facturer during preparation. We also failed to note any significant effect of reconstitution time upon Sn(II) content.

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## Reply

Kowalsky and Chilton have shown that the desired oxidation state of tin is maintained in Sn-PPi kits at various times after reconstitution and kits more than 90 days old. However, they have not shown that the Sn(II) is a chemically viable species. In fact their results would be similar whether stannous chloride, stannous oxychloride colloid, or stannous pyrophosphate were evaluated. Our *Journal* letter of October, 1982 (*1*), expressed concern regarding the status of pyrophosphate as an effective masking agent (*2*) in kits more than 90 days old. We did not base our comments on the oxidation state of Sn(II), but on its chemical state in Sn-PPi kits and on the incidence of hydrolytic by-products of Sn(II) secondary to ineffective masking.

Historically it has long been known (A. Reynoso-1852; H. Giren-1903) that pyrophosphate suffers from entropic doom and that this hydrolytic degradation is governed by pseudo-first-order kinetics (*2-4*). Lyophilization, unfortunately, does not remove all water. Furthermore, the effect of pH has been studied (*5,6*) as well as addition of neutral salts (*5-7*) and the influence of strongly coordinated cations (*7*).

For these reasons, and having investigated the properties of Sn-PPi kits (*8*), we limit Sn-PPi use to 30 days after manufacturing and 30 min after reconstitution with pertechnetate. This product is further limited to in vivo RBC labeling that requires a Sn(II) content of 15 μg to 20 μg per kg body weight. In vitro RBC labeling is performed using Sn-MDP or Sn-citrate. Myocardial infarction studies are performed very successfully using imidodiphosphonate (IDP).

Perhaps a more effective analytical technique to evaluate this compound would involve the use of HPLC and ion chromatography HPLC.

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## Perchlorate Blocking for Radioimmunodetection

Tumor detection using radiolabeled antibodies (RAID) is an important new technique (*1*) that is being applied in many centers. To date iodine isotopes have been the labels of choice because of their useful physical and biological characteristics and the simplicity of attaching them to proteins. However, the iodine is rapidly split from the antibody in the tissues, and unless preventative steps are taken it accumulates in the thyroid, stomach, salivary glands, and other organs, giving confusing results. Furthermore, damaging radiation doses to these organs may occur.

The usual technique for blocking radioiodine uptake is to give a large oral dose of stable iodide (KI) of ~ 500 times the normal daily intake of iodine (0.5 mg), but in practice, perhaps for reasons of poor intestinal absorption, this is insufficient to block all radioiodine uptake (*2*). Furthermore, pertechnetate ($TcO_4^-$), which is used as a subtracting tracer in RAID, has a much higher affinity (times 50 to 100) than iodine for the transport mechanisms (*3*) so blocking with iodide is even more difficult. This adds to the difficulties of both scan interpretation and radiation dose.

To overcome these problems we give potassium perchlorate ($KClO_4$) in addition to KI. $KClO_4$ has an affinity similar to $TcO_4^-$ for the iodide transport mechanisms, and can block stomach and salivary uptake of both radionuclides.

We use the following regimen: 30 min before the antibody injection, 420 mg of KI is given and thereafter 60 mg every 4 hr, with 120 mg at night, for 2 days; 2 hr before each study, 1 g of $KClO_4$ is given, which can split into four doses daily. After the last study the KI is reduced to 60 mg twice daily for 1 wk and no further $KClO_4$ is administered.

Using this regimen we have found that there is minimal radioiodine uptake in the stomach and salivary glands and little iodine accumulation in the gut. This gives us much greater confidence when interpreting images of the head and neck, stomach, pancreas, left lobe of the liver, and other abdominal organs. The only notes of caution are that the patients may experience nausea (in which case the $KClO_4$ doses should be divided) and allergic manifestations may rarely occur if the doses are extended beyond the period suggested.

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## Rhabdomyosarcoma Concentrating Tc-99m MDP

Extraosseous tissue uptake of bone-seeking agents has been observed in many malignant and benign conditions. This nonspecific phenomenon has not been found due to a single mechanism, and many causes must be taken into account. Cases of uptake by various neoplasms, such as ovarian (*1*), liver (*2*), breast (*3*), and other primary and metastatic carcinomas (*4*,*5*) have been reported.

