REPLY: In his letter commenting on our article (*1*), Dr. Seto says that he believes the numbers in Table 2 are incorrect. From the table, he calculates a total increase in group A of 28% and an 11% increase in group B. As reported in the footnote of our table, 4 patients were excluded. Therefore, we reported the total increase of only the included patients. This results in a 38% increase of the maximum standardized update value (SUV) for group A and a decrease of 11% for group B.

Dr. Seto's comments focus on 2 issues: first, the calculation of the averages; second, the reasons for excluding patients.

Regarding the first of these issues, the average increase in maximum SUV as presented in Table 2 of our article is the average increase in maximum SUV of the separate lesions per patient. Indeed, the increase in maximum SUV could also have been presented the way Dr. Seto suggests, as the increase between the averages of the initial maximum SUV and the averages of the maximum SUV at follow-up. Calculating the increases in this way would result in a 31% increase for group A and a 14% decrease for group B. Clearly this would not change our overall conclusion, that is, an increase in maximum SUV for the patients in group A, compared with the decrease in maximum SUV for the patients in group B.

Regarding the second issue, reports show that serial measurements of lesions with high initial ^{18}F-FDG uptake tend to be inaccurate because of rapid growth and the development of central necrosis (*2*). For this reason, we described in detail the exclusion of patients in whom most metastatic lesions showed SUVs greater than 10 on the initial ^{18}F-FDG PET scan. Dr. Seto suggests also excluding those patients who showed SUVs greater than 10 on the second ^{18}F-FDG PET scan. We do not completely understand this suggestion since these final scans do not need any serial measurement afterward.

Furthermore, Dr. Seto suggests that, rather than excluding patient 9, we selectively count those lesions with SUVs less than 10 at the initial measurement. In our opinion, it is more reasonable to exclude this patient than to selectively analyze only the 2 lesions with SUVs less than 10. If we were to include only these 2 lesions, the increase in maximum SUV for one lesion would be 15% (initial maximum SUV, 9.7; maximum SUV at follow-up, 10.2) and the increase in maximum SUV for the other lesion would be 16% (initial maximum SUV, 7.8; maximum SUV at follow-up, 9.1).

Therefore, we do not understand Dr. Seto's questioning the overall conclusions on the basis of the exclusion of patient 9, as inclusion of the 2 lesions of patient 9 is in line with our conclusions.

Moreover, Dr. Seto questions the exclusion of patient 17 in group B. We excluded this patient because a second liver metastasis had developed directly adjacent to the first metastasis. It proved to be impossible to reliably determine the separate SUVs on ^{18}F-FDG PET. Therefore, we believe that excluding this patient is valid.

Although significant differences between both groups were observed, we did not overestimate the results: our conclusions were qualified by our admitting that only a limited number of patients were studied. Thus, we are well aware that our findings may not be definitely conclusive for primary tumor–induced growth inhibition of metastatic disease. However, our results are in concordance with earlier reports (*3,4*) in which an increase in vascularization and growth of liver metastases was reported after resection of primary colorectal carcinoma.

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Can Delayed Cortical Transit Identify Those Kidneys Whose Function Is at Risk?

TO THE EDITOR: There is presently no single clinical study trying to estimate the predictive value of renal cortical transit in hydronephrosis, using as endpoint the deterioration of renal function during a longitudinal follow-up of patients treated conservatively. Therefore, we read with much interest the experimental study of Schlotmann et al. (*1*). We are, however, somewhat reluctant to accept entirely the conclusion that "a delayed [cortical transit] should identify those kidneys whose function is at risk."

Nineteen hydronephrotic kidneys were investigated before and after the creation of an experimental renal obstruction. Most had a striking decrease in split function on the first postoperative test, with a further drop at the second test, performed 2–4 wk after surgery. Such an evolution is not common in the clinical practice of antenatally discovered hydronephrosis. Split function, when measured some time after birth, can be either normal or abnormal, but in neither case is it common to observe such a huge deterioration of renal function during follow-up. The model of obstruction that the authors have created obviously corresponds to an extreme pattern of obstruction, close to subtotal or total obstruction.

When looking now to the detailed results of the study, one has to focus on only 12 of the 19 hydronephrotic kidneys, since 1 kidney was declamped between the first and second renograms, 2 others apparently underwent a second surgical intervention after the first renogram (to increase the degree of obstruction), and 4 were removed from the study because of technical problems. In only 9 of the remaining 12 kidneys was the first postoperative cortical transit predictive of the second postoperative split function. In 2 kidneys, cortical transit was delayed but split function remained stable. In 1 kidney, cortical transit was not delayed but split function deteriorated. Moreover, the same predictive value could be obtained in this study by replacing the first postoperative cortical transit by the first postoperative split function, or even by the first postoperative response to furosemide. Once again, this suggests that we are dealing here in most of the cases with a model