Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Please include a cover letter with a complete list of authors (including full first and last names and highest degree), corresponding author’s address, phone number, fax number, and e-mail address (if applicable). An electronic version of the communication should be included on a 3.5-inch diskette. Specific permission to publish should be cited in the cover letter or appended as a postscript. CHEST reserves the right to edit letters for length and clarity.

Lung Sonography in Pulmonary Embolism

To the Editor:

We read with interest the article of Reissig et al (December 2001). The main finding described was a small alveolar consolidation touching the lung surface and considered as a lung infarct. Our personal observations are contradictory at first sight, as we have never detected such lesions in pulmonary embolism. Two explanations are possible. First, the degree of severity of pulmonary embolism in the series of Reissig et al is not specified. Thus, patients enrolled in that study probably had nonsevere pulmonary embolism (ie, small emboli, which are known to cause more distal disorders than the severe forms). Another possibility is that our screening missed small lesions.

We report briefly 33 cases of patients admitted to our ICU for severe pulmonary embolism. None of these patients had the anterolateral pattern described by Reissig et al. We had previously found this pattern in ICU patients with severe infectious disorders, a pattern we called C lines; however, we can describe a pattern that is 91% sensitive to pulmonary embolism—a regular repetition of the lung-wall interface, a pattern we called A lines. This sign has no specificity, as it is the normal signal; however, in a patient with respiratory distress, the normality of the lung ultrasound signal is a crucial finding, as pneumothorax or pulmonary edema, for instance, give totally different patterns.

We fully agree with Reissig et al that there is a strong correlation between ultrasound and CT patterns, but we are still investigating the relationship of this ultrasound pattern to particular infectious or embolic processes.

Another point of agreement with Reissig et al is that lung ultrasonography in respiratory disorders should become routine, as it yields crucial bedside information that will be directly useful for the immediate management of the patient. We are confident this use will promote ultrasound to the status of a genuine stethoscope.

Daniel A. Lichtenstein, MD
Hôpital Ambroise-Paré
Boulogne, France
Yann Loubières, MD
Centre Hospitalier Intercommunal
Saint-Germain-en-Laye, France

REFERENCES

To the Editor:

We would like to thank Drs. Lichtenstein and Loubières for their interesting comments on our article in CHEST (December 2001) on the diagnostic significance of transthoracic parenchymal sonography of the lung in patients with pulmonary embolism (PE). We fully support their view regarding the role of comet tail artifacts in diagnosing pulmonary diseases. We also agree with Drs. Lichtenstein and Loubières that sonography of the lung should be performed as a routine investigation. Although the performance of transthoracic sonography requires some experience, this approach is extremely attractive since the technique is inexpensive, widely available, and immediately accessible to the physician at bedside, facilitating a swift diagnosis and onset of therapy.

Drs. Lichtenstein and Loubières failed to detect the characteristic multiple, hypoechoic, pleural-based, wedge-shaped or rounded parenchymal lesions, which generally are well-demarcated from the surrounding tissue that are observed in cases of PE and reported by us and others. The reasons for the
discrepancy are not immediately apparent but are likely to be due to the patient selection within an ICU setting, the sonographic procedure applied, and the inherent characteristics of the PE.

First, Dr. Lichtenstein examined patients with PE in an intensive care setting, whereas the patients enrolled in our study had their conditions diagnosed while on a pulmonary ward. The majority of our patients’ experienced isolated dyspnea and pleuritic pain. Circulatory collapse of no other apparent cause was observed in approximately 15% of all patients who experienced PE, and was only evident in four patients in our study. As suggested by Drs. Lichtenstein and Loubié, the explanation for the failure to detect characteristic parenchymal lesions may lie in the differences in the severity of disease. However, several studies have demonstrated that in PE peripheral lesions and the central obstruction of pulmonary artery branches occur simultaneously. After lodging at the bifurcation of the main pulmonary artery or the lobar branches, at least some thrombi disintegrate mechanically or under the influence of intrinsic fibrinolytic activity into smaller fragments and continue traveling distally, resulting in peripheral PEs.

Second, Dr. Lichtenstein and coworkers restricted their exploration to the anterolateral chest wall in supine patients, probably because of the difficulties in examining immobile and mechanically ventilated patients. In contrast, the data presented in our study are based on an ultrasound examination of all intercostal spaces (ie, ventral and dorsal). This may be particularly relevant to the diagnosis of PE. In a current investigation, only 18% of the parenchymal lesions were located at the anterior chest wall, whereas 82% could be detected in the lateral (14.5%) and dorsal (67.5%) intercostal regions. In addition, about 50% of the lesions were located within the lower lobes, 11% within the middle lobe, and only 9% within the upper lobes (unpublished data). Thus, for the detection of peripheral subpleural lesions, a thorough ultrasound examination of the complete intercostal area (ventral and dorsal) is indispensable. The uneven distribution of peripheral lung parenchymal lesions associated with PE with predilection for pulmonary emboli to the lower pulmonary section is in accordance with previous radiologic studies using CT scanning and with postmortem studies. The reasons for the predilection of pulmonary emboli to the basal and dorsal lung area are unknown. A possible explanation for the uneven distribution of embolic lesions may lie in the anatomic structure of the pulmonary arterial tree.

Third, venous thromboembolism is not a static disease but should rather be conceived of as a dynamic condition with rapidly changing features, with respect to all clinical, radiologic, functional, and laboratory findings. A number of animal studies (see the article by Kroegel and Reissig for a review) have suggested a gradual decrease in thrombus size within the first 2 to 4 days and a complete dissolution of the thromboembolic alteration within 48 h. The dynamic properties suggest that the results of diagnostic techniques performed to establish PE may directly be influenced by the period between the thromboembolic event and the diagnostic procedure.

Cyclooxygenase-2 Inhibitors in Aspirin-Sensitive Asthma

To the Editor:

As reported in CHEST (June 2002), several studies have suggested that selective cyclooxygenase-2 (COX-2) inhibitors, rofecoxib and celecoxib, are safe for use by patients with aspirin-induced asthma and who are intolerant to cyclooxygenase-1 (COX-1) inhibitors. However, there are less than 140 patients in all four of these studies. We wish to report a 78-year-old woman with asthma, well-controlled with benadryl and albuterol inhalers for at least 3 years, who developed an acute exacerbation of asthma symptoms after three doses of rofecoxib, each 25 mg. This patient had previously had an adverse reaction to aspirin and since then had avoided aspirin and all nonsteroidal antiinflammatory medications, using only acetaminophen for pain relief. The rofecoxib was stopped, and she responded to nebulized albuterol and prednisone therapy, and her symptoms improved.

Up to 10% patients with moderate-to-severe asthma are sensitive to aspirin. These patients may have life-threatening asthma if they take aspirin or nonselective cyclooxygenase inhibitors. The studies cited suggest that inhibition of COX-1 is the essential initiator of adverse outcomes in patients with aspirin-sensitive asthma. Therefore, highly selective COX-2 inhibitors should not affect these patients.

In our patient, one possibility is that she was unusually sensitive.

References

7. Stein PD, Henry JW. Clinical characteristics of patients with acute pulmonary embolism stratified according to their presenting syndromes. Chest 1997; 112:974–979.