

TABLE 1
Organ Masses Associated with MIRD "S" Factors
Organ Mass (grams)

Organ	MIRD phantom	Δ/S ³ H	Δ/S ¹⁴ C	Δ/S ³² P	Δ/S ³⁶ S	Δ/S ⁴⁵ Ca	Δ/S ⁹⁰ Sr	Δ/S ⁹⁰ Y
Adrenals	15.5	14	14	14	14	14	14	14
Kidneys	284	310	310	310	310	310	310	310
Liver	1,809	1,780	1,800	1,800	1,800	1,800	1,800	1,800
Lungs	999	1,000	1,000	1,000	1,000	1,000	1,000	1,000
Marrow (red)	1,500	1,500	1,600	2,280	1,600	1,700	2,000	2,300
Other tissue (muscle)	28,000	28,000	28,000	28,000	28,000	28,000	28,000	28,000
Ovaries	8.3	11	10.9	11	10.9	11	11	11
Pancreas	60	100	100	100	100	100	100	100
Spleen	174	178	180	180	180	180	180	180
Testes	37	35	35	35	35	35	35	35
Thyroid	20	20	20	20	20	20	20	20
Total body	69,880	70,000	70,000	70,000	70,000	70,000	70,000	70,000

The significance of organ mass disparity can be illustrated with the following example. Suppose a therapeutic radiopharmaceutical demonstrates homogeneous uptake in a 60-g tumor. In order to easily calculate the absorbed dose received by the tumor from self-irradiation due to particulate emissions, it would be tempting to use the "S" factor (source = target) for an organ of similar mass. In this example, one might choose the "S" factor for self-irradiation of the pancreas, since MIRD Pamphlet No. 11 implies that this "S" factor is based on a pancreas mass of 60 g. Since this "S" factor was actually based on a pancreas mass of 100 g, however, the absorbed dose thus calculated will be overestimated by 67%. Similarly, absorbed doses from self-irradiation for masses equivalent to ovaries, adrenals, kidneys, testes, and spleen will be in error by +33%, -10%, +9%, -5%, and +3%, respectively.

In conclusion, the masses of several organs incorporated in MIRD "S" factors differ from those listed for the MIRD phantom. Use of "S" factors for other purposes (e.g., calculation of self-dose to tumor) may result in substantial error if improper organ mass is assumed.

References

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Model to Evaluate Ventricular Insufficiency Utilizing First-Pass Radioventriculogram Component Analysis

TO THE EDITOR: In the recent article published in *Journal of Nuclear Medicine*, Philippe et al. presented a novel methodology to detect and quantify left side valvular regurgitations from first-pass radioangiography data (1).

Their approach deconvolves the left ventricle radiohistogram via pulmonary input in their valvular patients, thus obtaining unit impulse response functions (UIR) that are multimodal, owing to the long transit time of components associated with regurgitant flows. In order to quantify the degree of valve insufficiency Philippe et al. postulate that the areas under the first two UIR components are proportional to total and regurgitant flows, respectively.

I would like (1) to derive the above postulate in the model corresponding to mitral or tricuspid valve insufficiency, and obtain a new formula that enables more accurate regurgitant flow estimates; (2) comment on fitting the curves prior to deconvolution, and (3) discuss the limits of the method in aortic and pulmonary regurgitation.

1. Suppose N_0 indicator particles are injected proximally to an insufficient valve. In each contraction the ventricle ejects EF percent of its diastolic content, with part of EF ejected irreversible forward (FF), and RF as the regurgitant fraction: $EF = FF + RF$. Suppose that complete mixing of indicator with blood occurs in the ventricular cavity prior to each ventricular contraction. According to the number of ventricular passes N_0 particles are discriminated in the following groups:

$$\begin{aligned}
 N_1 &= (FF/EF) \cdot N_0 && 1 \text{ pass, 0 regurgitations} \\
 N_2 &= (RF/EF) \cdot (FF/EF) N_0 && 2 \text{ passes, 1 regurgitation} \\
 N_3 &= (RF/EF)^2 \cdot (FF/EF) N_0 && 3 \text{ passes, 2 regurgitations} \\
 \dots & && \\
 N_k &= (RF/EF)^{k-1} \cdot (FF/EF) N_0 && k \text{ passes, (k-1) regurgitations} \\
 \dots & &&
 \end{aligned} \tag{1}$$

The first part of Eq. (1) is easily comprehended if one imagines all particles initially situated in the ventricle, then $N_1 = FF \cdot$

$(1 + (1 - EF) + (1 - EF)^2 + \dots) \cdot N_o =$ (geometric series) = $(FF/EF) \cdot N_o$. Obviously, if an identical result holds for each part of N_o that enters the ventricle in the succeeding diastoles, then it holds for the sum N_o , too, irrespective of the tempo of indicator ventricular input. The second part of Eq. (1) then follows, by observing that the two-pass particles should first regurgitate one time and are then ejected forward, the same reasoning establishes the other parts of Eq. (1). The sum of all components N_i equals the total number N_o , as expected: $\sum_1 N_k = N_o \cdot (FF/EF) \cdot \sum_1 (RF/EF)^{k-1} = N_o \cdot (FF/EF) \cdot (EF/FF) = N_o$.

