Treating Pulmonary Emboli

To the Editor:

Doctor Leeper and associates are to be congratulated on their paper on the treatment of massive pulmonary emboli with low-dose streptokinase.1 These findings will probably be applied to patients in the future as an alternative to standard modes of treatment for pulmonary embolus, especially massive emboli. However, it should be pointed out that such treatment is not universally effective. Additionally, since the perfusion score of the contralateral, noninfused lung did not change significantly, one wonders about the efficacy of treatment of bilateral pulmonary emboli in such patients. Additionally, the concern over repeat pulmonary emboli from deep venous sources is present.

In 1983, I reported a patient who had massive pulmonary emboli of the right main pulmonary artery. A Swan-Ganz catheter was inserted next to the clot and standard streptokinase infusion was administered. Treatment was begun within 24 h and a standard dose of 250,000 units of streptokinase was infused over two hours, with an additional 100,000 per hour per hour over the subsequent 70 h. Despite the use of streptokinase directly on a (presumably) new clot, treatment was totally without effect. The patient died two hours after streptokinase infusion was terminated. Autopsy results indicated a fibrin clot with some suggestion of superficial lysis.4

If treatment is begun according to the protocol of Doctor Leeper and associates, clinicians should be advised that this may not be universally efficacious. Thus, alternative treatment protocols may be employed if treatment failure is suspected.

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Adenosine Deaminase in Bronchoalveolar Lavage Fluid

To the Editor:

We congratulate Dr. J. Fontan Bueso and colleagues for publishing the interesting article, "Diagnostic value of simultaneous determination of pleural deaminase and pleural lysozyme/serum lysozyme ratio in pleural effusions" in the journal Chest (1988; 93:303-07).1 Stimulated by their encouraging report, we would like to bring to the attention of international readers the results of our recent work on adenosine deaminase activity in bronchoalveolar lavage fluid.2 ADA determination in 59 pulmonary tuberculosis patients and 25 pulmonary carcinoma patients showed higher ADA levels in BALF of tuberculous subjects (3.96 ± 3.75 units/L) compared to those from lung carcinoma patients (0.47 ± 1.14 units/L, p<0.0001); whereas ADA levels in the blood from both patient groups (34.83 ± 14.76 units/L for tuberculosis and 28.12 ± 15.58 units/L for carcinoma) were not different statistically from each other (p = 0.55), although they were significantly higher than those of healthy subjects (12.78 ± 4.28 units/L, n = 225, p<0.0001).

Worthy of note is that, while determination of ADA in BALF has an unarguable advantage in the diagnosis of patients with or without pleural effusion, the procedure of obtaining BALF is considerably more complicated and less accessible to medical practice than that of pleurocentesis. Thus its clinical application is strictly reserved for cases undiagnosed by other means.

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Cardiac Findings in AIDS

To the Editor:

Raffanti et al1 have made some interesting observations on a relatively new topic, cardiac findings in patients with AIDS. But I think the significance of Raffanti’s observations is not straightforward and needs comment.

Raffanti and colleagues have demonstrated a high prevalence of ECG and cardiac contractile abnormalities in AIDS. Similar observations have been made in echocardiographic findings.2 But most of these abnormalities are probably incidental in that they do not distinguish patients with from those without clinically important heart disease (defined as heart disease that is frequently due to a treatable cardiac pathogen and, especially if untreated, results in decreased survival compared to AIDS patients without heart disease). In my experience, clinically important heart disease in an AIDS patient can be strongly suspected by the recent onset of cardiac symptoms and will often be present in patients with radiologic cardiomegaly. Additionally, although decreased systolic wall motion can be striking in some AIDS patients with congestive heart failure,3 in my patients diminished contractility without heart failure or other cardiac symptoms has not been associated with clinically important heart disease. Examined in this way, none of Raffanti’s patients had clinically important heart disease. Therefore, it is not surprising that the investigators were unable to find correlations between ejection fraction and disease class or survival. Also, although all four of Raffanti’s patients with normal ECG results had a normal ejection fraction, most of the ECG abnormalities noted by Raffanti were minor and nonspecific. In light of these observations and considering the costs involved, it seems ill-advised to recommend use of any ECG abnormality in an AIDS patient as the trigger for ordering more cardiac tests. Rather, it would seem more sensible to develop a heightened awareness for the possibility that dyspnea in an AIDS patient may be cardiogenic, and to order an echocardiogram or nuclear angiogram when an AIDS patient is found to have cardiac symptoms or even mild radiographic cardiomegaly.

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