not withhold any information from the patient or her physicians, and culture reports, which came from another institution, were not received by us until late in her terminal illness.

We feel qualified to comment on tuberculosis, but do not claim similar expertise for AIDS. One of our internal medicine residents contacted the CDC regarding this patient, and they classified her as AIDS without our knowledge. Drs. West and Tillinghast's points regarding the diagnosis of AIDS, however, do seem reasonable.

Patrick J. Seago, Maj, USAF, MC, FCCP, and R. Phillip Dellinger, Lt Col, USAF, MC, FCCP, Division of Pulmonary Medicine, USAF Medical Center Keesler, Keesler AFB, Mississippi

Estimation of Absolute Ventricular Volume

To the Editor:

We have read with interest the recent article by Thomsen et al entitled "Estimation of Absolute Left Ventricular Volume from Gated Radionuclide Ventriculograms." They have improved upon the method originally proposed by Links et al by using 2D echocardiography to determine the depth to the center of the left ventricle (LV) for attenuation correction and an automated edge detection algorithm for total LV counts. However, they assume the linear attenuation coefficient of 0.15 cm⁻¹ measured in water using narrow beam geometry, which is referred to by Slutsky in his editorial as a "workable" assumption.

Since scatter is inherent in clinical nuclear medicine imaging with window settings of 15-25 percent, an assumption of a narrow beam μ for attenuation correction is incorrect. The narrow beam μ can only be used in conjunction with the buildup factor which corrects for the contribution due to scatter. We have measured the change in counts as a function of depth in tissue-equivalent material for a source of activity approximately the size of the LV. Using the equation I(d)/I(0) = TF = B × e⁻μd where I(0) is the source count rate at depth d, I(d) is the source count rate in air, B is the buildup factor, and d is the source depth, the measured data can be plotted as shown in Figure 1. The slope of the straight line portion of this curve is the linear attenuation coefficient and is 0.13 cm⁻¹.

This agrees with the results of Nickoloff et al who have shown that the attenuation coefficient from the LV center to the chest wall was equal to 0.13 cm⁻¹ based on radiographic CT scans of the thorax. Thus, the assumption that the body habitus between the LV and the chest wall approximates that of water is a good one.

The puzzling aspect of this study is that accurate LV volumes could be obtained by using an automated region of interest since, according to Links, this always resulted in underestimation of true LV volume. Since the average depths were less in the Thomsen et al study (8.2 ± 0.05 cm compared to 10.2 ± 1.6 cm by Links et al) the correction factor for depth attenuation e⁻μd was higher (0.29 ± 0.02 cm⁻¹ compared to 0.23 ± 0.06 cm⁻¹). Therefore, Thomsen et al should also have underestimated volumes since e⁻μd appears in the denominator of the volume equation. One of the explanations for this is probably found in the method used for venous blood counting. The blood was counted in a 7 ml test tube which does not approximate the geometry of the LV and results in self-attenuation which leads to a decreased value for the venous blood counts. This lower number in the denominator of the calculation for LV volume may correct for the smaller LV counts obtained from using a tight, automated LV region of interest.

We agree with Thomsen et al that automated regions of interest should be used since they are more observer-independent and thus more reproducible. We obtained accurate LV volume measurements using automated regions of interest, but calculated volumes based on direct measurements of attenuation in each patient using an esophageal point source. A direct measurement of attenuation avoids the need to make accurate determinations of the LV depths and to assume a value for μ.

In summary, we believe that the major error in the technique of Links et al and Thomsen et al is the assumption of a narrow beam geometry μ. A refinement in the depth measurement will not correct this. It may be that when errors cancel each other in a number of parameters needed to measure LV volume, results can appear to justify the methodology.

