potheses and experiments designed to test them, whose goal is to show an effect of the caloric content of a test meal on gastric emptying. Because of this, I found the discussion of the nice study by Phillips and his colleagues of special interest (*1*).

The caloric content of a meal is measured by placing it in a calorimeter, oxidizing it completely, and measuring the heat produced. A living organism has no physiologic mechanism to measure a meal's calories, much less react to them. In fact, the calories of a meal are released over a long period of time after it is digested—probably days. An acute effect of an ingested meal's calories on gastric motility, or anything else for that matter, is preposterous.

The problem presented to investigators of this bankrupt idea is that it is impossible to prepare two meals that only differ in caloric content and do not differ in many already recognized and physiologically important variables. In summary, I find calories influencing gastric motility indigestable.

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REPLY: We agree with Dr. Hattner that gastric emptying is determined by multiple complex factors. The meal phase (*1*) (liquid versus solid), composition of the meal (*2*) (carbohydrate:protein:fat), weight (*2*) and volume (*3*) of the meal, stimulation of osmoreceptors (*4*), as well as energy density (*5,6*) (kcal/g) have all been reported to affect the rate of gastric emptying.

Although many of the articles in nuclear medicine and gastric motility literature emphasize the effect of the caloric content of a test meal on gastric emptying (*5–7*) (an increased number of calories is associated with slower gastric emptying), this theory is not always consistent. Our article (*J Nucl Med* 1991;32:377–381) described a 400-kcal glucose solution emptying almost twice as slowly as our 200-kcal glucose solution. However, we described in the same article a 400-kcal liquid glucose solution having a *longer* half-emptying time than the 600-kcal meal in the Velchik et al. study (107 min with the 400-kcal glucose solution versus 95 min with the 600-kcal solid meal). This difference, we believe, may be due to a slower rate of intestinal absorption of the solid meal, compared to the more rapid intestinal absorption of the glucose solution.

Thus, although we believe that all the variables mentioned above affect gastric emptying, in our opinion it is the rate of absorption of nutrients by the small intestine that is the most important factor controlling gastric emptying. The release of at least one gut hormone, gastric inhibitory polypeptide (GIP), has been shown to be dependent on the rate of absorption of nutrients by the small intestine (*8*). Another possibility is that circulating blood levels of nutrients could exert a strong control on gastric emptying. Several investigations have shown that intravenous infusion of glucose greatly slows gastric emptying (*9,10*). Obviously, the rate of absorption of nutrients by the small intestine and circulating blood levels are closely connected.

This rate of absorption, as Dr. Hattner has mentioned, can vary greatly between different types of foods (*11*). The absorption rate is dependent on the physical properties of each food.

The use of glucose solutions, as in our studies, allows for very rapid intestinal absorption after the glucose is emptied from the stomach. In this case, the gastric emptying time should be increasingly prolonged with increasing caloric content, caloric content being simply an indirect way of specifying the quantity of glucose.

If these ideas are correct, they have important applications in the performance of gastric emptying studies. Therefore, we believe that, in order to have reproducible gastric emptying studies, the physical properties and quantity (weight) of a gastric emptying meal must be rigidly controlled.

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A Novel In Vivo Red Cell-Specific Label

TO THE EDITOR: In his editorial (*1*), Ronald J. Callahan listed 13 radionuclides that have been used to label human red blood cells and commented on the in vitro and in vivo labeling protocols. We would like to add radioiodine to the list following our work on the selective labeling of human erythrocytes by para-iodobenzenesulphonamide (pIBS). The results of these studies have recently been published (*2–4*), and we simply wish to point out the advantages of our technique over current methodologies.

Our targeting method is based on the use of a facilely radioiodinated, lipophilic enzyme-inhibitor as a radionuclide "carrier." Red cells are ideal for this approach since they contain a high concentration of the target enzyme, carbonic anhydrase. Further-

more, potent inhibition of the target by aromatic sulphonamides is well understood, allowing the rational design of such carrier sulphonamides (*5* and references therein). In vitro incubation of whole human blood with radioiodinated pIBS gives quantitative uptake by red cells in less than 1 min at 37°C. In vivo administration of the radiopharmaceutical, involving a single intravenous injection, is just as effective, and it clears from the blood at least eight times more slowly than the standard technetium-red cell label. Consequently, blood-pool images can be obtained using far less activity and over longer periods than are routinely employed for ^{99m}Tc; moreover, the problems that are now occasionally encountered with patients on medication are unlikely to arise when using this selective approach.

While ^{123}I has excellent imaging properties and [^{123}I]pIBS is amenable to kit preparation, the cost and limited availability of the radionuclide vitiates its use in routine blood cell labeling. However, a combination of our approach with the use of ^{99m}Tc would present an ideal short-term blood-pool imaging agent. We recently have been investigating ^{99m}Tc-propyleneamineoxime derivatized sulphonamides in this regard (*6*), and other preliminary studies in this area have been reported by Subramanian and coworkers (*7*). Unfortunately our results have not yet matched those with radioiodinated pIBS, since there is significant plasma binding of the technetium complexes; however, an equally efficacious agent should eventually emerge.

The simplicity of the protocol, the selectivity of the carrier, and its persistent retention suggests that the enzyme-inhibitor approach to red cell labeling will not only be of value to short-term studies but could also offer significant advantages to longer term studies such as the determination of in vivo red cell survival.

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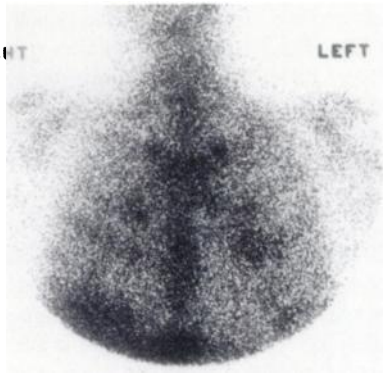

FIGURE 1. Anterior ^{67}Ga-citrate scan showing increased lung uptake and absence of deltoid muscle uptake.

Gallium Uptake in Eosinophilia Myalgia Syndrome

TO THE EDITOR: In a recent case report on gallium uptake in eosinophilia myalgia syndrome (EMS), Kim et al. (*1*) reported gallium lung uptake but absence of uptake in the muscle. The authors attributed the lack of muscle uptake to prior steroid therapy. We wish to report a patient with EMS, who had positive muscle biopsy but failed to take up gallium at that site even though he was not on steroid therapy.

A 64-yr-old male presented with fatigue, myalgia, muscle cramps, and swelling of his extremities. Examination showed non-pitting edema in the extremities and moribiliform rash on the abdomen. He had been taking L-tryptophan for 6 mo for insomnia. Laboratory data revealed increased sedimentation rate and absolute eosinophilia of 1170/mm^3. Chest x-ray demonstrated bi-basilar reticular infiltrates. A gallium lung scan showed bilateral basal uptake and normal uptake in the deltoid muscles (Fig. 1).

Muscle biopsies from both deltoid areas (including skin and fascia) were compatible with eosinophilic fascitis and EMS. Open lung biopsy showed diffuse interstitial fibrosis (desquamative interstitial pneumonia). This case points to the nonspecificity of gallium lung uptake and the need to pursue tissue diagnosis. Absence of gallium muscle uptake, as seen in this case, may be a feature of this disorder.

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REPLY: We wish to thank Dr. Rao et al. for their comments on our recent paper (*1*).

Before responding to their letter, we must note that we had difficulty in understanding their description of Figure 1, which they described as "bilateral basal uptake," though the image shown does not have increased uptake at the bases. In addition,