to the Editor:

The recent communication by Bense et al., concerning the occurrence of mitral valve prolapse (MVP) in nonsmoking patients with spontaneous pneumothorax, is rather provocative. However, until the cause-and-effect relation of smoking and MVP is found, three questions need to be answered.

First, the authors did not report the prevalence of MVP in patients with spontaneous pneumothorax who smoked. Therefore, one does not know whether the prevalence of MVP in their series of one in ten patients who never smoked is too high or too low.

Second, one would also like to know the prevalence of MVP in normal subjects in Stockholm, although the overall prevalence of MVP in Sweden was reported to be 7.4 percent. The prevalence of MVP varies among different countries, as well as among different cities in the same country.

Third, the authors did not mention how MVP was diagnosed in their patients—by auscultation, by echocardiography, or by angiocardiography. The prevalence of MVP could be different according to the method of diagnostic examination.

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To the Editor:

Professor Cheng has raised three problems regarding the possible effect of smoking as one factor in the pathogenesis of mitral valve prolapse (MVP). First, regarding the prevalence of MVP in spontaneous pneumothorax, we suggested, on the basis of our data, that smoking may have some role in the hitherto-unclarified pathogenesis of MVP. To our knowledge, the proportion of subjects who have never smoked in different studies of patients with spontaneous pneumothorax does not exceed 10 percent. In a study by Margaliot et al., 50 percent of their subjects had MVP, but no data on their smoking habits were published; we hypothesized that approximately 90 percent of them were smokers. In our study we investigated the prevalence of MVP in subjects who had never smoked and who had suffered spontaneous pneumothorax. The diagnosis of MVP could be established in one of ten patients.

Second, regarding the prevalence of MVP in the county of Stockholm, since the population of Sweden is essentially homoge-
neous, the prevalence of MVP in the south of Sweden, 7.4 percent, is considered to be valid also for the county of Stockholm. 3

Third, regarding how MVP was diagnosed in our patients, our description was obviously not sufficiently clear. Like Margalit et al., 4 we used echocardiography as the diagnostic method for detection of MVP.

Regarding Professor Cheng's point that a cause-and-effect relationship between smoking and MVP should be established, we believe that this can be done in two ways: (1) by comparing the smoking habits of MVP patients with the smoking habits of a large matched group of contemporary control subjects, stratified according to age and cigarette consumption in the same geographic area; and (2) by studying siblings who have never smoked and who have suffered familial spontaneous pneumothorax.

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Delayed Quinidine-Induced Diarrhea after Five Years of Treatment

To the Editor:

We recently encountered an unusual form of quinidine-induced diarrhea.

A 53-year-old hypertensive woman was found six years ago to have multiple, symptomatic, atrial premature beats. An echocardiogram showed mitral valve prolapse. The patient was started on a regimen of quinidine, 200 mg three times a day, and became entirely asymptomatic.

A year before the present visit, the patient developed diarrhea; she had numerous daily watery bowel movements. Stool examinations for pathogenic bacteria and parasites were negative. The patient declined endoscopic evaluation.

Other medical problems included thyroid carcinoma, treated by resection and radioactive iodine, and surgically induced hypoparathyroidism, which was controlled with oral calcium.

Although the possibility of quinidine-induced diarrhea seemed remote, since the patient had been taking the drug for five years before the diarrhea started, quinidine was discontinued and disopyramide was substituted. The diarrhea resolved within three days and did not recur for two weeks. The patient was challenged again with quinidine; daily diarrhea recurred within two days and lasted for the two weeks of the rechallenge period. When the quinidine was stopped again, the diarrhea disappeared. The patient remains free of diarrhea three months later. We have verified that no change was made in the quinidine preparation supplied to the patient during the past two years.

Diarrhea is the most frequent side effect of quinidine; in one large study,1 it was found to affect 3.6 percent of treated patients. We have found no good data on the time course of this side effect, but it is generally believed that quinidine-induced gastrointestinal side effects occur immediately after initiation of treatment; therefore, diarrhea in a patient who has been receiving quinidine for a long time (five years in our patient) is unlikely to be attributed to the agent.

We believe that the causal relation between quinidine and diarrhea in this case has been well established and conclude that quinidine may cause diarrhea after as long as five years of treatment.

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Erythromycin for the Treatment of Bronchial Hyperresponsiveness in Asthma

To the Editor:

In the March 1991 issue of Chest, Miyatake et al. 1 concluded that "erythromycin reduces the severity of bronchial responsiveness in patients with bronchial asthma." However, their observation was uncontrolled; there was no placebo treatment. It is possible that something other than erythromycin caused an increase in the provocation concentration producing a fall in FEV1, of 20 percent. For example, bronchial reactivity in the patients may have spontaneously decreased or some other factor may have contributed to the results.

Also, it is noteworthy that serum theophylline concentrations were not elevated after ten weeks of therapy with erythromycin. This is contrary to the findings in several previously published studies indicating that erythromycin results in an average 25 percent increase in serum theophylline levels when taken in such doses for more than five days.2

Given these concerns, it certainly would be premature for physicians to prescribe erythromycin for the treatment of asthma until benefit has been documented in a double-blind, placebo-controlled trial.

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Communications to the Editor