mentioned regimen in the absence of ethambutol. The details of the sequential changes are shown in Table 1. Thus, the circumstantial evidences strongly favored ethambutol as the incriminated agent for the hematologic disturbance. The main adverse reactions of ethambutol are toxicities to the eyes and peripheral nerves.2 The only reported hematologic adverse reaction for this drug is thrombocytopenia.3,4 Neutropenia and eosinophilia have not been, to the best of our knowledge, hitherto reported. The divergent influence of ethambutol on the neutrophil count and eosinophil count is intriguing. This has tempted us to conjecture that the hypoplasia of the neutrophils and its progenitors in the bone marrow in this patient could result from an immunologic or allergic mechanism with eosinophilia being its surrogate marker.

Chi Fong Wong, MBBS, FCCP, and Wing Wai Yew, MBBS, FCCP, Tuberculosis & Chest Unit, Grantham Hospital, Hong Kong

REFERENCES
1 Holdiness MR. A review of blood dyscrasias induced by the antituberculosis drugs. Tuber. 1987; 68:301-09
2 Girling DJ. Adverse Effects of Antituberculosis Drugs. Drugs 1982; 23:56-74
3 Prasad R, Mukerji PK. Ethambutol-induced thrombocytopenia. Tuber. 1989; 70:211-12

Mediastinal Sarcoidosis

To the Editor:

I have read with great interest the case report in the May 1994 issue of Chest by Cohen et al1 about a patient with adenopathy due to sarcoidosis causing ventilation-perfusion mismatch simulating pulmonary embolism. We have recently reported an almost identical case, which appeared as an abstract in Chest.2 This case has since been published in the Journal of Nuclear Medicine.5 This type of abnormality in sarcoidosis is probably fairly frequent and not often detected because patients with sarcoidosis rarely present with chest pain and therefore are not often referred for perfusion scanning.

The authors' other observation of pulmonary sarcoidosis developing suddenly is also very common. I have seen several such cases. The only reason this is not often appreciated is that, again, patients rarely have x-ray films done just before the onset of their illness.

Charlotte Colp, MD, FCCP, Beth Israel Medical Center, New York

PROBABLY FAIRLY FREQUENT

REFERENCES
1 Cohen LA, Murphy WD, Kelling JS. Mediastinal sarcoidosis presenting as ventilation-perfusion mismatch. Chest 1994; 105:1576-77

More Study Needed for Mechanisms Underlying the $^{67}$Ga-Pulmonary Leak Index

To the Editor:

We read with interest the report in the December 1993 issue of Chest by Rajmakers et al1 using transferrin, labeled with $^{65}$Ga, as a marker of pulmonary vascular permeability after cardiopulmonary bypass surgery. Pulmonary permeability in this setting has been attributed to neutrophil sequestration in pulmonary capillaries after activation of complement, and subsequent endothelial damage from neutrophil-derived proteolytic enzymes and oxygen radicals. The use of transferrin-bound gallium to indicate pulmonary permeability, in this setting, poses methodologic problems, which are not fully addressed by the authors. First, $^{67}$Ga will dissociate from transferrin in the presence of binding proteins with a higher affinity for gallium, such as lactoferrin. The secondary granules of neutrophils are rich in lactoferrin, and disruption of neutrophils in inflammation is known to cause leakage of this protein. Lactoferrin is also exocytosed by activated neutrophils. The complicated kinetics of $^{65}$Ga-transfer-