

SIMPLE OFFICE SPIROMETRY

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One of the most useful instruments for office practice is the spirometer. Today, the spirometer must find its rightful place alongside the sphygmomanometer, the electrocardiograph, and the ophthalmoscope. Abnormal spirometry is an indicator of increased risk for premature death from all causes. This indication has been known since the time of its invention in 1846 by John Hutchinson, a surgeon.²¹

Why has spirometry been so slow to be accepted in the mainstream of clinical practice? The author believes that spirometry has been couched in too much mystique. It also has required the careful study of structure-function relationships of the human lung to understand what causes alterations in spirometric measurements. Longitudinal studies have helped clarify the clinical significance of tests purported to indicate early stages of chronic obstructive pulmonary disease (COPD). Also, in the past, inexpensive and user-friendly devices were not available for office and clinic use. All of this is changing rapidly. Now the primary care physician and his or her assistant can learn the basics of spirometry easily, which provides two main values—the forced vital capacity (FVC) and the forced expiratory volume in 1 second (FEV₁). Reviewing how these values can be applied immediately to everyday practice is the purpose of this article.

THE ESSENCE OF SPIROMETRY

Spirometry simply measures airflow out of fully inflated lungs. The lungs are filled by muscular force, to expand the thorax. Full inhalation stretches the chest to its maximum. Following this, a full forced expiration rapidly empties the lungs into a device that records flow over time. Normal lungs empty in 6 seconds. Figure 1 represents the elastic forces, pulling outward from the thorax, balanced by the inward force of the lung's elasticity. The resting lung volume is known as *the functional residual capacity* (FRC). Although FRC can be determined indirectly by body plethysmograph or inert gas techniques, it is not measured by spirometry.

Expiratory airflow is a function of elastic recoil of lungs and thorax, small airways function, large airways function, and interdependence between small airway and the surrounding alveolar attachments. These fundamental concepts are presented in Figure 2 in an expression analogous to Ohm's law.

FLOW-VOLUME AND PRESSURE-FLOW RELATIONSHIPS

Studies on the relationship between flow and volume have shown that expiratory airflow is limited over most of the range of vital capacity.^{15,17} By producing a series of isovolume-pressure maneuvers, it has been

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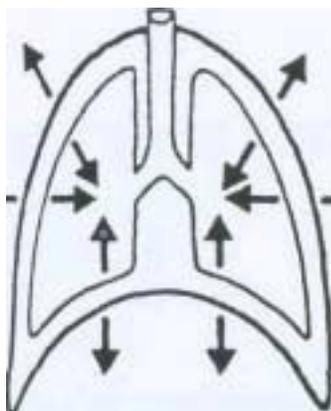


Figure 1. The equalization of forces between the inward retraction of the lungs and the outward recoil of the thorax. *Reproduced with courtesy of Thomas L. Petty, MD, from American Journal of Medical Quality.*

determined that exhalation from total lung capacity (TLC) is not limited by a flow maximum, but that below approximately 80% of the TLC, expiratory airflow plateaus and maximum flow is limited.²² The basic theory of flow limitation uses the equal pressure point concept of Mead et al.^{25,26} and the Starling resistor theory concept of Pride et al.³⁷ Both theories share a common concept of dynamic resistance to airflow and the flow-limiting segment where airway pressure resis-

tance exceeds alveolar pressure.⁸ Understanding these basic concepts reveals why simple results of airflow such as FEV_i are so useful. In brief, FEV_i is determined by intrinsic properties of the lungs and not simply by effort. For more detail and illustrations of these concepts, the reader may refer to the original reports or to a thorough review of the function of the normal human lung.²⁹

The chest radiograph representation of the full inspiration compared with a forced expiration is presented in Figure 3. The FVC has been expelled between the inspiratory and expiratory chest radiographs. A small amount of air remains in the upper part of the lungs. This remainder is the residual volume. The alveolar wall attachments surrounding the smallest airways (Fig. 4) tend to tether them, to prevent premature closure during forced expiratory airflow. It must be stressed that spirometric tests are effort-dependent. The quality of the effort to fill and to empty the lung forcefully affects the precision of expiratory and inspiratory airflow efforts.

The key spirometric tests are FVC (volume test) and FEV_i (flow test). Like systolic and diastolic blood pressure measurements, spirometry is a simple expression of a complex process. The determinants of expiratory airflow are compared with the determinants of

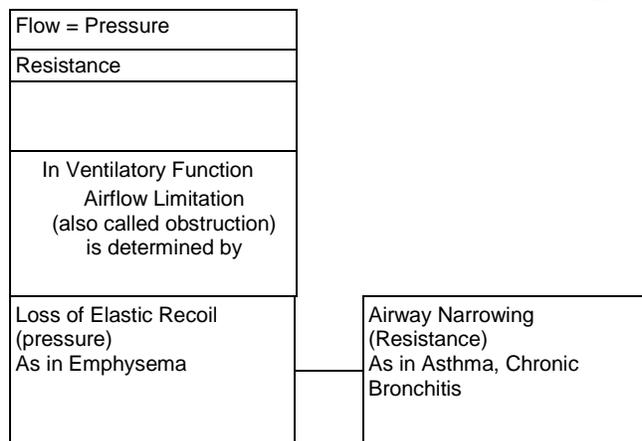


Figure 2. The determinants of expiratory airflow are the driving force that is the elastic recoil of the lungs and thorax against the resistance of the conducting airways. Muscular effort is required for a complete inspiratory breath and the forced vital capacity maneuver. *Reproduced with courtesy of Thomas L. Petty, MD, from American Journal of Medical Quality.*

