

Iwata's correction in Equation 3 may prove useful, especially for the right ventricle and the tight bolus, when rapid changes in indicator concentration occur.

Finally, Iwata simulated $c(t)$ by the gamma-variate function fitted to end-systolic first-pass points. Alternatively, one may use the cycle-averaged sampled curve, which requires acquiring the data in the list mode, allowing for multiple reformations. Therefore, the curve sampled in accordance with the heart rate is directly proportional to indicator concentration, there is no referring to any particular model, even multimodal bolus is allowed, and recirculation need not be distinguished from the first-pass data.

REFERENCE

1. Iwata K. Alternative method for calculating right ventricular ejection fraction from first-pass time-activity curves. *J Nucl Med* 1988; 29:1990-1997.

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REPLY: I think that the perfect mixing in the indicator within the right ventricle will not occur for the short period between diastole, ed , to the succeeding systole, es , in the case of the tight bolus in the first-pass method and rapid change in the indicator concentration.

I showed how the imperfect mixing influenced the determination of right ventricular ejection fraction, and that the influence could be attenuated by using Equation 3 in the Eterovic letter, when the spatially averaged concentration $c(t)$ in the right ventricle at time t was given. However, in the present stage, $c(t)$ is unknown. I assumed that $c(t)$ was represented by the gamma-variate function fitted to end-systolic first-pass points. Naturally, the possibility exists that other estimations of $c(t)$, better than my own, are found. If any, the error in my correction will be caused from the estimation of $c(t)$. Namely, if the indicator is well mixed with turbulent blood flow in right ventricle during the ejection phases, the modification of my estimation of $c(t)$ or $c(ed)/c(es)$ in Equation 3 will be needed. However, at least in my phantom experiments, ejection fraction after my correction agreed well with the known phantom ejection fraction.

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Splenic Dynamics of Indium-111-Labeled Platelets in Idiopathic Thrombocytopenic Purpura (ITP)

TO THE EDITOR: In a recent paper by Syrjälä et al. (1) on platelet kinetics in idiopathic thrombocytopenic purpura (ITP), the authors concluded that the closed two-compartmental model frequently put forward to represent platelet

exchange between blood and the splenic platelet pool was not valid for short-lived platelets.

Their data, however, do not in any way invalidate the two-compartmental model. It is widely accepted that this model is only considered to be effectively "closed" for platelet life spans measurable in days rather than hours, but that a "runoff" component (due to platelet destruction) becomes increasingly significant as the mean life span becomes shorter. Heat damaged red blood cells, for example, provide a special case for this model in which about half the cells entering the spleen on each pass fail to get out again while the other half transit the spleen with a mean time of ~ 15 min (2,3). Syrjälä et al. (1) make no reference in their paper to this "destruction" rate constant and imply that the two exponentials seen on the splenic uptake curve represent two separate pooling compartments with different equilibrium time courses, similarly to red cells in splenomegaly (4). It would be expected from this implication, i.e., that the true model has three compartments, that the blood-pool curve should also be bi-exponential. The fact that there was no correlation between the exponential components of the blood-pool curve and those of the splenic uptake curve is meaningless.

Fitting a bi-exponential function to splenic platelet uptake curves of a duration of only 40 min is of questionable reliability because of the uncertainty that a plateau value (i.e., equilibrium) has been reached. The illustrated examples in their paper could have been fitted with a single exponential approaching an asymptote, as would be the case for a two-compartmental model with insignificant "runoff." It would be interesting to know if a bi-exponential fit was significantly better than a monoexponential fit in all the cases that they describe as bi-exponential. We took their illustrated example and indeed showed no significant difference between the respective standard errors of monoexponential (plus asymptotic constant) and bi-exponential fits.

Finally, there is no reason why deconvolution analysis applied to the spleen should be invalidated when platelets are being taken up or exchanging at sites in addition to the spleen. It was for the very reason that such additional sites were likely with short lived platelets that we applied a deconvolution analysis in patients with reduced platelet survivals in order to measure mean platelet transit time through the spleen (5,6). Indeed, in patients with very short platelet survivals, we observed splenic retention functions that were monoexponential and approached an asymptote that we interpreted as representing the fraction of incoming platelets that were irreversibly removed in the spleen. The raw splenic uptake curves in such cases have precisely the bi-exponential appearance that Syrjälä et al. (1) described in their paper.

REFERENCES

1. Syrjälä MT, Savolainen F, Nieminen U, Gripenberg J, Liewendahl K, Ikkala E. Splenic dynamics of In-111-labeled platelets in idiopathic thrombocytopenic purpura. *J Nucl Med* 1989; 30:1546-1549.
2. Peters AM, Walport MJ, Elkon KB, et al. The comparative blood clearance kinetics of modified radiolabelled erythrocytes. *Clin Sci* 1984; 66:55-62.
3. Peters AM, Ryan PFJ, Klonizakis I, Elkon KB, Lewis SM, Hughes GRV. Measurement of splenic function in humans using heat damaged autologous red blood cells. *Scand J*