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Lung Scans in Hilar Lymph-Node Enlargement

Perfusion lung scans are performed most frequently in cases of suspected pulmonary embolism. The diagnostic accuracy of these scans is improved if they are combined with ventilation scans (*1*,*2*). The characteristic finding is mismatching, that is a region of abnormally high ventilation/perfusion ratio. The combined use of Tc-99m-labeled macroaggregates and the steady-state inhalation of Kr-81m allows detailed comparison of the relationship between ventilation and perfusion. Many centers use radio-xenon to detect regional abnormalities of lung ventilation. This radionuclide is a sensitive indicator of such defects, but we believe that Kr-81m is better at detecting areas of ventilation/perfusion mismatching because its gamma energy (190 keV) is better suited to direct comparison with the lung perfusion images obtained with Tc-99m. The radiation from Xe-133 (80 keV) is rather too low for that purpose (*3*). Ventilation/perfusion mismatching is not specific for pulmonary embolic disease. A number of conditions, ranging from relatively common ones such as bronchogenic carcinoma to rare ones such as pulmonary-artery sarcoma, are known to cause such mismatching (*4*). Fibrotic sarcoid has also been reported as a cause of ventilation/perfusion mismatching (*5*). We have encountered two patients with hilar lymph-node enlargement due to sarcoidosis with lung scan findings similar to those of pulmonary embolism.

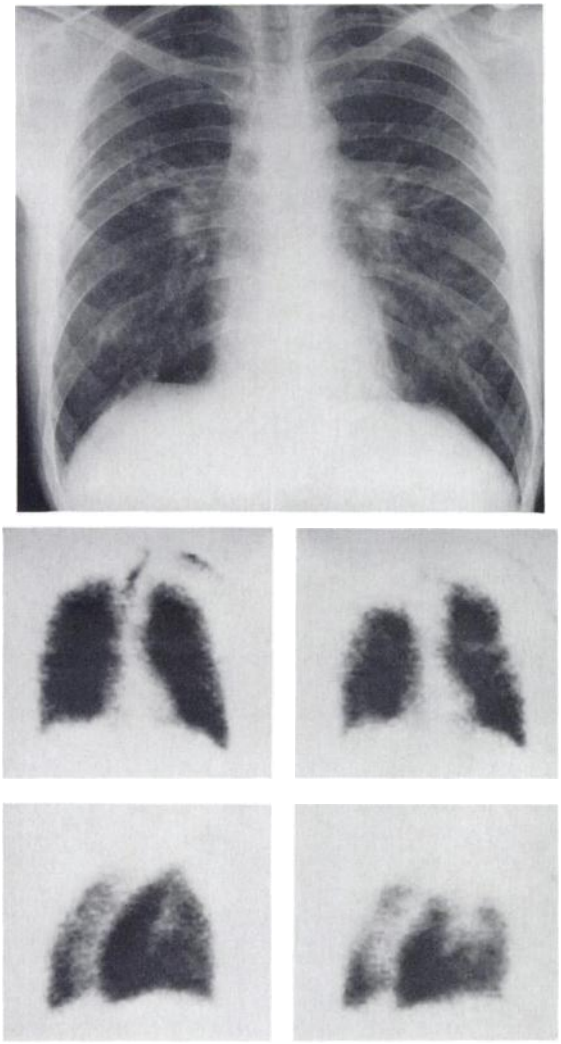

FIG. 1. Postero-anterior chest radiograph showing bilateral hilar lymph-node enlargement and patchy fibrosis in upper and mid zones. Anterior and right posterior oblique views of ventilation scans (top) and similar views of perfusion scans (bottom) show small matched defects in left lung and an area of high ventilation/perfusion ratio at right apex.

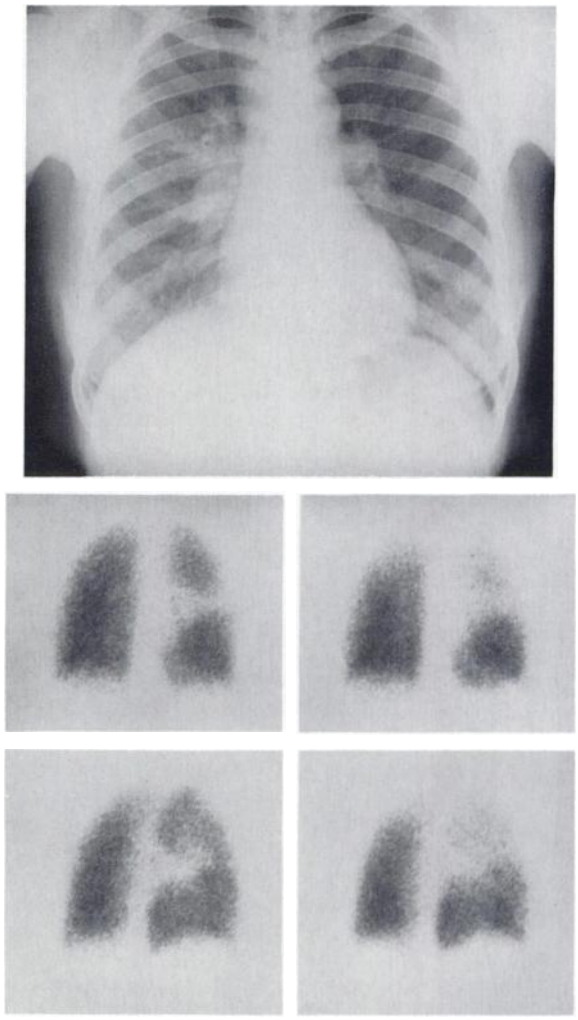

FIG. 2. Postero-anterior chest radiograph shows diminished activity over right mid zone and right upper zone. Posterior and right posterior oblique views of ventilation scans (top) show diminished activity over right mid zone. Similar views of perfusion scans (bottom) show diminished activity over right upper and mid zones.

