noncalcified, diffuse disease, which usually encompasses the entire trachea and may even extend outside the thorax to be palpable in the neck. This form of mediastinal fibrosis should be distinguished because it does appear to respond to corticosteroids or tamoxifen, at least in several published case reports.

We believe that attempts to stent vessels to palliate disease are clearly indicated in patients with bilateral pulmonary vascular disease from mediastinal fibrosis, in whom the outcome is universally poor and operative reconstruction risk is high. Stenting of vessels for patients with unilateral disease may be appropriate, but the risk/benefit ratio is less clear because many of these patients may have prolonged survival with acceptable functional status. Indeed, their functional status may be analogous in many respects to that of a single lung transplant recipient. Another consideration should include the possibility that the relative risk of a stent for a pulmonary vein (compared to that for a pulmonary artery) may be higher, because a stent complication of thrombosis might cause systemic embolism, and thereby possibly jeopardize extrapulmonary structures.

Finally, we were disappointed that the authors or reviewers did not use this opportunity to include any discussion regarding the physiologic mechanisms of dyspnea in their interesting patient or why the procedure might be associated with its resolution.

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To the Editor:

I would like to thank Drs. Robbins, Davis, Doyle, and Loyd for their interest in our article. Briefly, we felt confident, based on echocardiography and CT review, that the pulmonary veins were not involved in our patient, and bronchoscopy documented that the right mainstem bronchus was patent. We suspect that the patient’s improvement was due to a combination of reduced dead-space ventilation and some reduction in right-heart strain. The authors have succinctly reviewed the topic as well as their experience in stenting. We agree that each patient’s workup must be thorough and treatment individualized. Institutionally we managed two more patients in this manner, including one referred after a failed stent. Clearly, although more experience needs to be gained, interventional approaches are becoming recognized as a valuable tool in the management of these complex cases.

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Complications Following Percutaneous Tracheostomy

To the Editor:

We were interested to read the article by Briche et al (April 2001) concerning complications following percutaneous tracheostomy. However, in both cases, we are simply told that “percutaneous tracheostomy was performed.” There was no comment as to whether or not the procedure was aided by a separate operator performing simultaneous fiberoptic bronchoscopy of the trachea. The complication rate of percutaneous dilational tracheostomy may be reduced using endoscopic guidance, since experience does not seem to reduce the incidence of paramedian wire insertion. The study of Dexter, assessing blind wire placement, found that only 45% of wires entered the trachea at the intended level, 30% of wires pierced the thyroid isthmus, and only 15% of wires punctured the trachea centrally.

The proposed mechanism of stenoses in the two cases (a difficult perforation of the tracheal ring, thus creating an intraluminal tracheal flap) would surely have been noted if the fiberoptic bronchoscope had been used. We would advocate the use of bronchoscopy in all cases of percutaneous dilatation in order to minimize immediate and long-term complications resulting from this valuable bedside technique.

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REFERENCES


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To the Editor:

For 4 years, we have performed bedside percutaneous tracheostomy with two physicians, always with the same procedures.
The first physician, in each case a senior physician, is located at the head of the patient and performs fiberoptic bronchoscopy (Olympus NFT3 Rhino-Laryngo Fiberscope; Olympus Optical; Hamburg, Germany) to prevent complications. The second physician performs the procedure.

To prevent abnormal insertion and tracheal injury, the use of a single progressive conic dilator (Ciaglia Blue Rhino; William Cook Europe; Bjaeverskov, Denmark) is better than the use of several dilators with progressive size. In our experience, complications can occur even if we are satisfied with the immediate fiberoptic bronchoscopy result. I agree with Dr. Perkins’s opinion that fiberoptic bronchoscopy is required. Even better, it must be performed for a long time even after decannulation. Other methods can be performed to reduce tracheal impaction. A kit by Mallinckrodt (Tracheostomia translazingea Fantoni methods; Mallinckrodt Medical; Mirandola, Italy) uses a similar procedure as an endoscopic gastrostomy (internal to external procedure), but in our experience, this procedure is more complicated at bedside.

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Pleural Effusion Associated With the SAPHO Syndrome

To the Editor:

The acronym SAPHO (for synovitis, acne, pustulosis, hyperostosis, and osteitis syndrome) was first proposed in 1987 as a unifying concept to describe the association between musculoskeletal disorders and various dermatologic conditions. Since then, there has been an increasing interest in recognizing and characterizing this syndrome. Patients with pulmonary infiltrates and respiratory symptoms have been described occasionally, but, to our knowledge, pleural involvement has not been mentioned.

In November 1998, a 61-year-old man was admitted to our hospital because of a right pleural effusion. He reported pain in both shoulders and in the left hip, accompanied by stiffness of the chest wall and exertional dyspnea, all symptoms having been present for the previous month. The pleural fluid was a straw-colored, eosinophilic exudate with the following measurable characteristics: pH, 7.38; glucose, 93 mg/dL; protein, 5.1 mg/dL; and lactate dehydrogenase, 506 U/L. Rheumatoid factor, antinuclear antibody, adenosine deaminase, carcinoembryonic antigen, and microbiological and cytologic analysis results were all normal or negative. Complement studies were not performed. The patient had no history of exposure to asbestos, and an extensive diagnostic study (which included a CT scan, nuclear scintigraphy, and echocardiography) did not find the cause of the effusion, which resolved. During the following 12 months, the patient presented the features that established the diagnosis of the SAPHO syndrome (scap psoriasis, synovitis of the shoulder, pain in the anterior chest wall, and pubic osteitis), and he experienced a worsening of his exertional dyspnea. Pulmonary function tests showed a moderately restrictive defect. In the CT scan, a localized right pleural thickening was seen.

We speculate that the pleural effusion may be associated with the SAPHO syndrome because the pleurisy and the musculoskeletal symptoms started at the same time, and because no other cause for the effusion was found. Although it may be an incidental coincidence, we wish to alert clinicians to this possible diagnosis, bearing in mind that the SAPHO syndrome has been described recently and is still not widely known.

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Elevated Cardiac Troponin I Following Heavy-Resistance Exercise in Ostium Secundum Type-Atrial Septal Defect

To the Editor:

Raised concentrations of cardiac troponin, indicating poor short-term prognosis, have been reported in patients with dilated and secondary cardiomyopathy. It is assumed that these elevated levels of troponin reflect ongoing myocyte degeneration. Heavy-resistance exercise and strength training are associated with adaptive changes of cardiac structure and function. Furthermore, recently published data demonstrate that more than one third of patients who received a clinical diagnosis of pulmonary embolism had presented with elevated serum troponin concentrations due to minor myocardial damage in acute right ventricular dysfunction.

We report on a 71-year-old man with a 9-year history of ostium secundum type-atrial septal defect and increased right ventricular size. The patient was admitted to our hospital because of dyspnea and chest discomfort after heavy-resistance exercise, moving furniture. The patient’s BP was 120/80 mm Hg, his pulse rate was 82 beats/min, and his breathing rate was 18 breaths/min. The first heart sound was loud, the second heart sound was widely split without widening during inspiration, and a pulmonary systolic ejection murmur was present. ECG revealed complete right bundle-branch block and signs of right ventricular hypertrophy. No changes in the repolarization pattern could be documented compared with ECGs recorded previously. Echocardiography showed increased right ventricular size, moderate right ven-