primary concern was cost containment as companies and insurers sought ways to give patients and providers incentives to hold down costs.

Dr. Weinberg and I have transmitted to the officers of the American College of Chest Physicians the deliberations of the national seminar cited above. This information will be shared with the membership via a new ACCP bulletin entitled “Issues Affecting the Practice of Cardiopulmonary Medicine and Surgery.” ACCP is intimately involved in future governmental and industrial deliberations if we are to serve our patients well. Our Government Liaison Committee members, under the leadership of Dr. Joseph Ross and our distinguished consultants in Washington, D.C., Mr. Ray Cotton and Mr. Michael Romansky, are anxious to represent ACCP members in the turbulent months ahead.

We have been greatly distressed with recent rulings such as the one which makes it unnecessary for respiratory care services to be under the direction of a qualified physician. Our concerns were transmitted to the highest sources in government and we hope that the justifiable objections of the American College of Chest Physicians will be fully considered. In this and in other issues which lie ahead (such as mandatory assignment) we are determined that the quality of care shall not be affected deleteriously.

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Screening for Lung Cancer

An estimated 135,000 men and women developed lung cancer in the United States last year and 90 percent or more will die of that disease, most within two years. Among male cigarette smokers, deaths from lung cancer equal all other cancers combined. In women, the incidence of lung cancer soon will exceed that of carcinoma of the breast, to become the primary cause of cancer deaths.

At this time, the chest x-ray examination and sputum cytology are the only means available for detection of lung cancer at an asymptomatic stage. The Memorial Sloan-Kettering study of a lung cancer screening program, reported by us in this issue (see page 44) was designed to determine whether cytology screening as an adjunct to the annual chest x-ray examination could facilitate detection and reduce the mortality from lung cancer. All 10,040 participants in that study, who were cigarette smokers at high risk of lung cancer, were offered annual chest x-ray examinations; half, chosen at random, also were asked to submit sputum for cytologic examinations every four months.

Early lung cancer detected by cytology proved to be slow-growing epidermoid (squamous) carcinomas of major bronchi. The study showed that in a program of annual chest x-ray screening conscientiously carried out, these squamous carcinomas can be detected radiographically at a later time, but while still localized. Thus, the addition of cytologic examinations to the annual chest x-ray examination does not have an effect on overall mortality from lung cancer. In a single screening, however, cytology may be of value as an adjunct to the chest x-ray film since there are no subsequent examinations.

The Memorial Sloan-Kettering study was not designed to evaluate the effect of an annual chest x-ray examination on mortality from lung cancer. All of the participants were offered annual chest x-ray examinations. Thus, the data from this study cannot be used to compare radiologic screening with no screening. However, we have established that a program of annual chest x-ray examinations, with or without cytology, will identify approximately 40 percent of all lung cancers (46 percent of nonsmall cell carcinomas) in AJCC stage I. Furthermore, 70-80 percent of the men with localized (stage I) lung cancer treated by resection do not die of that disease. There are only two reasonable explanations for these facts. Either the screening is saving lives of some men who would otherwise have had disease undetected until it progressed to an advanced, asymptomatic and incurable stage, or these men could have lived for many years unaware that they had lung cancer. To advise against efforts to detect lung cancer early, by screening asymptomatic high risk populations, is to assume the second explanation is true. Since symptomatic lung cancer is more than 90 percent fatal, a decision not to screen is equivalent to a decision not to treat for cure.

We therefore recommend annual chest x-ray examinations for the detection of early lung cancer in asymptomatic subjects who are at high risk (eg, cigarette smokers over the age of 50 years). Sputum cytology should be included as an adjunct to the chest x-ray examination only if x-ray screening is to be performed on a single occasion or as infrequently as once every five years. Since it is difficult and expensive to select, recruit and motivate high-risk subjects to comply with a single site screening program, and since successful screening requires medical facilities for prompt investigation and resolution of suspicious findings, we recommend that screening for lung cancer in high-risk subjects be incorporated within a comprehensive health care system, through the private sector.
practitioner, health maintenance organization or general medical clinic rather than as a single site screening program. Certainly the weight of evidence at this time supports the prudent medical practitioner who recommends regular screening of the asymptomatic person at high risk of lung cancer.

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Increased Levels of Airways Responsiveness as a Risk Factor for Development of Chronic Obstructive Lung Disease

What Are The Issues?

Why do only 20 percent of cigarette smokers develop chronic obstructive lung disease (COLD)? Competing risks may be part of the answer. Smokers may die of conditions such as coronary artery disease and lung cancer prior to developing COLD. However, susceptibility also may be an important part of the problem. Early investigators formulated two theories about the origins of COLD. The first, which we call the British hypothesis, held that cigarette smoking led to inflammation and phlegm production. This mucus hypersecretion led to abnormalities in airway clearance and in certain persons susceptible to repeated infections. These infections destroyed lung tissue and led to a more rapid decline in lung function and to airflow obstruction.1 An alternative hypothesis, first proposed by the Dutch, held that in susceptible individuals, cigarette smoking led to increased levels of bronchial responsiveness which, through repeated acute increases in airflow obstruction, led to a more rapid decline in pulmonary function.5

Fletcher and others tested the British hypothesis in a landmark study of British transport and postal workers.1 This study demonstrated that respiratory infections bore no relationship to rate of decline in lung function, thus raising serious doubt about infection as a risk factor for the development of COLD. Another major contribution of this study was an exposition of the methodologic issues in the longitudinal assessment of changes in adult lung function. The Transport and Postal Workers Study suggested that initial levels of lung function at study onset was an important determinant of future rate of decline in lung function. This was due to two factors: the statistical concept of regression to the mean, and their finding that those subjects with low levels of lung function had greater rates of decline of lung function (the so-called horse-racing effect). Normally attained maximal level of lung function in early adult life may also serve as a surrogate for past history of environmental exposures in childhood and, thus, provides an important correlate of rate of decline in lung function.

Existing data bearing on the Dutch hypothesis are relatively sparse, but recently have been well reviewed." In cross-sectional data, increased levels of airways responsiveness have been associated with lower levels of pulmonary function in normal subjects5 and in subjects with chronic bronchitis.6,7 Several investigators have also reported that chronic cigarette smoking is associated with increased levels of airways responsiveness.4,9 Data from the Tucson group lend additional support for the Dutch hypothesis. These investigators have found that cigarette smoking in adults is associated with elevated serum IgE levels10 and the subsequent development of doctor-diagnosed asthma.18 Burrows et al11 have even suggested that the serum IgE measured in cigarette smokers may be related to microorganisms in the respiratory tract, thus possibly linking the British and the Dutch hypotheses. The methodologic issues raised in the British Transport and Postal Workers Study provide an important context for considering existing longitudinal data about airways responsiveness. Since both level of pulmonary function and cigarette smoking are major determinants of rate of decline in lung function, one would need to control for the effects of these (and possibly other) variables to establish that increased levels of airways responsiveness are truly an independent risk factor for decline in lung function. The work of Barter and Campbell,14 and the earlier work of Kanner et al16 support the Dutch hypothesis, showing increased levels of airways responsiveness in COLD patients with more rapid rates of decline in lung function. However, both studies failed to control for the initial level of pulmonary function.

The major contribution of the article by Kanner and associates in this issue of Chest (see page 54) is that airways responsiveness remains a predictor of rate of decline in lung function in patients with COLD even when controlled for initial level of lung function. The