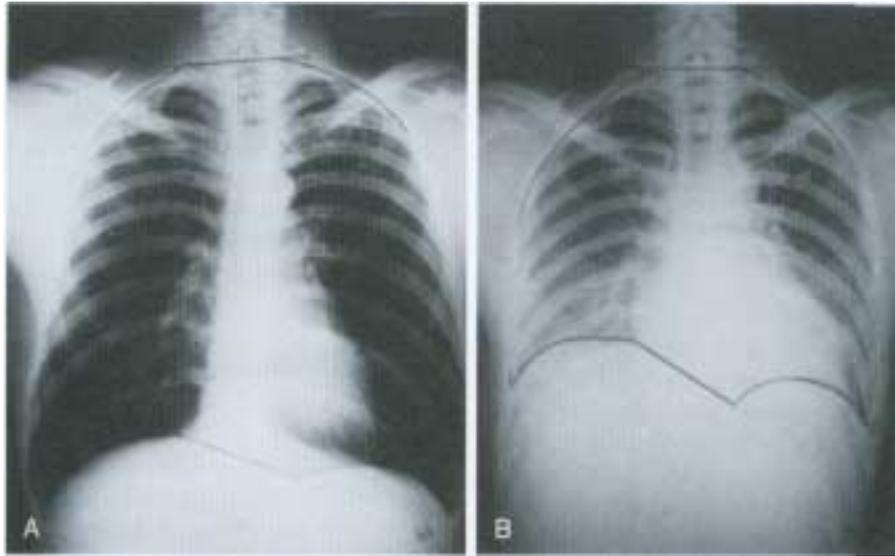


Figure 3. Inspiratory (A) and expiratory (B) radiographs revealing residual volume in the upper half of the lung. The forced vital capacity has been expelled during the inspiratory and expiratory films.

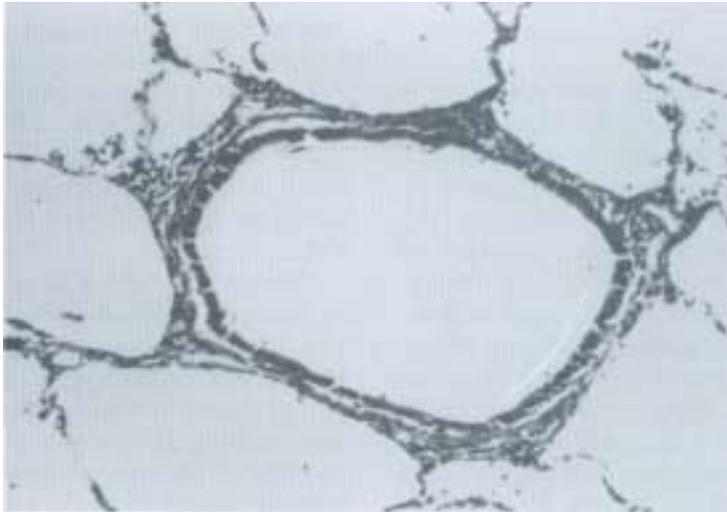


Figure 4. Alveolar attachments to small airways.

Table 1. DETERMINANTS OF EXPIRATORY AIRFLOW (LUNG FUNCTION) AS COMPARED WITH THE DETERMINANTS OF BLOOD PRESSURE

Blood Pressure (Sphygmomanometry) 120/80	Lung Function (Spirometry) 3.0 FEV ₁ /4.0 FVC
Elastic recoil	
Airways resistance	
Large airways	
Small airways	
Cardiac output	Interdependence, and so forth
Peripheral resistance	Muscular effort and
Blood volume	coordination, and so forth
Blood viscosity	
Renin-angiotensin axis	

blood pressure in Table 1. Spirometry measures elastic recoil, small airways function, large airways function, interdependence of airways and alveoli, and muscular effort. The net result is the FEV₁, the FVC, and the ratio between the two—that is, FEV₁/FVC.

Normal spirometric values are based on age, gender, and height. These values are programmed into modern electronic spirometers and are available as printed nomograms or formulae. The most complete normal values for a large, random US population are used by most spirometer manufacturers.¹⁸

THE TWO CONVENTIONS USED IN SPIROMETRY

Historically, Spirometry was presented as **the exhaled volume over time**. These volume-over-time curves are easy to visualize and allow the clinician to identify at a glance the FEV₁ and the FVC and the expiratory time. The advent of flow transducers provided another method of expressing airflow—that is, **flow-volume**. Although **the** so-called flow-volume convention gained popularity, it must be remembered that this method measures exactly the **same thing** as the volume-over-time method. The values are expressed in a different, sometimes more confusing, manner. All physicians should be able to use either method of analyzing airflow and volume in their practices. The flow transducer also allows a tracing of inspiratory flow, which is not available from most volume displacement devices. These two methods, flow-volume **and** time-volume are illustrated in Figures 5 through 8. These curves range from normal through the various progressive stages of air-