The first patient, a 32-yr-old white male, presented with a nodular erythematous rash over his legs and back and a palpable spleen. Microscopy of a skin biopsy was consistent with sarcoidosis. The chest radiograph showed bilateral hilar lymph-node enlargement and patchy fibrosis in the mid and upper zones (Fig. 1). Ventilation and perfusion lung scans were performed using Kr-81m

and Tc-99m macroaggregates. These showed small matched defects in the left lung, but there was a defect of perfusion at the right apex not matched by a defect of ventilation (Fig. 1).

The second patient, a 56-yr-old Negro woman, has occasional cough and retrosternal pain unrelated to exercise. There was enlargement of some lymph nodes in the left supraclavicular fossa. One of these was biopsied, and microscopy was consistent with sarcoidosis. The chest radiograph showed marked right hilar lymph-node enlargement with discrete shadowing scattered throughout the right lung (Fig. 2). Lung scanning showed matched defects of ventilation and perfusion in the right mid zone, related to the parenchymal lung lesions. In the right upper lobe, however, there was a greater diminution of perfusion than of ventilation (Fig. 2).

Sarcoidosis frequently causes hilar lymph-node enlargement. It also causes interstitial lung lesions that have been shown to be associated with perfusion defects on lung scanning (*6*). The pulmonary vasculature is frequently involved in this disease. It is thought that sarcoid granulomata surround small blood vessels, decreasing the alveolo-capillary surface available for gas exchange (*7*). Less frequently the granulomata involve the walls of peripheral blood vessels (*8*).

There have been two well-documented reports of major pulmonary-artery compression by enlarged hilar lymph nodes in sarcoidosis (*7,9*). Lung scanning performed on our second patient showed matched defects of ventilation and perfusion presumably due to parenchymal involvement by sarcoidosis. However, the lung scans of both our patients revealed areas that were ventilated but only poorly perfused. These we feel can be attributed to compression by enlarged hilar lymph nodes. Our evidence for suggesting such a mechanism in the two cases mentioned is based on the relation between the perfusion defects and the hilar changes, where it can be seen that there are enlarged nodes in close relationship to the compromised vessels. Neither patient had evidence of venous occlusion and both were ambulant at the time of examination.

Mediastinal malignant infiltration is a well-recognized cause of mismatching. Our findings suggest that benign lymphadenopathy may also cause changes in the regional distribution of pulmonary blood flow.

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Thyroid Trapping of Technetium-99m During In Vivo Labeling of RBCs

This letter describes the occurrence of thyroid trapping of Tc-99m during in vivo labeling of RBCs in six patients with toxic diffuse goiter.

We are currently performing a prospective study, with informed consent, of cardiac function in patients with toxic diffuse goiter. One parameter of this study is the radionuclide gated blood-pool study (GBS) performed with RBCs labeled with technetium in vivo. During the evaluation of the first seven patients, six were noted to have thyroid activity; the thyroid of the seventh patient was outside of the field of view. Five of the six patients showed marked increase of activity (Fig. 1) and one had moderate increase. The six patients are listed in Table 1, with brief clinical history, thyroid function tests, and I-131 uptake. All had elevated thyroid function tests and 24-hr I-131 uptakes. The longest time period between thyroid tests and gated blood-pool study was 14 days. The patient with the least activity in the thyroid had the lowest uptake. Patient 1 was on Premarin, but all other patients were off medication. All in vivo labeling of RBCs was performed using 4.5 to 7.5 mg of stannous pyrophosphate*, injected intravenously. This was followed ~ 15 min later by intravenous injection of pertechnetate (Tc-99m), with imaging initiated ~ 20 min after that. Review of the technician worksheet performed at the time of the GBS revealed no record of infiltration of stannous pyrophosphate.

We believe the Tc-99m activity in the thyroid is attributable to increased thyroid blood flow and blood pool, and most significantly to elevated thyroid uptake secondary to toxic diffuse goiter (*1,2*). Other mechanisms could include infiltration of stannous pyrophosphate, but this appears unlikely. No infiltrated doses were recorded on the technician worksheet, and no salivary activity was seen in any of the six patients. The method of labeling may be inadequate, but we have not observed this degree of thyroid activity in any other patients to date.

Thyroid uptake compromising in vivo labeling of RBCs has been described previously by Abdel-Nabi et al. (*3*). They stated that they had no satisfactory explanation and believed thyroid hyperfunction could not explain the thyroid activity. As noted above, we feel that it could, and propose that it also may account for the two cases described by Abdel-Nabi. As he discusses, his first patient may have had increased thyroid uptake of the "rebound

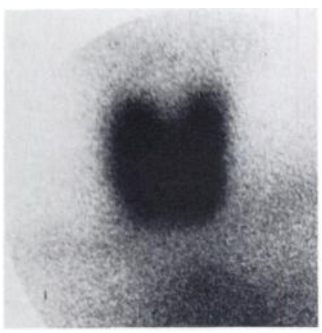

FIG. 1. Image of thyroid following in vivo labeling of erythrocytes with Tc-99m.