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\textsuperscript{5,6} and was only evident in four patients in our study. As suggested by Drs. Lichtenstein and Loubiéres, the explanation for the failure to detect characteristic parenchymal lesions may lie in the differences in the severity of disease. However, several studies\textsuperscript{1,3} 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 80% of the lesions were located within the lower lobes, 11% within the middle lobe, and only 9% within the upper lobes (unpublished data).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.\textsuperscript{1}

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\textsuperscript{8–9} and with postmortem studies.\textsuperscript{10–12} 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.\textsuperscript{10}

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\textsuperscript{11} 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.

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Cyclooxygenase-2 Inhibitors in Aspirin-Sensitive Asthma

To the Editor:

As reported in CHEST (June 2002), several studies\textsuperscript{1–4} 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 bencathasone 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\textsuperscript{1–4} 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...
to the minimal COX-1 inhibition in rofecoxib. In any case, we recommend caution when prescribing selective COX-2 inhibitors to patients with aspirin-sensitive asthma.

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To the Editor:

We appreciate the comments of Drs. Passero and Chowdhry regarding our study of selective cyclooxygenase-2 (COX-2) inhibitors in patients with aspirin-induced asthma (AIA), which was published in CHEST (June 2002).1 We agree that recent reports about the safety of such inhibitors in patients with AIA were carried out with a limited number of patients1–4; as we suggested in our article, further challenge procedure studies performed with higher doses of rofecoxib and other highly selective COX-2 inhibitors on larger series of patients with AIA are necessary in order to achieve the safety of such new drugs in patients with AIA.

The authors mentioned that the acute exacerbation of asthma developed after three doses of rofecoxib, each 25 mg. We supposed each rofecoxib dose was taken once per day on 3 consecutive days, and not all three doses in 1 single day. There are no published data at present about the safety of COX-2 inhibitors in patients with AIA receiving high doses, and we regard that studies concerning this point are necessary.

It is known that the degree of sufficient enzymatic inhibition to induce bronchial narrowing in patients with AIA is an individual hallmark; therefore, we believe that oral challenges with progressive doses of the drug (rofecoxib or celecoxib) would have been done until a therapeutic and tolerable dose was reached. Nevertheless, despite the safety demonstrated by the new highly selective COX-2 inhibitors, caution when prescribing any type of nonsteroidal anti-inflammatory drugs in patients with AIA should always be recommended.

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Noninvasive Ventilation Is More Than Mask Ventilation

To the Editor:

I read with discouragement the article by Markstrom et al (November 2002) comparing the quality of life for patients with neuromuscular disease treated by noninvasive ventilation (NIV) vs that for patients who have tracheostomies. It is discouraging that an article could be published that equates NIV only with mask ventilation; that does not indicate pulmonary function or the extent of the need for ventilatory support and, thus, makes no effort to match cohort groups; that does not indicate the type of ventilator used, or settings, indications, or approaches; that ignores the vital need for mouthpiece ventilation, or even pneumobelt use via portable volume-cycled ventilators, for patients with advanced disease who require NIV continuously; that makes no mention of manually or mechanically assisted coughing methods or their vital need during intercurrent chest infections; and then concludes that tracheostomies are considered desirable by many postpolio patients and postkyphoscoliosis patients.

It is obvious that patients who are not trained in air stacking, effective and convenient daytime aid methods, or mechanically assisted coughing would feel more secure having tracheostomy tubes for disease management during intercurrent infections. Indeed, patients who are limited to mask ventilation, quite possibly at low pressure spans or inadequate daytime volumes, might feel better with a tracheostomy tube, even as a nocturnal aid. We have already reported on >100 patients who used both NIV and tracheostomy ventilation for continuous ventilatory support for ≥1 month, and only a few of those changing from NIV to tracheostomy ventilation who were never taught mouthpiece ventilation, air stacking, or mechanically assisted coughing considered the tracheostomy tube to be more desirable. Furthermore, there are cohort-matched studies of the quality of life comparing patients using the noninvasive and tracheostomy methods that the authors never mentioned. I suggest that the authors obtain a recent book on noninvasive ventilation and learn that there is more to NIV than mask-only ventilation.

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Communications to the Editor