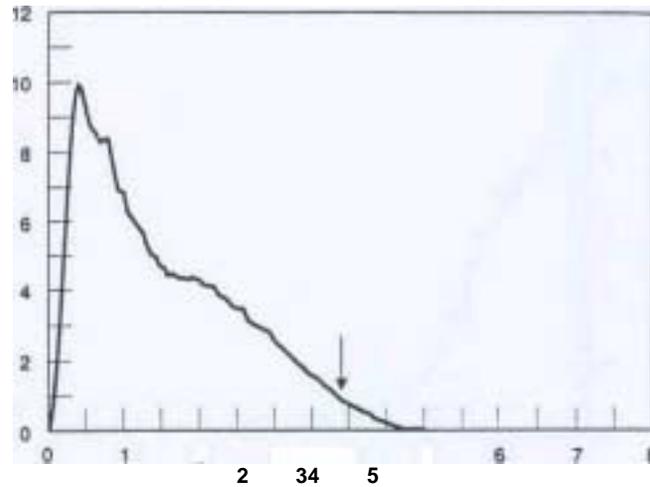
flow obstruction. Figures 9 and 10 are examples of the flow-volume and time-volume curves in moderate and severe ventilatory restriction. It should be remembered that airflow is a result of pressure against resistance (see Fig. 2). Pressure is generated by muscular effort and the elastic recoil in the lung. Airways resistance impedes airflow. Expiratory airflow can be limited by a reduced driving pressure from loss of elastic recoil, as in emphysema, in airways disorders (i.e., chronic bronchitis, asthma, which increases airways resistance), or both. Overlaps are common.

TESTS OF SMALL AIRWAYS DISEASE

Following the classic description of small airways disease in smokers by Hogg and associates,²⁰ an understandable focus on possible physiologic markers of airway inflammation, fibrosis, and mucous plugging emerged. At one time, the most popular spirometric index for small airways pathology was purported to be the forced expiratory flow between 25% and 50% of the FVC (FEF_{25-50%}).⁷ Additional tests of small airways pathology were the nitrogen washout test for closing volume² and flow-volume tests using gases of different densities⁴⁴ and frequency dependency tests.⁴⁴ Although abnormalities in the FEF_{25-50%} did correlate with small airways lesions as determined by surgical resectional specimens, the relationship was not significantly better than a relationship in FEV₁/FVC.⁷

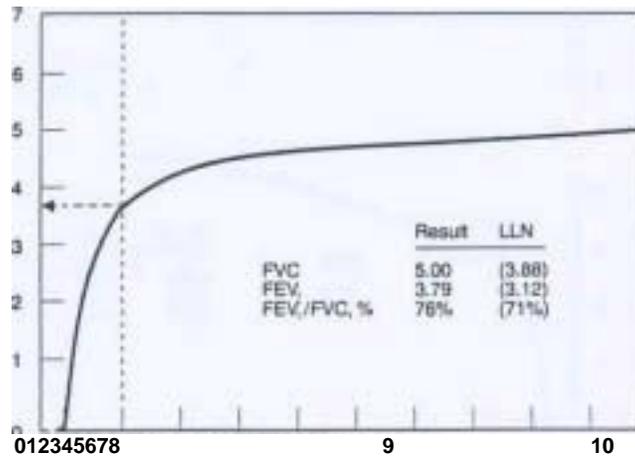
Nitrogen washout tests are not suitable for widespread application, such as office practice. Of greater significance is the fact that longitudinal tests of patients with an abnormal closing volume, as determined by nitrogen washout, did not predict a later reduction in FEV₁ or the emergence into clinically significant disease.^{31,40} In the author's laboratory, in studies of whole, fresh, excised human lungs, the FEF_{25-50%} was not better than the FEV₁/FVC at identifying small airways disease.³⁴⁻³⁶ Frequency dependency tests are also not relevant to office practice.

The author's main objection to the *TEF's* is that it is not only unnecessary, it is frequently misleading. To cite an example, the



Exhaled Volume (L, BTPS)

A



B

Time (s)

Figure 5. Normal flow-volume (A) and time-volume (B) expiratory curves.

author saw a 79-year-old woman for a preoperative evaluation during a work-up for a noncalcified solitary nodule that, on positron emission tomography, was strongly suggestive of malignancy. She had smoked a total of

50 pack-years but had stopped smoking 10 years before. Her father, also a heavy smoker, had died of lung cancer.

Her spirometric tests are presented in Table 2. It should be noted that this woman's FVC

Table 2. PREOPERATIVE PULMONARY FUNCTION TESTS

	Value	% Predicted		Value	% Predicted
FVC	2.87	115	TLC	4.54	100
FEV ₁	1.69	97	RV	1.54	80
FEV ₁ /FVC	59.0	70	DLco/VA	2.48	89
FEF _{25%-75%}	0.67	34	SaO ₂	RA	93
FET (sec)	9.3	<6.0			

FVC = forced vital capacity; FEV₁ = forced expiratory volume in one second; FEV₁/FVC = ratio; FEF_{25%-75%} = forced expiratory flow between 25% and 75% of forced vital capacity; FET = forced expiratory time; TLC = total lung capacity; RV = residual volume; DLco/VA = diffusion test corrected for alveolar volume; SAO₂ = arterial oxygen saturation.