Jeffrey A. Siegel, Ph.D., and Alan H. Maurer, M.D., Division of Nuclear Medicine, Department of Diagnostic Imaging, Temple University Hospital, Philadelphia

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To the Editor:

Better methods for correcting left ventricular (LV) counts for tissue scatter and depth attenuation should and probably will soon be developed. However, to assume that a linear attenuation coefficient (μ = 0.13 cm⁻¹) and a buildup factor (1.15) determined in a uniform
"tissue equivalent material" apply to human chests is likely fraught with at least as many assumptions and potential sources of error as empirically selecting a value for $\mu$ equal to the linear attenuation coefficient of water which is then applied to a limited range of depths. The actual value for $\mu$ determined from CT scans of the chest by Nickoloff et al. was 0.128 cm$^{-1}$±0.02 SD. The relationships of patient characteristics, eg, age, sex, height, weight, and body fat content to this range, were not given. It is possible that adult men as we studied with heavier bone and muscle structure than some other categories of patients may cluster above the mean.

One of the major points of emphasis of our article was that indexing the LV region of interest search threshold to the LV phase image resulted in a loose LV edge fit. Therefore, the LV counts which we measured are not at all comparable to those measured by the tight automated edge finders used by Links et al and also by Maurer et al. Therefore, it is not valid to assume that because our mean transmission factor (e$^{-0.0}$) was higher than that of Links et al that we also should have underestimated volumes. Without commenting on whether a horizontally positioned test tube or a petri dish more closely approximates the geometry of the left ventricle, I will say we have found in ten cases that counts emanating from a 7 ml test tube average only 3.69 percent (range: 2.49-4.91 percent) less than those emanating from a petri dish (9 cm in diameter) that contains the same volume of blood (5 ml).

Finally, Siegel and Maurer suggest that they have solved the problem of LV count attenuation by directly measuring attenuation from a point source in the esophagus. Assuming all LV counts arise far posterior from within the esophagus, behind the left ventricle, and at times behind the left atrium as well, will result in an erratically marked overestimation of LV count attenuation. If one assumes the following: 1) the center of the left ventricle is the best place from which to calculate attenuation, 2) $\mu$ of 0.13 cm$^{-1}$ and a buildup factor of 1.18 (as suggested by Siegel and Maurer), and 3) normal adult values for LV internal dimensions and posterior wall thickness determined echocardiographically, then one can estimate that with a heart size at the lower limits of normal, the esophagus is approximately 2.95 cm behind the center of the left ventricle (1.85 cm for one-half the LV internal end diastolic dimension, 0.60 cm for the posterior LV wall thickness, and another 0.50 cm to reach the esophageal lumen). Adding this cumulative distance to any initial measurement from the camera collimator to the center of the left ventricle will result in a 31 percent overestimation of attenuation of LV counts. A similar calculation for an adult heart at the upper limits of normal in size would result in approximately 43 percent overestimation of attenuation. We have also had experience with Medical Data Systems nuclear cardiology software and conclude that the tight automated edge finder must have largely compensated for this error. Therefore, the last sentence of Siegel and Maurer's letter aply applies to their own method of estimating LV volumes.

We made it clear in our article that we felt current methods, including our own, being applied to LV volume determinations provide estimations only. Advantages of our method include excellent interobserver and intraobserver variability, a loose automated LV edge finder, and good correlations with volumes calculated from standard angiographic methods.

James H. Thomsen, M.D., Chief, Cardiology Section, William S. Middleton Memorial VA Hospital and Associate Professor, Department of Medicine, University of Wisconsin-Madison, Madison, WI

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Complex Pulmonary Function Data

To the Editor:

We read with interest the article by Cottrell et al and the accompanying editorial. The article describes the presentation of the results of pulmonary function tests in graphic form. The editorial highlights this and suggests that it would also be useful to present flow/volume loops plotted at absolute lung volume and to allow the comparison of the predicted, the pre- and the postbronchodilator loops.

We have developed a system similar to that proposed in the editorial, which has been working in a routine patient laboratory for over a year. Presented on the lung function report are patient details, the results of pulmonary function tests in numerical form, and flow/volume loops. The results are tabulated to indicate the range predicted for the patient's age, sex and height, the measured

RESPIRATORY FUNCTION REPORT

Figure 1

Comparison of baseline & predicted flow/volume loops

Comparison of baseline & post-bronchodilator flow/volume loops

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