The Importance of Spirometry in COPD and Asthma*

Effect on Approach to Management

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COPD is characterized by airflow limitation. The diagnosis is suggested by history and physical examination and is confirmed by spirometry (*ie*, a low FEV_1 level that is unresponsive to bronchodilators). Once diagnosed, there is no widely accepted staging or severity scoring system. COPD presently is graded using a single measurement such as FEV_1, which, unlike the case with asthma, has a limited role in disease management. A more comprehensive staging system is required incorporating, for example, age, arterial blood gases, dyspnea, body mass index, and distance walked, in addition to FEV_1. These criteria should allow for more evidence-based recommendations for management of this condition. Asthma is an inflammatory disease also characterized by airflow limitation. But in contrast with COPD, the airflow limitation is highly reversible either spontaneously or with therapy. Repeated lung function measurements using portable peak flowmeters have resulted in improved outcomes. Therefore, frequent flow determination is recommended in the routine management of asthma. Treatment with anti-inflammatory agents and close monitoring of lung function should help decrease the morbidity and mortality associated with asthma. ***(CHEST 2000; 117:15S–19S)***

Key words: COPD; disease management; evidence-based medicine; spirometry; staging

COPD is characterized by airflow limitation caused by chronic bronchitis or emphysema. Reversible bronchoconstriction often plays a role in the cause of COPD, but its true magnitude remains to be determined.[1] Chronic bronchitis is manifested by cough and excessive sputum production. Patients with emphysema exhibit progressive and eventually crippling shortness of breath. It is estimated that COPD affects 14 million adults in the United States and that, of these, 2 million individuals suffer from symptomatic disease. COPD is the fourth most frequent cause of death and was a primary or contributing cause of 8% of all deaths in 1985.[2] Between 1979 and 1991, mortality from COPD rose by 33%.[1–3] In 1995, COPD was the primary cause of $>$ 100,000 deaths[2] and produced substantial disability because of chronic dyspnea. In the same year, COPD was responsible for $>$ 500,000 doctor's office visits and 60,000 hospital discharges.

Diagnosis of COPD

A diagnosis of COPD is suggested by history and physical examination and is confirmed by spirometry (*ie*, reduced FEV_1).[1] The residual volume and total lung capacity are increased in most cases. A chest radiograph may suggest emphysema, and the diagnosis can be confirmed with a CT scan, which is especially useful in the selection of patients for lung volume reduction surgery.[4] Gas exchange is usually impaired and is frequently reflected by systemic hypoxemia with and without hypercapnia.[5]

Pathophysiology of COPD

The basic pathophysiologic process in COPD consists of increased resistance to airflow, loss of elastic recoil, decreased expiratory flow rate, and overinflation of the lung.[6–10] The alveolar walls frequently rupture (emphysema) in the process. The hyperinflated lungs flatten the curvature of the diaphragm and enlarge the rib cage. The altered configuration of the chest cavity places the respiratory muscles, including the diaphragm, at a mechanical disadvantage and impairs their force-generating capacity.[11–13] Consequently, the metabolic work of breathing increases, and the sensation of dyspnea heightens.[14–16] The alterations in regional ventilation and blood perfusion result in hypoxemia and, in some cases, the increased dead space, decreased alveolar volume, and hypoventilation that is observed in hypercapnia.

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Natural History of COPD

Little is known about the natural history of COPD since the initiation of the modern era of treatment. Knowledge about the natural course of severe COPD is based on old studies and is linked entirely to changes in lung function.[16–18] We know that the FEV_1 in nonsmokers without respiratory disease declines by 25 to 30 mL per year beginning approximately between the ages of 25 and 30 years. The rate of decline of FEV_1 is steeper for smokers than for nonsmokers (Fig 1). It is also steeper for heavy smokers than for light smokers. The decline in lung function occurs along a slowly accelerating curvilinear path. In most persons, the loss occurs uniformly, but in some it develops in stages with relatively steep declines. There is a direct relationship between the initial FEV_1 level and the slope of FEV_1 decline. There is also a somewhat stronger association between a low FEV_1/FVC and a subsequent decline in FEV_1 in men but not in women. Age, which is correlated with the number of years of cigarette smoking, is clearly a risk factor for more rapid decline of lung function, as are lifetime smoking history and the number of cigarettes currently smoked per day. Individuals with COPD have more frequent acute chest illnesses that invariably decrease lung function for at least 3 months.[1] In some cases, lung function never returns to baseline, and this process may accelerate lung function decline in a rather abrupt stepwise manner.

Role and Value of Spirometry in COPD

As we have seen, postbronchodilator spirometry is required to confirm the diagnosis of COPD.[1] Once diagnosed, there are no widely accepted staging or severity scoring systems for patients with COPD. At present, we grade the disease based on a single objective physiologic measure such as FEV_1.

