our own study using an F+8 protocol, which is not ideal for MTT derivation, a monotonic relationship between MTT and ROE was found (*1*). This suggests that a similar diagnostic decision would have been made on most of the renograms, using either parameter. In addition, Piepsz and Ham claim that MTT is limited by the duration of the acquisition. It is true that in situations in which the maximum transit time is not reached, the parameter calculated will not actually be the MTT. However, it represents the area-to-plateau-height ratio of the retention function, which is still a useful empiric measure of transit through the kidney. Thus, we feel that MTT may still have a useful role to play in clinical assessment of response to a diuretic.

Piepsz and Ham also claim that the presence of a spectrum of transit times will reduce the influence of clearance on ROE compared with the assumption of a single transit time. They correctly point out that our theoretical derivation of a relationship between MTT and ROE was based on the simplifying approximation of there being a single transit time through the kidney. However, given the systematic effect of clearance on ROE over a range of transit times, it seems likely that the relationship between MTT and ROE would be similar in the situation in which there is a spectrum of transit times present. Therefore, we question the claim that the influence of clearance will be reduced in this case. The clinical importance of the effect of clearance is, of course, another issue.

We feel that in the absence of firm evidence to the contrary, our original conclusion that both MTT and ROE have merits and limitations that should be considered in interpretation of renography is still valid. However, 1 caution on the use of MTT is worthy of mention. A recent interdepartment audit carried out in the United Kingdom (*2*) has shown large variation in the values of MTT obtained at different centers for some of the renograms studied. This reflects the lack of standardization of deconvolution software available on different nuclear medicine imaging computer systems. Thus, at present we consider that deconvolution analysis should be used only for routine clinical analysis if careful validation and testing of the software has been carried out. Unfortunately, relatively few centers in the United Kingdom use the ROE parameter, and it was not possible to obtain a measure of its interdepartment variability.

REFERENCES

1. Fleming JS, Kemp PM. A comparison of deconvolution and the Patlak-Rutland plot in renography analysis. *J Nucl Med.* 1999;40:1503–1507.
2. Houston AS, Whalley DR, Skrypniuk JV, et al. UK audit of quantitative parameters obtained from nuclear medicine renography [abstract]. *Nucl Med Comm.* 1999;20:469.

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Attenuation Correction in Dosimetry

TO THE EDITOR: In their article, "Pharmacokinetics and Dosimetry of an α-Particle Emitter Labeled Antibody: ^{213}BiHuM195 (anti-CD33) in Patients with Leukemia," Sgouros et al. (*1*) used the following formula to correct for attenuation of geometric mean of counts obtained over a source organ (Eq. 6 in their article):

$$\text{AttCorr} = e^{[\mu(T_{WB}-T_{ORG})]/2} \times \frac{\mu \times T_{ORG}}{2 \times \sinh(\mu \times T_{ORG}/2)}. \quad \text{Eq. 1}$$

However, as stated in the article, attenuation consists of 2 elements: self-attenuation within the source organ and attenuation by the surrounding tissue. Self-attenuation can be calculated as the mean attenuation over the source volume and, therefore, is equal to:

$$\text{SelfAtt} = \frac{1 - e^{-\mu T_{ORG}}}{\mu \times T_{ORG}}. \quad \text{Eq. 2}$$

Combining this with the attenuation in the overlying tissues, the correct formula to correct for total attenuation is:

$$\text{AttCorr} = e^{[\mu(T_{WB}-T_{ORG})]/2} \times \frac{\mu \times T_{ORG}}{1 - e^{-\mu T_{ORG}}}. \quad \text{Eq. 3}$$

An alternative form of the same formula uses the total attenuation in the body thickness, including the source thickness, instead of the attenuation in the overlying tissues only. This form has to be used if total attenuation is determined by transmission:

$$\text{AttCorr} = e^{(\mu T_{WB})/2} \times \frac{\mu \times T_{ORG}}{2 \times \sinh(\mu \times T_{ORG}/2)}. \quad \text{Eq. 4}$$

As can be seen, Equation 1 underestimates total attenuation by a factor $e^{\mu T_{ORG}/2}$. Organ activities obtained using Equation 1 will be underestimated by the above factor. Even for an organ thickness of only 10 cm, the factor already amounts to more than 1.6 and, therefore, will have considerable impact on the dosimetric results.

REFERENCE

1. Sgouros G, Ballangrud ÅM, Jurcic JG, et al. Pharmacokinetics and dosimetry of an α-particle emitter labeled antibody: ^{213}Bi-HuM195 (anti-CD33) in patients with leukemia. *J Nucl Med.* 1999;40:1935–1946.

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Reply: De Geeter correctly points out that the attenuation equation used in reference 1 has introduced a factor of $\exp(-\mu T_{ORG}/2)$. This term was introduced to better account for photon scatter and is equivalent to an organ-specific reduction in the μ value that reduces the attenuation correction to compensate for scatter. We found this necessary because even after the background correction, unrealistically high values (>10 %ID) were obtained for individual organs.

REFERENCE

1. Sgouros G, Ballangrud ÅM, Humm JL, et al. Pharmacokinetics and dosimetry of an alpha-particle emitter labeled antibody: ^{213}Bi-HuM195 (anti-CD33) in patients with leukemia. *J Nucl Med.* 1999;40:1935–1946.

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