1). Acute respiratory failure was defined based on the classical clinical and biological criteria for requiring admission to the ICU. All patients had an ultrasound test by investigators (D.L., G.M.) who did not participate in the patient's management, which was undertaken by other ICU members blinded to the ultrasound results. The ultrasound test was performed without interrupting management at the time of ICU admission (*ie*, within 20 min) and lasted < 3 min. The internal review board of the hospital approved this study and waived the requirement for informed consent.

Table 2—*Comprehensive Results**

Anterior Pattern	Bilateral-Predominant A Lines				Bilateral-Predominant B + Lines				Alveolar Consolidation				Predominant A Lines on One Side, and Predominant B + Lines on Other Side				A Lines
Lung sliding	+	−	+	−	+	+	−	−	+	+	−	−	+	+	−	−	− plus lung point
PLAPS	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Any
Pulmonary edema	2	0	0	0	54^{1}	8	0	0	0	0	0	0	0	0	0	0	0
COPD	2	1	38	4	2	1	0	0	1	0	0	0	0	0	0	0	0
Asthma	1	0	33^{1}	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Pulmonary embolism	10^{8}	0	10^{9}	0	0	0	0	0	0	0	1^{0}	0	0	0	0	0	0
Pneumothorax	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	8
Pneumonia	34	1	3	0	4	2	9	0	7	2	9	0	7	1	4	0	0
	A and A′ profile plus PLAPS		Normal profile, and A′ profile without PLAPS		B profile		B′ profile		C profile				A/B profile				Pneumothorax profile

*Exponent indicates No. of cases with venous thrombosis (datum without exponent means negative venous exploration).

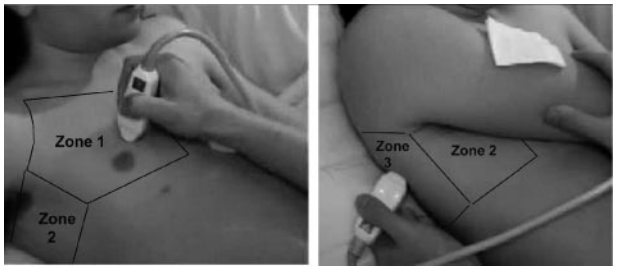

FIGURE 1. Ultrasound areas. Stage 1 defines the investigation of the anterior chest wall (zone 1) in a supine patient (1′ in this semirecumbent patient). Stage 2 adds the lateral wall (zone 2) [left panel]. Stage 3 adds the posterolateral chest wall using a short probe, moving the patient only minimally (zone 3) [right panel]. Each wall is divided into upper and lower halves, resulting in six areas of investigation. Note the shape of the microconvex probe, which allows satisfactory analysis of the intercostal space, and satisfactorily controlled compression maneuvers at the veins investigated in this study: internal jugular, subclavian, iliofemoropopliteal veins, and as far as possible, inferior vena cava and calf veins.

Ultrasound Approach

Ultrasound was performed (Hitachi-405; Hitachi Medical; Tokyo, Japan) with a 5-MHz microconvex probe (Fig 1). Patients were investigated in a semirecumbent position, or were supine if intubated (n = 35). Scans were longitudinal. The pleural line, sought between two rib shadows, indicates the pleural layers. The normal lung[14] displays lung sliding, a movement in rhythm with respiration at the pleural line, indicating sliding of the visceral pleura against the parietal pleura,[15] and A lines (Fig 2), these repetitive horizontal artifacts arising from the pleural line generated by subpleural air, which, either intraalveolar or pure (pneumothorax), blocks ultrasound waves. Normal interlobular septa are not detected. Three signs with dual answers were assessed, as follow.

Artifact Analysis: A or B Lines: The B line is the name given to an artifact with seven features: a hydroaeric comet-tail artifact; arising from the pleural line; hyperechoic; well defined; spreading up indefinitely; erasing A lines; and moving with lung sliding when lung sliding is present (Fig 3). It reflects the coexistence of elements with a major acoustic impedance gradient, such as fluid and air. Fluid at the subpleural interlobular septum surrounded by air-filled alveoli (*ie*, septal edema) fulfills this condition. Three or more B lines in a single view are called B + lines. B + lines indicate the subpleural part of interstitial syndrome.[16] Other comet-tail artifacts can be seen; none has B line characteristics.[14]