Paradoxically, we define COPD by a low FEV_1 value that fails to respond to bronchodilators, a characteristic that differentiates it from asthma, and then we use the change in FEV_1 to evaluate the effect of therapy. This contrary approach is unique in medicine. It would be the equivalent of defining essential hypertension as an increase in BP that must not respond to antihypertensive therapy, and then testing antihypertensive agents using the unmodifiable BP as the only outcome. Given this paradigm, it is no wonder that many well-designed trials have failed to document significant benefits from the use of bronchodilators or inhaled corticosteroids.[19] This contrary approach is highlighted further by the widespread use of bronchodilators despite the marginal effect that they seem to have on disease progression.[1,10,20,21]

It follows that there is a need for a more comprehensive staging system that would allow categorization of the heterogeneous population of patients with COPD for epidemiologic and clinical studies, health resource planning, and prognosis. Such a system

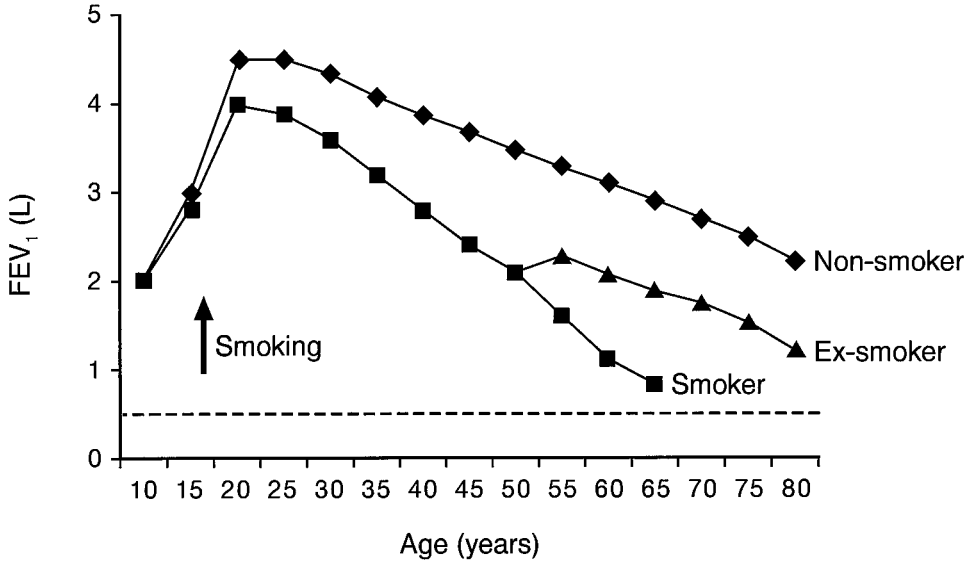

Figure 1. FEV_1 decreases with age. The rate of decrease is steeper for smokers. The rate of change in ex-smokers will approximate that of nonsmokers. Smoking cessation is associated with a small but significant increase in FEV_1.

would also greatly simplify and facilitate the application of clinical information and allow more evidence-based recommendations.

Two examples of such staging or scoring systems that have gained wide applicability in medicine include the TNM staging system for cancer and the ARDS severity scoring system. These relatively simple tools have proven useful to characterize disease severity, extent of disease, and response to therapy when one single variable fails to adequately represent the disease itself.

FEV_1 as a percentage of its predicted value is the best single correlate of mortality in COPD.[1–4] However, it is not until values fall to < 50% of predicted that mortality begins to increase.[1] Health-related quality of life also has shown a small but significant correlation with the severity of airflow obstruction and, hence, supports the continued use of FEV_1 in the evaluation of patients suspected of having the disease. Once patients reach a very low value of FEV_1, this measurement has little predictive value, but no other measurements have been thoroughly validated. Anthonisen[22] compared 3-year survival data from the intermittent positive-pressure breathing and the nocturnal oxygen therapy trials. The survival rate among nonhypoxic patients in the intermittent positive-pressure breathing trial was similar to that of hypoxic patients receiving continuous oxygen therapy in the nocturnal oxygen therapy group.[22,23] Survival was also lower than that of both these groups in hypoxic patients receiving only nocturnal oxygen therapy in the same nocturnal oxygen therapy group. Therefore, with new effective therapy and with the capacity to handle acute exacerbations, including the use of noninvasive mechanical ventilation, factors different from FEV_1 must influence survival. On the other hand, death is not the only outcome attributable to COPD, and the impact of COPD on the ability of patients to perform the normal activities of a vocation or of daily living are incompletely described by measuring the levels of FEV_1 and arterial blood gases. There are other easily determined measurements that have been shown to predict outcomes such as mortality or utilization of health-care resources.[24–27] There is a need to evaluate systematically these and other variables, including biological markers,[28] in an attempt to better characterize patients with COPD (Table 1).

