Cardiac Sarcoidosis

To the Editor:

In their report on cardiac sarcoidosis, Smedema et al (July 2005)1 remarkably conclude that patients without cardiac symptoms do not require more sensitive studies to detect heart disease. This report, while of interest, is limited by a patient follow-up that varied widely from 3 to 54 months. Since this study does not well characterize the natural history of asymptomatic cardiac sarcoidosis and, as related in the accompanying editorial by Judson,2 sudden death is a tragic occurrence in seemingly healthy young patients, the conclusions of Smedema et al are not reassuring. The authors list “heart failure” as the symptom of cardiac involvement in 14 of their 19 patients, and only 7 patients are listed as having “probable arrhythmias.” Thus, it appears that the cause of morbidity and mortality from cardiac sarcoidosis has shifted from death due to fatal malignant arrhythmias to death due to progressive loss of left ventricular function. Presumably, the loss of ventricular function and arrhythmias are due to granulomatous inflammation or myocardial scarring. We think that the early recognition of active myocardial sarcoidosis such as with MRI, as found in 12 of the 82 patients who were deemed not to have cardiac sarcoidosis, or utilizing other sensitive tests such as positron emission tomography scans might have alert the physicians to the presence of treatable myocardial disease.

The awareness of myocardial involvement allows anticipatory monitoring. Such monitoring permits intervention prior to the evolution of clinical symptoms. Smedema et al1 have reaffirmed the conclusions of prior studies that have emphasized the poor prognosis of patients with clinically apparent cardiac sarcoidosis. They also have reaffirmed the fact that the severity of lung disease offers ambiguous clues with more advanced radiographic stage occurring in their clinical cardiac group, but worse pulmonary function in the noncardiac group. They have not addressed the more pressing problem of how to prevent this tragic complication. There is little comfort in knowing that your patient has not yet developed life-threatening cardiac symptoms.

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Importance of Appropriately Adjusting Diffusing Capacity of the Lung for Carbon Monoxide and Diffusing Capacity of the Lung for Carbon Monoxide/Alveolar Volume Ratio for Lung Volume

To the Editor:

I thank Traynor et al (May 2005)1 for providing data showing the importance of appropriately adjusting the diffusing capacity of the lung for carbon monoxide (DLCO) and DLCO/alveolar volume (VA) ratio for measured lung volume.

DLCO falls and DLCO/VA ratio increases with smaller VA,2,3 so an unadjusted DLCO underestimates diffusion and an unadjusted DLCO/VA markedly overestimates diffusion at low lung volumes. A method for adjusting DLCO and DLCO/VA ratio for lung volume, which fits the theoretical and empiric data well,2 multiplies predicted DLCO by (0.38 + 0.42*VAfr), and predicted DLCO/VA ratio by (0.42 + 0.53*VAfr), where VAfr is the measured VA/predicted VA ratio. Daco (ie, DLCO adjusted for VA) and KACO (DLCO/VA or KCO adjusted for VA) have the same percentage of the predicted value.

At baseline, the patients of Traynor et al1 with refractory systemic lupus erythematosus had a DLCO of 54% predicted (12% were >80% predicted), a DLCO/VA ratio of 84% predicted (53% were >80% predicted), and Daco and KACO values of 62% predicted (12% were >80% predicted). Two patients increased DLCO by 27% and 1%, but decreased DLCO/VA ratio by 41% and 54% during sustained remissions, with Daco increasing by 15% and 10%. Very different interpretations of lung function occur using percent predicted values for DLCO or DLCO/VA ratio. Daco and KACO provide better assessments of diffusion capacity.

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2 Judson MA. Cardiac sarcoidosis: there is no instant replay [editorial]. Chest 2005;128:3–6