Lung Sliding: Present or Abolished: Abolition (Fig 4) occurs when the visceral pleura does not slide against parietal pleura (inflammatory adherences, loss of lung expansion, atelectasis, apnea, chronic symphysis) or is separated (pneumothorax, pneumonectomy). If abolished lung sliding is associated with A lines, the search for pneumothorax is mandatory. The lung point is a specific sign of pneumothorax, alternating lung sliding and abolished lung sliding plus A lines at the same location.[17]

Alveolar Consolidation and/or Pleural Effusion: Absent or Present: Pleural effusion classically yields an anechoic-dependent pattern (Fig 5),[18] an inconstant criterion. The roughly quadrangular shape with a regular lower border (the visceral pleura, called the lung line) was required for the diagnosis. The inspiratory shift of the lung line toward the pleural line is called the sinusoid sign. The sensitivity of these signs is 92%, and specificity is 97%.[5,19] Alveolar consolidation[20] results in fluid-filled alveoli. The alveolar-interstitial interfaces generate reflections yielding a tissular pattern, absence of the lung line, absence of the sinusoid sign. Ultrasound sensitivity is 90%, and specificity is 98%.[21]

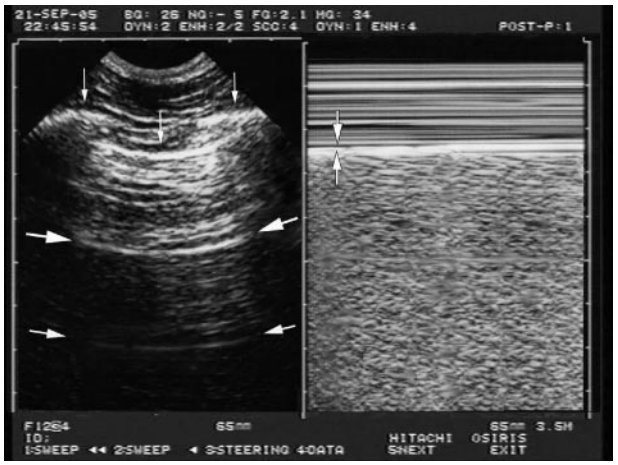

FIGURE 2. Normal lung surface. Longitudinal scan of an intercostal space. *Left panel*: Pleural line and A line (real-time). The pleural line is located 0.5 cm below the rib line in the adult. Its visible length between two ribs in the longitudinal scan is approximately 2 cm. The upper rib, pleural line, and lower rib (vertical arrows) outline a characteristic pattern called the bat sign. The horizontal lines arising from the pleural line (horizontal arrows) are separated by regular intervals that are equal to the distance between the skin and the pleural line. These were called A lines. A lines are usually large (see upper line) but can be shorter (lower line), which has no clinical significance. *Right panel*: M mode. An obvious difference appears on either side of the pleural line (arrow). The motionless superficial layers generate horizontal lines. Lung dynamics generate lung sliding (sandy pattern). This pattern is called the *seashore sign*.

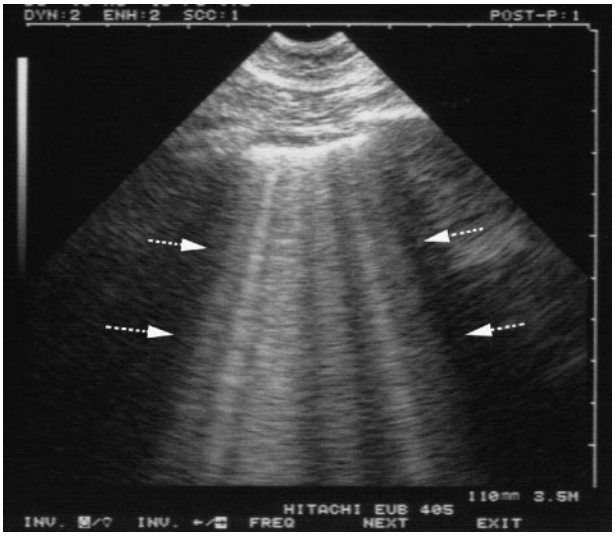

FIGURE 3. Interstitial syndrome. These vertical comet-tail artifacts arise strictly from the pleural line, are well defined (laser-like), hyperechoic, move with lung sliding, spread to the edge of the screen without fading, and erase A lines (dotted arrows indicate their theoretical location). This pattern defines B lines. Several B lines in a single view, reminiscent of a rocket at lift-off, are called lung rockets, or B + lines (featuring here, B3 lines). Diffuse lung rockets indicate interstitial syndrome. One or two B lines in a single view, referred to as the *b line*, have no pathologic significance. This patient had cardiogenic pulmonary edema.