Table 1–*Potential Variables to Establish a New COPD Staging System*

Variables
Evidence-based
Age[1,17–20]
FEV_1[1–3,17–20,23,29]
Arterial blood gases[1,22,23,29]
Time walked distance[24–26]
Sensation of dyspnea[30]
Body mass index[28]
Possible
Biomarkers
Inspiratory capacity
Genetic markers
Sex

Spirometry in Asthma

Asthma is a chronic inflammatory disease of the airways. In the United States, it afflicts approximately 14 million people. It is the most common disease of childhood and causes close to 500,000 hospitalizations a year. It is estimated that 5,000 people die from asthma every year. Many more develop acute respiratory failure and require mechanical ventilation. The death rates from asthma have remained stable for the past decade.

Inasmuch as asthma is a chronic inflammatory disorder, many cells play a role, and, in the susceptible individual, inflammation causes recurrent episodes of variable degrees of wheezing, dyspnea, chest tightness, and cough. The episodes are associated with widespread but variable airflow obstruction that is often reversible either spontaneously or after treatment. The inflammation also causes an associated increase in the existing bronchial hyperresponsiveness to different stimuli.[29] Recent evidence suggests the development of sub-basement membrane fibrosis that may lead to persistent abnormalities in lung function.[30,31]

The diagnosis of asthma is based on a medical history of episodic symptoms of cough, chest tightness, and dyspnea. The physical examination may reveal wheezes. Airflow obstruction is determined with spirometry. The obstruction is usually reversible with bronchodilator use, and the lung function may return to normal spontaneously.[29] In contrast with COPD, a normal result does not exclude asthma.[1,29] In patients suspected of having the disease, it may be necessary to complete a bronchoprovocation test to establish the diagnoses. It is important to exclude alternative diagnoses and to identify precipitating factors.

Asthma frequently begins in childhood and is associated with atopy. Even though the prevalence of atopy is high in the general population (30 to 50% of tested children), the presence of atopy is the strongest predisposing factor for the presence and development of asthma.[32] Less well understood is the development of asthma in adults. Atopy may play a

role, but the possible action of environmental agents at home or in the workplace is a well-recognized cause in some cases.[33,34] Interestingly, removal of a susceptible individual from the offending environment may not totally reverse the symptomatology and lung function alterations. An important feature of asthma is the presence of airway hyperreactivity. This response is commonly quantified using inhalation challenge testing with histamine or methacholine. It can be elicited also with cold air, with the inhalation of hyper- or hypotonic saline solution, or after exercise. The variability of morning and evening peak flow values may help measure airways responsiveness. Asthma is a treatable, reversible disease that has a relatively good prognosis. The acceptance of inflammation as the primary mechanism for disease progression has resulted in the development of several effective pharmacotherapeutic agents capable of improving the overall outcome of these patients.[35] Despite this, some patients will develop progressive airflow obstruction that may become irreversible. These patients may be indistinguishable from patients with COPD.

Expert consensus has recommended that spirometric testing be completed at the initial assessment, after treatment is initiated and symptoms have stabilized, and at least every 1 to 2 years.[29] The use of FEV_1 is necessary for the diagnosis. The measurement of peak expiratory flow rates is recommended for monitoring the patient who has received a diagnosis of moderate to severe asthma. The use of peak expiratory flow seems appropriate inasmuch as the disease is largely reversible and the spontaneous or treatment-induced variations are reflected by changes in expiratory flow. In addition, patients with the most severe asthma tend to underestimate their symptoms and may present with very severe obstruction with little perception of any clinical change.[36] Data from studies in which peak expiratory flow monitoring was one component of a comprehensive program indicate favorable outcomes.[37]

Conclusion

Spirometry remains essential for the diagnosis and monitoring of both asthma and COPD (Table 2).

Table 2–*Pulmonary Function Indications in Disease Management Using FVC and FEV_1*

Disease	Definitive	Probable	Possible
COPD	Diagnosis	Smoking cessation	Screening
	Prognosis		
Asthma	Diagnosis	Prevention	
	Prognosis	Screening	
	Disease management		

The use of spirometry in patients at risk for the development of both diseases or with respiratory symptoms could help detect cases at an early stage when intervention may prevent further deterioration. Because of the reversible component of asthma, the use of peak flowmeters to determine airflow on a continued basis is practical and seems to have resulted in improved outcomes. In contrast, in patients with progressive COPD, the use of frequent peak flow measurements and spirometry has not been shown conclusively to influence outcomes. In patients with the most severe degree of obstruction, the use of other tools needs to be explored and validated.

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