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.

References

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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.

References

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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