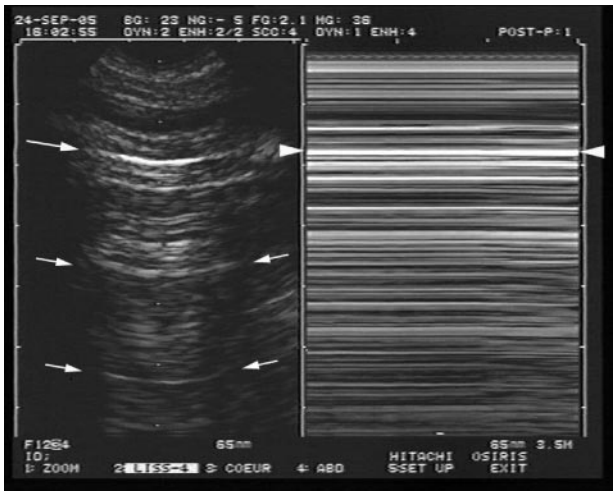

FIGURE 4. Pneumothorax. *Left panel* (real-time): one significant item is the complete absence of the B line. Lower arrows: A lines; upper arrow: pleural line. *Right panel* (M mode): this succession of horizontal lines indicates complete absence of dynamics at, and below, the pleural line (arrowheads). This pattern is called the stratosphere sign. The lung point (not featured here) confidently rules in the diagnosis.

Deep venous thrombosis was sought using the same probe.[22] Visualization of anatomic echoic intraluminal thrombosis or absence of compressibility was considered as a positive finding (Fig 1). An examination combined an anterior approach (analyzing artifacts, lung sliding, alveolar consolidation), a lateral subposterior search for posterolateral alveolar and/or pleural syndrome (PLAPS), and venous analysis.

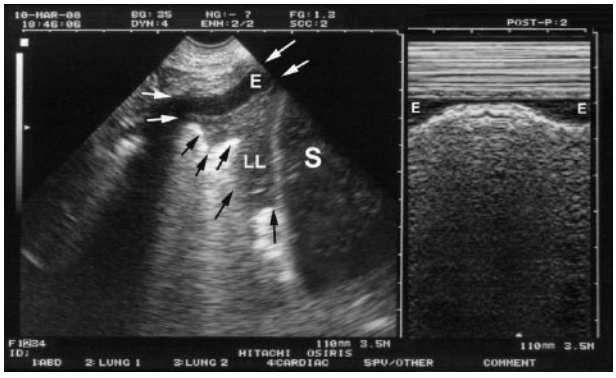

FIGURE 5. Pleural effusion and alveolar consolidation; typical example of PLAPS. *Left panel*: real-time, stage 2. The quad sign: a pleural effusion on expiration (E) is delineated between the pleural line (upper white arrows) and the lung line, always regular, which indicates the visceral pleura (lower white arrows). The shred sign: a lower-lobe alveolar consolidation (LL) yields a tissular pattern, characteristically limited by the lung line (or the pleural line when there is no effusion) and in depth by an irregular border (black arrows), the shred line, as in connection with aerated lung. Below, air artifacts are displayed. Between consolidation and spleen (S) is the diaphragm, a basic landmark in stage 2. *Right panel*: time-motion demonstrates the sinusoid sign, a basic dynamic sign of pleural effusion. The sign will not be generated by alveolar consolidation, which behaves like a solid lesion.

Study Design

The signs observed in each disease were methodically collected; then the ultrasound data were compared with the diagnosis established by the ICU team.

RESULTS

This study included 260 patients with a definite diagnosis: 140 men and 120 women (mean age, 68 years; range, 22 to 91 years; SD, 16 years).

Signs Observed

Pulmonary Edema: Pulmonary edema was observed in 64 patients. Anterior-predominant bilateral B + lines were observed in 62 cases (diffuse in 59, predominant involvement of lower halves in 3). Anterior-predominant bilateral A lines were seen in two cases. Anterior lung sliding was always preserved. In 56 cases, PLAPS was detectable. One patient (with B + lines) had internal jugular vein thrombosis.

