diagnosis can be easily made with the combination of clinical history, pulmonary function tests, and chest radiography. Occasionally CT may be requested for symptomatic patients with abnormal gas transfer (decreased carbon monoxide diffusing capacity [DLCO]) without evidence of airway obstruction on pulmonary function tests. Klein et al reported the cases of ten patients with impaired gas transfer (single-breath DLCO <80 percent predicted), normal flow rates (FEV/FVC >80 percent predicted), normal chest radiographs, and evidence of emphysema on high-resolution CT. All were smokers. In such patients, high-resolution CT can be of value in clinical management by differentiating emphysema from pulmonary vascular disease.

Another indication for CT is in the preoperative assessment of patients with large bullae being referred for bullectomy. Good outcome following surgical treatment is predicted by the presence of large bullae, a rapid onset of dyspnea, restrictive lung function due to compression of more normal areas of lung, and absence of generalized emphysema. The presence of crowding of vessels surrounding the bulla and the presence of emphysema surrounding the bulla or in the contralateral lung can be easily assessed. The less invasive nature of CT compared with angiography makes CT the examination of choice in the assessment of these patients.

Another indication, perhaps, may be in the assessment of patients with recurrent spontaneous pneumothorax. However, while it would demonstrate emphysema in the vast majority of these patients, what difference would that make? It is unlikely that it would change patient management. More exciting at the moment is the use of CT in research to increase our understanding of the pathogenesis and evolution of lung disease. There the information hopefully will be not only accurate but also relevant.

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Environmental Tobacco Smoke and Asthma

Many cities now oblige smokers to leave enclosed spaces, such as office buildings and bus and train stations, if they wish to indulge their addiction. There can be little argument that the accumulation of stale tobacco smoke is unpleasant and annoying to both the nonsmoker and the ex-smoker, and this will undoubtedly remain the main reason why a smoke-free environment is guaranteed in public places. Nevertheless, there is also some evidence that the concentrations of smoke likely in the absence of legislation have adverse consequences for health, including small but unacceptable increases in the risks of bronchial carcinoma, chronic obstructive lung disease, and coronary heart disease. There has been considerable discussion as to whether exposure to tobacco smoke provokes asthma. The tobacco plant undoubtedly produces powerful allergens, as witnessed by skin lesions in tobacco harvesters, and a proportion of subjects with a history of asthma demonstrate wheezing and signs of small-airway spasm with smoke exposure. However, such responses have been inconsistent. Some authors have also argued that no specific allergens have been demonstrated, although immune reactions to tobacco smoke have been observed in basophil leukocytes and immunoglobulin E antibodies. One major source of difficulty in conducting more conclusive experiments is that the odor of tobacco smoke is well known, so that exposure can create psychological reactions including not only tachycardia, but possibly also bronchospasm. If the smoke cloud is dense, it is less easy to detect relative levels of exposure, and one potential way of distinguishing biological from psychological reactions is to demonstrate a dose-response relationship. The technique adopted by Danuser and associates and described in this issue of Chest (see page 353) is attractive in this regard: the major eye and nasal symptoms that could precipitate psychological reactions are avoided, and nicely graded doses of smoke can be administered to the airways.

Like a number of previous investigators, Danuser
and colleagues used carbon monoxide as an index of smoke concentration. Carbon monoxide is one of the more readily measured constituents of the gas phase, but if the smoke is circulated through pumps and ducting, it is less certain how well carbon monoxide levels correlate with the effective concentrations of irritant constituents (which are likely absorbed upon particulate matter).

The choice of "realistic" carbon monoxide readings is also very debatable, with complications from urban background levels of carbon monoxide. The subjects studied by Danuser et al had an average blood carboxyhemoglobin concentration of 1.5 percent, which suggests either that some of the subjects were unadmitted smokers or that there was a substantial carbon monoxide exposure in the center of Zurich.15 In small enclosed spaces, such as a car with the windows closed, smoke-related carbon monoxide levels of 70 to 100 ppm have occasionally been recorded,14 although a ceiling of about 30 ppm is more likely under real-life conditions.15 Concern about the health hazards of environmental tobacco smoke has greatly reduced the smoke concentration in many public buildings where smoking is still permitted (at a considerable cost to heating in winter and cooling in summer), but in crowded and poorly ventilated areas, such as bars, readings of 30 ppm have been observed.16 The range of 0 to 32 ppm of smoke-related carbon monoxide adopted in the experiments of Danuser and associates thus seems quite appropriate.

Unfortunately, the key question of the relative contributions of a biological and a psychological response is not completely answered by the experiments that they report, for while the symptoms show a rather nice dose-response relationship, the physiologic reactions do not. Although they postulate a threshold, it is difficult to envisage a biological mechanism that would yield this type of response. Is the smoke so irritating that it provokes a secretion of fluid, which dilutes the initial stimulus? If so, why do the symptom scores remain dose-related? Plainly, more data are required: a larger sample of subjects, varying durations of exposure, and possibly measurements of small-airway function in addition to standard spirometry.

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Mitrail Stenosis and Left Atrial Thrombus
Role of Transesophageal Echocardiography

The article by Vigna and co-workers in this issue of Chest (see page 348) examines the role of transesophageal echocardiography in patients with mitral stenosis. The authors evaluated a group of 59 patients with mitral stenosis, in 12 of whom previous arterial embolization had occurred. All 12 of these patients were found to have left atrial spontaneous contrast, indicating a low-flow state in the left atrium. Although spontaneous contrast in the left atrium was found by transthoracic echocardiography in only two patients, the authors demonstrate that left atrial spontaneous contrast could be predicted to a large extent by clinical parameters, including atrial fibrillation and left atrial enlargement, in the vast majority of patients in their series. Furthermore, left atrial thrombus was found in 12 patients by transesophageal echocardiography, in only four of whom it was identified by transthoracic echocardiography. On the basis of these findings, the