ter a percutaneous lung biopsy in 13 of 52 cases (25%) in which the FEV1 was less than 2 L/m vs 3 of 41 with FEV1 of 2 or more liters per minute (7%, p<0.03, one-tailed Fisher's exact test).

Their finding of no difference in the total incidence of pneumothorax by computed tomography (CT) scan differs from the previous work of Fish et al. They allude to the most likely source of this discrepancy by noting the work of Murphy et al, who indicate that pneumothoraces detected only by CT scan are clinically inconsequential. Instead of elaborating on the size of the pneumothoraces detected in their study, they attributed their difference with Fish et al to selection bias in that work.

It is straightforward to estimate the volume of a pneumothorax by CT scan, which they did in these cases by Riemann sum integration of serial cut areas. What is the result of these data in these pneumothoraces vs FEV1 status? This test is necessary to remove the possible effect that the knowledge of the patient's pulmonary function may have had on the clinician's decision regarding chest tube placement. Until these data are available, we suggest that the authors consider renaming their article, "Risk of Significant Pneumothorax Increased 2.5-Fold by Obstructive Lung Disease in Percutaneous Needle Biopsy" to avoid misleading simple clinicians like ourselves.

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3. Murphy FB, Small WC, Wichman RD, et al. CT and chest radiography are equally sensitive in the detection of pneumothorax after CT-guided pulmonary interventional procedures. AJR 1990; 154:45-6

To the Editor:

We appreciate the comments by Drs. Dogra and Smeltzer concerning our article "Risk of Pneumothorax Not Increased by Obstructive Lung Disease in Percutaneous Needle Biopsy" (Chest 1994; 105:1705-08). We did choose our title to emphasize differences from previous studies. Another recent study also failed to show the fourfold increase in pneumothorax in patients with obstructive lung disease as previously reported by Fish et al (AJR 1988; 150:71-4). Chest tube requirements were not addressed in this study.

We have a computed tomography (CT) scanner (GE 9800). To obtain a biopsy, the area questioned is imaged, the patient is then slid out of the scanner on a gantry, and skin preparation and needle placement are done outside of the scanner. The patient is then advanced in and out of the scanner to check and adjust needle location. The aspiration is done out of the scanner. The patient is again advanced into the scanner and an image is obtained to look for a pneumothorax. This does not significantly add to the time or expense of the study. Obtaining a chest x-ray film after the study requires moving the patient to another area of the radiology department. We think that our experience is fairly typical, but institutions with CT scanners that are more recent in design and faster may have an advantage.

As only one section was scanned, we did not attempt to estimate the volume. We believe that there were only two cases of pneumothorax detected only on CT scan in our series. Neither of these patients showed an identifiable pneumothorax on subsequent chest x-ray films or required a chest tube, in agreement with the experience of Murphy et al (AJR 1990; 154:45-6). The most common indications for inserting a chest tube were respiratory distress with a pneumothorax immediately after the procedure or increase in size of the pneumothorax on sequential chest films. We admit that knowing a patient's pulmonary function status can bias a decision for chest tube placement. We only reported what happened at our institution over a 4-year period. Calling a pneumothorax significant only if it required chest tube placement may be more misleading than the title we chose.

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REFERENCE

Buprenorphine Causes Pulmonary Edema Just Like All Other Mu-Opioid Narcotics

Upper Airway Obstruction, Negative Alveolar Pressure

To the Editor:

Please allow me to correct some misinformation about buprenorphine. This drug is not a congener of butorphanol (Stadol), termed an agonist-antagonist. Buprenorphine has the clinical features of a most potent narcotic, with avidity and great potency of action on the mu (morphine) receptor. When introduced into the neuraxis via the epidural space it has produced respiratory inhibition unresponsive to naloxone reversal, necessitating prolonged intubation and ventilation of such patients. Nevertheless, in the postanesthesia care unit it offers effectiveness without unoward effect when judiciously given for postsurgical analgesia. It is not used as a component of general anesthesia. Essentially, it acts as a mu opioid, not as a kappa agent.

In the operating room, does morphine or buprenorphine or fentanyl or sufentanil cause noncardiogenic pulmonary edema? No! Potent mu opioids given to patients across a dosage range from slight analgesia to profound narcosis cause neither pulmonary alveolar toxicity nor "allergic reaction." But the illicit use of narcotics has long been recognized as a cause of noncardiac pulmonary edema, sometimes called neurogenic pulmonary edema, with misplaced emphasis. The pertinent pathophysiology of this phenomenon is the same as the occasional complication of upper airway obstruction with negative alveolar pressure.