COPD: COPD was observed in 49 patients. In 38 cases, anterior-predominant bilateral A lines with lung sliding and no PLAPS were observed. In five cases, the same pattern with abolished lung sliding (without lung point) was seen. Anterior-predominant bilateral B lines were present in three cases, anterior consolidation in one. PLAPS was seen in six cases.

Status Asthmaticus: Status asthmaticus was observed in 34 patients. Asthma gave anterior-predominant A lines with lung sliding in all cases, posterior consolidation in one, and calf thrombosis in another.

Pulmonary Embolism: Pulmonary edema was observed in 21 patients. Twenty patients had anterior-predominant A lines with lung sliding. One had anterior consolidation with absent lung sliding. PLAPS was found in 11 patients. Seventeen patients had venous thrombosis.

Pneumothorax: Pneumothorax was observed in nine patients. Abolished anterior lung sliding was associated with anterior-predominant A lines in all cases. Lateroposterior lung point was present in eight cases. PLAPS was found in five cases.

Pneumonia: Pneumonia was observed in 83 patients. In 75 cases, PLAPS was present. In six cases, an anterior-predominant bilateral B + pattern was associated with lung sliding (with PLAPS in four cases). In nine cases, anterior-predominant bilateral B + lines were associated with abolished lung sliding;

Table 3—*Combined Results

Diagnoses	A Profile Plus PLAPS	Normal Profile, and A' Profile Without PLAPS	B Profile	B' Profile	C Profile	A/B Profile	Lung Point
Pulmonary edema	2	0	62^{1}	0	0	0	0
COPD or asthma	4	75^{1}	3	0	1	0	0
Pulmonary embolism	10^{8}	10^{9}	0	0	1^{0}	0	0
Pneumothorax	0	1	0	0	0	0	8
Pneumonia	35	3	6	9	18	12	0

*Exponents indicate No. of cases with venous thrombosis (datum without exponent means negative venous exploration). To simplify this Table, COPD and asthma are considered together; three columns in Table 2 were combined because analysis showed no loss in performance. One patient with pneumonia and the A' profile plus PLAPS was inserted in the A profile plus PLAPS column. The term *lung point* implies abolished anterior sliding associated with anterior A lines.

PLAPS was always associated. In 12 cases, anterior-predominant B + lines in one lung coexisted with predominant A lines in the contralateral lung; PLAPS was seen in 11 cases. In 18 cases, anterior consolidations were observed; lung sliding was abolished in 9 of them; PLAPS was associated in 16 cases. In 34 cases, an anterior-predominant A pattern with lung sliding was associated with PLAPS. Lung sliding was abolished in 28 cases. Three patients had a normal examination.

Ultrasound Accuracy

We retained characteristic combinations of signs that produced specificities > 90% (Tables 3, 4). We suggest a practical nomenclature that avoids repetitive descriptions (Fig 6). The *A profile* designates anterior predominant bilateral A lines associated with lung sliding (with possible focalized B lines). *The A' profile* is an A profile with abolished lung sliding and without lung point. The *B profile* designates anterior-predominant bilateral B + lines associated with lung sliding (with possible focalized A lines). *The B' profile* is a B profile with abolished lung sliding. *The A/B profile* designates anterior-predominant B + lines on one side, predominant A lines on the other. *The C profile* designates anterior alveolar consolidation(s). *PLAPS profile* is described in the Appendix. *The normal profile* associates the A profile without PLAPS (regardless of posterior A or B lines) [online document 1].

Ultrasound Accuracy Rates

For pulmonary edema, the B profile had 95% specificity and 97% sensitivity. For COPD and asthma (considered together for purposes of simplicity), the normal profile had a 97% specificity and a

