Interpreting Lung Ultrasound B-Lines in Acute Respiratory Failure

To the Editor:

The article in this issue of CHEST (see page 1586) by Bataille et al.\(^1\) on thoracic and lung ultrasonography (LUS) is of high interest. This study has the great merit of going in the right direction. To date, scientists explored the potential of LUS alone because it represents a new paradigm. Now, it is time to move on and explore how LUS may further improve diagnostic workup by combining it with other ultrasound modalities and, even more, with other diagnostic tools.

Regarding the interpretation of B-lines, there are two points of this study that need to be addressed. The first is the high number of false-positive diagnoses of pneumonia (33%). I imagine that the typical consolidations were not visualized by LUS. It is well known that pneumonia and other consolidative conditions of the lung are surrounded by interstitial involvement.\(^2\) Sometimes, the early phase of pneumonia is only visible by interstitial patterns, which often are radiooccult.\(^3\) Moreover, deep consolidative lesions not abutting the pleural surface may be visualized as surrounding interstitial pattern. All these situations are known as focal interstitial syndromes, which cannot be misdiagnosed for congestion because of the different distribution and, as said, the focal limited extension.\(^4\) Did the authors consider these aspects?

The second point is the high percentage of false-negative findings of cardiogenic edema (37%). The interstitial syndrome is nothing more than the ultrasound effect of a partial loss of aeration and increase in density, which involves the periphery of the lung. The sensitivity was very high in previous studies. If congestion occurs, B-lines should be bilateral, homogeneously distributed, and multiple in the anterior and lateral chest. In my opinion, there are only three possible explanations for false-negative findings. The first explanation is the effect of early treatment preceding LUS. B-lines have been shown to be very fast to appear but just as quick to resolve following medical and ventilator treatment.\(^4\) A second explanation is that congestion may spare the lung periphery. In this case, normally aerated lung interposes between the probe and the congested lung, thus giving a false-negative pattern. This would be contrary to physiology and the anatomic distribution of the secondary pulmonary lobules.\(^5\) The third explanation is respiratory distress not due to congestion in acute heart failure, which is a novelty worth consideration and full discussion.

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References


Response

To the Editor:

I thank Dr Volpicelli for his insights on our article published in CHEST\(^6\) and agree with the fact that we probably are on the verge of a paradigm shift in this field. Pioneering studies that have described thoracic ultrasonography semiotics\(^7\) must now be followed by research aiming to integrate the totality of physiologic data at our disposal in a comprehensive and clinically relevant way. We could expect from this not only to improve the diagnosis accuracy of ultrasound tools but also to allow patient bedside study of interorgan interactions, an
indispensable step to further expand our understanding and potentially lead to the design and monitoring of new individually tailored treatment strategies.3

Dr Volpicelli’s pertinent comments converge to the moot issue of the potential utility of lung ultrasonography, not only to detect but also to quantify extravascular lung water. We completely agree with the idea that B-lines are nothing more than reverberation artifacts through edematous interlobular septa within the lung (a phenomenon also observed in physiologic conditions).4 Nevertheless, it remains to be shown to what extent this pattern could be used to accurately explore pulmonary edema topography and, additionally, to track the impact of therapeutic interventions across time.

In the current study,1 we used supervised machine-learning methods to integrate lung ultrasound and echocardiography data as independent variables in a predictive mathematical model. Remarkably, we identified several cases of lung consolidation (C and posterolateral alveolar and/or pleural syndrome profiles),2 but their detection was not significantly correlated with any component of the model (e-Fig 1, e-Table 1 in our article). In other words, we confirm that we observed several cases of lung consolidation in patients without pneumonia. We interpret these data relative to the value of such ultrasound patterns in patients on mechanical ventilation in the ICU and highlight the interest of concomitant echocardiography recordings in this setting.

Regarding cardiogenic edema and its spatial and temporal dynamics, it is worth noting that we used previously well-described lung ultrasound criteria, which do not take into account B-line spatial distribution.2 Furthermore, we note that both thoracic and lung ultrasound recordings were performed during reproducible clinical conditions at the time of ICU admission, aiming to avoid confounding factors.

Overall, we suggest that B-lines seem to be sensitive markers of extravascular lung water per se, regardless of its etiology. The technical revolution in which we are now immersed does not make us forget to integrate such valuable information in a broader medical reasoning. Substantial progress has been made in the description of a standardized lung ultrasound semiotics. We can speculate that further advances will come from the complementary study of thoracic ultrasonography, alongside described methods for the assessment of pulmonary edema in human3 and animal6 models. For thoracic ultrasonography, it is time to move beyond the lines.

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References