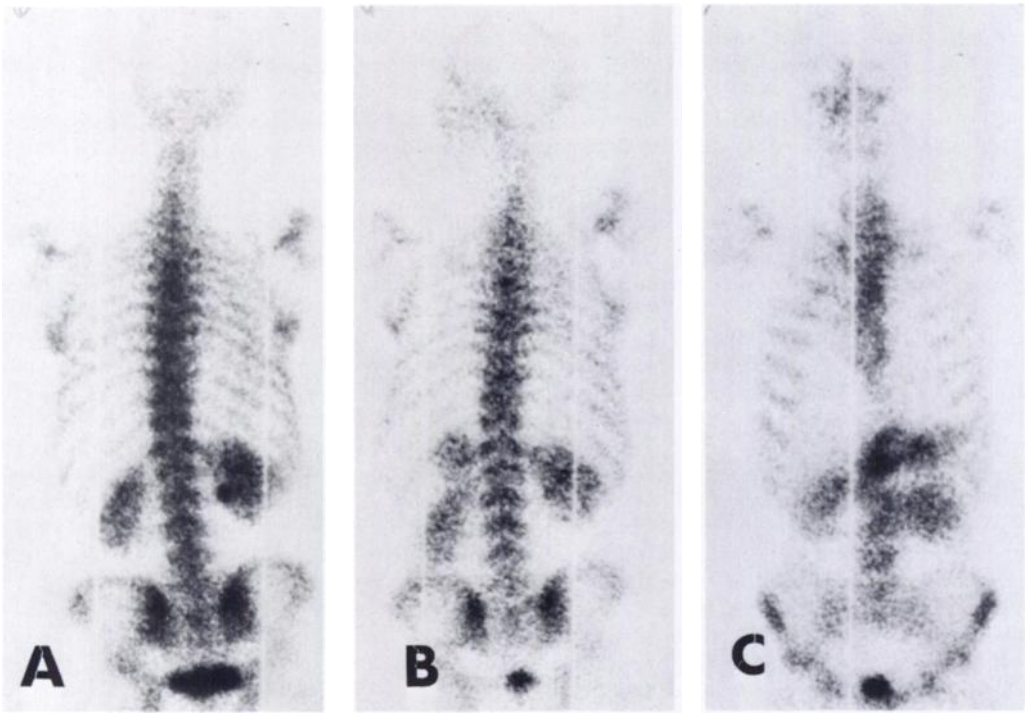

FIG. 2. (A) Uptake of pertechnetate by stomach after injection of 2 mCi of pertechnetate in case of normal scintigram. (B) Demonstration of stomach artifact in the posterior and (C) anterior views.

out to be stomach artifacts. This uptake was found with both Tc-99m diphosphonate (eight cases) and Tc-99m methylene diphosphonate (three cases). There was no extraosseous uptake in other patients investigated on the same day using the same vial of diphosphonate (DIP) or methylene diphosphonate (MDP). Only two of the 11 patients also showed thyroid uptake.

In eight patients it was possible to perform a repeat study, and it showed no gastric uptake. Four other patients with no abnormal uptake were injected with 2 mCi of [^{99m}Tc] pertechnetate after the completion of routine bone scintigraphy. Sequential scintigrams of the stomach region at 5-min intervals were made. The uptake in the stomach area appeared at 15 min and reached its height at 50 min. It corresponded in all aspects to the uptake observed in the 11 patients where it appeared unexpectedly. A normal bone scintigram after the injection of pertechnetate is shown in Fig. 2A.

Pertechnetate is known to accumulate in gastric mucosa (*1*) and this is assumed to be the reason for the appearance of a stomach artifact in bone scintigraphy. It is also possible that DIP or MDP accumulate in the stomach, but there is no proof for this.

Watson et al. have described metastatic visceral calcification in the stomach and lungs in a patient with marked hypercalcemia (*2*). Patients with chronic uremia show metastatic calcification that resolves after treatment (*3*). We have no information concerning the urea and calcium levels of our patients, but all of them were outpatients and in good clinical condition, and the eight patients on whom a repeat study was performed received no treatment.

Regardless of the mechanism involved, the possibility of stomach artifact should be recognized. It may mimic lesions in the ribs, or uptake in tumors of soft tissue (*4–6*). It is not a common finding, but when unrecognized may lead to inappropriate management of the patient.

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Vocal Cord Paralysis after Radioiodine Therapy

Although vocal-cord paresis may be seen in association with malignant or benign (*1*) thyroid disease or may follow

thyroid surgery, injury to the vocal cords or recurrent laryngeal nerves is not anticipated as a complication of radioiodine therapy for hyperthyroidism (*2*). Described below is a patient who developed a right vocal-cord paresis 1 wk after receiving radioiodine therapy for Graves' disease.

