Correction of a Relationship That Assesses Beta-Adrenergic Receptor Concentration with PET and Carbon-11-CGP 12177

TO THE EDITOR: In 1991, we proposed a graphical method that allows estimation of beta-adrenergic receptor concentration using PET and two injections of ^{11}C-CGP 12177 (*1*). Receptor concentration is estimated from an equation (Eq. 18 of ref. 1) using administered ligand doses and two graphical measurements. This relationship assumes that the two labeled ligand doses (D_0^* and D_1^*) can be considered as tracer doses (hypothesis 4 of ref. 1).

The general relationship, without the tracer hypothesis, was also provided for information and without proof (Eq. 19 of ref. 1). We have recently noticed an unfortunate error in the published formula: in the second term of this equation, D_1 in the numerator has to be replaced by $D_1 + D_1^*$. Therefore, the correct equation of the general case is:

$$(B'_{max} - C^*(T_1 - \varepsilon))(1 - e^{\left(\frac{D_1^* + D_1}{D_0^*}\right)} \log\left(\frac{B'_{max} - C_0^*}{B'_{max}}\right) - C_1^* \frac{D_1 + D_1^*}{D_1^*} = 0. \quad \text{Eq. 1}$$

I am grateful to Chris Rhodes of the Hammersmith Hospital for pointing out this error.

The proof of this relationship was not given in Reference 1 because it is similar to the proof of the relationship applied to tracer doses (Eq. 18 of ref. 1): it is only necessary to take into account that the percentage of the occupied receptor sites after the first labeled ligand injection is not negligible, which leads to an equation giving C_0^*, similar to the equation corresponding to C_1 (Eq. 15 of ref. 1).

All the results of the paper are retained since, for the dog studies, the labeled ligand doses can be considered as tracer doses (*1*). However, in human studies (*2*), it may be necessary to use the general case equation in the correct form presented here.

REFERENCES

1. Delforge J, Syrota A, Lancon JP, et al. Cardiac beta-adrenergic receptor density measured in vivo using PET, CGP 12177 and a new graphical method. *J Nucl Med* 1991;32:739–748.
2. Merlet P, Delforge J, Syrota A, et al. Positron emission tomography with ^{11}C-CGP 12177 to assess β-adrenergic receptor concentration in idiopathic dilated cardiomyopathy, *Circulation* 1993;87:1169–1178.

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Assessment of Myocardial Viability by Sestamibi Scintigraphy

TO THE EDITOR: Marzullo et al. investigated the potential of sestamibi scintigraphy at rest to discern viable from nonviable myocardial tissue (*1*). They report two limitations of sestamibi scintigraphy in this respect. First, sestamibi uptake is reduced in a number of segments that have preserved function. Second, dysfunctional myocardial segments may recover normal function after revascularization, even if they show sestamibi uptake below normal limits. These points deserve some comments.

Reduced sestamibi uptake in segments with normal function is hardly a problem, since the question of viability arises only in dysfunctional segments. Besides, sestamibi may provide information on regional function, either by means of first-pass studies or by means of gated SPECT.

We tend to disagree with the authors' point of view that "assessment of residual viability using ^{201}Tl is less dependent from the assessment of regional function since this tracer has been reported to be more accurate in the detection of normal, viable and necrotic areas with more exact threshold values." In the reference cited, no analysis was made of regional wall motion, so this study can hardly serve as a case in point (*2*). Moreover, the data presented in this reference relate to exercise-redistribution-reinjection ^{201}Tl studies and do not allow for a comparison between rest ^{201}Tl and rest ^{99m}Tc-MIBI studies. On the other hand, it was found that irreversible mild (60%–84% of peak activity) or moderate (50%–59% of peak activity) thallium defects often do not fill in at reinjection, whereas most of them are viable by PET criteria. So, if abnormal activity at reinjection is used as the single criterion, it seems that ^{201}Tl, too, would overestimate myocardial scarring.

The whole issue may boil down to the definition of thresholds. Marzullo et al. have set the lower limit of normal perfusion at 2.5 s.d. below normal values for regional relative perfusion, i.e. at 55% of peak activity. Although this may indeed represent the lower limit of normal in a statistical sense, there is no reason to presume that this value would concur with the ideal cutoff between viable and nonviable tissue. Instead, it would be of interest to determine the optimal cutoff level by means of ROC analysis.

In summary, the data presented by Marzullo et al. do not warrant definitive conclusions as to the limitations of sestamibi in assessing viability. Further analysis of the excellent material in this paper, however, could be very helpful indeed to solve some of the problems to which these authors have pointed.

REFERENCES

1. Marzullo P, Sambuceti G, Parodi O. The role of sestamibi scintigraphy in the radioisotopic assessment of myocardial viability. *J Nucl Med* 1992;33:1925–1930.
2. Bonow RO, Dilsizian V, Cuocolo A, Bacharach SL. Identification of viable myocardium in patients with chronic coronary artery disease and left ventricular dysfunction: comparison of thallium scintigraphy with reinjection and PET imaging with ^{18}F-fluorodeoxyglucose. *Circulation* 1991;83:26–37.

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REPLY: Dr. De Geeter states that the detection of abnormal sestamibi uptake in normally contracting segments is not a "hard" problem. In our opinion, the occurrence of a sestamibi defect at