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Correction Factor for Left Ventricular Volume Measurement

TO THE EDITOR: The articles by Melin et al. (*1*) and Verani et al. (*2*) published in the December 1985 issue of the Journal of Nuclear Medicine note a systematic deviation in radionuclide volume determinations.

My impression is that both papers start from a systematic error when assuming a relation between counts at the camera and counts originating at the target volume. To be more specific, in the simple experimental arrangement considered in the referred papers for phantom measurements, with a counting area defined at the camera by a two-dimensional region of interest (ROI) and a preferential direction along an axis perpendicular to the detector and going through the target volume (attenuation direction), we can assume a counting geometry based on very thin (dx) slices of the target volume. Those slices are of areas circumscribed by the ROI, located at positions starting at $x = 0$ up to $x = d$ along the attenuation axis. From $x = 0$ up to the detector interface the distance is L. If the counts originating at slice x are dS (x) and they are recorded at the camera as dC counts, then:

$$dC_{ROI} = K \cdot d\,S\,(x) \cdot \exp(-\mu\,(L - x).$$

Now it is important to note that both members of this relation are in different coordinate systems (the left one at the camera, the right one at the target volume). K is an efficiency counting factor that relates both counting coordinate systems. Then it is obvious that you can not move terms from one side to the other before integrating: this is the systematic error incurred by the referred authors.

To produce simple expressions, let us assume a rectangular or cylindrical geometry, for the target volume and detector, along the attenuation axis; then:

$$d\,S\,(x) = s\,A \exp(-\mu dx)\,dx$$

$$= s\,A\,dx.$$

where we assumed that attenuation through the slice is negligible, s is the radioactive (volumetric) concentration (homogenous) and A the cross-section of the target volume. Then

$$d\,C_{ROI} = K\,s\,A \exp(-\mu\,(L\text{-}x)\,dx.$$

Integrating for an interval of time across the ROI is the camera system and from $x = 0$ up to $x = d$ in the target system, we have

$$C_{ROI} = K\,s\,A\,\frac{1}{\mu} \exp(-\mu L)\,(\exp(\mu d) - 1).$$

Then, for the physical volume of the target (a number independent of both coordinate systems) we can write

$$V_t = Ad$$

$$= R\,C_{ROI}\,\frac{\exp(\mu L)}{\exp(\mu d) - 1},$$

where $R = \mu d/(sK)$. By doing measurements under similar geometrical conditions for a target sample (blood standard), using the same ROI at the camera, we can estimate μ (broad geometry), s and K:

$$C_{ROI}\,(\text{standard}) = K\,[s\,A\,\frac{1}{\mu} \exp$$

$$(-\mu L)\,(\exp(\mu d) - 1)]\ \text{standard}.$$

From this standard measurement, we can estimate R, required for the estimation of Vt.

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REPLY: Dr. Vergara correctly points out that the rigorous relation between ventricular volume and region counts differs from the simplified expression utilized by ourselves and others:

$$V = KC_{ROI}\,e^{\mu T},$$

where K = constant; C_{ROI} = counts in LV region of interest; μ = attenuation coefficient; T = distance of the volume centroid from camera. As previously pointed out by Links et al. (1), the rigorous expression obtained by integrating the extended source volume is:

$$V' = KC_{ROI}\,e^{\mu T}\,\frac{\mu d}{e^{\mu d/2} - e^{\mu d/2}},$$

where, T = distance of volume centroid from camera and d = mean thickness of radioactive volume. By replacing $L = T + d/2$ in expression 5 mentioned by Vergara, this same expression is obtained.

The correction factor imposed by extended source geometry is then:

$$\frac{V'}{V} = \frac{\mu d}{e^{\mu d/2} - e^{-\mu d/2}}.$$

Links et al. (*1*) have pointed out that this correction is quite small for clinical LV volumes. For example, if for simplicity one assumes a spherical ventricle which is not far from correct for a large dilated heart, the mean LV thickness for a 500-ml volume is 6.56 cm and V′/V is 1.02, that is, a 2% error. Thus, even for the largest volumes encountered clinically, this source of "systematic" error is negligible compared with other sources of error in the technique, such as the depth estimation, background subtraction and edge detection.

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Mathematic Models to Assess Platelet Kinetics

TO THE EDITOR: In a recent study Lötter et al. (*1*) have compared 12 different mathematic functions used to calculate the mean platelet survival time. The functions were fitted to experimental data derived from the decay in activity of autologous platelets labelled with indium-111. Quite correctly they indicated that the mathematic functions which they used were simply a device for "smoothing" the experimental data, so that the slope and initial value of the activity curve (after splenic pool equilibration) could be calculated based on all the experimental evidence available. Hence, the mathematic functions are not necessarily derived on the basis of any physiological assumptions.

Unfortunately, the approach of simple curve fitting without physiological basis neglects one of the most powerful attributes of a "true" mathematic model: its predictive nature. By trying to identify the dominant biologic mechanisms which lead to the removal of platelets from the circulation it is possible to construct mathematic equations which describe this consumption process. The solution of these equations then yield mathematic functions which *predict* the decay in activity of isotopically labeled platelets. Such an approach (*2*) suggests that cell aging and random platelet destruction are equally important in hemostatically normal human subjects. The apparently linear decay is due to the size of the exponent in the exponential function which is predicted by the mathematic model. In this case cell aging associated with a linear decay function (*1*) is not the predominant destruction mechanism although a straight line may provide a good fit to the experimental data.

Mean platelet lifetime is an important parameter associated with platelet kinetics. However, the processes which determine this lifetime are as equally important, especially in pathological conditions. The fitting of arbitrary mathematic functions to the experimental data that have not been derived from a biologic base will never allow identification of these processes.

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REPLY: We agree with Dr Trowbridge that the ideal would be to apply mathematic models to identify the biologic mechanisms which cause the removal of platelets from the circulation. His efforts in this regard are, therefore, laudable and should be pursued (*1*).

However, the first priority of our group is the optimum fitting of the experimental data to a platelet survival curve. If this is attained, it should at least enable investigators to standardize the procedure and to adequately interpret and compare data. The present situation where a plethora of mathematic models, which have not been compared or evaluated, are advocated is not satisfactory.

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Utility of Total Body Gallium-67 Scintigraphy in a Patient with AIDS Related Complex

TO THE EDITOR: We report the unusual presentation of a malignant lymphoma in the appendix and cecum of an ARC patient detected by gallium-67 (^{67}Ga) uptake scintigraphy prior to the onset of abdominal symptoms and signs. This case illustrates the utility of including total-body images when a ^{67}Ga scan is requested in the evaluation of pulmonary symptoms.

A 43-yr-old homosexual man, with a 3-year history of diffuse lymphadenopathy, fever, chills, night sweats, and weight loss, was diagnosed as having AIDS related complex (ARC) in October 1984. A gallium scintigraphy study, performed at that time to rule out early *Pneumocystis carinii* pneumonia (PCP), revealed mild focal mediastinal uptake