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 translazinglea Fantoni method; Mallinckrodt Medical; Mirandola, Italy) uses a similar procedure as an endoscopic gastrotomy (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. Complete 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 (scalp 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-
tricular dysfunction, and tricuspid regurgitation, indicative of increased right ventricular stroke volume. A pulmonary-to-systemic flow ratio of 2.1, due to an ostium secundum type-atrial septal defect, could be accurately diagnosed by Doppler echocardiography. Symptoms disappeared spontaneously within a few minutes after hospital admission without therapy.

Laboratory examination revealed elevated serum concentrations of cardiac troponin I (2.2 ng/mL; normal < 1.6 ng/mL) and myocardial band (MB) isoenzyme of creatine kinase (15 U/L, normal < 5 U/L). MB isoenzyme of creatine kinase peaked (17 U/L) 4 h after hospital admission, and the level of troponin I raised to a peak concentration of 6.4 ng/mL. Other laboratory findings, including inflammatory markers and eosinophils, were within normal ranges. Coronary angiography showed normal epicardial coronary vessels with no signs of atherosclerotic lesions, and a cineangiogram revealed no wall motion abnormalities.

For several reasons, we do not believe that ischemia caused the elevation of cardiac enzymes. First, a normal coronary angiogram finding does not definitely rule out previous coronary artery occlusion, but it makes myocardial ischemia or myocardial infarction unlikely, particularly in the absence of known risk factors for atherosclerosis. Second, neither echocardiography nor cineangiography revealed any wall-motion abnormalities indicative of segmental myocardial damage. Third, elevated concentrations of cardiac troponin I have been reported in patients with dilated cardiomyopathy, secondary cardiomyopathies, and acute right ventricular dysfunction.

To our knowledge, this is the first report in which heavy-resistance exercise induced a raise in levels of serum cardiac troponin I and MB isoenzyme of creatine kinase in a patient with preexisting dilatation and dysfunction of the right ventricle due to an ostium secundum type-atrial septal defect with left-to-right shunt.

The management of atrial septal defects in older patients is debated persistently, and clinical trials have demonstrated conflicting data. We do not have absolute proof to guide our decisions; therefore, we must rely on our experience as well as on the available evidence in deciding how to treat these patients. Further studies are needed to determine whether the management of these patients should be guided by clinical signs or by biochemical markers, like troponins.

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Effects of Cardiac Glycosides on Atrial Fibrillation
To the Editor:

The experimental investigation of Friedman et al (October 2000) demonstrated that acetelystrophanthinid (AS) exerts a favorable effect on atrial fibrillation (AF)-induced atrial stunning, action that was not related to its effects on the duration of AF. In this study, the effect of cardiac glycosides on the duration of AF deserves further analysis.

Following atrial stimulation, the authors observed that “although the baseline duration . . . of AF in control dogs was the same as that found in . . . dogs before the administration of the glycoside (102 ± 70 s vs 106 ± 72 s). AF duration in the control dogs at a time comparable to that at which the glycoside had been administered was sharply lower (106 ± 72 s vs 7 ± 3 s; p = 0.12 . . . ). Thus, the lack of a difference in AF duration following AS might actually indicate an enhancement of the arrhythmia by the glycoside.”

Friedman et al further stated that “randomized controlled studies . . . have demonstrated little benefit of these drugs for alleviation AF. When compared to controls, neither IV nor oral cardiac glycosides have been found to increase the number of cardioversions or to shorten the time it takes for the cessation of AF,” and also “the administration of IV or oral cardiac glycosides does not appear to be effective in converting or shortening episodes of AF,” findings that were considered “consistent with the effects of AS on experimental AF observed in this study.”

Digoxin had been considered to have no efficacy for conversion of AF to sinus rhythm. However, trials have shown the advantage of digoxin over placebo for conversion to sinus rhythm in patients with shorter AF duration (< 72 h). As the AF induced in the study had a very short duration, their experimental model probably was very susceptible to the action of digoxin in the conversion of AF. Furthermore, the reported reduction in AF duration, in comparison with the dogs that received AS, did not reach statistical significance.

I do believe that the favorable effect of cardiac glycosides on AF-induced atrial stunning might be important in clinical practice and should be established in clinical investigations. However, the role of the cardiac glycosides in the duration of AF deserves further investigation to clarify the differences between clinical and experimental studies.

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CHEST / 120/5 / NOVEMBER, 2001 1753