If general requirements of stationary and linear indicator ventricular kinetics are satisfied, it can be shown (2) that the area under the indicator ventricle time-number curve (a) equals the product of the total indicator input (N_{tot}) and the mean transit time of indicator particles through ventricular cavity (MTT):

$$a = N_{tot} \cdot MTT. \quad (2)$$

In regurgitation, $N_{tot} = N_o$ since each particle renewal is the new count. In terms of the numbers N_i defined above: $N_{tot} = \sum_2 k \cdot N_k$, where the partition coefficient k stands for the number of particle renewals in the ventricle. Summing the above series: $N_{tot} = (FF/EF) N_o \sum_1 k \cdot (RF/EF)^{k-1} = (FF/EF) \cdot N_o (\sum_0 r \cdot (RF/EF)^r) \cdot (\sum_0 s \cdot (RF/EF)^s) =$ (geometric series product) = $(EF/FF) \cdot N_o N_o$.

Suppose that the renewal of the particle in the ventricle after each systolic regurgitation is not guaranteed in the succeeding diastole, but on average lasts longer. Then the ventricular radiohistogram could be made up of the components that are successively broader as the number of regurgitations increases. In other words, N_{tot} is partitioned in the following components:

$$\begin{aligned} C_1 &= \sum_1 N_i = N_o && \text{0th regurgitation} \\ C_2 &= N_o - N_1 = N_o \cdot (RF/EF) && \text{1st regurgitation} \\ C_3 &= N_o - (N_1 + N_2) = N_o \cdot (RF/EF)^2 && \text{2nd regurgitation} \\ \dots &&& \\ C_k &= N_o - (N_1 + N_2 + \dots + N_{k-1}) = N_o \cdot (RF/EF)^{k-1} && \text{kth regurgitation} \end{aligned} \quad (3)$$

The first component C_1 is made up of the particles that did not regurgitate yet: it thus comprises the single transit particles and all multiple transit particles during their first transit. The component C_2 consists of the particles that regurgitate for the first time: excluding the single transit particles, it comprises all multiple transit particles during their second transit, or first regurgitation. The succeeding components C_i present the number of indicator particles in the ventricle in higher order regurgitations. The sum of all components C_i must equal the total indicator input N_{tot} : $C_i = N_o (RF/EF)^i = N_o (EF/FF) = N_{tot}$.

Denote by a_i the i^{th} component radiohistogram area. Using the number-area theorem expressed in Eq. (2) one finds:

$$a_2/a_1 = RF/EF. \quad (4)$$

This concludes the derivation of the postulate of Philippe et al. (1). Alternatively, one may use the total radiohistogram

area a to arrive at the same goal:

$$(a - a_1)/a = RF/EF. \quad (5)$$

Equation (5) enables a more accurate RF/EF assessment since it obviates the need for part-by-part fit to determine the area a_2 , which is an uncertain procedure at noise levels typical for the first-pass studies (3).

2. As documented by Philippe et al. in Figure 2B of their article (1), the longer transit time components are present even on the original radiohistogram of insufficient ventricles, and are pronounced on UIR obtained by deconvolution. This implies that avoiding recirculation by replacing the original curves with unimodal models prior the deconvolution, as done by Philippe et al. may have unfavorable results. Still, the deconvolution procedure is exactly valid if recirculation data of both LV and pulmonary curves are taken in account.

I reemphasize that an insufficient ventricle radiohistogram can sustain multimodality subject to quantitation only if the renewal of each regurgitant volume is extended in time. This may be the case in mitral or tricuspid insufficiency when, following rapid ventricular emptying the regurgitant particles dissolve in large, weakly contracting atria. This agrees with the successful demonstration of the method of Philippe et al. in the experimental model where the atria is an elastic balloon of 20-40 ml and the ventricle is the pump of 20 ml in end diastole.

3. In aortic and pulmonary insufficiency the regurgitant flow completely returns to the ventricle in the same cycle, where it mixes with the residual systolic volume and it is not possible to distinguish between an insufficient and competent, but slowly emptying ventricle. It should be appreciated in this context that the data sampling frequency used in the study of Phillippe et al. allow monitoring of the changes from cycle to cycle, not within the cycle.

In conclusion, although Phillippe et al. propose their method for the left side; i.e., mitral and aortic regurgitations, the principles of their methodology can also be derived in the model of mitral and tricuspid regurgitation.

References

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REPLY: We thank Eterović for his interest in our work. We appreciate his comments and the elegant mathematic derivation of the regurgitation quantitation formula.

In his comments, RF does not represent the regurgitant fraction but the regurgitant ejection fraction: the usual definition for RF is the ratio of the regurgitant stroke volume by the total stroke volume.

We do not, however, see the purpose of the formula 5