Three unambiguous terms can be defined:

1. "Carrier-free" (CF) should mean that the radionuclide or stable nuclide is not contaminated with any other stable or radioactive nuclide of the same element.

2. "No carrier added" (NCA) should apply to an element or compound to which no carrier of the same element has been intentionally or otherwise added during its preparation.

3. "Carrier added" (CA) should apply to any element or compound to which a known amount of carrier has been added.

It should be clear that these terms refer to a specific position or positions when applied to a molecule.

The term "carrier-free" (CF) is the most difficult to quantitate. It should only be used when evidence is provided that the element or compound is indeed carrier free. There are very few examples at present where this might even be possible. It may be useful to modify this term slightly by using "near carrier-free" (NCF), followed by an obligatory quantitation, such as ^{18}F-NCF (1% of all fluorine atoms in the compound at assay time are stable fluorine).

The term "no carrier added" (NCA) applies to the vast majority of elements and compounds to which the term "carrier-free" is incorrectly applied. The problems in technetium chemistry are well known. Whereas many of the preparations of ^{11}C-carbon monoxide are of "high" specific activity, none in the literature today are demonstrably CF, indeed they are far from it! If known, the term "NCA" can also be qualified with a statement of precise or approximate dilution.

The term "carrier-added" (CA) is obvious and again should be qualified with precise data if available

The use of these three simple terms CF, NCA, and CA would immediately identify for the reader the extent of dilution of the tracer and simplify the evaluation of utility in those cases where precise nuclidic, radioactive, or other composition is *necessary* information for the study. If nothing else, their use would remove a source of confusion and misdirection in the scientific literature.

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Re: Simultaneous Treatment of Toxic Diffuse Goiter with I-131 and Antithyroid Drugs: A Prospective Study

Recently in the *Journal* Steinbach, Donoghue, and Goldman reported a decreased incidence of hypothyroidism after I-131 therapy for diffuse toxic goiter in patients who were receiving antithyroid drugs compared with those who were not (*1*). They reported that the estimated thyroid weights for determination of the I-131 doses were obtained from thyroid scans "using the Bauer-Blahd formula," but did not report the actual estimated weights. Application of their tabulated data and their dosimetry formula appears to show an average estimated thyroid weight of 91 g for patients who received I-131 alone and 77 g for patients who received both I-131 and antithyroid drugs.

In another recent *Journal* article, Tamagna, Levine, and Hershman reported a considerably lower average estimated thyroid weight of 43 g in 12 patients with diffuse toxic goiter "based on planimetry of the thyroid scan" (*2*). Their series also consisted of mostly male patients, although their patients were presumably from California rather than from New York.

The discrepancy between the average estimated thyroid weights in the two studies suggests the possibility that Steinbach, Donoghue, and Goldman may consistently derive higher estimates of thyroid weight than at least some other physicians might. If so, perhaps they could decrease the incidence of hypothyroidism after I-131 therapy in their patients by merely revising their technique for estimating thyroid weight rather than by adding antithyroid drugs to their regimen.

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2. TAMAGNA EI, LEVINE GA, HERSHMAN JM: Thyroid-hormone concentrations after radioiodine therapy for hyperthyroidism. *J Nucl Med* 20:387-391, 1979

Reply

Dr. Hegge comments on an apparent discrepancy in the thyroid gland weight between our study (*1*) and that performed by Tamagna et al. (*2*). He then concludes that by revising the calculation of the thyroid weight alone the incidence of hypothyroidism could be reduced.

Dr. Hegge has quite reasonably arrived at the mean thyroid weights in our series. Our data show this mean weight to be 85 g (range 24-190 g) for Group A and 72 g (range 33-239 g) for Group B.

Our data quite obviously differ from those of Tamagna et al. (*2*), and any of the following could account for the differences: (a) patient selection—random (*1*) contrasted with selective (*2*); (b) type of patients—all male (*1*) contrasted with 21% female (*2*); (c) geographical location—Great Lakes (*1*) contrasted with West coast (*2*); (d) type of goiter—toxic diffuse only (*1*) contrasted with toxic diffuse and nodular (*2*). In addition, the number of patients in either study is too small to be representative of any particular population.

