attention. Indeed, the consensus among experts in pulmonary medicine, pulmonary physiology, and epidemiology a few years ago was that we were not ready to screen populations for abnormalities of pulmonary function, yet all available current information suggests that the screening for abnormalities of FEV₁ could prove to be an outstandingly effective means of preventing the serious consequences of chronic limitation of airflow in a large number of subjects. We do not know whether this is true; but even more important, we do not know that it is not true! The message seems clear: We must encourage the use of the spirometer as part of the routine examination of nearly all adult patients, and we must encourage the development of controlled studies in populations to determine the effectiveness of evaluation of pulmonary function as a means of preventing the serious consequences of chronic limitation of airflow.

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Screening for Early Obstruction of the Airways
A 1978 Reappraisal

Ten years ago, Hogg and his associates made an important observation that the principal site of increased resistance in chronic airflow obstruction occurred in the peripheral airways of the lung and not in the more proximal bronchi, as had been believed. These same studies suggested that resistance to airflow in the peripheral airways of normal lungs was negligible. A novel concept concerning the natural history of chronic airflow obstruction evolved from these observations. It was suggested that the peripheral airways were functionally a “quiet zone,” such that extensive disease might exist at these sites and yet not be detected by the usual tests of pulmonary function, particularly the forced expiratory volume in one second (FEV₁). It was further suggested that more sophisticated tests of ventilatory function, which were sensitive to disease in the small airways, might allow chronic airflow obstruction to be diagnosed at an earlier and more reversible stage. Because of the dismal experience in attempting to arrest the progression of clinically overt chronic airflow obstruction, this idea had enormous appeal to pulmonary physicians. Subsequently, considerable effort has been expended in developing and evaluating so-called tests of “disease of the small airways,” which might be suitable for purposes of screening.

In 1973, the Division of Lung Diseases of the National Heart, Lung, and Blood Institute and the American Thoracic Society sponsored a workshop on early diagnosis of obstruction of the airways in relation to mass screening programs. This conference concluded that mass screening for early chronic airflow obstruction should not be undertaken until two major questions were answered: (1) Do the proposed tests of early chronic airflow obstruction predict the development of frank obstruction at a later time? (2) If detected at an early stage, can the subsequent course of chronic airflow obstruction be modified by treatment or removal of risk factors? In the five years since that conference, a fairly definite answer to the second question and a tentative answer to the first question have emerged.

The usefulness of different tests of pulmonary function in diagnosing the early stages of chronic airflow obstruction can be determined only by prospective studies which allow sufficient time for disabling chronic airflow obstruction to develop. This particular type of study has not yet been completed, but other types of studies have raised questions about the original premises which indicated the need for more sophisticated testing methods. Early studies purporting to demonstrate the greater sensitivity of the newer tests (such as the closing volume [CV]) to detect early chronic airflow obstruction proceeded on the assumption that an abnormal FEV₁ was evidence of advanced disease, and individuals to be studied were selected on the basis of a normal or nearly normal FEV₁. Studies of this type are interesting because they demonstrate that physiologic abnormalities can occur in the face of a normal FEV₁, but these studies provide no basis for stating that any test is more sensitive than the FEV₁.

An independent judgment as to the presence of early chronic airflow obstruction (by clinical assess-
ment or by association with a known risk factor, such as cigarette smoking) allows a more valid basis for comparing the relative sensitivity of various tests of pulmonary function. In a study with rigorous guards against bias in selection of the population, Knudson and his associates\(^6\) found that variables obtained from flow-volume curves were superior to the slope of phase 3 and the CV in separating asymptomatic nonsmokers from smokers both with and without symptoms. In a similar cross-sectional study restricted to older men and with fewer subjects, Oxhoj and his co-workers\(^7\) reported that the slope of phase 3 appeared to be the most sensitive test in separating smokers from nonsmokers but that the FEV\(_{1.0}\) was equally as sensitive as the CV and other spirometric indices. Additionally, studies of structure and function in lungs at autopsy have challenged the prevailing concept that the peripheral airways are a “quiet zone” in the normal lung.\(^8\) These studies indicate that spirometric data, including the FEV\(_{1.0}\), should be quite sensitive to minor degrees of pathologic narrowing in the peripheral airways. Thus, on the basis of available information, there is little reason to believe that any test is appreciably more sensitive to early chronic airflow obstruction or to “disease of the small airways” than is the FEV\(_{1.0}\) and even less reason to believe that any test would be more useful for screening purposes than routine spirometric studies.

