Free-floating Thrombi: Effects of Venography on Choice of Therapy for Recurrent Pulmonary Embolism

To the Editor:

Akers et al.1 used venography to look for thrombosis in the leg of a patient with suspected pulmonary embolism. The venogram of the right leg showed extensive thrombosis of the iliofemoral system with a free-floating (nonadherent) thrombus. Since large, free-floating thrombi might embolize despite conventional anticoagulant therapy,2 and since thrombotic therapy can cause fatal pulmonary embolism in these patients,3 they treated the patient successfully by placing a Greenfield filter percutaneously to interrupt the inferior vena cava. In this report we describe a similar case treated in the same way.

A 54-year-old man was admitted to our hospital (October, 1987) for pleuritic chest pain and acute dyspnea. The patient had been hypertensive for ten years and was being treated with nifedipine. Ten days before, he had had surgery for left varioccele and right inguinal hernia. Physical examination revealed decreased breathing sounds at the base of the right lung with pleural friction rubs. The lower extremities were normal. No parenchymal infiltrates were seen on an x-ray film. Arterial blood gas values on room air were: pH 7.43, PaO2 80 mm Hg, and PaCO2 36.6 mm Hg. A radionuclide perfusion scan revealed multiple subsegmental defects in both lungs. Because of suspicion of acute pulmonary embolism, heparin therapy was started (30,000 to 40,000 Units/h) to keep PTT at 1.5 to 2.5 times baseline PTT. Despite this therapy, the patient had two new bouts of pleuritic chest pain and acute dyspnea in the following days. Chest x-ray film showed parenchymal infiltrates, and a radionuclide perfusion scan revealed new subsegmental defects. Two-dimensional echocardiography showed no intracavitary thrombi.

With contrast iliofemoral venography, a free-floating thrombus in the inferior vena cava, extending cranially 12 cm from the conjunction of iliac veins, was seen. Because of the recurrence of pulmonary embolization in spite of appropriate anticoagulant therapy, a Greenfield filter was placed percutaneously via the right internal jugular vein; oral anticoagulant therapy was started and heparin discontinued. Routine follow-up for four months did not reveal any symptomatic evidence of recurrent pulmonary embolism.

Pre-clotting Techniques in Thoracic Surgery

To the Editor:

I read with interest the article by Hoover et al. on "Left-to-Right Shunts in Control of Bleeding Following Surgery for Aneurysms of the Ascending Aorta" (Chest 1987; 91:844-49). While the authors described the type of material used for creating their left-to-right

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Correction

To the Editor:

It has recently come to my attention that there is an error in our paper in Chest 1987; 92:310-12 entitled "Effect of Inorganic Phosphate in Hypoxic Chronic Obstructive Lung Disease Patients during Exercise". The error is on page 310 under "Patients and Methods", paragraph 2 which begins "From 0 to 3 hours . . . " The second line of this paragraph should read: phosphate (0.228 mmol/kg/h) instead of 2.28. I am sorry that we missed this in the galley proofs.

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shunts, there was no mention of the type of graft material or the technique of pre-clotting used for the ascending aortic graft itself. Certainly, massive bleeding has been described frequently in the early post-bypass period following ascending aortic aneurysm resection in many series and it has been seen at times by virtually all of us who have occasion to perform these procedures. Undoubtedly, the source of this bleeding is occasionally from proximal or distal anastomoses or from the coronary orifices or vein grafts. However, it is usually the case that the majority of early post-bypass bleeding is not from the suture lines but rather through the interstices of the graft material itself. In the latter case, bleeding can be virtually eliminated by effective pre-clotting of the ascending aortic graft using a technique in which the graft material is soaked in albumin and then autoclaved (J Thorac Cardiovasc Surg 1966; 92:691-705). This technique is entirely applicable to composite valve-graft conduits which use a mechanical valve.