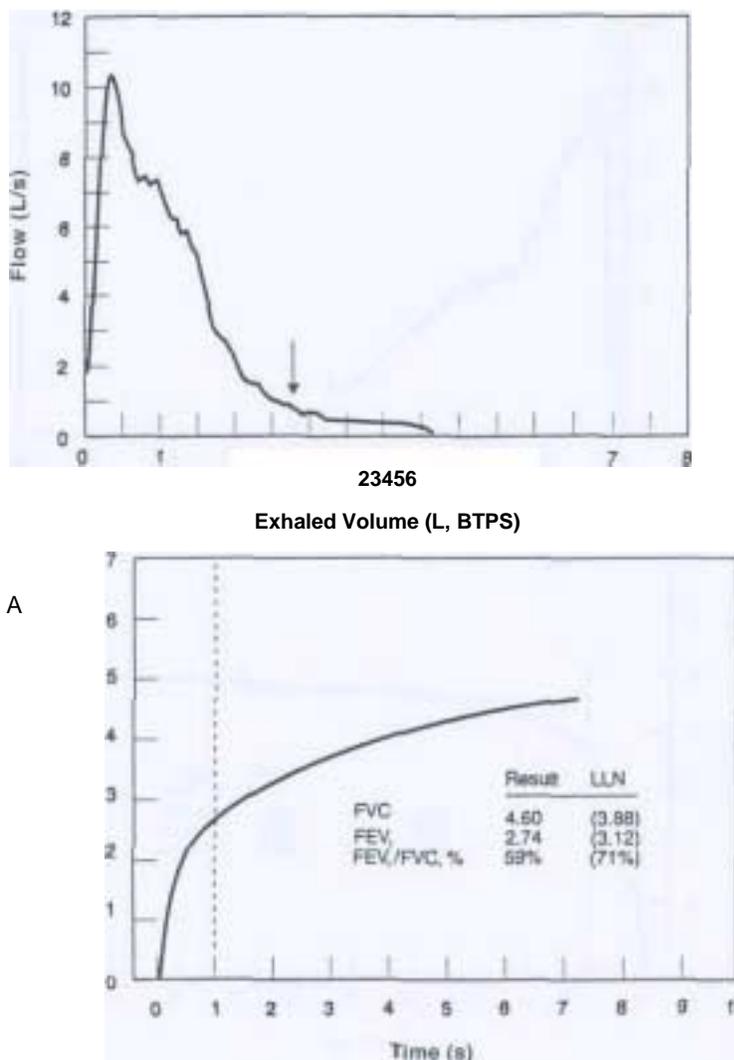
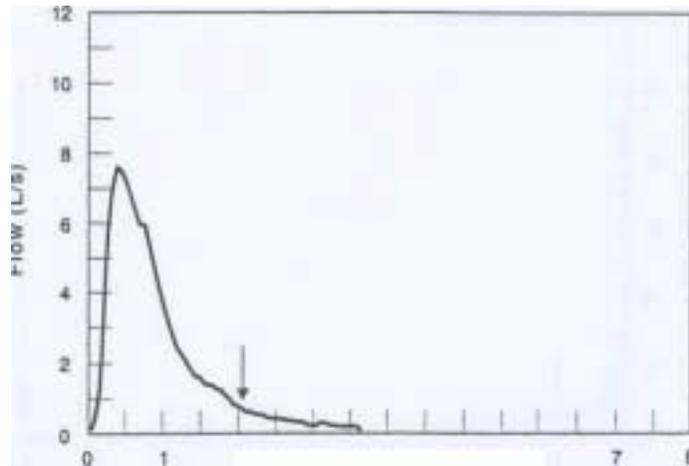


Figure 6. Mild airflow obstruction demonstrated in flow-volume (A) and time-volume (B) curves. FEV₁/FVC = 59%.

is supernormal (115% of predicted) and her FEV₁ is exactly normal for her age, gender, and height, (97%). Her FEF_{25-75%} is low because it is taken from midexpiration, which is in the middle half of the total expiratory time of 9.3 seconds—that is, a prolonged expiratory time because of an exceptional effort. Normal lungs usually empty in 6 seconds. This patient has the ability to empty her lungs to below her normal predicted residual volume (80% of predicted). On resection, the solitary nodule proved to be a benign carcinoid tumor. Her small air-

ways and alveolar structures in the wedge resection revealed no pathology.

This striking example demonstrates the trap the clinician can fall into if not aware of the complexities of spirometry. Unfortunately, because a flow transducer can yield many numbers and derived values from the expiratory volume-time versus flow-volume curve, an excess number of extraneous values often are printed out, only to confuse the practitioner. Clinicians should focus only on the two primary parameters—FEV₁ and FVC—and the ratio between the two. As this



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Exhaled Volume (L, BTPS)

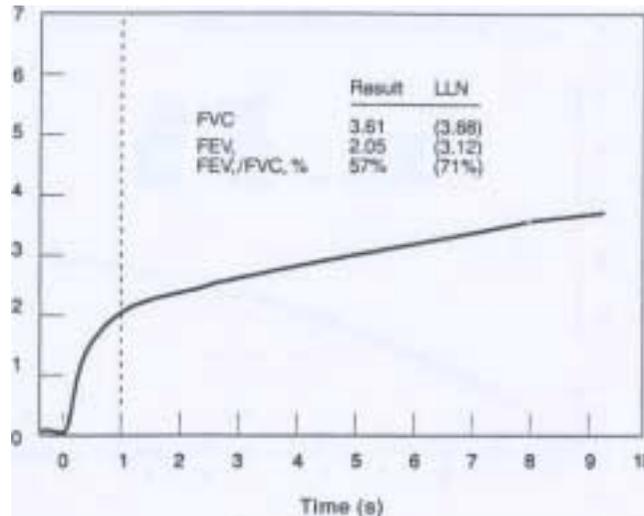


Figure 7. Moderate airflow obstruction seen in flow-volume (A) and time-volume (B) curves.

case example demonstrates, however, even the FEV₁/FVC ratio may be spuriously low when the FVC is high.