Table 4—*Accuracy of the Ultrasound Profiles

Disease	Ultrasound Signs Used	Sensitivity, %	Specificity, %	Positive Predictive Value, %	Negative Predictive Value, %
Cardiogenic pulmonary edema	Diffuse bilateral anterior B+ lines associated with lung sliding (B profile)	97 (62/64)	95 (187/196)	87 (62/71)	99 (187/189)
COPD or asthma	Predominant anterior A lines without PLAPS and with lung sliding (normal profile), or with absent lung sliding without lung point	89 (74/83)	97 (172/177)	93 (74/79)	95 (172/181)
Pulmonary embolism	Predominant anterior bilateral A lines plus venous thrombosis	81 (17/21)	99 (238/239)	94 (17/18)	98 (238/242)
Pneumothorax	Absent anterior lung sliding, absent anterior B lines and present lung point	88 (8/9)	100 (251/251)	100 (8/8)	99 (251/252)
Pneumonia	Diffuse bilateral anterior B+ lines associated with abolished lung sliding (B' profile)	11 (9/83)	100 (177/177)	100 (9/9)	70 (177/251)
	Predominant anterior B+ lines on one side, predominant anterior A lines on the other (A/B profile)	14.5 (12/83)	100 (177/177)	100 (12/12)	71.5 (177/248)
	Anterior alveolar consolidation (C profile)	21.5 (18/83)	99 (175/177)	90 (18/20)	73 (175/240)
	A profile plus PLAPS	42 (35/83)	96 (170/177)	83 (35/42)	78 (170/218)
	A profile plus PLAPS, B', A/B or C profile	89 (74/83)	94 (167/177)	88 (74/84)	95 (167/176)

*Data in parenthesis indicate No. of patients (total).

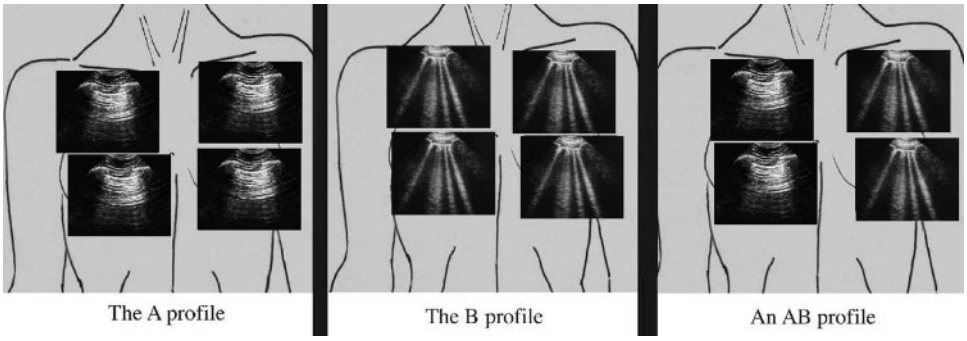

FIGURE 6. Ultrasound profiles. *Left panel*: The A profile is defined as predominant A lines plus lung sliding at the anterior surface in supine or half-sitting patients (stage 1/1′). This profile suggests COPD, embolism, and some posterior pneumonia. Pulmonary edema is nearly ruled out. Middle: The B profile is defined as predominant B + lines in stage 1. This profile suggests cardiogenic pulmonary edema, and nearly rules out COPD, pulmonary embolism, and pneumothorax. *Right panel*: an A/B + profile, massive B lines at the left lung, A lines at the right lung. This profile is usually associated with pneumonia.

89% sensitivity. For pulmonary embolism, the A profile plus venous thrombosis showed 99% specificity and 81% sensitivity. For pneumothorax, absent anterior lung sliding, anterior A lines, and a positive search for lung point yielded 100% specificity and 88% sensitivity. For pneumonia, specificity and sensitivity were, respectively, 100% and 11% for the B' profile, 100% and 14% for the A/B profile, 99% and 11% for the C profile, and 96% and 42% for the A profile plus PLAPS. These four profiles indicated pneumonia with 94% specificity and 89% sensitivity. For all patients, lung ultrasound yielded correct diagnoses in 90.5% of cases.

DISCUSSION

Briefly, the B profile (anterior interstitial syndrome with lung sliding) indicated pulmonary edema. The B' profile (lung sliding abolished) indicated pneumonia. The A/B profile (asymmetric anterior interstitial syndrome) and the C profile (anterior consolidation) indicated pneumonia, as did the A profile plus PLAPS. The A profile plus venous thrombosis indicated pulmonary embolism. A normal profile indicated COPD/asthma.

These results correspond to physiopathologic patterns, particularly echoed by ultrasound artifacts, that have been in clinical use since 1994.[23] The pleural line is superficial. Most acute disorders reach it: acute interstitial changes involve deep as well as subpleural areas[16,24]; most (98.5%) cases of acute alveolar consolidation abut the pleura[21]; pneumothorax and pleural effusions always abut the wall.[14] The high acoustic impedance gradient between air and fluid generates artifacts. Air stops ultrasounds, and fluid facilitates their transmission. The air-fluid ratio is 1 in pneumothorax; roughly 0.98 in asthma, COPD, and normal lungs[25]; roughly 0.95 in interstitial syndrome[24]; near zero in alveolar consolidation; and zero in pleural effusion (online document 2).

