

FIG. 1. Perfusion lung scan showing large perfusion defects in both bases seen on lateral and oblique views.

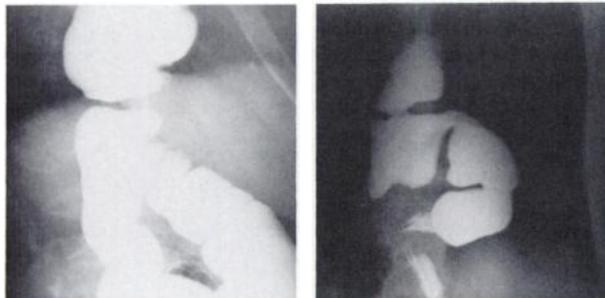


FIG. 2. Lateral view of barium enema showing mid portion of transverse colon in chest (left); Upper GI series demonstrating entire stomach and duodenal bulb in chest cavity (right).

esophageal hiatus hernia (2). We present a case of ventilation-perfusion mismatch secondary to intrathoracic stomach and colon.

A 68-yr-old white female was admitted for evaluation of anemia and fever. Her past medical history was remarkable for a left mastectomy for infiltrating ductal carcinoma. Physical examination revealed a temperature of 100.4°, a regular pulse of 80, and respirations of 16. The lungs were clear to auscultation. On cardiac examination a grade II/VI systolic ejection murmur was noted. The abdomen was negative. A chest radiograph showed evidence of old granulomatous disease and intrathoracic stomach.

During the patient's hospital course, she developed shortness of breath and chest pain, and a pulmonary embolism was suspected. A perfusion lung scan using 3 mCi of Tc-99m MAA demonstrated a large defect in both pulmonary bases (Fig. 1). The defects corresponded in location to a large hiatal hernia/intrathoracic stomach observed on chest roentgenogram. No segmental or subsegmental defects were noted, and the ventilation scan did not show a matched defect. Subsequently the patient demonstrated angina pectoris. As part of the evaluation of her anemia, a barium enema and upper gastrointestinal series (Fig. 2) were performed, and an intrathoracic stomach and colon were seen corresponding to the area of the perfusion defect on the lung scan.

Many causes of perfusion defects on lung scans have been described (1), and the above case illustrates two possible nonembolic etiologies.

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Re: Concerning the Labeling of DTPA-Coupled Proteins with Tc-99m

In their contribution describing the labeling of DTPA-coupled antibodies and fibrinogen with Tc-99m, Khaw et al. (1) do not claim that the label is attached to the DTPA moieties, although this could be inferred since DTPA-coupled proteins were used in their study. In this respect, the article may be misleading. In our attempts to label free DTPA in the presence of proteins, including antibodies and fibrinogen, we find that Tc-99m normally attaches primarily to the proteins. Following a faithful reproduction of the dithionite method described by Khaw et al., we were unable to label free DTPA with Tc-99m even in the absence of antibody, and at much higher DTPA concentrations (up to 1 mg/ml).

It is unlikely that a Tc-99m labeling method that successfully chelates the DTPA groups on proteins would result in colloids such as that described by Khaw et al., nor would we expect the rapid blood clearance and large liver accumulation. It is surprising that the authors have not compared the in vitro and in vivo behavior of the Tc-99m-labeled proteins with and without the attached DTPA groups.

Although we feel that the attached DTPA groups are not involved, we agree that proteins are labeled with Tc-99m by the described method. Furthermore, there is little question that this and other reported methods provide a protein label that is stable during in vitro analysis by gel chromatography, affinity chromatography, etc. It is the stability of the label in vivo that is not adequately established. In studies such as these, it would be helpful if the biodistribution of the Tc-99m-labeled proteins were compared with that of the same protein labeled by alternative methods such as radioiodination or, in the case of DTPA-coupled proteins, with In-111. It might then be possible to distinguish liver activity due to the uptake of labeled proteins from that due to the accumulation of colloids and other Tc-99m species of low molecular weight.

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