INTERPRETATION OF SPIROMETRIC ABNORMALITIES

First the time-volume or flow-volume curves should be scrutinized carefully for evidence of a good effort. This evidence means there must be a smooth curve, without cough or premature cessation of airflow from an obviously poor effort. One of the advantages

of the flow-volume convention is that peak flow can be visualized directly. It cannot be seen on the time-volume curve. Peak flow should occur within 0.1 second of initiation of forced expiration.

The FEV₁/FVC percent should be above 0.7. When this value falls significantly below 70% and the FVC (i.e., denominator) is normal, this is indicative of an obstructive ventilatory defect. By contrast, in restrictive ventilatory defects, the FEV₁/FVC ratio increases, often above 80% to 90%. This increase is because fibrotic lungs have increased elastic recoil and airways are patent. In restrictive dis-

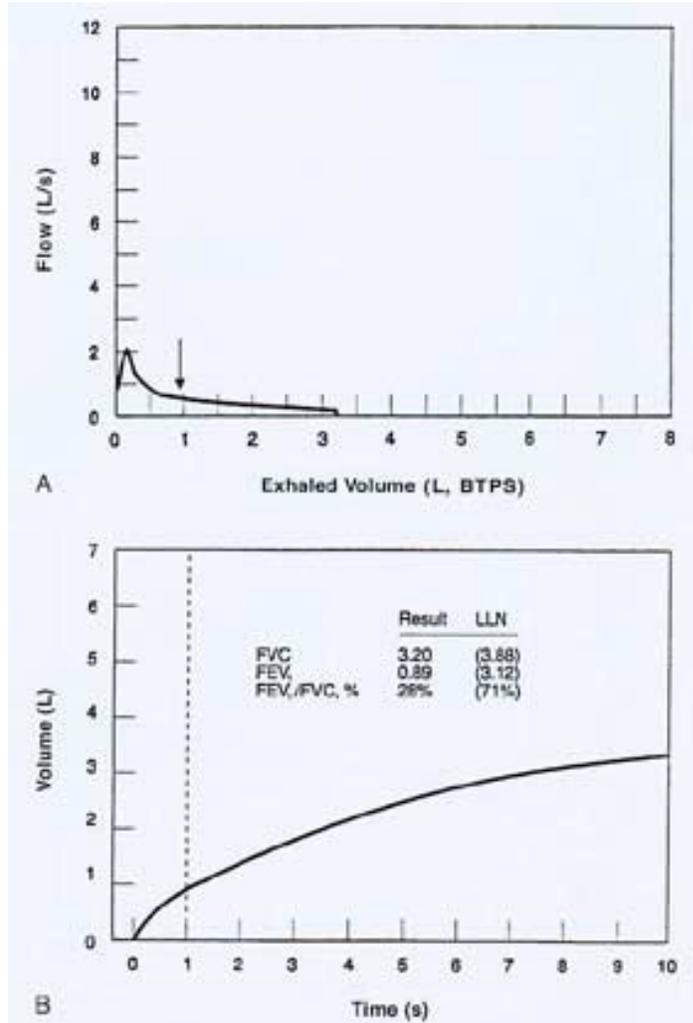


Figure 8. Severe obstruction seen in flow-volume (A) and time-volume (B) curves.

ease, therefore, if the FVC is less than 80% predicted and the FEV₁/FVC is greater than 80% predicted, a restrictive ventilatory defect is likely to be present. Figure 11 shows an interpretation algorithm that may be useful to differentiate obstructive and restrictive ventilatory disorders.

The common obstructive ventilatory disorders are:

- Asthma
- Asthmatic bronchitis
- Chronic obstructive bronchitis
- Chronic obstructive pulmonary disease (COPD; a generic term that includes asth-

matic bronchitis, chronic bronchitis, bronchitis, and emphysema; these states commonly overlap)

- Cystic fibrosis
- Emphysema

Where major reversibility is present, this is indicative of asthma or possibly asthmatic bronchitis, a form of chronic bronchiectasis in which airway hyperreactivity is increased and reversibility may be significant. The common restrictive ventilatory disorders are:

- Idiopathic fibrosing alveolitis
- Interstitial pneumonitis and fibrosis associ-

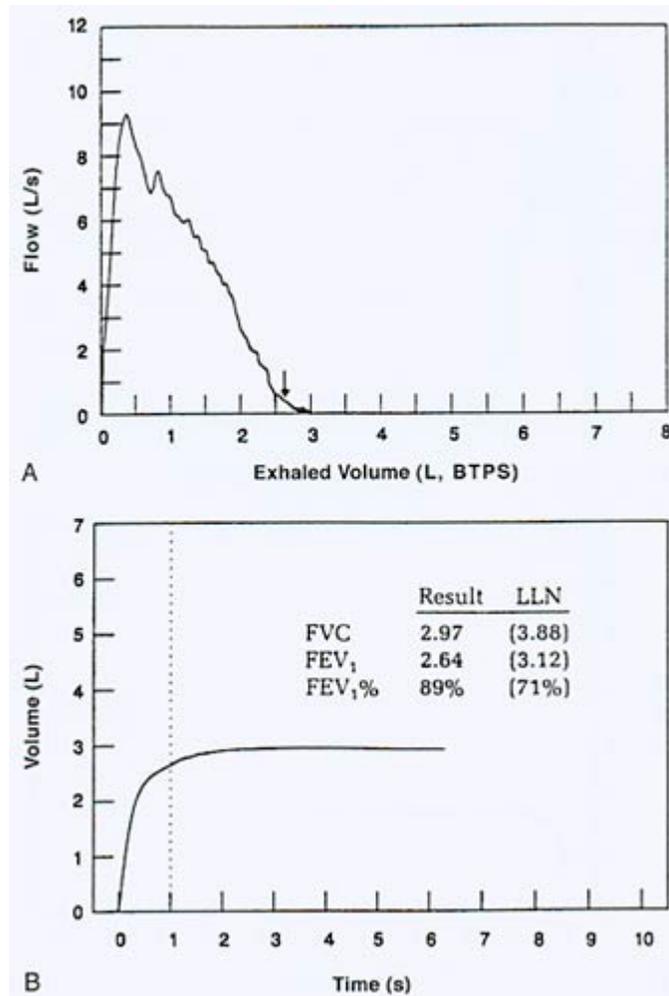


Figure 9. Moderate restriction seen in flow-volume (A) and time-volume (B) curves.