COPD and asthma are bronchial diseases assumed to yield a normal lung surface. This explains the ability of ultrasound to distinguish these entities from pulmonary edema.[26]

In pulmonary edema, the transudate under pressure is pushed along interlobular septa against gravity, up to the anterior wall, explaining the quasiconstant anterior, symmetric interstitial patterns (indicating anterior Kerley lines). Edema of interlobular septa is constant and early.[27,28] The B profile (with or without PLAPS due to gravitational filling of dependent alveoli) characterizes pulmonary edema with high accuracy. Posterior interstitial syndrome was not sought, since gravitational interstitial changes are physiologic.[24] Pulmonary edema produces transsudate, which is not supposed to generate inflammatory adherences (a factor that may hinder lung sliding, see below).

Pulmonary embolism does not yield interstitial change. A normal anterior lung surface was usually seen, as previously reported.[29] None of 92 patients with anterior interstitial patterns had pulmonary embolism. The positive predictive value of deep venous thrombosis was 89%, but 94% if associated with the A profile, suggesting that the search for venous thrombosis should be associated with lung analysis (Table 2). Pneumothorax features have been extensively described.[14,15,30]

Pneumonia yields numerous signs. The frequent abolition of lung sliding (B' profile) is explainable by inflammatory adherences due to exudate.[31] Abolished lung sliding again shows low specificity for pneumothorax (22% positive predictive value here). Pneumonia can be found in a wide variety of loca-

tions, which explains the asymmetric patterns (AB profile), anterior consolidations (C profile), or lack of anterior interstitial patterns (A profile). Note that among seven patients initiating ARDS from pneumonia, only one had the B profile. Briefly, ultrasound highlighted distinctions between pneumonia and pulmonary edema. If confirmed by further data, this may provide a potential means of differentiating hemodynamic from permeability-related pulmonary edema.

Suggested Algorithm

Anterior lung sliding is checked first. Its presence discounts pneumothorax. Anterior B lines are sought. The B profile suggests pulmonary edema. The B', A/B, and C profiles suggest pneumonia. The A profile prompts a search for venous thrombosis. If present, pulmonary embolism is considered. If absent, PLAPS is sought. Its presence (A profile plus PLAPS) suggests pneumonia; its absence (normal profile) suggests COPD/asthma (Fig 7).

This algorithm, using ultrasound alone, would have retrospectively given an accurate diagnosis in 90.5% of cases. Its routine integration into the clinical approach would give even better results. This algorithm was called Bedside Lung Ultrasound in Emergency—the BLUE protocol. When your patient is blue, promptly perform a BLUE protocol. The absence of echocardiography in this algorithm stems from the fact that, even if yielding data of primary importance, it gives indirect arguments, whereas lung ultrasound provides a direct approach to acute respiratory failure. In practice, a cardiac analysis completes our approach.

Clinical Implications

Using lung ultrasound saves time and decreases the need for CT, whose drawbacks include delayed-care implementation, irradiation,[32–34] cost (therefore available only in resource-rich countries), and the required supine position. Lung ultrasound is nearly equivalent to CT in detecting most disorders,[5] can be repeated at will, and provides additional information.[35]

Online document 1 shows the number of erroneous initial diagnoses using conventional tools. One fourth of the patients in the first 2 h had erroneous or uncertain initial diagnoses. Many more received inappropriate therapy.

Lung ultrasound generates standardized, reproducible patterns, explaining the high interobserver agreement.[5,11,21] Feasibility is high. Lung ultrasound may appear complex at first sight but simply requires a change in thinking.[36] Once the process has been learned, a step-by-step use will make it routine.[37–44]

Limitations

The operators in this study have several years of experience. They were not blinded to the patient's clinical presentation, yet ultrasound profiles were established based on objective signs.

Among the erroneous results (9.5%), some resulted from limitations of this simplified ultrasound approach: problems distinguishing pulmonary edema and interstitial pneumonia, or embolism without thrombosis. Others can be explained by possible flaws in the reference tests: "decompensated COPD" associated with the B profile or PLAPS, or "pulmonary edema" without the B profile.

