accessible obstructions of more than 50% of the pulmonary vascular bed.³

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Reply to the Editor:

We appreciate the attention that Dr Riedel has paid to our article. He brings up several points that deserve clarification.

First, selection criteria for pulmonary endarterectomy are multiple, and determination of candidacy for pulmonary endarterectomy is based on a composite of factors. Our selection criteria for surgery include but are not limited to just New York Heart Association (NYHA) class III and IV patients. We offer pulmonary endarterectomy to patients with NYHA class I and II disease, although only 1 in our series was in NYHA class I. The long-term survival of patients with untreated chronic thromboembolic pulmonary hypertension, irrespective of symptoms, is poor.¹ In our hands, the mortality associated with pulmonary endarterectomy (4.5%) is much less than the published mortality associated with untreated thromboembolic pulmonary hypertension (32.6% in 1 series with an average follow-up of 18.7 months²). In our experience, pulmonary endarterectomy is not only associated with im-

proved long-term survival but also a better quality of life for patients who previously suffered from this disease.³ Furthermore, we have become increasingly aware that the remaining open pulmonary vascular bed after thromboembolic occlusion can become irreparably damaged by prolonged exposure to the flow and pressure changes occasioned by vascular occlusion in other areas.⁴ For these reasons, we believe that pulmonary endarterectomy should be offered to all patients with thromboembolic pulmonary hypertension, including those with less clinical symptomatology.

Second, patients in group 1 of our study were comprised of individuals with chronic pulmonary hypertension who had fibrotic thromboembolic obstruction of their main and/or lobar pulmonary arteries as well as fresh clot that occurred either as a result of recurrent pulmonary emboli or as a secondary consequence of this obstructive process (see Figure 1). No amount of anticoagulation or thrombolytic therapy would have altered the distal fibrotic obstruction that was responsible for the patients' pulmonary hypertension. Incidentally, proximal clot propagation occurs even in the setting of therapeutic anticoagulation, as more than 80% of the group 1 patients referred to us for operation had been on coumadin for a period of longer than 3 months. We do not treat patients with acute pulmonary embolism with pulmonary endarterectomy; this is a different disease.

Third, although we have performed more than 1600 pulmonary endarterectomies in the last 10 years at the University of California, San Diego, we still do not have a completely foolproof method of determining location of pulmonary vascular obstructive disease in all patients. We have found that pulmonary angiography is the imaging modality that provides the best preoperative assessment of thromboembolic obstruction. However, using a combination of assessments, including angiography, angioscopy, ventilation-perfusion scanning, spiral computed tomography (CT) scanning, and 3-dimensional CT angiography, there are still a few patients in whom disease location is not diagnosed with certainty prior to surgery. It is precisely this small group of patients (with distal small vessel

disease, or "primary" pulmonary hypertension, type 4) that represent our treatment failures and in retrospect would not have been operated on. These patients, incidentally, had failed other therapeutic measures to reduce their pulmonary pressures.

Fourth, Table 3 in our paper addresses 1-month survival data, while Table 4 lists complications we have seen in the perioperative period after this operation. We report complications we have seen with this operation, and they represent a rate similar to (or even less than) the complication rates seen for other major cardiovascular operations. Rather than operating upon lower risk patients, in recent years we have expanded the eligible patient population for pulmonary endarterectomy to include older, sicker individuals, often with other concomitant heart disease. In any event, postoperative complications per se are not a reason to exclude patients from a lifesaving operation; rather, they represent an accurate depiction of our clinical experience with the surgical treatment of chronic thromboembolic disease over a wide spectrum of patient age groups and disease severity.

Finally, although comparisons between medically managed chronic thromboembolic disease and pulmonary endarterectomy have not been systematically made in prospective randomized trials, we believe that such trials would essentially be unethical, given the disparity in published survival statistics. The cumulative data from our institution leave no doubt that pulmonary endarterectomy is the superior treatment choice for management of pulmonary hypertension caused by chronic thromboembolic disease to the lungs.

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Tuberculous aneurysm of the aortic arch

To the Editor:

We read with interest the recent article of Hatem and colleagues.¹ We report here an additional surgical case of tuberculous aneurysm of the thoracic aorta with some resemblance to their case report.

A 52-year-old man was admitted to the hospital. He was previously healthy and reported a 6-week history of fever, malaise, and weight loss. On admission, results of cardiothoracic examination were normal, and all upper and lower extremity pulses were palpable. The chest radiograph showed a diffuse micronodular pattern in both lung fields. Bronchoscopic lung examination and biopsy revealed granulomatous pneumonitis with the presence of Langhans cells. The Ziehl-Neelsen test result was positive. The diagnosis was tuberculosis, and subsequently treatment with antituberculous agents was administered. Results of blood and urine analyses were normal. Investigation for HIV infection had negative results. Computed tomographic scan (Figure 1, A) and digital aortography (Figure 1, B) disclosed a localized aneurysm in the aortic arch. A diagnosis of mycotic aneurysm of the thoracic aorta was reached. The patient underwent operation on May 3, 1994. A left thoracotomy through the fourth intercostal space was accomplished, and an inflammatory aneu-

Figure 1. A, CT scan shows aneurysm in aortic arch. B, Digital aortography demonstrates saccular aneurysm in aortic arch. Remainder of thoracic aorta is angiographically normal. C, Arterial wall of aortic arch and aneurysm showing inflammatory signs with lymphocytes, histiocytes, plasmatic cells, and giant cells of Langhans type (hematoxylin and eosin, original magnification $\times 200$).

rysm of the distal aortic arch was exposed. The left upper lobe of the lung was involved in the process and was dissected off the aneurysm. The left subclavian artery, distal descending thoracic aorta, and part of the aneurysm were isolated. During dissection of the proximal aortic arch, the aneurysm ruptured, with massive cataclysmic hemorrhage. Despite clamping of the aortic

arch, rapid transfusion, and other maneuvers, the patient exsanguinated and died in the operating room.

The upper left lung, aortic arch, and mediastinal lymph nodes were sent for pathologic study. The histopathologic examination of the lung and nodes was consistent with tuberculosis, as was that of the walls of the aorta and aneurysm, which showed an inflammatory pattern with lymphocytes, histiocytes, plasmatic cells, and giant cells of Langhans (Figure 1, C).

We agree with Hatem and colleagues¹ regarding the extreme rarity of tuberculous aneurysm of the thoracic aorta. In 1996 Izekawa coworkers² published a case report and in a review collected 32 additional surgical cases. Since then, a few more cases have been reported.^{3,4} We believe that our case reported here may be useful and of interest to complete the scarce bibliographic list of this uncommon entity.

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