ated with drug reactions (e.g., bleomycin [Blenoxane]) or occupational exposures (e.g., asbestosis) or with collagen diseases (e.g., rheumatoid arthritis)

Fibrotic residue of disseminated granulomas (e.g., tuberculosis, histoplasmosis)

Sarcoidosis

Thoracic deformities

Congestive heart failure

SPIROMETRY IS AN IMPORTANT DATABASE

Normal spirometry predicts good chance of survival; abnormal spirometry indicates an ad-

verse prognosis. Simple spirometric measures provide an important database for the primary care physician. One example is the patient who comes to the physician with cough and dyspnea thought to be associated with a certain occupation. Knowledge of prior spirometry will give a baseline for comparison.

SPIROMETRY IN PATIENT MONITORING

Simple office spirometry is key to the identification of many disease states and to the objective monitoring of responses to therapy

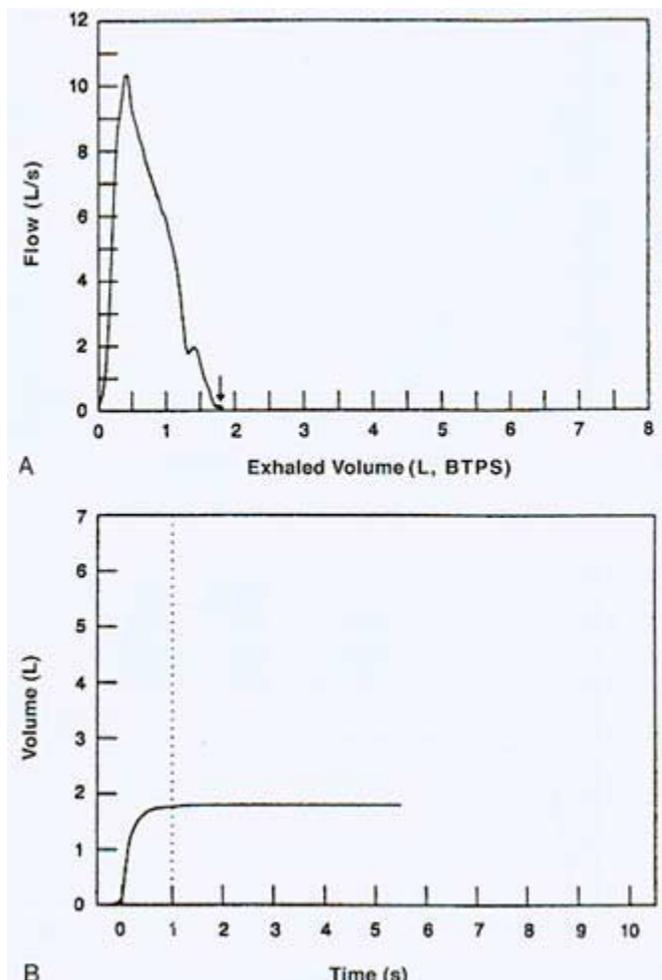


Figure 10. Severe restriction seen in flow-volume (A) and time-volume (B) curves.

for these heterogeneous conditions. It should play a central role any time a physician prescribes potent bronchoactive and anti-inflammatory drugs. A clinician would not treat hypertension without measurements of blood pressure, give insulin or an oral hypoglycemic agent to a diabetic without measurements of blood sugar, treat cardiac arrhythmias without ECG monitoring, or use warfarin anticoagulation without monitoring prothrombin times and INR. Nonetheless, spirometry is still not part of the primary care practice of most physicians who regularly prescribe drugs designed to improve airflow, volume, or both.

LUNG CANCER SPIROMETRIC ABNORMALITIES AND SMOKING-RELATED DISEASES

Lung Cancer is the most common fatal malignancy among men and women. Death rates remain high because the disease usually is discovered late in its course, with symptoms of cough, chest pain, or hemoptysis bringing the patient to the physician. The frequent use of chest radiographs and sputum cytology in screening large populations for lung cancer has been studied in three major centers, with disappointing results.^{13/14,27/42} Although earlier diagnosis through screening was possible

