We are confused by the statement that radiolabeled monoclonal antibodies "produce some undesirable activity in certain organs, such as the liver, spleen, and bone marrow." It is more probable the nature of the isotope, not the antibody, that results in this radioactivity. The bone marrow is the critical dose-limiting organ in radioimmunotherapy, and this is likely due more to red marrow targeting of blood-borne radioactivity than to nonspecific antibody accretion. Excessive liver and spleen radioactivity usually is seen when indium-111 labels are used.4

In summary, although locoregional administration of monoclonal antibodies is another intriguing possibility for cancer radioimmunotherapy, there is still a great dependence on the choice of antibody, antibody form, radionuclide, and dosage schedule.5

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Thromboembolism Associated with Acquired Immunodeficiency Syndrome

To the Editor:

Noninfectious thromboembolic disease is infrequently considered in the differential diagnosis of acquired immunodeficiency syndrome (AIDS)-related pleuropulmonary complications. Circulating antiphospholipid antibodies, though commonly detectable, are usually not associated with thrombotic complications in patients with human immunodeficiency virus (HIV) disease.1 Stimulated by the report of a case of unexplained pulmonary embolism,2 which appeared in the September 1991 issue of Chest, we describe a second case, in which the perceived rarity of this condition in AIDS led to a delay in diagnosis.

A 37-year-old Haitian man presented with a three-day history of dyspnea, right-sided pleurisy, fever (maximum, 39.5°C), and scant hemoptysis. He had advanced HIV disease (CD4+ lymphocytes, 6/cu mm; prior cerebral toxoplasmosis) but had recently been stable and ambulatory on a regimen of zidovudine. A chest roentgenogram showed a moderate right pleural effusion, consolidation of the underlying lung, and a small left effusion. The right-sided exudate contained 17,500 erythrocytes and 8,500 leukocytes (88 percent polymorphs) per cubic millimeter. Penicillin and cefotaxime were administered for suspected bacterial pneumonia. Cultures of blood, sputum, and pleural fluid were unremarkable.

The patient's symptoms and fever diminished over several days, but on day 10 he developed acute left pleurisy, tachycardia (120 beats per minute), and tachypnea (30 breaths per minute), followed by recurrence of fever (38.5°C) and recurrence of the left-sided effusion. Uncontrolled infection remained the focus of diagnostic thinking for another week, until radionuclide scans showed multiple, bilateral segmental perfusion defects in regions with normal ventilation, indicating a high probability of pulmonary embolism. Venous Doppler examination and computed tomography of the abdomen revealed no source of emboli. Routine coagulation studies on admission were normal, as were protein C, protein S, and antithrombin III levels. Circulating lupus anticoagulant was not detected. The patient recovered with anticoagulant therapy alone.

We believe that pulmonary emboli accounted for this patient's entire presentation, although an initial right-sided bacterial pneumonia cannot be excluded. No antecedent risk factor for venous thrombosis was identified. The predisposing role of HIV disease itself, if any, remains speculative. This and other recent cases3,4 emphasize the need to consider thromboembolism in AIDS patients, despite their young age and the usually infectious nature of pleuropulmonary disease with fever in this setting.

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Pulmonary Intubation with Nasogastric Tubes

To the Editor:

I read with interest the "Roentgenogram of the Month" feature1 in the November 1991 issue of Chest, in which pulmonary intubation with nasogastric tubes was discussed. This excellent discussion does not, however, address methods to prevent this common complication.

Roubenoff and Ravich2 describe a two-step method of feeding tube insertion, whereby the tube is first advanced to a point estimated to be at the ziphoid process and its position is then confirmed by an anteroposterior chest roentgenogram, which should show no lateral deviation from the midline, as occurs with bronchial intubation. The tube is then advanced into the stomach, and the position is confirmed with an abdominal x-ray film.

At the University of New Mexico Hospital Intensive Care Unit, we encountered eight episodes of pulmonary intubation in 170 instances of attempted nasogastric feeding tube placement (4.7 percent incidence) over a two-year period. We have modified our method of nasogastric feeding tube placement by rotating the patient's head to either shoulder. This simple maneuver causes deviation of the feeding tube tip from the midline laryngeal opening; this can be verified by visualization of the pharynx. With reeducation of physicians and nursing staff in the passage of fine-bore feeding tubes and meticulous documentation by a single abdominal x-ray film of appropriate tube location, this problem has not recurred

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over a one-year period of observation. While this cannot eliminate
the risk of pulmonary intubation, we recommend that this safe and
simple modification be used for insertion of all fine-bore feeding
tubes.

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Inadvertent Pericardiacophrenic Vein
Catheterization

To the Editor:

A unique complication arose during an attempted percutaneous
right subclavian vein catheterization. After insertion of the catheter,
guide-wire withdrawal was met with resistance, followed by the
unraveling of the outer coil-spring sheath. A chest radiograph
demonstrated a portion of the wire in a seemingly intracardiac
position. Upon exploration, the wire was found protruding through
the distal branches of the pericardiacophrenic vein (Fig 1).

The pericardiacophrenic vein, the main vein of the pericardium,
courses along the phrenic nerve and drains into the superior vena
cava through the internal mammary (thoracic) vein. Its course
parallel to the vena cava makes cannulation unlikely, although not
impossible. In this case, a slight angulation of the guide wire caused
by contact with the clavicle may have caused it to enter the vein.
The coil-spring, anchored in the pericardium, unraveled upon
withdrawal, exposing the broken-off piece of inner core, which does
not have recoil and will break if bent too often. This case emphasizes
again the element of chance associated with blind percutaneous
procedures.

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Diagnosis of Tuberculous Pleural
Effusion by the Detection of
Tuberculostearic Acid in Pleural
Aspirates

To the Editor:

I wish to comment on the article by Yew et al., which appeared
in the November 1991 issue of Chest. The authors considered
examination of pleural fluid for tuberculostearic acid (TBSA) to be
unhelpful, mainly because of its low specificity (52 percent). This is
in marked contrast to the results of our prospective study on the
diagnosis of sputum smear-negative pulmonary tuberculosis by
TBSA assay in bronchoscopic aspirates and bronchoalveolar lavage
(BAL) specimens. This study was performed by the Department
of Medicine, Chinese University of Hong Kong, in collaboration
with the Department of Microbiology, which carried out the TBSA
assay for both studies. Although the sensitivities of 52 percent and
68 percent for bronchoscopic aspirate and BAL specimens, respec-
tively, are similar to the sensitivity for pleural aspirates in the study
by Yew et al (69 percent), the specificities for bronchoscopic aspirate
and BAL specimens were both 100 percent. We therefore concluded
that TBSA assay of bronchoscopic specimens was useful for the
rapid diagnosis of sputum smear-negative TB.

What is the reason for the remarkable difference in specificity
for the different types of specimens processed in the same labora-
tory? Yew et al suggested that this might be related to release of
TBSA from old subvisceral pleural foci in their nontuberculosis
patients. I would like to suggest an additional possibility. Conditions
within a sizable pleural effusion rapidly become anaerobic. Since
TBSA is also present in some anaerobic organisms, such as actino-
mycetes, it is possible that some false-positive results may be due
to the presence of such organisms. In contrast, bronchoscopic
aspirate and BAL specimens are almost inevitably obtained from
well-aerated regions of the lungs, where anaerobes are unlikely to
be present, so that the specificity is high. Therefore, the diagnostic
value of TBSA in pulmonary tuberculosis seems to depend on the
type of specimen assayed, being good for bronchial aspirates and
BAL fluid but unsatisfactory for pleural aspirates.

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