we believe this is due largely to limitations in high-frequency response caused by our data-collection frequency of 2.5 frames/sec (multipeak boluses contain a greater proportion of high frequencies than prolonged boluses). Faster sampling rates may improve our ability to deconvolute a fragmented bolus.

In our clinical trials of deconvolution analysis we have been purposely injecting a slow, smooth bolus. This minimizes the chances for a fragmented, multipeak injection and maximizes the ability of our current deconvolution algorithm to provide accurate shunt quantitation. Our initial clinical experience with the algorithm in both adult and pediatric patients has been excellent, and we hope to report it in the near future.

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Regarding Venography and Lung Scanning

After I read this superb article (*1*), I remained frustrated with regard to several points:

1. Of 19 patients with abnormal venograms and normal perfusion lung scans, eight were said to have had pulmonary embolism. In my experience no patients with pulmonary embolism have had a normal perfusion lung scan when performed within 24 hr of the occurence of embolism. Nor am I aware of any reports in the literature describing such a case. The time of study after onset of suspected pulmonary embolism was not stated in the article.

2. The article states that "47 of 102 patients were serially studied on two to four occasions," but there was no discussion of those repeat studies. When the perfusion lung scan is delayed 24 to 48 hr after occurence, if the lesion is small, all evidence of pulmonary embolism may be gone. (I have seen only one such case; however, I rarely have the opportunity to repeat lung scans after one day.) The authors report normal venograms with abnormal perfusion lung scans in five patients with pulmonary embolism. Although it certainly is possible that embolism originated at a site not amenable to diagnosis by lower-extremity venography, evidence of thrombosis and/or phlebitis may have disappeared if the study were delayed too long following onset of the pulmonary lesion. Here again it is important to know the timing of the study in relation to the clinical situation, and both results and timing of any follow-up studies that may have been obtained. I have had the opportunity to do follow-up venograms on only two patients with definite evidence for thrombosis-phlebitis at initial examination. Both had perfusion lung scans that showed high probability for pulmonary embolism. Follow-up radionuclide venography and perfusion lung scanning was carried out on one patient after 6 days and on the other after 7 days. Both showed partial regression of abnormality in lung scan but entirely normal venogram. I am sure the authors can shed further light on this problem, which deserves systematic evaluation. I am confident that the eight false-normal lung scans will be found to have been done at least 24 hr following onset of the clinical problem, and I believe delay in performing venography probably accounts for many of the false normal results described in this paper (*1*) and in previous reports.

3. "Emission venograms were interpreted as abnormal if one of the following criteria were met: (a) venous occlusion with or without collaterals; (b) intraluminal defects in ileofemoral segment with stasis distal to the partially occluded segment." How many abnormal venograms met criteria (a) only, (b) only, or both? Venous occlusion may represent permanent residual of old thrombophlebitis. Since the criteria for final diagnosis of pulmonary embolism were primarily clinical, I wonder if any of the "false-normal" lung scans were actually correct with diagnosis inferred from venogram abnormality representing sequalae of previous disease.

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Reply

In Table 1 of our article (*1*), perfusion studies with high probability of pulmonary embolus were considered positive, whereas normal nondiagnostic, or low probability perfusion lung scans were handled as negative. The note at the bottom of this table ("+ lung interpreted as low probability for pulmonary embolus") applies to negative lung scans in that column.

We agree with Dr. Wolfstein that pulmonary embolus is highly unlikely if the perfusion study is normal. None of the patients considered to have pulmonary embolus in this study had normal perfusion.

We emphasize the importance of follow-up studies in patients who have evidence of thromboembolism (*2*), and of a simultaneous repeat emission venogram. The latter increases the diagnostic accuracy of acute venous thrombosis, since it is invariably associated with evolutionary changes, whereas chronic venous disease without superimposed acute thrombi remains unchanged.

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Scintigraphic Findings in Angioimmunoblastic Lymphadenopathy

Angioimmunoblastic lymphadenopathy (AIL) is a lymphoproliferative syndrome first described by Lukes and Tindle in 1973 as immunoblastic lymphadenopathy (*1*). The syndrome is characterized by fevers, sweats, weight loss, rash, pruritus, lymphadenopathy, hepatosplenomegaly, and hypergammaglobulinemia. The clinical course is usually rapid and fatal (*2*). The lymph-node architecture is distorted by infiltration with immunoblasts and a peculiar proliferation of aborizing postcapillary venules (*4*). The disorder usually appears between the third and fifth decades and is slightly more common in males. Its initial clinical presentation often suggests malignant lymphoma, and histologically it resembles Hodgkin's disease (*1*). Radiographic findings in AIL had been reported (*2,5*), but its scintigraphic characteristics have not been described in detail. We discuss here the scintigraphic findings in two patients with AIL. The first case had a malignant course; the