Much of our new information about the natural history of chronic airflow obstruction and the potential benefits that might be derived from screening studies comes from the study carried out by Fletcher and his colleagues.\(^9\) This prospective study of working men both confirmed some older data and provided some highly significant new information on the value of FEV\(_{1.0}\) in predicting eventual disability from chronic airflow obstruction and on the effects of cessation of smoking. Their findings can be summarized as follows: First, obstruction of the airways is virtually nonexistent in people who have never smoked cigarettes. This is also true of emphysema, as determined by an earlier study based on autopsies.\(^10\) Secondly, although cigarette smoking is a prerequisite for the development of obstruction of the airways, only a minority (approximately 28 percent in the study by Fletcher et al\(^9\)) of cigarette smokers will develop clinically significant obstructive pulmonary disease. Third, cigarette smokers destined to develop clinically significant obstruction of the airways will, in most instances, have a reduced FEV\(_{1.0}\) by the age of 40 years and will display a more rapid rate of decline of their FEV\(_{1.0}\) than nonsmokers and than smokers who will not develop significant obstruction of the airways. Finally, and most significantly, cessation of smoking will restore the accelerated rate of decline in FEV\(_{1.0}\) in susceptible smokers to the normal age-dependent decline in FEV\(_{1.0}\) observed in nonsmokers, even though the actual FEV\(_{1.0}\) does not return to its original level. Thus, the potential benefits of cessation of smoking is established in those persons who already have evidence of early chronic airflow obstruction.

These recent studies go a long way toward answering the original questions posed by the 1973 workshop on screening programs for chronic obstructive pulmonary disease, but they still do not establish the value of mass screening programs. Cigarette smoking remains the only clearly identified risk factor for chronic obstruction of the airways that can be eliminated with definite beneficial effects in pulmonary function. The public is already aware of the dangers of this habit, yet cigarette smoking is actually increasing in some groups of the population.\(^11\) It may be that it will be easier to persuade an individual to stop smoking once he or she has been placed in that 26 percent minority of smokers who are susceptible to cigarette smoke, but that remains to be proven. Furthermore, what will happen to the other 74 percent of cigarette smokers once they are tested and found to not be at risk for developing chronic obstruction of the airways? Will this knowledge deter their efforts to stop smoking? As was the case in 1973, more research needs to be done before mass screening for the early detection of obstructive pulmonary disease becomes worthwhile.

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Exercise Testing in Variant Angina

In the August 1978 issue of Chest, Weiner and associates reported the case histories of four patients who developed ST-segment elevation following exercise. One of their patients (case 1) had previously experienced recurrent pain in the chest at rest, which was associated with transient ST-segment elevation, arrhythmias, and heart block; this patient clearly meets the criteria for variant angina pectoris. This patient is unique in that the ST-segment changes that developed immediately after exercise were remarkably similar to those recorded during spontaneous attacks. The other three patients did not have documented ST-segment elevation during spontaneous pain prior to the exercise test; and in this respect, they are similar to previous reports of patients with “variant angina” and ST-segment elevation associated with an exercise test. Even though these three patients did not meet a strict definition of variant angina prior to the exercise test, they may still represent part of the spectrum of variant angina which has not previously been published.

As will be seen, a careful definition of variant angina and a clarification of its relation to the other subsets of patients with ischemic heart disease are of signal importance. Failure to achieve such clarification has already led to unnecessary controversy regarding the place of surgery in the management of patients with variant angina.

In 1974, we reviewed our experience with the surgical management of variant angina and concluded that the results of aortocoronary bypass surgery in patients with variant angina were not as good as in those with classic angina. Later, Shubrooks and associates concluded that “most patients [with variant angina] with significant fixed coronary lesions will do well after coronary bypass surgery; however, many of their patients simply exhibited ST-segment elevation during a period of unstable angina and did not meet the definition of variant angina.

Because our initial report contained data from only 26 patients and because there was a lack of agreement on the place of aortocoronary bypass surgery in the treatment of variant angina, we again reviewed the literature with respect to surgically treated patients with variant angina during the period of 1974 through 1975. These results (in over 75 patients) were remarkably similar to those in our initial report; perioperative infarction and mortality were excessive, and symptomatic improvement (in the absence of infarction) occurred in less than 50 percent.

Similarly, Wiener and associates reported suboptimal surgical results in patients with carefully documented coronary arterial spasm; only 50 percent showed improvement, 22 percent had myocardial infarction, and the early mortality was 22 percent. In contrast, Johnson et al reported better results in a heterogeneous group of patients with ST-segment elevation during pain (some of which clearly were in an unstable phase of classic angina).

While these reports have contributed to a continuing controversy, most of the disagreement regarding the place of surgery in treating variant angina is unnecessary. Certainly, there is little reason to attempt aortocoronary bypass surgery in patients with normal coronary arteriograms or in those with insignificant obstructive lesions, even in the presence of severe and disabling symptoms. Except under unusual circumstances, single-vessel disease is not considered a surgical lesion in classic angina, and this is probably also true in variant angina. Unfortunately, the results of surgery in those patients with carefully defined variant angina and fixed lesions remain inferior to the results obtained in patients with classic angina; thus, it would seem that we need more refined criteria for the selection of surgical candidates.

While disabling symptoms and fixed coronary lesions are essential, we do not have an ideal set of criteria for the selection of candidates for surgery. The exercise test may be useful in this regard. Weiner and associates reviewed the literature on exercise tests through January 1977 and concluded that “contrary to the prevailing belief, over half of the patients with Prinzmetal’s variant angina have electrocardiographic changes diagnostic of ischemia during exercise testing.” Our experience has been more in keeping with the popular concept that although an abnormal exercise test is not rare, exercise tolerance is generally preserved in patients with variant angina.

A typical example of ST-segment elevation (during pain at rest) and a normal exercise test are