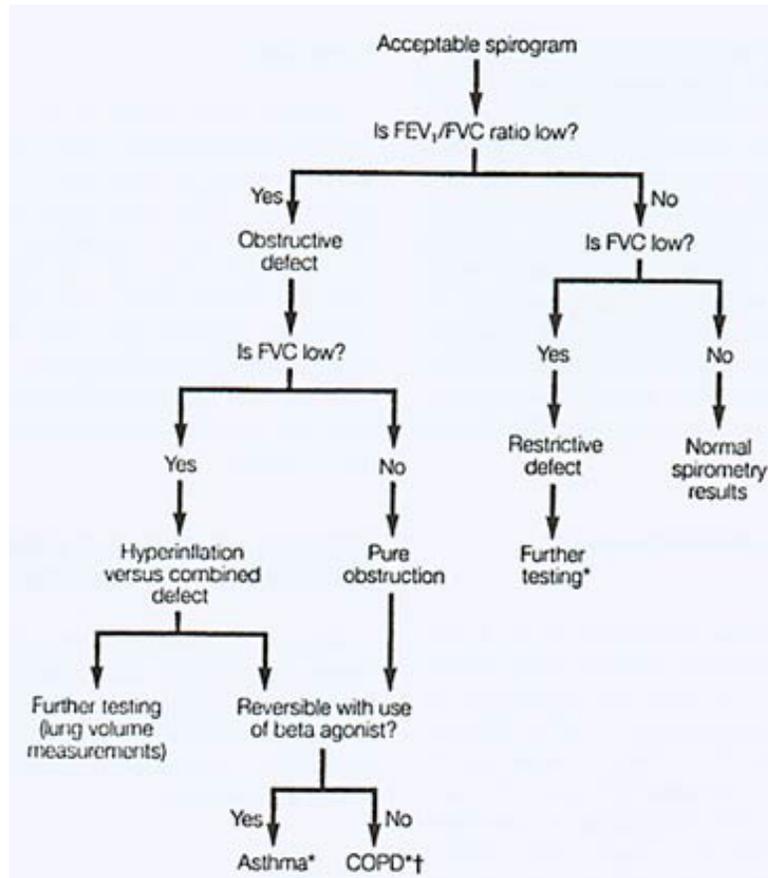


Figure 11. Nomogram algorithm for separating obstructive from restrictive airflow obstruction. *If clinical correlation present. †Some COPD may have reversible component.

in men who smoked, the resectability and cure rates were not substantially improved by screening.¹³ " This lack of improvement has led to the widespread belief that screening for lung cancer is not worthwhile.^{10,42} The author believes that in light of new findings using spirometry (described subsequently), this advice must be reconsidered.^{31,32}

Lung Cancer

Several epidemiologic studies have indicated a common denominator between COPD and lung cancer.^{5,6} In one study, nine lung cancers were found in 113 patients with airflow obstruction, compared with only two patients in 113 in whom airflow was normal over a 10-year follow-up period.³⁸ These two groups were matched for age, gender, smok-

ing history, and occupation. The presence of airflow obstruction therefore is a powerful predictor of the subsequent development of lung cancer.^{23,38} Furthermore, the presence of mucus hypersecretion seems to be an independent indicator of lung cancer.²³

These new observations may reopen the thought of using sputum cytology to look for lung cancer in high-risk patients—for example, smokers who have airflow obstruction or a family history of lung cancer. Chest radiographs or CT scans also should be used regularly for cancer surveillance in patients with these high risks.³²

Myocardial Infarction

The well-known and often-quoted Framingham Heart Study showed a close nega-

tive correlation between reduction in FVC and risk for death from heart disease."³⁹ The reason for this association is probably multifactorial. Patients who are at risk for myocardial infarction are commonly obese and in a state of poor physical fitness. In addition, they may have cardiac hypertrophy or occult congestive heart failure. Any or all of these abnormalities could lead to reduced FVC in proportion to increased myocardial infarction risk. More recent studies have confirmed that a decline in spirometric function is an independent risk factor for cardiovascular disease and death.⁴³

Chronic Obstructive Pulmonary Disease

By far, the greatest risk factor in the development of emphysema, asthma, and chronic bronchitis—that is, the full spectrum of COPD—is smoking. In early COPD, FEV₁ begins to fall before FVC. This decrease results in a reduced ratio between FEV₁ and FVC (less than the normal 70%). Population studies have shown clearly that this single value (FEV₁/FVC) can identify patients who are at risk for undergoing accelerated lung function loss. Abnormalities in FEV₁ and FEV₁/FVC herald the onset of clinically significant COFD.⁴

Lung Age

Another way to look at the effect of smoking on lung function is the concept of "lung age,"²⁸ which is computed by matching an individual's FEV₁ value with the age at which that FEV₁ value is considered normal based on predicted values. If a six-foot, 47-year-old man has an FEV₁ of 2.2, for example, his lung age is 72, because 2.2 is the FEV₁ measurement considered normal for a 72-year-old man of that height. Calculating a patient's lung age can be a potent motivator in smoking cessation.²⁸

EFFECTS OF SMOKING CESSATION ON SPIROMETRIC ABNORMALITIES

Because of these powerful correlations between spirometric abnormalities and smoking-related diseases, it follows that smoking cessation should be the key intervention whenever spirometric abnormalities are found in a smoker.

