cases, pulmonary arterial pressure may not completely normalize after mitral valve surgery if more marked structural abnormalities of the interstitium involve pulmonary arteries.

There have been many reports on the reversibility of the ventilatory and hemodynamic abnormalities after mitral valve surgery. However, any beneficial change induced by surgery is offset by the adverse effects of thoracotomy or cardiopulmonary bypass. Thus, ventilatory function may not improve for more than ten weeks after surgery. The study by Yoshioka et al (see page 290) on the other hand, suggests that ventilatory function improves much more rapidly after percutaneous mitral valvuloplasty than after surgery. The most likely mechanisms for improvement are either decreased mucosal edema or reversal of hyperresponsiveness of the airways to endogenous bronchoconstrictors. These beneficial changes in ventilatory function cannot be explained by formation of an acute procedure-related atrial septal defect that leads to hyperkinetic pulmonary hypertension and a decreased lung compliance.

Can we expect improvement in pulmonary function in all patients who undergo mitral valvuloplasty? Unfortunately, a benefit may not be expected in patients who have had long-standing elevation of left atrial pressure and irreversible changes in pulmonary anatomy. Since no patient in the present series had New York Heart Association class 4 symptoms, we cannot determine from this study how much improvement in ventilatory function would be seen after valvuloplasty in patients with advanced complications of mitral stenosis. The current study suggests that ventilatory improvement is related to hemodynamic benefit. Thus, we may speculate that patients with advanced complications of chronic severe mitral stenosis who show less improvement in hemodynamic status after mitral balloon valvuloplasty will experience less improvement in ventilatory function than those with mild to moderate mitral stenosis.

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Diagnostic Studies in Catamenial Hemoptysis

Cyclical hemoptysis occurring during menses (catamenial hemoptysis) is a rare entity; fewer than 20 cases have been reported since Lattes et al first described endometriosis of the lung in 1956. In 1966, Yeh suggested that hematogenous spread from the pelvis was responsible for parenchymal lesions, and in several case reports, a gynecologic operation preceded the onset of symptoms, lending support to this theory. The diagnosis of pulmonary endometriosis is usually made on the basis of the clinical history and the exclusion of other causes of recurrent hemoptysis including tuberculosis, bronchial carcinoma, pulmonary infarction, chronic bronchitis, congenital abnormalities and carcinoma. Bronchoscopy, ventilation/perfusion lung scans, angiography, and computed tomograms (CT) of the chest have been used to support the diagnosis.

Of the reported cases of pulmonary endometriosis in the English literature, all but one underwent bronchoscopic examination. The diagnostic yield from bronchoscopy in these patients would be expected to be small, since most endometrial metastases involve distal pulmonary parenchyma rather than the mucosa of large bronchi. In the one patient who underwent bronchoscopy in whom a definitive diagnosis was made, the tissue was obtained from a transbronchial biopsy. On the other hand, serial computed tomograms of the chest during and in the interval between menses has proved to be a useful confirmatory test. This was the only diagnostic procedure which indicated the etiology of bleeding in recent reports.

The diagnosis of pulmonary endometriosis is based upon the history and changes in the character of the lesions as documented on radiographic studies of the chest performed during and in the interval between
menses. When appropriate clinical and radiographic findings are present, further tests, including fiberoptic bronchoscopy, are not indicated.

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Occupational Asthma in the Egg Processing Industry

Smith and associates call attention to yet another occupation where workers become allergic to airborne allergens and develop asthma (see page 398). Considering that egg is one of the classic potent allergens, and that for years allergists have heard egg allergic patients say that beating an egg makes them wheeze, it is not surprising that workers develop asthma in egg-processing plants where the air contains large amounts of egg protein. The magnitude of the problem is surprising, however. Smith and colleagues used conservative criteria to estimate that about 10 percent of workers in egg processing plants develop asthma, an unusually high prevalence rate. There are about 100 such plants in the United States, each employing about 100 workers, so at least 1,000 workers are affected. Those who develop occupational asthma have a chronic disease and are at risk for permanent respiratory impairment. In other industries with occupational asthma, asthma has been found to remit in only about half the workers two years after their exposure ends. The others continue with chronic asthma indistinguishable in its progressive course from common cases of adult onset asthma of unknown cause.¹

In treating occupational asthma in the egg processing industry, two distinctly separate kinds of "patients" need attention: the workers who become ill, and the plants they work in. Physicians treat the workers; industrial hygienists, engineers and occupational medicine specialists treat the plants. Both "patients" must be treated to "cure" the problem and further development is needed in both aspects of treatment in the egg processing industry.

Physicians consulted by a patient with asthma who works in an egg processing plant must first decide whether egg allergy is causing the disease, or whether the patient is simply one of the 5 percent of the general population with asthma who happens to work in the egg industry. Several criteria help make the diagnosis of occupational asthma, particularly early in the course of the disease: (1) onset of asthma after employment; (2) increase in symptoms during work or the night after work and improvement on weekends and holidays; (3) reduction in peak flow and increase in the diurnal variation of peak flow associated with work; (4) presence of IgE antibody to egg protein demonstrated by skin test or in vitro tests; and (5) provocation of symptoms and change in spirometry by deliberate exposure at a time when airway obstruction is minimal and medications are not required. These last two special procedures are simple in principle, but fraught with pitfalls in performance and interpretation, so are perhaps best delegated to centers that have experience with them. Accurate diagnosis is important to the patient because continued exposure may cause irreversible disease, but unnecessary restrictions or job termination based upon an erroneous diagnosis may have equally disastrous socioeconomic consequences. A priori, skin or in vitro test for IgE antibody should be expected to be highly reliable in this particular situation of occupational asthma due to a classic allergen. However, reliability, the relationship between the prevalence of the disease, and specificity and sensitivity of the test, must be considered here as it is in all laboratory tests. In their epidemiologic study, the investigators found that neither the sensitivity nor specificity of these tests was as good as the medical history and peak flow measurement. This is not the place to discuss technicalities of preparation and standardization of reagents and procedures for tests of IgE antibody. Suffice it to say that there is