A 61-year-old white woman was first seen in December 1975, and stated that she had never regained the 20 lb she had lost after her husband's suicide in the fall of 1974. She suffered from chronic anxiety but it had not worsened. In the past year, her eyes had become more prominent. She denied heat intolerance or hyperdefecation. Pertinent physical examination revealed a blood pressure of 170/90, pulse 84, prominent and puffy eyes, globe and lid lags, a mild resting tremor, and a smooth goiter with bruits present over it, enlarging the gland symmetrically to twice its normal size. A thyroxine (T_4) was 16.6 μg/dl and the free thyroxine index 560 (normal 150 to 350). A 2-hr uptake of I-131 was 31%, 24-hr uptake 93%, with homogeneous distribution. In January 1976, she was treated with 7.3 mCi of I-131. One week later she suddenly became hoarse. She did not seek medical attention for 7 wk, but when seen, indirect laryngoscopy revealed a true right vocal-cord paralysis in the paramedian position. A repeat T_4 was 2.0 μg/dl. A chest x-ray and tomograms of the larynx were unrevealing. L-thyroxine therapy was started. She has been followed regularly in the ENT clinic, and as of August 1977 the vocal-cord paresis had remained.

Only once before has injury to the recurrent laryngeal nerves been reported following radioiodine therapy for hyperthroidism (*3*), and in that instance the treatment was for a multinodular goiter. Hypothyroidism, and very rarely hypoparathyroidism (*4*), are recognized complications of radioiodine therapy. However, the recurrent laryngeal nerves seem resistant to the high levels of radiation that accompany radioiodine treatment of Graves' disease. This case suggests that the resistance is not absolute.

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Splenic Accumulation of Tc-99m Diphosphonate in Thalassemia Major

Extraosseus accumulation of Tc-99m phosphate compounds occurs fairly commonly (*1–5*). We wish to report a case with thalassemia major who had splenic accumulation of Tc-99m methylene diphosphonate (MDP) without radiologic evidence of splenic calcification. Splenic concentration has been reported in patients with sickle cell disease (*6–8*) and Hodgkin's disease (*9*).

A 19-year-old Italian girl with thalassemia major was admitted to the hospital for investigation of her persistent nonspecific lower-lumbar pain. She was diagnosed at the age of 4 yr when she presented with anemia. On her initial admission her hemoglobin level was 4.2 g/dl and the blood film was hypochromic with marked anisocytosis, poikilocytosis, and polychromasia. The reticulocyte count was 7.8% and hemoglobin electrophoresis revealed a fetal hemoglobin of 47% but no other abnormal hemoglobins. A sickle-cell preparation was negative. Serum iron was elevated at 193 μg/dl. Radiographs of the skull and wrists showed classical changes of advanced thalassemia major. There was no family history of thalassemia or blood dyscrasias. She was managed on a hypertransfusion regimen, and before her last admission was having biweekly transfusions. She frequently developed congestive cardiac failure and her course was complicated by hypersplenism for which her parents refused a splenectomy. On her last admission she had the typical malar facies, hyperpigmented skin, and short stature of thalassemia major. She had mild cardiomegaly but no heart failure. Her massive spleen was palpable for 13 cm in the midclavicular line and her liver extended 8 cm below the costal margin. Spinal radiographs demonstrated osteoporosis and compression of vertebral bodies L2, 3, 4, and 5 and massive splenomegaly was obvious, but with no calcification. Her serum alkaline phosphatase and SGOT were elevated and her hemoglobin was 7 g/dl. Excretion pyelography showed an enlarged left kidney without obstruction. A Tc-99m methylene diphosphonate scan (8.3 mCi/m²) was performed on a high-resolution gamma camera. A large left kidney and enlarged splenic blood pool were observed. There was dramatic concentration of radiopharmaceutical throughout the spleen, but with a markedly irregular pattern (Fig. 1). There were focal areas of increased uptake in the right femoral trochanter, T10, 11, and 12 thoracic and L1, 2, and 3 lumber vertebrae.

Radiopharmaceutical integrity was confirmed by thin-layer chromatography, with greater than 99% of the Tc-99m being bound to the diphosphonate. Physiologic performance was satisfactory, since no thyroid or stomach activity was seen.

Many different types of nonosseus tissues may accumulate Tc-99m phosphate bone-scanning radiopharmaceuticals, but in none of these is the precise mechanism known. This phenomenon has been reported in normal breast tissue, carcinoma of the breast, malignant lymphoma, rectal adenocarcinoma, ovarian carcinoma, neuroblastoma (*1–3,10*), cerebral (*4*), and myocardial infarction (*3*), soft-tissue inflammation (*5,10*), and in association with intramuscular injections (*11,12*). Splenic accumulation has been reported in patients with sickle cell disease (*6–8*) and Hodgkin's disease (*9*). Goy and Crowe's (*6*) patient with sickle cell disease had radiographically obvious calcification in the region of the abnormal radiopharmaceutical concentration. Fischer et al. (*7*) described a 16-year-old boy with sickle cell disease who had functional asplenia and splenic accumulation without radiographic calcification. It has been postulated that the mechanism of tracer accumulation is due to splenic infarction with subsequent calcification. Byun (*11*) and VanAntwerp (*12*) described radiopharmaceutical localization in muscles where iron dextran had been injected, and postulated that a complex of iron dextran with Tc-99m phosphate might have been formed. Excessive iron deposition in the spleen may cause the dissociation of Tc-99m from the ligand carrier, with exchange to another ligand or complex and ultimate local accumulation (*18*). The presence of macroscopic and microscopic calcification has also been proposed as a cause of Tc-99m phosphate