In soft-tissue sarcomas of adults and in neuroblastomas in children, the uptake of bone-seeking agents by the neoplasm is not an uncommon event and could probably play a clinical role (*6*). Howman-Giles et al. (*7*) have even suggested that neoplastic uptake of Tc-99m phosphate compounds in a pediatric patient would be almost pathognomonic of a neural-crest tumor. However, Magill and Strang (*8*) reported a case of a paraspinal isolated metastasis from a Wilms' tumor taking up Tc-99m MDP, and suggested the nonspecificity of this finding.

Accordingly, we present here a similar case of rhabdomyosarcoma in support of this statement.

A 5-yr-old boy was admitted to a pediatric ward because of the presence of a paravertebral spindle-shaped mass. The mass was observed after a right lumbar contusion. The child complained continuously about a "left-sided" lumbar pain. A chest radiograph confirmed the presence of a paravertebral mass. Hematochemical data showed increased levels of CPK (382 U/l) and LDH (305 U/l). The level of urinary catecholamines was within normal range. Bone imaging with Tc-99m MDP was done for clinical staging. It showed uptake in a paravertebral mass in both anterior and posterior views (Fig. 1, left). The mass seemed to increase activity of the left tenth rib. No hyperactivity was observed in other skeletal structures. The radiological survey showed no bone abnormalities. A needle biopsy of the mass was performed and the histological diagnosis was "embryonic rhabdomyosarcoma" with dispersed infiltration of the skeletal muscle and diffuse necrosis.

The patient was treated according to AIEIP RMS 79 schedule: endoxan, vincristine, and actinomycin-D, combined with radiotherapy (44 Gy), with full radiological remission of the paravertebral mass. Repeat bone images performed at 6 mo after the end of the therapy showed no evidence of extraosseous uptake (Fig. 1, right).

Many factors have been suggested to account for soft-tissue tumor uptake: (a) tumor neovascularity with altered capillary permeability (*9*); (b) binding of phosphate compounds by mitochondria in damaged (particularly necrotic) tissue (*10*); (c) binding of the radiopharmaceutical to soluble proteins resulting from denatured macromolecules (*11*). In our case it may be that the second mechanism was the most important cause of the uptake.

This case is a further demonstration of the nonspecificity of soft-tissue tumor uptake in pediatric patients.

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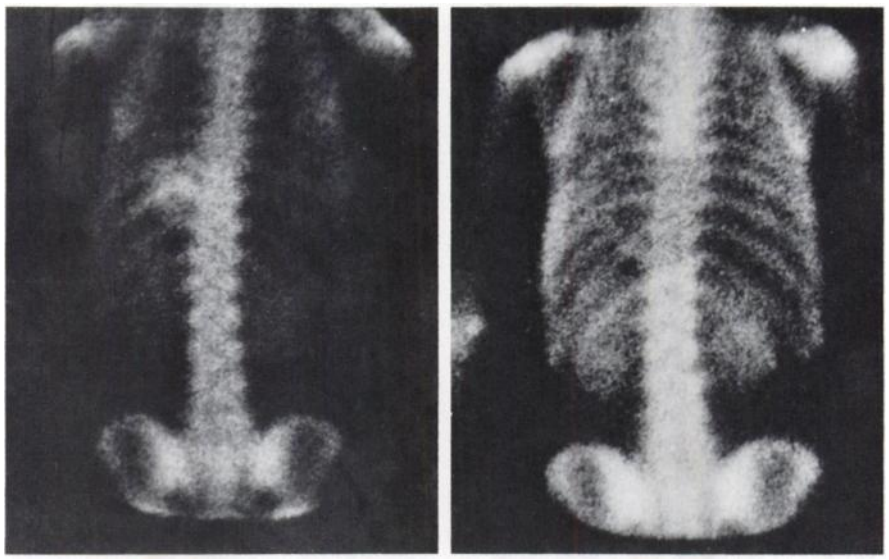

**FIG. 1.** Posterior bone image showing uptake in paravertebral mass (left): Bone study performed 6 mo after radiochemotherapy demonstrating no evidence of mass (right).