found only three brief mentions of possible treatment (theophylline, nebulized bupivacaine, and nonsteroidal anti-inflammatory agents).

The cause of cough is not known, but increased bronchial reactivity, as in asthma, is probably involved. Because cromolyn sodium reduces airway reactivity, a trial of cromolyn inhaled administration in usual dosage was undertaken in six patients in whom dry cough had developed within two weeks of starting therapy with enalapril or captopril. The patients, all of whom suffered from well-documented congestive heart failure, were aged 55 to 85 years. Three patients were also hypertensive.

After two weeks of therapy, three patients had complete cessation of cough, and two had greater than 90 percent improvement. The latter two patients described the residual cough as minor and were able to continue ACE inhibitor therapy. The single patient who did not improve had immediate relief of cough when enalapril was discontinued.

In this small series of patients, use of a cromolyn inhaler appeared to be helpful for this difficult problem. A more thorough study, perhaps employing a crossover design and a placebo inhaler device, is needed.

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Hyperventilation Challenge Test for Myocardial Ischemia

To the Editor:

I enjoyed reading the article by Magarian and Mazur, which appeared in the January 1981 issue of Chest, but I have considerable doubts about the utility of the hyperventilation challenge test. Similar doubts may explain why the test has been used infrequently in the United States. Hyperventilation itself, as Dr. Magarian knows, frequently precipitates atypical chest pains that are not due to changes in myocardial blood flow. Recognizing this fact, it becomes hard to believe that induced hyperventilation can have significant utility in defining coronary artery spasm on any consistent basis in patients with "insignificant obstructive coronary artery disease." Even given a "positive" test (a 1.0-mm change in the ST segment), it remains uncertain that coronary artery spasm was the cause.

In clinical practice, having patients hyperventilate has its major utility in precipitating symptoms that they recognize and in reassuring them of the benign nature of the symptoms generated by their anxiety-produced breathing disturbance.

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To the Editor:

We appreciate the letter from Dr Miller expressing concerns over the utility of using a hyperventilation challenge test in patients with undiagnosed chest pain in whom coronary vasospasm is considered a potential cause. Indeed, hyperventilation can cause nonischemic chest pain, probably from a variety of causes. In many instances the associated chest pain is quite atypical of angina pectoris. However, in other patients, hyperventilation can be associated with chest pain that has many features suggestive of myocardial ischemia.

For many years it has been recognized that hyperventilation can cause not only anginalike chest pain but also electrocardiographic (ECG) changes consistent with ischemia. These observations were made in patients who had such ECG changes induced by hyperventilation but who had insignificant fixed, obstructive, atherosclerotic disease of their coronary arteries. Therefore, these changes were thought to represent false-positive responses and therefore were labeled "pseudoischemia." Unfortunately, coronary vasospasm was not excluded in these patients by currently recognized means of testing with ergonovine.

Since then, it has been demonstrated by many investigators that hyperventilation, like ergonovine, can provoke coronary vasospasm sufficient to produce ischemic ECG changes (with or without chest pain), myocardial infarctions, and ventricular arrhythmias. In patients with active coronary vasospasm, the hyperventilation challenge test, when performed as described, has a sensitivity that approaches that of ergonovine. In one recent study of 83 consecutive patients with stable angina pectoris, 16 had coronary vasospasm induced by hyperventilation. In all but one, coronary vasospasm became noninducible after administration of a calcium channel blocker (nifedipine or felodipine). Further, only these patients experienced improved exercise tolerance when treated with calcium channel blockers but not those without a propensity for hyperventilation to induce coronary vasospasm. In other studies, it has been demonstrated that early ischemic responses before completing the hyperventilation challenge in patients with coronary artery disease are due to increased myocardial oxygen demand from an increased rate-pressure product associated with the effort of the test, and not from coronary vasospasm, as in those with the classic delayed (posthyperventilation recovery phase) response.

