Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

**Disseminated Infection Due to Mycobacterium avium-intracellulare Complex**

To the Editor:

The letter by Savage and Dellinger entitled "Disseminated tuberculosis caused by M intracellulare" (Chest 1982; 82:900-01) describes a woman whose care in her terminal illness was directed by us. We would like to clarify their report and comment on the diagnosis of the acquired immune deficiency syndrome (AIDS) which was retrospectively made by others. Omitted from their letter was that on January 1, 1982, three days prior to discharge from Keesler Air Force Base, she was started on therapy with prednisone, 40 mg daily. This dose of prednisone was continued for six weeks, accompanied by the use of isoniazid and rifampin. When the dose was reduced to 30 mg daily, about February 26, 1982, fever recurred. She tapered the prednisone. Because of severe febrile and systemic symptoms, she sought care and was admitted to Schumpert Hospital (Shreveport, LA) on March 22, 1982 by one of us (AJT), two days after stopping prednisone. The positive culture for Mycobacterium intracellulare from the transbronchial lung biopsy and bronchial washings done in December, 1981 at Keesler Air Force Base Hospital was not known to the patient until after her admission to Schumpert Hospital.

During her final admission, bone marrow and liver biopsies showed granulomas with definite acid-fast bacilli. Some of the bone marrow granulomas were caseous. These specimens, as well as lung tissue cultured post-mortem, grew M avium-intracellulare complex. Her hospital course was inexorably downhill, despite therapy with isoniazid, rifampin, amikacin, cycloserine, pyrazinamide, ethambutol, and ethionamide, instituted shortly after admission. In addition, she received indomethacin, levamisole, and lymphocyte-rich leukocyte transfusions, most from donors with stable or resolved M avium-intracellulare complex infections, in an attempt to improve her cell-mediated immunity. None of these measures had a clinically significant effect upon her course and she died April 7, 1982. The post-mortem examination showed acid-fast bacilli in the lungs, lymph nodes, liver, and bone marrow. Dr. Albert Hand, pathologist, reported the granulomatous disease to be in a pattern of largely nonreactive, rapidly disseminated mycobacteriosis.

It is of note that there was no history of either bisexual behavior or drug abuse in her husband or the patient, as has been described for some other cases of disseminated M avium-intracellulare complex.1 This patient's illness might have ended fatally without therapy with steroids, but there is no doubt that the steroids are implicated in the progressive dissemination of her disease. She represents case 2543 of AIDS, registered at the Center for Disease Control (Atlanta, GA). While this case meets the general definition of a case of AIDS, it causes us to reflect that a fatal infection in a person who is otherwise normal with regard to cellular immunity should not be adequate for the diagnosis of AIDS, and that using this definition will culd all cases of certain serious or disseminated infections of a variety of causes as AIDS, not leaving any without the AIDS designation. The result of this practice is predictable: the number of cases of AIDS will increase until all such cases are included, at which point the rise in the number of new cases will plateau, thus terminating the portion of the "epidemic" created in part by the case definition. The other result will be a loss of regard for the natural course of certain disseminated infections, the diagnosis of which automatically equates with AIDS. Accepting the present case as one of AIDS seriously challenges the conception of the case definition.

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**REFERENCES**


2 CDC. Update on acquired immune deficiency syndrome (AIDS)—United States. MMWR 1982; 31:507-14

To the Editor:

Drs. West and Tillinghast make several points that deserve response. It is often difficult to understand reasons for therapeutic decisions without all the facts leading to those decisions. For the sake of brevity in our brief communication, many significant aspects of the patient's care were omitted. She was discharged 4 January 1982 after an extensive multisystem evaluation. Transbronchoscopic and open lung biopsies showed marked acute inflammation compatible with hypersensitivity pneumonitis. We never had any bacteriologic evidence of tuberculosis from these biopsies, sputum or other materials. A therapeutic trial of steroids was initiated. However, because of her positive PPD, radiographic findings and one noncaseating granuloma demonstrated on bone marrow biopsy, we felt obligated to cover for tuberculosis with isoniazid and rifampin, at least until results of all cultures were negative. Even if cultures were negative, as long as significant steroid dosages were given, we recommended continuing isoniazid. There was never any reason to suspect disease caused by atypical mycobacteria. The suggestion that steroid therapy was continued despite disseminated disease with Mycobacterium intracellulare is inaccurate, since culture results were not available at the time of her discharge. We agree that a decision to add steroids to an antituberculosis regimen for disseminated tuberculosis would be controversial.

All culture material for tuberculosis is sent to our state laboratory for identification. We were notified of positive identification of M intracellulare in mid-March 1982, and our patient's referring military physician was contacted. Subsequently, when we learned from that physician that she was again hospitalized, we contacted her civilian physicians who informed us of her terminal stage of illness. We did
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.

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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, the assumption of a narrow beam \( \mu \) for attenuation correction is incorrect. The narrow beam \( \mu \) 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 \( I/dV = TF = B \times e^{-2d} \) where \( I(d) \) is the source count rate at depth \( d \), \( I_s \) 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\(^{10} \) 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 \( e^{-2d} \) 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 \( e^{-2d} \) 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 \( \mu \).

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 \( \mu \). 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.

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3 Slutsky RA. On the analysis of left ventricular volume from gated radionuclide ventriculograms. Chest 1983; 84:2-3
5 Wu RK, Siegel JA. Absolute quantitation of radioactivity using the build up factor. Med Phys 1983 (in press)

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 \( (\mu = 0.13 \text{ cm}^{-1}) \) and a buildup factor (1.18) determined in a uniform

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Figure 1. Transmission factor, TF, as a function of depth obtained in a phantom of tissue-equivalent material. The equation of the straight line portion of the curve (which occurs after a build-up depth of 3.6 cm), the correlation coefficient (\( r \)), and the standard error of the estimate (SEE) are shown.