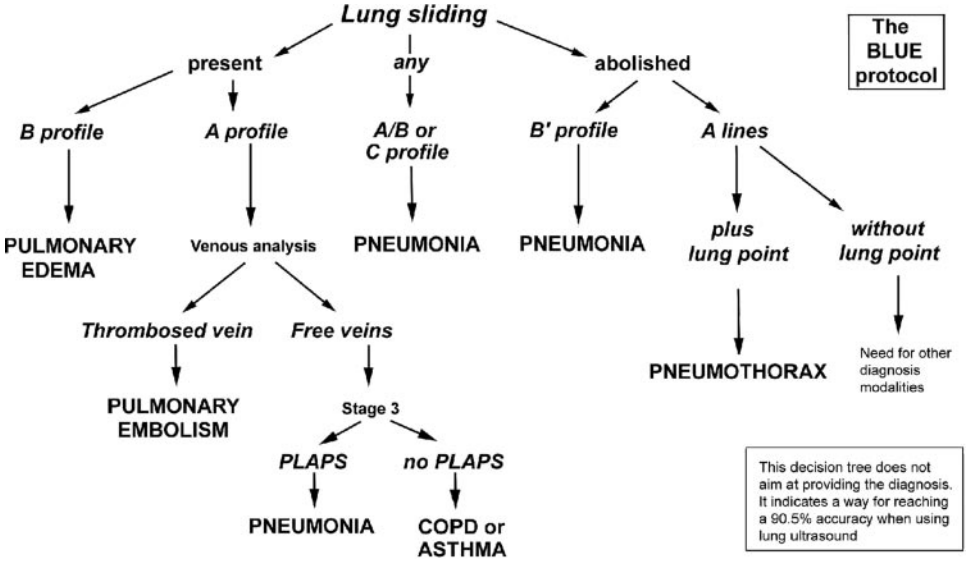

FIGURE 7. A decision tree utilizing lung ultrasonography to guide diagnosis of severe dyspnea.

As regards the excluded patients, among rare causes of dyspnea, massive pleural effusion was not a diagnostic problem. Chronic interstitial diseases produce B lines; the solution deserves a subtle approach that cannot be discussed herein. Among undefined official diagnosis, note that all patients had one characteristic ultrasound profile. Among patients with several official diagnoses, their inclusion would require an accurate way to determine the respective role of each mechanism involved in respiratory distress.

The choice of the material can be decisive. Cumbersome echocardiographic units with cardiac probes usually have insufficient resolution for the lung. Recent ultracompact technologies (not a mandatory requirement in hospital settings), if technologically designed for cardiac investigations, with no consideration for the lung, will not solve this problem. Both systems usually present additional drawbacks: cost, switch-on time, keyboard design that prevents rapid disinfection, and modes that seek to remove artifacts. The choice of the probe is critical. Vascular probes usually prevent deep analysis and artifact recognition. Abdominal probes have inappropriate ergonomics but are perhaps the least problematic in terms of resolution. The authors use a low-cost, 31-cm large, hybrid machine with a compact design and flat keyboard, available since 1992 and still being manufactured, without Doppler.[45,46] Their microconvex probe, the optimal type of probe for the lungs, is also ideal for emergency whole-body analysis.[22]

Conclusions

Lung ultrasound immediately provided diagnosis of acute respiratory failure in 90.5% of cases. It can therefore be added to the armamentarium of critical care.[47] The additional value of saving time should provide prompter relief for these severely dyspneic patients.

Appendix

Lung Artifacts Nomenclature

A lines indicate horizontal repetitive artifacts originating from the pleural line. *B lines* indicate vertical, comet-tail artifacts originating from the pleural line, long, hyperechoic, well-defined, dynamic, erasing A lines.

Ultrasound Profiles

A profile represents anterior-predominant bilateral A lines associated with lung sliding. *A' profile* represents A profile with abolished lung sliding and without lung point. *B profile* represents anterior-predominant bilateral B lines associated with lung sliding. *B' profile* represents B profile with abolished lung sliding. *A/B profile* represents anterior-predominant B + lines at one side and predominant A lines at the other. *C profile* represents anterior alveolar consolidation(s). *PLAPS* represents posterior and/or lateral alveolar and/or pleural syndrome. All these definitions are based on the patient being supine or semirecumbent.

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