Lung Cancer

Smoking cessation reduces the risk for developing lung cancer,²⁴ and the risk decreases

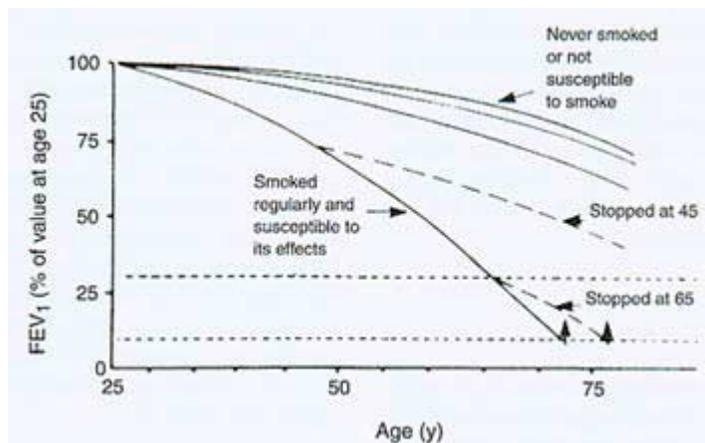


Figure 12. Accelerated losses of ventilatory function in susceptible smokers, and the beneficial effect of stopping smoking. (From Peto R, Speizer FE, Cochrane AL, et al: The relevance in adults of air-flow obstruction, but not of mucus hypersecretion, to mortality from chronic lung disease. Results from 20 years of prospective observation. *Am Rev Respir Dis* 128(3):492, 1983; with permission).

with each year of smoking cessation. After 10 years of abstinence, the risk for developing lung cancer drops considerably but never to that of a nonsmoker in patients who have been heavy smokers—that is, more than 30 pack-years.

Ischemic Heart Disease

Studies show that the survival rate of patients with prior myocardial infarction or angiographically proved ischemic heart disease is far better in patients who stop smoking than in those who continue to smoke cigarettes." The benefit from smoking cessation has been greatest in patients with moderate to marked manifestations of ischemic heart disease, and least evident in the most cardiac-disabled persons. It therefore behooves physicians to develop a treatment plan to educate patients about smoking cessation as early as possible.

Chronic Obstructive Pulmonary Disease

The course and prognosis of COPD are improved greatly in patients who stop smoking. In a study conducted in the United Kingdom, patients who stopped smoking at age 45 and whose FEV₁ was 70% of that predicted at age 25 had a decrease in deterioration of lung function within a few years, so that their lung-function loss paralleled the normal age-related change, or approximately 30 mL/year for a normal-size man.³⁰ Even patients who stopped smoking at age 65 and whose FEV₁ was reduced to 30% of that predicted at age 25 had improved survival compared with individuals who continued to smoke (Fig. 12).³⁰ The message of this study is that it is never

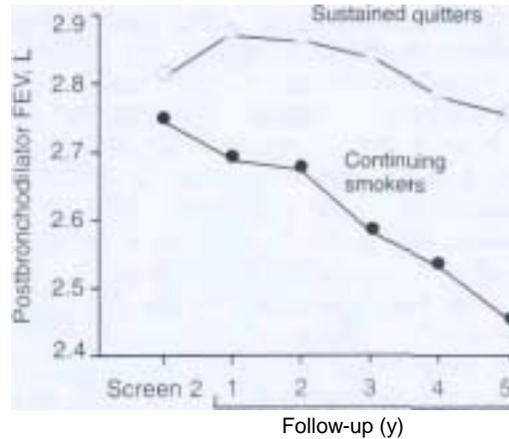


Figure 13. Improved ventilatory function in sustained quitters in the Lung Health Study, compared with continued smokers. (From Anthonisen NR, Connett JE, Kiley JP, et al: Effects of smoking intervention and the use of an inhaled anticholinergic bronchodilator on the rate of decline of FEV₁. The Lung Health Study. JAMA 272(19): 1502, 1994; with permission).

too late to stop smoking, although it is better if patients stop smoking earlier in the natural course of COPD.

The 1994 Lung Health Study also showed a reduced rate of decline in FEV₁ over 5 years in patients who stopped smoking. Those with the mildest abnormalities had a slight improvement in airflow before the mild, age-related decline occurred (Fig. 13).¹ It is highly noteworthy that the most common cause of death in the middle-aged participants in the Lung Health Study was lung cancer, with an incidence much higher than heart attack and stroke. This finding is remarkable since these patients' average age was 48.5 years (Table 3).

THE NATIONAL LUNG HEALTH EDUCATION PROGRAM

The National Lung Health Education Program (NLHEP) is a new national health care

Table 3. THE LUNG HEALTH STUDY: DEATHS WITHIN FIVE YEARS'

Cause	Smoking Intervention + Ipratropium	Smoking Intervention + Placebo	Usual Care	Total
Lung cancer	18	20	19	57
Cardiovascular disease	18	7	12	37
Other	18	17	20	55
Total	54	44	51	149

initiative aimed at the early diagnosis of COPD and related disorders.³³ The Spirometer Committee of the NLHEP recommends spirometry in all smokers older than the age of 45 years and in anyone with cough, mucous hypersecretion, dyspnea, or wheeze.¹² Because the forced expiratory volume in 6 seconds (FEV₆) can be taken as a surrogate for the FVC, new office-based devices use the FEV₆ in the denominator of the FEV₆/FVC equation.^{18,41} This use is because an FEV₆/FEV₆, of less than 70% is as good a predictor of a rapid decline in FEV₁ as the full ratio. Because it is difficult for many patients to exhale for 12 to 15 seconds to complete the full expiratory maneuver, it is practical and reasonable to limit the forced expiratory airflow measurement to 6 seconds.^{18,41} A new family of spirometers has been developed in direct response to the NLHEP initiative. Most of these devices sell for \$500 or less. They can store up to 300 tests. They can be interfaced with a printer to record time-volume curves or flow-volume curves.

SUMMARY

Spirometric measurements are as fundamental to medicine as are measurements of pulse, blood pressure, temperature, height, and weight. Spirometric measurements should be considered important vital signs. Any deviations from "normal" measurements can point primary care physicians toward the use of behavioral modification or effective pharmacologic agents to prevent or forestall their patients' premature morbidity and mortality from many disease states, including premature deaths from all causes.

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