volume is low compared with tissue volume, and this assumption is reasonable. However, in the lung, plasma volume is nearly equal to tissue volume, and plasma activity could be a substantial fraction of total activity in a region of interest, even late during imaging. This consideration may be particularly problematic in regions with slow uptake, such as the diluent lobe in our experiment, for which the error may be magnified when SUV is used.

Using dynamic ^{18}F-FDG scans rather than late, static PET scans is more complicated, as it requires a rapid, smooth injection; multiple blood draws; and a single–bed-position acquisition. However, our laboratory has developed a method to reduce the number of arterial samples to derive an input function (*2*). With this method, we can take as few as 2 venous samples (we often take 5 or 6 in case of problems with sampling), which are used to "calibrate" an input function measured with PET from a region of interest defined over the right heart or the aorta. Granted, we still have to image continuously during injection, thus reducing the imaging field to a single bed position. Newer scanners are able to acquire larger fields of view, or alternatively, one can move the subject back and forth between 2 or 3 bed positions in order to acquire dynamic datasets.

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Differentiated Thyroid Carcinoma: Is There Any Evidence for the Use of Recombinant Human TSH in Thyroid Hormone–Secreting Metastasis?

TO THE EDITOR: We are thankful for the interesting and highly relevant article by Douglas Van Nostrand and colleagues, published in the March 2012 issue (*1*). The objective of this study was to compare administration of recombinant human thyroid-stimulating hormone (rhTSH) versus thyroid hormone withdrawal for the identification of metastasis in differentiated thyroid cancer (DTC) on ^{131}I planar whole-body imaging and ^{124}I PET. The authors observed that ^{131}I planar whole-body scans and ^{124}I PET scans identified significantly more foci of metastasis in patients after preparation with thyroid hormone withdrawal than with rhTSH injections. The conclusion drawn by the authors that "physicians should be cautious in using rhTSH for patient preparation before diagnostic scanning for the detection of DTC or treating distant metastasis secondary to DTC with ^{131}I" appears well founded on the data presented. Furthermore, such a study is important, because recently there has been a shift toward the use of rhTSH in increasing numbers of indications in patients with DTC.

However, we have several concerns regarding the authors' conclusion that "the use of rhTSH is appropriate for patients. . .[to] increase their endogenous TSH because their metastases are producing significant thyroid hormone." First, none of the patients in the evaluated study cohort was identified as a patient with thyroid hormone–secreting metastasis. Second, there is no corresponding discussion to support this conclusion. Furthermore, DTC with thyroid hormone–secreting metastases is exceedingly rare. Only a few cases have been reported since the first patient with adenocarcinoma of the thyroid with thyroid hormone–secreting metastasis and postoperative thyrotoxicosis was described by Leiter et al. in 1946 (*2*). Because of the small number of cases reported so far, patients with hormone-producing metastasis represent a challenge for the further treatment of DTC. Recently, we reported the case of a patient with thyroid hormone–secreting metastasis leading to persistent TSH suppression after thyroidectomy and radioiodine remnant ablation. As suggested by Van Nostrand et al., we assumed that rhTSH was the appropriate preparation to elevate the TSH level before ^{131}I whole-body imaging. However, we observed that when applied before the second radioiodine treatment, rhTSH increased the ^{131}I uptake into the thyroid hormone–secreting metastasis and prolonged the effective half-life of ^{131}I in relation to measurements from the first radioiodine therapy without rhTSH (*3*). Compared with the original therapy without rhTSH, the ^{131}I uptake after rhTSH increased from 8.4% to 39% and the effective half-life increased from 2.2 to 4.1 d. Subsequent radiation exposure caused bone marrow toxicity with myelosuppression. To prevent grade IV neutropenia, the patient was successfully treated with pegfilgrastim, a long-acting granulocyte-stimulating growth factor.

On the basis of the data presented by van Nostrand et al. and our own experience, we cannot agree with their recommendation regarding the use of rhTSH in the subgroup of patients with thyroid hormone–secreting metastasis. We would propose adding the following clarifications to the article: rhTSH might not be necessary for diagnostic ^{131}I whole-body imaging in patients with TSH suppression due to thyroid hormone–secreting metastasis, because thyroid hormone–secreting metastases generally show a high ^{131}I uptake. Furthermore, in patients undergoing ^{131}I treatment, the use of rhTSH needs to be handled carefully as it can increase ^{131}I uptake and prolong the effective half-life of ^{131}I, leading to an increased exposure to radiation and bone marrow toxicity. Because this subgroup of patients is extremely rare, further studies regarding their optimal diagnostic work-up and treatment should be performed before any general recommendation is given.

