firmed. As one who believes that if carefully researched, most things touted as new will be found to be old items repackaged, I was gratified to learn that all of the work on this question done in the past 133 years has not added substantially to the common-sense conclusions arrived at by Hutchinson in 1846.

Other sources of spurious variation in spirometric results between pulmonary epidemiologic surveys are (1) the adequacy of spirometric instruments and (2) the skill and experience of the persons administering the test. The recommendations of the Snowbird Workshop on Standardization of Spirometry represent a laudable effort to set minimum standards of performance for equipment measuring volume and flow. However, the larger issue, of which the questions answered by the report of Nathan et al are but small (although vital) components, is the comparability of data between pulmonary epidemiologic studies done by different investigators on different populations. The recently published report of the Epidemiology Standardization Project represents a major advance toward attaining the standardization of methods necessary for the maximal utilization of data from present and future epidemiologic efforts in the respiratory field. This project, which is administered by the American Thoracic Society, participated in by representatives from the American College of Chest Physicians, and funded by the Division of Lung Diseases of the National Heart, Lung, and Blood Institute, developed and tested improved respiratory questionnaires and made further cogent recommendations on the standardization of tests of pulmonary function and the use of chest x-ray films in epidemiologic studies. This report should be required reading for anyone with a serious interest in the epidemiology of the common disabling chronic pulmonary diseases.

As is the case with so many current health problems, the key to controlling and eventually eradicating the common chronic pulmonary diseases is prevention rather than treatment. Knowledge gained from epidemiologic surveys has already contributed much information relating to risk factors and the course of progression of these diseases over time. Increasingly, future efforts will study the effects of various forms of active intervention in sizable populations at risk. This type of large-scale experiment has already been undertaken in the closely related field of cardiology. In pursuing such future investigation the three reports referred to herein will form a useful and important basis for more accurate collection and interpretation of data.

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Rapid Progression in Valvular Aortic Stenosis

The isolated calcific aortic stenosis of adults, whether due to secondary degeneration of a congenitally deformed bicuspid valve or to a primary process in an originally normal valve, is clearly a progressive disorder, arising in valves unobstructed at birth. In this condition, increasing calcification and rigidity of the valve is related to increasing stenosis. Few data are available on the rate at which this process occurs in individual patients, but it has usually been assumed to be a slow one. This view was first expressed by Thomas Peacock in 1853: "Notwithstanding the absence of any symptoms of the heart till short a period before death, there can be little doubt that the change in the valves had been long in progress."

The report of Bogart et al which appears in this issue of Chest (see page 391), as well as two earlier studies on the same subject, have clarified certain aspects of the hemodynamic natural history of this disorder. Using serial cardiac catheterization studies, these observers have clearly documented that while progression may, as Peacock postulated, be slow, it is quite variable and, in certain patients, may be remarkably rapid. In all three series, instances are noted of progression from mild to critical disease occurring in as little as two to three years. In the study of Gradman and Hancock, more than half of the patients demonstrated progression from non-critical to critical disease in less than a five-year period.

The incidence of this type of rapid progression cannot be readily assessed. Patients generally undergo repeated cardiac catheterization studies only if there is strong clinical suspicion, usually based on progressive changes in symptomatology indicating that severe stenosis has developed. Serial observations in an unselected group of patients with mild aortic stenosis are not available for comparison. The present observations serve principally to indicate that progression does occur, that progres-
sion from noncritical to critical aortic stenosis can occur in less than five years, and that this is very likely to be found when patients with progressive symptoms and electrocardiographic changes are restudied.

Why is it that certain patients enter a period of accelerated hemodynamic and presumably pathologic progression of their disease? Edwards has adopted the unifying hypothesis that calcific aortic stenosis, regardless of etiology, is really the result of trauma to the valve leaflets. This trauma, whether initiated early in life by rheumatic inflammation or by the abnormal stresses sustained by a bicuspid valve, or much later by many years of normal valve motion, leads to the lipid deposition and subsequent calcification which restrict leaflet mobility and obstruct blood flow. It is reasonable to extend this hypothesis further: once some degree of stenosis has developed, turbulent blood flow patterns and increases in flow velocity occur at the site of obstruction. This results in an increased amount of trauma to which the valve leaflets are subjected with every heart beat, and leads to further calcification. A self-perpetuating cycle is set up, capable of rapidly transforming mild into severe valvular stenosis. Rodbard has shown evidence that similar mechanisms may be operative in the progression of stenotic vascular lesions.

The natural history of aortic stenosis has often been viewed as one of a lengthy period during which the stenosis is severe but well-compensated and asymptomatic, followed by a symptomatic period which is relatively brief and downhill until death (unless interrupted by aortic valve replacement). In this view, the left ventricle is considered to be able to carry out a greatly increased pressure work for some years before symptoms develop; the symptoms then represent ventricular deterioration secondary to long-standing overwork. Experience with aortic stenosis in children, who are often asymptomatic with severe stenosis and are more likely to die suddenly than to have disabling symptoms, is consistent with this view. The picture may be different in adults, however, who rarely present severe aortic stenosis without any symptoms at all. Oleson and Warburg showed that while survival is short after the onset of overt right-sided congestive heart failure, the average survival after the onset of the first symptoms is 8.8 years, and even after the onset of angina and syncope the average survival rate is three to four years. It is possible that the valvular stenosis is progressing during these years of gradually increasing symptoms.

Recognition that a rapidly progressive phase sometimes occurs in the natural history of valvular aortic stenosis should serve to alert the clinician to the fact that progressive, though often subtle, changes in the symptoms, electrocardiograms and physical exams of patients previously found to have mild or moderate aortic stenosis may herald the development of critical and life-threatening valvular obstruction.

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Interpretation of Pulmonary Function Tests

The article by Cary and colleagues (see page 389) raises the question: How should one interpret pulmonary function tests?

In the case of pure restrictive diseases, the situation is relatively straightforward. Restriction can be defined as a reduction in total lung capacity (almost invariably reflected in vital capacity as well), and the severity can be gauged quantitatively according to guidelines laid down in the article. But what of the occasional patient with pulmonary fibrosis due to, for example, scleroderma, whose lung volumes may be entirely normal even in the face of marked reduction of diffusing capacity? Is this restrictive lung disease without restriction, and if so, of what severity?

In attempting to classify patients with obstructive lung disease, the situation is more complex. As pointed out by the authors, the timed vital capacity (FEV1%) may be quite deceptive, since it depends upon the vital capacity. Patients with severe airway