I have personally found this pre-clotting technique to be so effective that virtually no bleeding has occurred after ascending aortic aneurysm replacement, even in cases where the aortic graft was not wrapped by aneurysm wall at all. I doubt very much that the authors used this pre-clotting technique for the patients described in their series. Had they done so, I cannot imagine that they would have had so many cases in which massive bleeding required the creation of left-to-right shunts. If meticulous care is taken with suture placement and if albumin/autoclave pre-clotting is used, it should be a rare case in which there is sufficient bleeding to require the shunt technique which the authors describe.

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To the Editor:

Dr. Berger brings up an extremely important point regarding the pre-clotting techniques used in ascending aortic grafting, which is quite pertinent to surgeons performing these procedures. Our failure to mention our pre-clotting technique in the methods portion of our paper was an inadvertent omission. Dr. Griep is a recognized expert in the field of thoracic aneurysm surgery and insures that our group incorporates state-of-the-art techniques and concepts in the conduct of these procedures. In fact, we have used the pre-clotting technique of soaking low-porosity woven grafts with 50 ml of 25 percent albumin solution followed by autoclave sealing at 270°F for five minutes as described by Thurer et al (Circulation 1992; suppl 1; 143-46) in all of the patients in this report. Five of our nine patients had acute dissections with the usual friable vascular tissue. However, we offer this as an observation only and not an excuse or explanation. Finally, this particular subset of patients were treated well into a long and successful series of these procedures, all of which are attended by the senior author, so there was no element of a "learning curve" involved.

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Eosinophilic Alveolitis

To the Editor:

We read with interest the article by Davis et al on eosinophilic alveolitis. The authors described two immunosuppressed patients with acute respiratory failure and bilateral, interstitial and alveolar infiltrates initially assumed to be secondary to lung infection. Infectious etiologies were excluded in both, but bronchoalveolar lavage (BAL) fluid analysis showed eosinophilia in each case. Both patients responded favorably to glucocorticoid therapy. The authors concluded that eosinophilic alveolitis in this setting is a useful clinical marker for noninfectious, steroid-responsive lung injury.

This conclusion is not supported by our BAL findings in patients with AIDS and P carinii pneumonia. We reviewed BAL cell populations in 39 patients with AIDS and P carinii infection. BAL eosinophilia (>5 percent) was found in 16 (41 percent) patients. The latter group had a mean eosinophil count of 6.65 percent (range 2 to 16 percent). No patient had significant blood eosinophilia or apparent allergic disease. Although the role of the eosinophil in P carinii pneumonia is unclear, we observed previously that the presence of a neutrophil-eosinophil alveolitis in AIDS patients with this infection is associated with greater abnormalities of gas exchange and a more severe clinical course.

Thus, BAL eosinophilia is not necessarily a marker of a noninfectious lung process in patients at risk for AIDS and opportunistic infection. With respect to the effect of glucocorticoid therapy, it is possible, although unproven, that severe P carinii infection may respond favorably to adjunctive steroid therapy, particularly in patients with an associated BAL eosinophilia.

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2 Smith RL, El-Sadr WM, Lewis ML. Correlation of bronchoalveolar lavage cell populations with clinical severity of Pneumocystis carinii pneumonia. Chest 1988; 93:60-64

To the Editor:

We thank Drs. Smith and Berkowitz for their interest in our article. It seems that we and they are describing BAL eosinophilia in totally different patient groups. Our two patients were immunocompromised. However, exclusion of infectious causes of lung disease served as a starting point for our paper. When we serendipitously noted BAL eosinophilia, we concluded that the eosinophilia was probably occurring in the setting of noninfectious lung injury. We have since had phone calls from several centers noting the same syndrome in normal individuals, and we have now studied four additional such patients, two of whom required mechanical ventilation. A well-documented case of a previously healthy patient with similar findings was recently reported from Denver.1 In all of the above cases, BAL eosinophilia has been 40 percent or more, far greater than reported in AIDS patients. I agree with Smith and Berkowitz that AIDS patients with PCP constitute a completely different patient population and that BAL eosinophilia and eosinophil-neutrophil interactions have different implications in these patients.