We have suggested consideration of use of this test in patients in whom coronary artery vasospasm is thought to be triggering myocardial ischemia and anginal chest pain, especially if ergonovine challenge testing is unavailable or otherwise not performed during coronary arteriography. As with ergonovine challenge testing, there are potential risks, as noted in our review; therefore, this test should be done only in a setting in which appropriate responses to myocardial ischemia can be made. When done as outlined in our review, a positive posthyperventilation recovery phase response, even in the presence of coronary artery disease, can be considered to represent myocardial ischemia. It is critical to maintain ECG monitoring for 10 min after the 6-min hyperventilation trial because of the delayed coronary vasospasm response to hyperventilation. In others who develop exact replication of their chest pain with hyperventilation but without ECG changes suggestive of ischemia, it is very unlikely that their chest pain is from coronary vasospasm-induced myocardial ischemia. It is important to keep in mind that in patients with coronary artery disease, hyperventilation...
also can induce ischemia, due to increased myocardial oxygen demand and/or coronary vasospasm. The frequency of true false-positive hyperventilation-induced ischemic-appearing ECG changes warrants further research to clarify this important issue. For now, such changes, especially if occurring in the posthyperventilation recovery phase, should be considered to be secondary to ischemia from coronary vasospasm.

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Atopy and Primary Lung Cancer

To the Editor:

I read with interest the report by Dr McDuffie1 on an inverse relationship between atopy and lung cancer risk, which appeared in the February 1991 issue of Chest. I recently investigated the involvement of malignancy in 249 asthmatic patients admitted to our hospital during the past nine years.4

Eight (3.2%) patients (mean age, 68 years) had primary lung cancer (group A): five with squamous cell carcinoma, two with adenocarcinoma, and one with small cell carcinoma (all stage III or IV). Eight (3.2%) patients (mean age, 68 years) had extrapulmonary malignancy (group B): three with gastric cancer, two with malignant lymphoma, one with bladder cancer, one with laryngeal cancer, and one with prostatic cancer (all stage I, III, or IV). In group A, the mean duration of asthma was 19 years, and all cases were associated with respiratory tract infections. Of the eight patients, three had mild-type asthma, and five had moderate-type asthma. In group B, the mean duration of asthma was 20 years, and all cases were associated with respiratory tract infections. Five of the eight patients had mild-type asthma, and three had moderate-type asthma. There were no patients with pure atopic-type asthma. The mean smoking (Brinkmann) index in group A (1.194) was significantly higher than that in 241 asthmatic patients without lung cancer (1.063) or that in group B (1.063). The mean survival duration in group A (more than 26 months) was significantly lower than that in group B (more than 77 months), but was significantly higher than that of 114 patients with stage III or IV lung cancer but without asthma (12.7 months).

These results suggest that in many asthmatic patients with primary lung cancer who have an adult-type infectious asthmatic history, smoking exposure and age risks are deeply related to the development of lung cancer.

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Development of an Air-Fluid Level after Bronchoalveolar Lavage in Fibrocystic Lung Disease

To the Editor:

The clinical role of bronchoalveolar lavage (BAL) in adults with pulmonary diseases was summarized recently in an American Thoracic Society statement. It is generally a safe procedure. In this communication we describe a rare complication of BAL—fluid accumulating in a preexisting large fibrocystic space within the lung.

A 59-year-old woman, in whom a diagnosis of sarcoidosis had been made in 1964 after a left upper lobe resection, presented with a history of anorexia, weight loss, fever, and a nonproductive cough. A chest radiograph showed fibrocystic disease in the left lung. Bronchial brushing, biopsy, and BAL were performed in the left lower lobe. Approximately 350 ml of normal saline in 40- to 50-ml aliquots was instilled because of minimal return. Immediately following BAL the patient became acutely short of breath, and a chest radiograph showed an air-fluid level in the cystic space on the left side (Fig 1). Brushings and biopsy specimens were unremarka-