In another study by Blahd and Hays (*3*) done on 241 male hyperthyroid patients at the Wadsworth VA Hospital in California, the mean thyroid gland weight was 51 ± 28 g.

Dr. Hegge's suggestion that reduction in thyroid weight used for calculation—i.e., reduction of the dose administered to the thyroid—will result in decreased incidence of hypothyroidism is not a novel one. A vast amount of literature is available on this subject (some of it referred to in our original paper). Dr. Hegge, however, has ignored the difference in results for the two groups we reported. Group A had a very low incidence of hypothyroidism (8%) and Group B had an incidence (36%), which is no different from other reported series (*4*,*5*). Since gland weight was calculated in the same manner for both groups, it could not account for the difference in outcome.

Finally, Dr. Hegge has suggested revision of our method of calculation, implying that the method used by Tamagna et al. is the preferred one. That method, however, is the same as the one we have used, and the difference is only the semantics of reporting.

Our paper examined the effect of a 5000-rad dose on the clinical

outcome of toxic diffuse goiter. We have shown a clear difference in outcome when patients are treated simultaneously with antithyroid drugs. The superiority of our method to the various so-called low-dose methods has not been established.

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Mechanism of Pertechnetate and Iodine-123 Localization in Warthin's Tumor

We read the letter by Moinuddin and Rockett (*1*) with interest. They describe a patient whose Warthin's tumor in the parotid gland was visualized on 24-hr iodine-123 scan. We have encountered a similar patient (Fig. 1). A follow-up pertechnetate (Tc-99m) salivary gland scan was virtually diagnostic: there was increased radioactivity in the parotid tumor and the activity failed to wash out following administration of potassium perchlorate (*2-4*). Surgical confirmation was obtained.

A possible explanation of the reaction of Warthin's tumor to pertechnetate and iodine could lie in its histology. These tumors (and oncocytomas) arise from the ductal epithelium and retain their ability to extract and secrete large anions (iodides, pertechnetate, etc.) (*5,6*). The presence of large numbers of mitochondria in the tumor cells also confirms their secretory capabilities (*7*). There is no communication between the cystic spaces within Warthin's tumors and the ductal system; therefore, pertechnetate and iodine continue to accumulate without being discharged.

The failure of perchlorate to wash the pertechnetate out of the Warthin's tumor in our patient is against the theory that the uptake of iodine in such tumors is related to abberant thyroid tissue.

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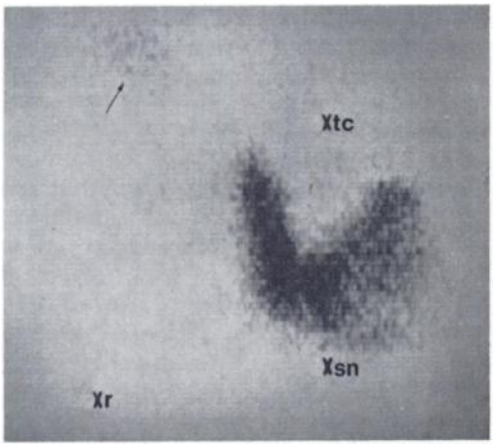

FIG. 1. Twenty-four hour iodine-123 scan showing cold nodule in left lobe of thyroid and accumulation of radioiodine in right parotid mass. Thyroid cartilage (tc); suprasternal notch (sn); right side of the neck (r).

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Re: Development of I-123-Labeled Amines for Brain Studies: Localization of I-123 Iodophenylalkyl Amines in Rat Brain

I read with great interest the paper on "Development of I-123-labeled amines for brain studies: Localization of I-123 iodophenylalkylamines in rat brain" by Drs. Winchell et al. (*1*). I bring to the attention of the authors and readers that the compound identified as 4-iodoantipyrine in the text is actually 4′-iodoantipyrine (as shown in Table 1A). These two compounds have different chemical formulae and chemical characteristics. The interchange of names might create some confusion because only 4-iodoantipyrine has been accepted as a true tracer for the blood flow measurements in the brain.

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