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REPLY: I would like to thank Schneider et al. for their compliments regarding our article (*1*), and I value and appreciate their time in submitting their concerns regarding our recommendations at the end of the article.

For the reader, the correct quotation of our recommendation from our article was "the use of rhTSH is appropriate for patients who cannot. . .increase their endogenous TSH because their metastases are producing significant thyroid hormone." As Schneider et al. point out correctly, our article does not address the appropriate or inappropriate use of recombinant human thyroid-stimulating hormone (rhTSH) in those rare patients with differentiation thyroid cancer whose endogenous thyroid-stimulating hormone (TSH) cannot be increased because their metastases are producing a significant amount of thyroid hormone. In retrospect, I believe that I could have chosen a better phrase to have communicated my original intent in that "the use of rhTSH injections is still appropriate to *consider* in patients who cannot increase their endogenous TSH because their metastases are producing significant thyroid hormone." Again, I appreciate the time and effort of Dr. Schneider et al. in bringing this to the readers' and my attention.

Having said that, I believe an even more important point is noted by Schneider et al. in their original case report, in which they emphasize that "even standard activities of 7.4 GBq (200 mCi) [of] ^{131}I may constitute a crucial dose in the rare combination of thyroid hormone secreting metastases and rhTSH-stimulation. . ..(*2*)" And as they further state, "higher standard [fixed] activities of ^{131}I should not be used without pretherapeutic dosimetry in patients with such large functioning metastases." I certainly agree with and support this comment. In addition, I believe that pretherapeutic dosimetry should not just be performed in a patient, like theirs, who is being considered for a fixed prescribed activity higher than 7.4 GBq (200 mCi), but pretherapeutic scans and pretherapeutic dosimetry should also be performed in all patients who are being considered for ^{131}I therapy and have documented or suspected functioning metastatic differentiated thyroid cancer. As has been reported by multiple authors, including Leeper (*3*), Tuttle et al. (*4*), and Kulkarni el al. (*5*), as many as approximately 10%–20% of patients may receive over 200 cGy (rad) to the blood (e.g., bone marrow) if prescribed activities of ^{131}I ranging from 3.7 GBq (100 mCi) to 7.4 GBq (200 mCi) are administered. (Additional restrictions apply, including not administering a prescribed activity of ^{131}I that would result in more than 4.44 GBq [120 mCi] of ^{131}I whole-body retention at 48 h in patients without pulmonary metastases and 2.96 GBq [80 mCi] of ^{131}I whole-body retention at 48 h in patients with pulmonary metastases.) In fact, as reported by Schneider et al. and using the OLINDA/EXM software, they calculated that the patient's blood-absorbed dose was 320 cGy (rad). If full dosimetry is not available, then the use of one of the simplified dosimetric alternatives such as percentage 48-h whole-body retention as proposed by Hänscheid et al. (*6*) or Van Nostrand et al. (*7*) should be considered in order to identify those patients whose prescribed activity of ^{131}I should be reduced. These simplified methods can be performed in almost any nuclear medicine facility.

Again, I thank Schneider et al. for their compliments, comments, and time.

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Palliation and Survival After Repeated ^{188}Re-HEDP Therapy of Hormone-Refractory Bone Metastases of Prostate Cancer: A Retrospective Analysis

TO THE EDITOR: We read with great interest the article by Biersack et al. published in the November 2011 issue (*1*). Because our group shares with the authors a similar interest in the potentials of therapeutic bone-seeking radiopharmaceuticals not only for palliation of bone pain but also for some objective antitumor activity (especially when administered in combination with other therapies) (*2,3*), this article constitutes for us an additional source of inspiration and stimulates further impetus to our ongoing investigations in this field.

Considering this evolving scenario, we believe that clarifying somewhat further some of the issues addressed by Biersack et al. would contribute to enhancing the value of the overall information that the nuclear medicine community (as well as the medical