

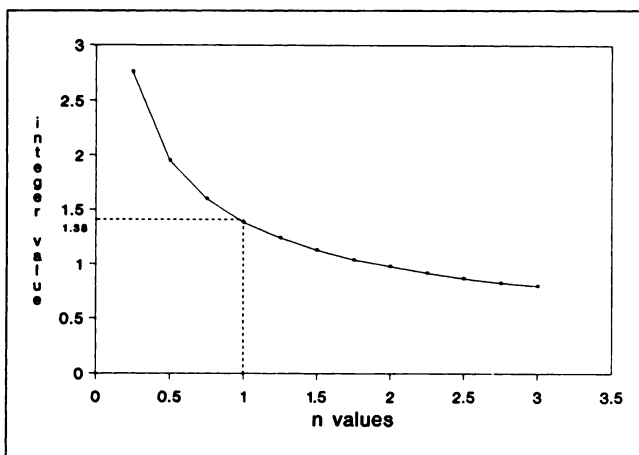
## Count-Based Ratios for Determining Left Ventricular Volume

**TO THE EDITOR:** We read with great interest the recent article by Massardo et al. (1), which described a count ratio based method for the determination of left ventricular volumes. We note, however, that they *assumed* a sphere for the derivation of their volume ( $V_s$ ), which is given by:

$$V_s = 1.38 M^3 R^{3/2}, \quad \text{Eq. 1}$$

where  $M$  is the pixel size (in cm) and  $R$  is the ratio of the total counts in the left ventricle to the maximum pixel count in the left ventricle. It is straightforward to show that Equation 1 above may be generalized for computing the volume ( $V_e$ ) of any ellipsoid using the following equation:

$$V_e = k M^3 R^{3/2}, \quad \text{Eq. 2}$$



**FIGURE 1.** The constant  $k$  in Equation 2 plotted as a function of  $n$ , the ratio of major to minor axis for an ellipsoid.

where  $k$  is a constant that is only dependent on  $n$ , the ratio of the major to minor axis. The behavior of  $k$  as a function of  $n$  is shown in Figure 1. Note that for  $n = 1$  (i.e., a sphere) we obtain Equation 1.

In MUGA studies, a LAO 40° view generally makes possible the determination of  $n$  from the ratio of the long-axis to the short-axis in end-diastole. Use of this empirically-determined ratio  $n$  for *each patient* permits the determination of the “best” constant  $k$  for use in Equation 2. Given the relative ease with which the major/minor axes may be determined in MUGA studies, we obtained left ventricular volumes for 101 consecutive patients using both the Massardo method (Equation 1) and also Equation 2. Each patient undergoing a MUGA study was placed into one of three ventricular “wall motion” categories corresponding to (a) normal ventricular wall motion; (b) regional ventricular wall motion abnormalities; and (c) global ventricular wall motion abnormalities. The resultant end-diastolic ventricular volumes obtained using Equations 1 and 2 are presented in summary form in Table 1. For those patients who had a normal wall motion and a normal ejection fraction, the computed mean end-diastolic left ventricular volumes using both Equations 1 and 2 are summarized in Table 2.

These results show that the use of Equation 2 results in left ventricular volumes that are generally about 20% lower than those obtained using Equation 1. Comparison with “normal” left ventricular volumes in the literature (2) would suggest that use of Equation 1 generates better agreement for males, but Equation 2 generates better agreement for females. Thus, given the well-established fact that the heart more clearly resembles an ellipsoid than a sphere, we would propose that the determination of left ventricular volumes using Equation 2 merits further investigation.

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**TABLE 1**  
Mean Values of End-Diastolic Volumes (EDV) of the Left Ventricle Volumes Obtained for 101 Consecutive Patients Undergoing MUGA Studies at the Health Sciences Centre in Winnipeg

Patient category	Number of cases	Mean EDV (ml) (Equation 1)	Mean EDV (ml) (Equation 2)	Ratio Equation 1/ Equation 2
Normal wall motion	62	115 ± 29	95 ± 24	1.21
Regional wall motion abnormality	35	171 ± 56	144 ± 48	1.19
Global wall motion abnormality	4	210 ± 52	187 ± 55	1.12

**TABLE 2**  
Mean Values of End-Diastolic Volumes (EDV) of the Left Ventricle Volumes Obtained for 39 Consecutive Normal Patients Undergoing MUGA Studies at the Health Sciences Centre in Winnipeg

Patient category	Number of cases	Mean EDV (ml) (Equation 1)	Mean EDV (ml) (Equation 2)	Ratio Equation 1/ Equation 2
Males (normals)	23	118 ± 31	98 ± 22	1.20
Females (normals)	16	104 ± 26	84 ± 21	1.24

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## Maximizing Thallium Stress/Redistribution Scans

**TO THE EDITOR:** In an effort to maximize the utility of the thallium scan, the subject of the reinjection of thallium has become an issue of recent concern (1). The rationale of the reinjection procedure is the observation that a stress/reinjection comparison does a better job of identifying viable myocardium than a stress/redistribution scan.

Some authors propose performing stress/redistribution scans with reinjection of thallium in those patients with a fixed defect on the redistribution scan (2,3). The problem with this technique is that it involves a third set of images and is disruptive of the imaging schedule. Some laboratories prefer a 24-hr delayed imaging session, but this is also disruptive to a busy schedule as well as inconvenient for outpatients.

Some authors (4) propose a reinjection of thallium 20 min before the performance of the redistribution scan. The problem with this approach is the fact that a very tight stenosis of a coronary vessel (the type that causes 'pseudo-fixed' stress-induced defects) can cause defects on rest studies that 'fill-in' over time (5). Thus, some viable regions will still be considered as areas of myocardial scarring.

To avoid these problems we propose the following sequence:

1. Perform a stress thallium scan in the standard manner. Leave the injection line in the patient's arm in place during the scanning procedure.
2. At the end of the stress images (about 35-40 min after the termination of exercise), inject the booster dose of thallium and remove the i.v. line.
3. Obtain a 4-hr redistribution scan later that day.

We find that this procedure gives us the maximum clinical information with a minimum disruption to the department's function. As far as the patient is concerned, it does not even involve having an extra needle stick.

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**REPLY:** We wish to thank Drs. Makler, Schwartz, Shapiro, and Scheff for their concerns in the limited value of current technique of stress-delayed thallium scan for assessing tissue viability (1-3). Many scientists are now pursuing alternative methods for enhanced detection of "redistribution" in the ischemic myocardium. The 24-hr delayed scan (4) or reinjection thallium scan (5-9) have been proven to be useful for identifying additional ischemia which often fails to show redistribution on the routine thallium-201 scan.

The reinjection of thallium immediately after the stress scan seems to work well based on the concept of increasing plasma concentration of thallium, which may redistribute during post-exercise hyperemia (10). However, since majority of ischemic segments already show redistribution on the 3-4-hr delayed scan, it may be difficult to delete the delayed scan. At present, we think that reinjection may not be necessary when the redistribution is already observed on 3-4-hr delayed scan. Such a new technique seems to be valuable only when the routine scan shows a persistent defect, although the third set of images might be disruptive to the imaging schedule. Perhaps, we need more clinical information on the reinjection scan before eliminating the 3-4-hr delayed scan. We do hope that the clinical investigations of Dr. Makler et al. will demonstrate that their procedure will really enhance detection of redistribution in the ischemic myocardium and that these areas will be reversible in cardiac function after restoration of blood flow.

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